



MONASH University

Accident Research Centre

**INFLUENCE OF CHRONIC ILLNESS ON CRASH
INVOLVEMENT OF MOTOR VEHICLE DRIVERS:
2ND EDITION**

by

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Abstract:

A significant issue for consideration in road safety is the impact of medical conditions on crash involvement and risk of injury. This aim of this project was to update the first edition report: *Charlton et al. (2004). Influence of Chronic Illness on Crash Involvement of Motor Vehicle Drivers. Report No. 213. Clayton, Australia: Monash University Accident Research Centre*. The report reviews the evidence for the influence of chronic illness and impairments on crash involvement of motor vehicle drivers for the period May-2003 to mid-2009, builds on previous research and provides an updated review of evidence since the last report. A number of methodological issues are discussed and recent research findings are critically evaluated. A risk rating system was applied to all medical conditions of interest. This provided a means of identifying those conditions that presented the greatest risk. Based on both new evidence and pre-May 2003 evidence, eight conditions were found to have at least a moderately elevated risk of crash involvement (relative risk greater than 2.0) compared with their relevant control group. These comprised the same conditions identified in the 2004 report: alcohol abuse and dependence, dementia, epilepsy, multiple sclerosis, psychiatric disorders (considered as a group), schizophrenia, sleep apnoea and cataracts. Guidelines regarding fitness to drive from selected jurisdictions were also considered in the light of evidence for crash risk. These comparisons revealed a number of differences across the jurisdictions and highlighted some inconsistencies with the available evidence for crash risk. A number of conclusions are presented which may contribute to the formulation of recommendations for managing the risk of injury crashes associated with medical conditions. The findings of this review also highlighted the need for a cooperative international approach to future research using population-based, prospective studies to advance scientific knowledge linking impairment from medical conditions and crash risk.

Key Words:	Disclaimer
Chronic illness, medical conditions, disorders, functional impairment, risk, motor vehicle, automobile, accident, crash, drivers, driving performance, injury, safety, fitness-to-drive, licence restrictions, training, rehabilitation, treatment, self-regulation, medications, cardiovascular, cerebrovascular, cognitive impairment, comorbidity, metabolic, musculoskeletal, neurological, psychiatric, sleep, respiratory, vestibular, vision.	This report is disseminated in the interest of information exchange. The views expressed here are those of the authors, and not necessarily those of Monash University or the Swedish Road Administration

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PREFACE

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EXECUTIVE SUMMARY

Aim of the review

The focus of this report is the influence of chronic illness and impairments on crash involvement of motor vehicle drivers. The review assesses the current state of knowledge in regard to the size of the problem in Western countries, taking into account the prevalence of specific medical conditions and the evidence for crash involvement and other measures of driver risk. A number of conclusions are presented which may contribute to the formulation of a set of recommendations for managing the risk of injury crashes associated with medical conditions.

Changes to the report

This report is the second edition of Charlton et al. (2004)¹ and provides a review of evidence on chronic illness and crash risk, including evidence reviewed in the first edition report as well as relevant studies published between May 2003 and June 2009.

The search strategy used to identify relevant literature followed the same fundamental principles as for the original report. The critical review process also followed the methods applied in the original report, with one noteworthy addition being the use of a checklist to provide more structure to reviewing relevant studies task. Key areas addressed in the critical review checklist included:

Definition of Condition: Was there an adequate method of defining/detecting the condition/disease?

Definition of key outcome measures: Was there an adequate method of assessing the outcome?

Study Design: Was the method of recruitment adequate to attract an unbiased sample? Were controls adequately recruited & matched? Were sample numbers large? Are data sources adequately described & an indication of data quality? Was there adequate control of other potential confounds (e.g. exposure)?

Results: Are the analyses/statistical techniques explained & justified? Is there a precise statement of the association between illness & outcome?

Discussion: Are interpretations of results & conclusions clear & justifiable?

Rate the empirical strength of study.

Sections of the report (3.2 to 3.13) documenting evidence for the relationship between selected medical conditions and safety outcome measures (crashes, infringements and performance) have been updated extensively. These sections cover the following conditions: *cardiovascular disease, cerebrovascular accident, cognitive impairment, diabetes mellitus, epilepsy and seizure disorders, musculoskeletal disorders, neurological disorders, psychiatric illness, respiratory disorders, sleep apnoea and related disorders, vestibular disorders and vision disorders*. It was not possible to include an updated review of risk associated with alcohol abuse and dependency (section 3.1) within the scope of the available project

¹ Charlton, J., Koppel, S., O'Hare, M., Andrea, D., Smith, G., Khodr, B., Langford, J., Odell, M. and Fildes, B. (2004). *Influence of Chronic Illness on Crash Involvement of Motor Vehicle Drivers*. Report No. 213. Clayton, Australia: Monash University Accident Research Centre.

resources. It is recommended that this be reviewed as a separate task to do justice to the vast body of recent literature on this topic. Guidelines on fitness to drive from selected jurisdictions were also reviewed where available for the purpose of comparison with evidence for crash risk.

Risk ratings were assigned for each medical condition using the same criteria and rating scheme as the original report. New risk ratings have been assigned and Chapters 1, 2 and 4 have been revised in line with the updated evidence presented in Chapter 3.

Crash risk

Licensing authorities are presented with the need to formulate policy to manage road safety within their jurisdiction. The challenge for licensing policy is to accommodate acceptable risk while balancing the societal and individual need for driving mobility. In particular, decisions must be made about the extent to which safety might be compromised for individuals with a specific medical condition. How much risk should be tolerated is a fundamental issue for policy development. At what point does the risk outweigh the need for mobility and other social and employment opportunities? The review provides authoritative, evidence-based guidance to enable policy development in the area of fitness-to-drive.

Methodological issues

In the review of evidence on medical conditions and crash risk, only one study was found which used a population-based, prospective design (see Skurveit et al., 2009, section 3.5). Generally, the best studies reviewed employed retrospective, case-control design, with adequate sample size, reliable diagnosis of condition and valid measures of crash involvement. However, most studies were found to have some level of bias, such as recruitment of non-representative cases (including severity, type of disorder, time since onset), and a lack of control of confounding variables such as comorbidity and driving exposure.

Influence of medical conditions on crash involvement:

A risk rating (RR) system was applied to all medical conditions of interest. Ratings were based on evidence for crash involvement only, since this was deemed to be of more direct relevance in assessing crash risk than both citations and driving performance. This provided a means of identifying those conditions that presented the greatest risk. The overall risk for each condition was rated as 'higher', 'not different' or 'inconclusive' compared with relevant control groups. Three levels of ratings for 'higher' risk conditions were applied:

Information on post-treatment risk was also considered. Overall post-treatment crash risk was rated as 'higher', 'lower' and 'inconclusive'.

Based on the evidence from studies reviewed, eight conditions were found to have at least a moderately elevated risk of crash involvement compared with their relevant control group (see Table 1). Specifically, these were alcohol abuse, dementia, epilepsy, multiple sclerosis, psychiatric disorders (considered as a group), schizophrenia, sleep apnoea and cataracts. A large number of other conditions was examined and found to have inconclusive evidence or evidence for only a slight elevation of risk. These conditions are detailed within the body of the report.

Table 1 Summary of crash risk associated with high-risk medical conditions

Condition	Prevalence (Population-based) %	Overall Crash Risk	Post-Treatment Crash Risk
Alcohol Abuse Alcohol Dependence ²	0.82%	Slightly to moderately high (2004 rating)	Inconclusive (2004 rating)
Dementia	1.0%	Moderately high	Inconclusive
Epilepsy	0.7%	Slightly to considerably high	Inconclusive
Multiple Sclerosis	0.03%	Moderately high	Inconclusive
Psychiatric disorders (as a group)	0.4% of licensed drivers, (Vernon et al., 2002) 25% (total population; at some time in life; includes substance abuse)	Slightly to moderately high	Benzodiazepine – Higher compared with controls without the condition (Methodological problems prevent the separation of risk associated with drug vs. condition.) Antidepressants (tricyclics) – Higher compared with controls without the condition (Methodological problems prevent the separation of risk associated with drug vs. condition.)
Schizophrenia	1%	Moderately high	Inconclusive
Sleep apnoea	0.3-7.5%	Moderately to considerably high	CPAP – Lower compared with controls without sleep apnoea
Cataracts	2-5% (40-49 yr olds)	Moderately high	Cataract surgery – Lower compared with un-treated cataract; – Inconclusive compared with those without the condition

² Not included in the post-May 2003 review.

Comparison of risk estimates for medical conditions and other driving groups

It is instructive to note that when the risk associated with young drivers (under 20 years) and alcohol impaired drivers (BAC 0.05%+) is compared with that of the high-risk medical condition population, the risk of the young driver group overwhelms all of the medical condition groups to such an extent that medical risks seem relatively minor.

Assessing fitness to drive

The review of evidence for crash risk was compared with guidelines regarding fitness to drive from selected jurisdictions. These comparisons revealed a number of inconsistencies across the jurisdictions and in some cases the guidelines did not appear to reflect the available evidence for crash risk.

Managing crash risk associated with medical conditions

Information about management of medical conditions was also reviewed. Intuitively, it would be reasonable to expect that well-established treatments might reduce risk. Indeed, the treatment of sleep apnoea using Continuous Positive Airways Pressure (CPAP) was shown to significantly reduce crash risk to the same level as that of drivers without the condition. However, in the case of treatments for psychiatric disorders, benzodiazepines and at least one type of antidepressants (tricyclics) were found to increase risk.

Other methods of management include special licensing conditions or restrictions. For example a driver who has lost a limb may be permitted to drive only whilst wearing a prosthesis. However, for most conditions there was extremely limited evidence available on these approaches to crash risk management.

Self-regulation is also a potentially useful management approach. This strategy is only likely to be effective if the driver has insight into the factors that place him or herself at risk. However, there is little evidence that specifically addresses the benefit of self-regulation in reducing crash risk.

Recommendations

In the light of the available information presented in this review, a number of recommendations can be made to improve safety outcomes associated with the influence of chronic illness and impairments on crash involvement throughout western countries:

- Develop reliable methods of identifying and referring those who are potentially at risk as a result of medical conditions;
- Promote public awareness, particularly amongst the driving population, about the known crash risks and effective management for particular medical conditions or impairments. This is important particularly because most jurisdictions are reliant on self-referral or voluntary reporting of medical conditions. Hence the onus is on the driver to determine whether they have a condition that affects their driving;
- Improve knowledge within the health profession about the known crash risks and effective management for particular medical conditions or impairments;

- Develop and implement valid and standardised assessments to identify the functional impairments of drivers with specific medical conditions at an increased risk;
- Review licensing guidelines for fitness-to-drive in the light of all available evidence regarding crash risk;
- Investigate the capacity for the use of medical technologies for more effective monitoring of driver risk (e.g., in-vehicle blood glucose monitoring system);
- Investigate the capacity for the use of adaptive technologies and intelligent transport systems (ITS) to enhance driver safety (e.g., safe following distance devices and rear collision warning and avoidance systems);
- Include appropriate licensing conditions/restrictions (e.g., alcohol interlocks for drivers with alcohol problems);
- Review of chronic alcohol and drug abuse in a broader framework, including drugs and alcohol abuse and high level dose/usage;
- Advance scientific knowledge linking medical conditions and crash risk in order to improve the evidence base for formulating policy about licensing and fitness to drive;
- Investigate and educate drivers with non-insulin dependent about hypoglycaemic awareness.

Future research

It is recommended that a cooperative international approach to future research be adopted. This should take the form of a large scale, prospective study (or group of studies) using a population-based or case-control design to investigate the following:

- Underlying impairments or mechanisms that contribute to crash risk for particular medical conditions;
- The effectiveness of treatments, rehabilitation and countermeasures, including ITS and other advanced technologies, in reducing crash risk;
- The effectiveness of mandatory and voluntary reporting and assessment of medical conditions;
- Risk and risk reduction strategies for targeted high-risk sub-groups, particularly with multiple medical conditions prevalent in the ageing population;
- The social, health and economic consequences of licensing restrictions in at-risk populations.

CHAPTER 1 INTRODUCTION

1.1 AIM OF THE REVIEW

The aim of this review is to critically review the literature identifying the relationship between medical conditions and crash risk. The report considers the influence of chronic illness and other enduring complications of illness and associated impairments on involvement in motor vehicle crashes and other indicators of driving risk. The current state of knowledge is assessed in regard to the size of the problem, taking into account the prevalence of specific conditions, evidence for crash involvement and other estimates of driver risk. A number of conclusions are presented which may contribute to the formulation of a set of best practice recommendations for managing the risk of injury crashes associated with medical conditions.

1.2 BACKGROUND

A significant issue for consideration in road safety is the impact of chronic illnesses on crash involvement and risk of injury. While much of the research on this topic has focused on specific medical conditions, there have been a relatively small number of reviews that have synthesised these findings. Much of the evidence considered in previous reviews is now at least two decades old and there is a need to review the evidence again, in the light of significant developments in a range of relevant areas.

Recent advances in the areas of medicine, applied health sciences and disability studies have led to a better understanding of underlying mechanisms of many chronic illnesses and associated impairment. Significant developments in pharmacological and other treatments are also likely to have had an impact on level of impairment, mobility and quality of life of individuals with chronic illness. Generally, this is likely to have a positive effect on driving experiences and crash risk (Macleod, 1999; Veneman, 1996), although some interesting exceptions have been discussed suggesting negative outcomes associated with new treatment regimes for some conditions such as tighter self-monitoring of blood glucose for diabetes and laser treatment for diabetic retinopathy. The impact of these and other treatment effects is considered further in Chapter 3.

Since the early 1980s, beginning with the international year of the disabled in 1981, there has been a considerable shift in philosophical thinking about disability, disability rights, equal opportunity and access to employment, education and resources. This is expected to have impacted on the mobility of people with disabilities including driving. Developments in information technology and improved access to educational materials are also likely to have led to greater public awareness about chronic illness and impairments and in turn, this may have influenced self-regulatory behaviours of drivers with medical conditions (e.g. Cox, Gonder-Frederick, Julian & Clarke, 1994).

Since publication of the first edition of the Charlton et al. report (2004) many hundreds of studies have been published on medical conditions and driver risk. It is therefore timely to update the review risk estimates in the context of recent advances in medicine and other scientific developments and the significant shifts in philosophical perspective relating to disability, impairment and driving skill.

1.3 THE AGEING POPULATION AND CHRONIC ILLNESS

A particular issue of relevance to the impact of chronic illness on crash involvement is the predicted pattern of ageing of western society. By the year 2030, it is estimated that in many OECD countries, one in every four persons will be aged 65 years or older. This shift in the population distribution is attributed largely to the ageing of the 'baby boomer' cohort. Current estimates suggest that approximately one third of those over the age of 65 years have a disability of some kind (OECD, 2001).

A critical issue relevant to ageing and chronic illness, is the co-existence of multiple conditions, which tends to be more common, but not exclusive to older age groups. While there have been relatively few studies that have considered the effect of comorbidity on crash risk, intuitively there is a strong likelihood that multiple conditions will carry a higher risk than that associated with any of the individual component conditions alone; that is, it is possible that they will have a non-linear, negative influence on risk. This is also complicated with general age-related frailty and decline in various cognitive, sensory and physical capacities. While it is true that from a scientific perspective, it is possible to tease apart the *independent* contributions of age and co-existing medical conditions using appropriate methodological and statistical procedures, it is also of interest to understand how these factors might interact in their impact on crash risk. Indeed, this will have important implications for policy and practice in guiding decisions in road safety.

In the past decade there have been several papers that have focused on crash risk and medical conditions of *older drivers* in particular (e.g. Hakamies-Blomqvist, 1993; 1994; 1996; Hu, Jones, Reuscher, Schmoyer & Truett; 2000; Janke, 1994; Dobbs, 2001; Dobbs 2005; Marshall, 2008; Staplin, Loccoco, Stewart & Decina, 1999). This review takes a broader view of the driving population, considering the relative risk associated with chronic illness across the age span, including those conditions that are more prevalent in older age groups.

1.4 HEALTH, CHRONIC ILLNESS AND FUNCTIONAL IMPAIRMENT

While there is a widely held view that overall health per se is a poor predictor of driving ability (Janke, 1994; Dobbs, 2001), there is some recent evidence that draws this into question. Using the Cornell Medical Index, derived from the total number of self-reported medical conditions, Rabbitt and Parker showed that drivers (n=362), aged 49-90 years, reporting a relatively poor health score (95th percentile) had a crash liability about 1.66 times that of those who reported a relatively good health score (5th percentile) (2002). Notwithstanding the equivocal evidence for a contribution of health status, what is likely to be of more interest to licensing authorities is a more sensitive analysis of those conditions that lead to the greatest compromise in driving skill and those which pose the greatest threat to safety. On this issue, there is some evidence that specific medical conditions have an impact on driving performance and crash involvement, although the literature is by no means in agreement for all conditions. In the case of sleep apnoea, for example, the evidence reviewed in Chapter 3 is relatively consistent in identifying an elevated risk. In contrast, findings for Parkinson's disease, traumatic brain injury and diabetes are less definitive and to a large extent, are influenced by disease progression, severity and associated complicating conditions.

Clearly, not all medical conditions affect injury risk on the road system to the same extent and not all individuals with the same condition will be affected in the same way. The severity of the condition and other characteristics of the disorder are likely to be important determinants of crash risk. Indeed, it is not necessarily the medical condition and/or medical complications per se that affect driving, but rather the *functional impairments* that may be associated with these conditions. In discussing the merits of focussing on impairments in assessing risk, Marottoli comments that functional impairments are “the common pathway through which ... medical conditions affect driving capability and ... can be relatively easy to test” (2001, p.11). Moreover, the extent to which individuals may be able to adapt or compensate for their impairment while driving will undoubtedly have some bearing on their likelihood of crash involvement. More research is needed to better understand the link between crash risk, medical conditions and specific types and levels of functional impairments and the impact of compensatory strategies in moderating this risk.

The OECD report on Ageing and Transport (2001) proposes the following approach to the understanding of the relationship between medical and health conditions, functional impairment and crash risk:

- Determine which health and medical conditions have functional consequences that affect driving and walking;
- If there are functional consequences, determine whether they necessarily lead to increased crash risk or whether the individual can compensate for them;
- If there is substantial injury risk, identify as appropriate, and implement countermeasures to reduce the risk;
- If there are no countermeasures, balance the costs of crash risk against the cost of any consequent reduction in mobility (OECD, 2001, p. 25).

This approach has a broader relevance beyond the older driver safety problem and has potential for the assessment of fitness to drive in people with chronic illness. However, in order for this model to be of any practical value, reliable ways of assessing functional impairment and crash risk must be established. The majority of studies identified in this review have addressed the question of risk associated with medical conditions rather than functional impairments. Some notable exceptions can be seen in the three key areas of cognition, psychomotor functions and vision, where researchers are endeavouring to understand underlying mechanisms of impairments and how these impact on driving skill and crash risk (e.g. Fitten et al., 1993; Ball, Owsley, Sloane, Roenker & Bruni, 1993). A potential problem with this approach, however, is that there is generally not one single method for assessing a given functional impairment. This is particularly evident in the case of cognitive impairment, where a very large number of neuropsychological functions may be affected and a profusion of assessments are available. More effort should be directed towards identifying a set of sensitive and reliable assessments of impairments that impact on driving skill.

In addition to deciding what are appropriate outcome measures for identifying impairments and driving risk, the question remains: *What is an acceptable level of risk?* Various studies have reported *statistically significant* or *non-significant risks* associated with specific chronic illnesses. However, what is less clear is how a statistically

significant risk translates into real-world road safety risk. Ultimately, it is this measure of real-world crash risk that is critical for the licensing authority in determining policy to protect the safety of its road users.

1.5 EVIDENCE BASED DECISION-MAKING

While the determination of risk may finally lie with the licensing authorities, in practical terms, medical and health practitioners are called upon to make decisions about whether individuals with medical conditions should be permitted to continue to drive; with or without restrictions. In some jurisdictions (e.g. the Netherlands) specialist medical practitioners are nominated to undertake such assessments. However, in the majority of countries, this responsibility lies with the general practitioner. Frequently, this decision-making places the clinician in a difficult ethical dilemma. Health care professionals report that they do not wish to make these decisions, which have such potential to impact negatively on the general well being and mobility of their patients. Moreover, general practitioners have indicated that they need more objective tools to assess potentially at-risk drivers for referral to licensing authorities (Andrea, Charlton, & Fildes, 2001; Charlton, Fildes, Koppel, Andrea, Newstead & Pronk, 2002). Other studies suggest that family physicians may not have sufficient knowledge to assess fitness to drive. Hakamies-Blomqvist reported that fewer than 10% of former drivers were advised by a physician to stop driving and only 20% of those individuals had received advice in the official context of mandatory medical control of older licence holders (in Finland) (Hakamies-Blomqvist & Wahlström, 1998). This highlights the need for guidelines for assessment of risk that are informed by scientific evidence.

1.6 APPROACHES TO MANAGEMENT

There is a wide range of approaches to the management of vulnerable road user groups with chronic illness. These include various practices for assessing medical fitness to drive, provisions for issuing conditional and restricted licences, and rehabilitation and driver re-training. To date, there has been little attention directed to how these approaches might best be coordinated and evaluated to optimise their effectiveness in reducing driver risk. This review presents a number of strategies for identifying and managing drivers with medical conditions who are potentially at risk. A particular focus is a comparative analysis of international practice in assessing fitness to drive and consideration of the extent to which these guidelines are informed by available scientific evidence. This interaction between science and policy is critical for the advancement of evidence-based practice in the road safety arena.

1.7 DISABILITY AND DISCRIMINATION

In managing the safety of road users, licensing agencies face difficult decisions about personal and public safety. On the one hand they are obliged to produce regulations and guidelines that provide optimal protection of the community. Yet, at the same time they must ensure that such regulations are not overtly restrictive on the rights and opportunities of the population, particularly in regard to the capacity of individuals to earn a living (Helbach, 1991).

1.8 PRIVATE AND COMMERCIAL LICENCES

In most licensing guidelines a distinction is drawn between licensing criteria for private and commercial licences. Due to the higher danger potential to the public and the environment that driving commercial vehicles carries (eg transporting dangerous goods, larger freight loads and passengers for hire, and the longer periods spent driving as well as the size and weight of the vehicle), drivers of these vehicles are required to undergo a more rigorous assessment prior to licensing. In comparison, the daily driving habits of a private licence holder may only involve driving to the shops or work and, hence, a less rigorous approach is indicated.

In addition, some countries allow scope to apply differing degrees of latitude when licensing both commercial and private drivers, depending on the driving circumstances. For example, a farmer may require a commercial licence to drive heavy vehicles on the farm, rather than on the open road. Such a scenario would not present a grave threat to public safety and less strict criteria could be applied. In addition, “grandfather rights” (less stringent test standards) apply to those who have held commercial licences prior to certain dates in the UK, Sweden and the USA. Conversely, a more rigorous approach may be called in the case of more onerous responsibilities associated with passenger transportation. For example, in the UK, the House of Commons Transport Select Committee has recommended that all people seeking a taxi licence should be required to pass a medical exam, and that relevant authorities may impose licensing and medical requirements over and above that set out in the guidelines (DVLA, 2003).

Regardless of whether considering decisions for private or commercial drivers, it is essential that guidelines for assessing fitness to drive are in line with legislation relating to disability and human rights and do not unfairly discriminate against individuals with a disability. This underlines the importance of establishing guidelines that are informed by sound scientific evidence.

1.9 BALANCING MOBILITY AND SAFETY

Policy makers need to set reasonable standards with due consideration not only to safety but also to mobility of both individuals and all road users in their jurisdiction. For example, while a decision to restrict all licence holders with epilepsy might be effective in greatly reducing crashes associated with seizures, the decision would have a massive impact on the mobility of this group. The outcome must also be considered in the context of the *prevalence* of the disease and what this would mean for the overall reduction in crashes within the jurisdiction.

A number of authors have argued that decisions about licence status need to be individually determined and indeed for many conditions (particularly where cognitive decline is implicated), specify that licensing privileges should be issued on a case-by-case basis, as distinguished from blanket restrictions for a given medical condition. Conditional licences may be particularly relevant for those who live in areas poorly serviced by public transport. Arguably, the decision-making process should incorporate a range of relevant issues including individual nature of the condition (co-morbidity; level of severity) as well as individual drivers’ capacity for rehabilitation, as well as their lifestyle and mobility needs (proximity to services; access to alternative transport, etc).

1.10 STRUCTURE OF THE REVIEW

The review is structured as follows:

Chapter 2 covers methodological considerations relevant to the evaluation of crash involvement and chronic illness. Issues include sampling methods and biases, identification of chronic illness and impairment, outcome measures of risk and statistical procedures for determining risk. The chapter concludes with a description of the literature search method and the review process.

Chapter 3 is presented in thirteen sections with each section devoted to one of the selected medical conditions, associated functional abilities, crash risk and other indicators of road safety risk. Management issues including assessment of fitness to drive, rehabilitation and training (where appropriate) and self-regulation of driving behaviour are also reviewed. Evidence from studies reviewed for the periods 1980 to May 2003 and post-May 2003 to mid-2009 are presented in separate sub-sections for each condition. A summary is provided for each condition, drawing on all the evidence reviewed across the two stages of the review.

Chapter 4 provides a summary of the main findings relating to crash involvement and medical conditions. Conclusions are presented which may contribute to the formulation of a set of best practice recommendations for managing the risk of injury crashes associated with medical conditions.

References

- Andrea, D., Charlton, J.L. & Fildes, B. (2001). *Assessing Older and Disabled Drivers: Evaluation of Referral Assessment Tools and a Modified Hazard Perception Test*. Research and development report GR/2001-, VicRoads.
- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Charlton, J., Koppel, S., O'Hare, M., Andrea, D., Smith, G., Khodr, B., Langford, J., Odell, M. and Fildes, B. (2004). *Influence of Chronic Illness on Crash Involvement of Motor Vehicle Drivers*. Report No. 213. Clayton, Australia: Monash University Accident Research Centre.
- Charlton, J.L., Fildes, B., Koppel, S., Andrea, D., Newstead, P.E. & Pronk, N. (2002). *Evaluation of a GP Referral Assessment Tool for Assessing Older and Functionally Impaired Drivers*. Research and development report GR/2002-, VicRoads.
- Ball, K., Owsley, C., Sloane, M. E., Roenker, D. L. & Bruni, J. R. (1993). Visual attention problems as a predictor of vehicle crashes in older drivers, *Investigative Ophthalmology & Visual Science*, 34, 3110-3123.
- British Columbia (Superintendent of Motor Vehicles) v. British Columbia (Council of Human Rights). (1999) 3 S.C.R. Accessed on 28th of May, 2003 from <http://www.lemum.umontreal.ca/>
- Cox, D.J., Gonder-Frederick, L., Julian, D.M. & Clarke, W. (1994). Long-term follow-up evaluation of blood glucose awareness training. *Diabetes Care*, 1, 1-5.
- Dobbs, B. M. (2001). *Medical conditions and driving: current knowledge*. Technical Report, National Highway Transportation Safety Administration and The Association for the Advancement of Automotive Medicine Project.
- Dobbs BM. (2005) *Medical Conditions and Driving: A Review of the Scientific Literature (1960–2000)*, Technical Report for the National Highway and Traffic Safety Administration and the Association for the Advancement of Automotive Medicine Project, Washington, DC.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Fitten, L.J., Perryman, K.M., Wilkinson, C.J., Little, R.J., Burns, M.M., Pachana, N., Mervis, R.J., Malmgren, R., Siembieda, D.W. & Ganzell, S. (1995) Alzheimer and Vascular dementias and driving: a prospective road and laboratory study. *Journal of the American Medical Association*, 273(17), 1360-6.
- Hakamies-Blomqvist, L.E. (1993). Fatal accidents of older drivers. *Accident Analysis and Prevention*, 25(1), 19-27.

- Hakamies-Blomqvist, L.E. (1994). Ageing and fatal accidents in male and female drivers. *Journal of Gerontology: Social Sciences*, 49, S286-290.
- Hakamies-Blomqvist, L. (1996). Medical screening of older drivers as a traffic safety measure – A comparative Finnish-Swedish evaluation study. *Journal of the American Geriatric Society*, 44: 650-653.
- Hakamies-Blomqvist, L. & Wahlström, B. (1998). Why do older drivers give up driving? *Accident Analysis & Prevention*, 30(3), 305-312.
- Helbach, D. (1991). A legislative view of driving and legal restriction. *Wisconsin Medical Journal*, 90(3), 119-120.
- Hu, P., Jones, D., Reuscher, T., Schmoyer, R. & Truett, T. (2000). *Projecting fatalities in crashes involving older drivers* Report for the National Highway Traffic Safety Administration, Oak Ridge National Laboratory Tennessee: ONRL
- Janke, M. (1994). *Age-related disabilities that may impair driving and their assessment. A literature review*. Sacramento: California Department of Motor vehicles. Report RSS-94-156.
- MacLeod, K.M. (1999). Diabetes and driving: towards equitable, evidence-based decision-making. *Diabetic Medicine*, 16, 282-290.
- Marottoli, R. (2001). Health issues for older road users. *Transcript of proceedings of the Mobility and Safety of Older People Conference*, Melbourne, August.
- Marshall, Shawn C. (2008). The Role of Reduced Fitness to Drive Due to Medical Impairments in Explaining Crashes Involving Older Drivers. *Traffic Injury Prevention*, 9 (4), 291-298.
- OECD (2001). Ageing and Transport. Mobility needs and safety issues. Paris: OECD Publications.
- Rabbit, P. & Parker, D. (2002). *The ageing driver: a programme of research*. Road Safety Report No 29. C.G.B. Mitchell and G. Maycock (Eds.). Department for Transport: London.
- Staplin, L., Loccoco, K.H., Stewart, J. & Decina, L.E. (1999). *Safe Mobility for Older People Notebook*. National Highway Traffic Safety Administration Model Driver Screening and Evaluation program. DOT HS 808 853.
- Vaa T. (2003) *Impairment, Diseases, Age and Their Relative Risks of Accident Involvement: Results from Meta-Analysis*, TØI Report 690 for the Institute of Transport Economics, Oslo, Norway.
- Veneman, Th. F. (1996). Diabetes mellitus and traffic incidents. *The Netherlands Journal of Medicine*, 48, 24-28.

CHAPTER 2 **METHODOLOGICAL CONSIDERATIONS IN IDENTIFYING CRASH RISK ASSOCIATED WITH CHRONIC ILLNESS**

This section considers methodological issues and difficulties in the research literature examining crash risk and medical conditions. Lack of agreement in the literature about the role of chronic medical conditions, impairments and medications in crash involvement can be attributed at least in part, to differences in study methodology (McGwin et al., 2000). Some of the key issues impacting on this topic are considered below.

As discussed in Chapter 1, this review builds on previous evidence presented in Charlton et al. (2004) and has focuses on studies conducted between 2003 and mid 2009. The review highlights current knowledge and practice relating to medical conditions, and considers driver risk within the current road safety context. However, even across this relatively short time frame, diagnostic methods for many conditions have been refined and treatment and management strategies have changed. In many cases, the capacity of individuals to maintain a stable medical status with minimal impairment or to compensate for impairments with new technologies has been greatly enhanced. This has lead to a lack of uniformity in study methodologies and characteristics of study groups across this review period and makes valid comparisons between various studies difficult.

2.1 MEASUREMENT OF RISK

One of the difficulties in interpreting research findings on crash risk and chronic illness is that there is no standardisation of measures of driving performance or of crash risk. This makes it difficult to compare findings across different studies. Risk can be expressed in absolute or relative terms. *Absolute risk* refers to the risk associated with a population of interest such the frequency of ‘driving events’ amongst drivers with epilepsy. Generally, while it is informative to know the absolute risk associated with a particular medical condition, it is more instructive to understand this in the context of known risk for other groups, such as the population of all drivers or other relevant comparison groups such as drivers without the condition of interest. Estimates of risk expressed as a ratio with such comparison groups are referred to as *relative risk*. Issues related to selection of appropriate control groups are discussed in the following section.

Measures of risk generally fit one of three categories: crashes, citations (driving infringements and violations) and driving performance. These categories are discussed further below.

Crash involvement

The most direct and frequently used method of assessing crash risk is by determining crash involvement. For this reason, in drawing conclusions in this review about overall risk associated with specific conditions, greater emphasis has been placed on evidence from crash risk studies.

The specific unit of measure of crash involvement reported in the literature varies. Some examples are:

- Crash involvement or non-involvement;
- Number of crashes (and or ‘near misses’);
- At-fault crashes vs not at-fault;
- Crash types;
- Severity of crash (e.g. fatality vs injury crashes vs property damage crash vs all crashes).

Involvement in motor vehicle crashes may be determined from various sources including:

- Hospital and other injury databases;
- Official crash databases such as police records (which have different criteria for inclusion);
- Self-report;
- Report by “significant other” (e.g. carer/vehicle occupant etc).

There is also considerable variation in the way that crash measures are determined. For example, crash involvement may or may not be corrected for exposure in a variety of ways. Some methods of correcting for exposure of drivers include:

- Crashes per kilometres driven;
- Crashes per licence holder;
- Crashes per head of population;
- Crashes per year of driving.

Driving citations

Official records of driving citations (violations and infringements) are maintained in most jurisdictions and offer a useful source of information about driver performance. However, the extent to which driving infringements may be predictive of future crashes is a matter of some debate. Particular types of infringements that are most relevant to crash risk are:

- speeding;
- ‘dangerous’ driving;
- alcohol and drug related infringements.

Adequacy of official records and self- reports of road safety outcome measures

Official records of crashes and driving citations are a critical source of data for understanding the relationship between medical conditions and road safety risk. It is

important to point out, however, that these records are not without bias. Indeed, in terms of estimation of crash fault, for example, the validity of the data is dependent on judgements made by the attending police usually at the time of the crash. It is possible that police reports of 'fault' may be biased towards the young and older drivers. In addition, estimates of injury severity at the time of the crash may be relatively crude (e.g. hospitalised/non-hospitalised) and subject to inaccuracy, given that the severity of injury may not become clear until sometime after the event. There is also an inherent age bias when considering injury severity as older crash victims will incur more severe injuries and higher death rates from a given incident compared to younger drivers. Driving citations are also subject to bias. The most obvious example here is that, given the ratio of police to drivers on most of our road systems, not all driving violations come to the attention of the police. Moreover, frequency of reporting of driving offences tends to be influenced by specific enforcement policy.

Some researchers have claimed that self-reports are likely to show greater levels of involvement than official records. This is attributed to a propensity for reporting minor crashes, usually in which injuries, if any, do not require hospitalisation or medical treatment and generally do not come to the attention of police nor are they recorded on official databases. However, another consideration is that individuals with medical illness may be less likely to report crashes for fear of licence revocation. Self-report is also potentially limited because it is only as accurate as the reporter's memory of the event and this is likely to change with time since the event. In some cases, the nature of the driver's impairment may diminish the reliability of the reporting (e.g. where cognitive impairment is implicated), hence the use for corroborative evidence from carers. It is also true that in some cases, driver injury is determined only *after* the official crash event is recorded and so the 'true severity' of the crash may be underestimated in the official crash database.

Another area for potential bias in study findings is the duration and timing of the study period during which crashes and citations are recorded. The majority of studies use retrospective designs so that crash records for a designated period *prior to* recruitment into the study are analysed. Often the study period is up to 5 years duration. This approach fails to take into account any changes in severity of impairments across the period of study. This is particularly relevant for progressive or degenerative conditions. Similarly, such retrospective approaches fail to take account of other important variables such as changes in compensatory or self-regulatory behaviours across the study period.

There are also methodological issues in establishing whether drivers with a specific medical condition are more vulnerable to injury in the event of a crash, because of the pre-existing condition. Predisposition or vulnerability to injury should be distinguished from crash risk; however, the literature rarely addresses this issue.

Driving performance

A less direct method of assessment of risk can be obtained by examining driving performance, either in real world on-road environments or in a simulated environment. Use of simulators is an increasingly popular method for assessing driver abilities. There are many advantages of using driving simulation to measure risk. For example, the effects of disease and treatments such as hypoglycaemia and sleep deprivation can be studied in a safe, off-road environment. The simulated vehicle also provides opportunity to manipulate aspects of the road environment and to record objective measures of

driving performance including steering, braking, near misses and crashes. Previous studies have shown that driving behaviour in simulators is very similar to that in the real world for selected performance measures (Mullen, Charlton, Devlin & Bedard, in press). On-road testing has the drawbacks of being expensive and difficult to replicate. In addition, performance measures of interest are difficult to observe in the real world driving setting, even with instrumented vehicles, largely because particular conditions of interest cannot be controlled and events such as near misses and crashes are comparatively rare. Importantly, as already noted, on-road experimental work may predispose participants with severe impairments to a level of risk that may be considered unethical. The one distinct disadvantage of simulator driving performance relates to the inherent compromise in ecological validity of the simulated road and traffic environment and the ability to make generalisations to real-world crash risk. As yet, there is little research into the predictive validity of simulator evaluation, although some studies do suggest a correlation between simulator behaviour and actual driving performance (Galski, Bruno & Ehle, 1993). Recent improvements in technology have led to increasingly better simulators being available to researchers, and the findings from future studies are likely to be a lot more reliable and valid due to these improvements. That is to say the ecological validity of the simulator experience will be greater.

2.2 DEFINITION OF THE STUDY POPULATION

The vast majority of studies examining medical conditions and driver risk are cohort or case-control studies, in which a comparison is made between two groups: ‘cases’, or individuals with the medical condition of interest, and one or more control groups, who are matched with cases on key variables. Critical variables may include age, sex, marital status, socio-economic group, ethnicity and place of residence. If appropriate matching is not applied at the time of recruitment, the unmatched variables may confound findings unless adequate post-hoc controls are applied using statistical adjustments.

Recruitment of participants

Bias may arise as a result of inadequate or inappropriate recruitment protocols. Population-based studies that include either the entire population of interest (e.g. all drivers in a particular jurisdiction known to have a specific medical condition) or random selection of a large number of participants from the population of interest provide the strongest recruitment approaches with least potential for bias. Examples of recruitment methods that are likely to result in bias include advertising for volunteers in a local newspaper or recruitment from a single clinic or limited geographic locality that is not representative of the population of interest. Furthermore, recruitment could be affected by an element of selection bias, in that individuals who elect to volunteer for studies are typically more able and healthier than the general population, and consequently more confident about driving.

A problematic method observed in some studies is case recruitment of individuals with a medical condition who are referred for poor driving performance (e.g. by physician, family or police). This is likely to yield a more severely impaired group of cases who are pre-selected for their ‘poor driving’. This approach excludes other potential cases in the population of interest who may not have come to the attention of the referring parties. This approach is likely to bias the findings towards an over-estimation of crash risk in the population of interest.

Diagnostic criteria

Lack of agreement about how cases are defined or diagnosed makes it difficult to compare findings in the research literature. Some medical conditions are difficult to diagnose, such as Alzheimer's disease, and in some cases there may be no standard diagnostic criteria and/or lack of uniformity across studies in applying standard criteria. Lack of precise sample inclusion criteria and a failure to use standardised diagnostic criteria may result in inherent biases in some studies. Importantly, in many studies, the 'purity' or homogeneity of both 'cases' (those with the medical condition) and controls (those without the condition) is, at best, questionable.

A further complication is that medical conditions may remain undetected in the general population. For example it is estimated that only 50% of cases of type II diabetes mellitus are detected (Australian Institute of Health and Welfare, AIHW, 2002). This is likely to mean that cases will be under-representative and controls (those without the disorder) are contaminated with undiagnosed cases.

Other methodological shortcomings in this field of research include the failure to account for:

- Severity of the disorder(s);
- Disease progression;
- Comorbidity (i.e., co-existing conditions).

The severity of a given condition (e.g. mild vs severe cerebrovascular disease) as well as the presence of other conditions (e.g. diabetes, heart disease, epilepsy) may result in an increased risk of crash over and above the risk associated with any one of these conditions.

Inclusion of participants with comorbid conditions is not in itself a problem. Rather, this must be addressed using appropriate methodological procedures. For example, in some studies those with and without comorbid conditions are included and appropriate statistical adjustments are made for these 'other' conditions when determining risk. In other studies, sampling procedures are used to exclude individuals with 'other' conditions from cases and control groups. What is worrisome is the failure to identify the presence of comorbid conditions in the sample.

Adequacy of official records and self-reports of medical conditions

Detailed investigation of crash involvement and medical conditions has been hampered by a paucity of data in crash and injury databases on preexisting medical conditions of drivers. Potentially, crash databases are a rich source of information regarding crash causality, crash type and severity, injury type and severity. In reality, these databases have numerous shortcomings, some of which were discussed in the previous section. Notwithstanding the inherent problems associated with these databases, much has been learned about driver characteristics (age, sex, BAC etc) and crashes from a detailed interrogation of crash databases. However, information about driver medical conditions is rarely recorded in such databases. Instead, researchers have had to rely on multiple alternative sources that record various data of interest. These data sources must then be linked retrospectively, usually by matching the cases in independent databases to

licence holders. In this way, crash events for licence holders (including those with 'restricted' or 'conditional' licences) can be matched with records of driver medical status held by the licensing authority.

Many of the studies reviewed here have relied on participant questionnaires to elicit information such as the presence of a particular medical condition or impairments as well as the type and severity of the condition and time since onset. This method of identification of cases is less reliable and likely to be biased towards under-reporting compared with clinical diagnosis.

Official databases are also subject to bias. Some driver licensing databases rely on drivers to report that they have a diagnosed medical condition (e.g. Vernon et al., 2000). There is little doubt that not all drivers report their medical condition to the licensing authority. Hansotia and Broste (1991) also note that drivers with medical conditions who come to the attention of the licensing authority are also likely to be those with the most severe forms of the disease. This may lead to under-representation of crash risk because mild forms of the condition are not included. The consequence of this is that not only are 'case' samples likely to be under-representative of the true population of individuals with the condition; but also control groups of individuals who are assumed not to have the condition may indeed include true cases.

Chronic illness and functional impairment

As noted in Chapter 1, studies on this topic primarily have addressed the question of risk with reference to specific medical conditions, diseases or illnesses (see Chapter 3). Few studies have addressed the risk associated with functional impairments directly although exceptions are noted in the area of vision (e.g. the association between crashes and visual field loss) and dementia, where more careful assessment of cognitive functions may be conducted. One important study that does address this issue, conducted by Vernon and colleagues (2002) studied a large sample of drivers in the State of Utah in the US. The study sample included drivers with specific medical conditions, known to the authorities, and who were rated on a 12-point scale for severity of impairment. Other than this, few studies have considered whether drivers are able to adequately compensate for their condition/impairment. While disease severity is an important factor, it is also important to consider the extent to which individuals with a given disorder are able to compensate for impairments through various treatments and strategies. For example, an individual with severe arthritis may be unable to safely operate vehicle foot controls, but with appropriate modifications to the vehicle (hand controls), the driver's crash risk may not be affected.

Defining an appropriate control group

In the same way that there is wide variation in the body of literature in selection criteria for cases with medical conditions and impairments, so too, there is little uniformity in the selection of control groups. Examples include:

- Drivers without the medical condition of interest;
- Drivers without any medical conditions;
- Population of all drivers from which cases are selected;

- Spouses and other samples of convenience without the disorder;
- The same case group during/after a particular treatment (i.e. cases act as their own controls, off and on treatment).

2.3 CHRONIC VERSUS ACUTE EFFECTS OF MEDICAL CONDITIONS

Another important consideration is the risk associated with *chronic* illness, which may permanently impair drivers' ability, versus the risk associated with *acute* illness and temporary/acute incapacitation in traffic.

Methodologically, it may be difficult to tease apart the chronic effects of the condition that underlie the effect of acute incapacitation. A pertinent example can be seen in diabetes:

- Chronic effects might include the effects of complications such as neuropathy and associated sensory loss, retinopathy and associated vision impairment, or cognitive impairment from multiple hypoglycaemic reactions;
- These effects can be contrasted with the acute effects of a severe hypoglycaemic reaction, which may result in temporary cognitive impairment, loss of alertness or a loss of consciousness.

2.4 STATISTICAL ANALYSES

A wide variety of statistical procedures have been used throughout the research literature linking crashes to medical conditions. The two most frequently reported statistical measures are:

- Odds ratio (OR);
- Relative risk (RR).

Relative risk has become a standard measure of risk in epidemiological and medical research and usually refers to the "risk of the outcome in one group compared with another group and is expressed as the risk ratio in cohort studies and clinical trials. When the risk ratio cannot be obtained directly (such as in a case-control study), the odds ratio is calculated and often interpreted as if it were the risk ratio" (Zhang & Yu, 1998, p. 1690).

In both cases, a ratio of 1 indicates no difference, whereas a ratio greater than 1 indicates an increased risk in the group being studied and a ratio less than 1 indicates a lower risk in the group being studied. The following computational descriptions summarise how RR and OR computations are calculated:

	Outcome occurred	Outcome did not occur	Total
Exposed	A	B	A+B
Not exposed	C	D	C+D
Total	A+C	B+D	A+B+C+D

The risk ratio is equal to $(A/(A+B)) / (C/(C+D))$. The odds ratio is equal to $(A/C)/(B/D)$ or, equivalently, AD/BC .

Odds ratios are often erroneously interpreted as relative risks in the research literature. Odds ratios can be approximated to relative risks – that is, taken as a direct probability of crash involvement (or risk ratio) – only when three conditions are met Gordis, (2004):

- The cases are representative of the population being studied;
- The controls are representative of the population being studied;
- The outcome measure being studied (eg crash involvement) is rare.

Other statistical procedures used in the literature include simple bivariate statistical procedures for comparison of case and control groups (eg. Chi Square, t-test and analysis of variance) and more sophisticated regression modelling in which crashes and other road safety outcome measures are predicted, with adjustments for factors such as age, gender, comorbidity and exposure may or may not be included. These differences in application of statistical procedures add to the complexity of comparisons across research studies.

As noted above, the majority of studies described in this review used a case-control design, where a comparison is made between the rates of road safety outcomes (e.g. crashes) of those with the condition of interest with drivers without the condition. However, several studies have considered the question of risk from the inverse perspective. That is, by examining the prevalence of a particular medical condition amongst drivers who are pre-selected on the basis of their road safety outcome; for example, drivers with and without a crash record. Hence, RR findings from these studies refer to the likelihood of finding a driver with the medical condition of interest amongst crash-involved cases relative to non crash-involved controls. It is important to note that the RR of a medical condition amongst crash cases cannot be compared with the RR of a crash amongst cases with a medical condition, although it is possible to draw common conclusions about the relationship between crashes and medical conditions from both types of studies.

2.5 SCOPE AND LIMITATIONS OF THE REVIEW

The review provides a brief overview of the nature of selected medical conditions and prevalence in selected developed countries and regions of interest (e.g. Europe, US, Australia). Medical complications and functional impairments associated with the

disorder, disease or condition are highlighted. It is not intended that the review cover detailed medical information about the condition or in-depth discussion of current treatments and management strategies. Rather the focus is on driver risk associated with specific conditions and approaches to management. In particular, when evaluating risk, emphasis is given to studies measuring crash involvement rather than citations or driving performance, which, as previously discussed, provide less convincing evidence of risk of future crash involvement.

While there are innumerable medical conditions and a vast array of associated functional impairments worthy of inclusion, of necessity, this review was limited to the following selected medical conditions:

- 3.1 Alcohol abuse and Alcohol dependence³;
- 3.2 Cardiovascular disease (including syncope, arrhythmias, coronary artery disease);
- 3.3 Cerebrovascular accident (CVA or stroke);
- 3.4 Cognitive impairment (including Alzheimer's disease and traumatic brain injury (TBI));
- 3.5 Diabetes Mellitus;
- 3.6 Epilepsy and seizure disorders;
- 3.7 Musculoskeletal disorders;
- 3.8 Neurological disorders (including Parkinson's disease, Multiple Sclerosis, cerebral palsy and spina bifida);
- 3.9 Psychiatric illnesses (including schizophrenia, depression, anxiety disorders, personality disorders, attention deficit and hyperactivity disorder);
- 3.10 Respiratory disorders;
- 3.11 Sleep apnoea and related disorders;
- 3.12 Vestibular (balance) disorders;
- 3.13 Vision disorders.

Selection of conditions was based on:

- Key medical conditions that were identified by the Expert Panel;
- Conditions that were identified in a number of medical fitness to drive guidelines from Europe, Australia, USA and New Zealand;
- Availability of scientific evidence;

³ Not updated in the current edition of the report due to resource limitations.

- Available time and resources for the review process.

2.6 LITERATURE SEARCH STRATEGY

Keywords

In consultation with an expert panel of medical practitioners and licensing policy professionals, a list of keywords and phrases was generated for searching databases of scientific literature (see Appendix A).

Search databases

The following databases were used to identify relevant scientific literature for both the 2004 report and the current edition:

- PsychInfo;
- Medline;
- The Cochrane Library;
- Australian Transport Index (ATRI);
- Transport CD Rom, a database combining the
 - Transportation Research Information Services (TRIS) database (US), and
 - International Transport Research Documentation (ITRD) database;
- Bibliography of research of medical and cognitive conditions affecting driver fitness (British Columbia Ministry of Transportation and Highways, 2000).

Additional sources included in the literature search for the 2009 update were:

- Pubmed;
- ScienceDirect.

The search for the 2004 report was conducted for relevant publications in the interval from 1980 to May-2003. The current search covered the period from January 2003 until mid-2009. Search terms included terms for the specific diseases, conditions and impairments, driving, crashes, and accidents with some searches having additional qualifiers, e.g., prevalence, later than 1980. In addition, cross-referencing was conducted, to include all relevant studies that appeared in reference lists of papers identified in the original search and which met the specification. Only full articles, with an emphasis on empirical studies and not abstracts of papers were reviewed. Some review papers and editorials were also included to capture historical context and current opinion. Searches were restricted to English language publications. Searches were also performed on authors' names that were well published in the area. In addition, web sites of reputable organisations were searched for general information on medical conditions.

Search results

The search strategy described above yielded in excess of 600 references for the 2004 report, of which approximately 530 were included, after reviewing for relevance. For the current report, 800 references were identified in the initial search with approximately 600 deemed relevant based on title and abstracts. The final number of relevant articles reviewed was 147. The conditions attracting the most new research relating to road safety risk were: dementia (27), Parkinson's disease (18), psychiatric disorders (12), ADHD (14), sleep disorders (16) and vision disorders (17). The majority of these references were papers in scientific journals, which described studies relating to risk. Other documents included review papers, editorials and other brief notes or commentaries in scientific journals as well as textbooks, reports and websites of reputable organisations.

Critical review of scientific literature

In this review, we included research papers meeting the above-described eligibility criteria (type of condition, date of publication, database source, publication type and addressing the specific research question on risk associated with selected medical conditions). Papers were reviewed using broad principles underpinning evidence-based science as specified by National Health and Medical Research Council (NHMRC, 1995). Higgins and Green (2008) highlight the importance of a clearly defined and reproducible methodology with pre-defined eligibility criteria and a method for assessing bias and a systematic way of synthesizing and presenting the findings. In this review, the quality of evidence was rated by examining papers for:

- Avoidance of systematic bias (any procedure that distorts comparison between groups or erroneously influences conclusions about groups) in:
 - Recruitment procedures, inclusion/exclusion criteria, control for confounding variables;
- Use of valid outcome measures;
- Adequacy of sample size for high chance of detecting a difference if it truly exists.

To assist the critical reviewing process for the 2009 update, a checklist was provided to reviewers/authors (see Appendix B for details). Reviewers were asked to note the type of study (case-control, cross-sectional, cohort, review or other) and note whether the definition of the condition used was consistent with the definition accepted within the medical field. Next, authors/reviewers were asked to consider whether the method used to assess outcome of the study was adequate, giving consideration to potential methodological biases which may have been present. Aspects of the study design were also considered, including potential bias in sample; adequacy of control group matching; adequacy of the sample size to draw relevant inferences; adequacy of the description of data sources; and adequacy of control for potential confounds. Review of the results sections involved critique of the statistical and analytic techniques used, and use of Relative Risk or Odds Ratios to identify the risk status associated with each medical condition. Finally reviewers were asked to judge whether the interpretations and conclusions made by the article authors were justifiable; whether the limitations of

the study were addressed adequately and subsequently, to rate the empirical strength of the study on a three-point scale.

References

- Australian Institute of Health and Welfare (2002) *Diabetes: Australian Facts 2002*, www.aihw.gov.au/publications/cvd/daf02/daf02-c01.pdf.
- Gordis, L. (2004). *Epidemiology*. Elsevier, Philadelphia, Pennsylvania.
- Galski, T., Bruno, R.L. & Ehle, H.T. (1993). Prediction of behind-the-wheel driving performance in participants with cerebral damage: A discriminant function analysis. *American Journal of Occupational Therapy*, 47, 391-396.
- Hansotia, B. & Broste, S.K. (1991). The effect of epilepsy or diabetes mellitus on the risk of automobile accidents. *The New England Journal of Medicine*, 324(1), 22-26.
- Higgins, J.P.T. & Green, S. (2008). *Cochrane Handbook for Systematic Reviews of Interventions*. The Cochrane Collaboration and Wiley-Blackwell, Chichester, England.
- McGwin Jr, G., Sims, R.V., Pulley, L. & Roseman, J.M. (2000). Relations among Chronic medical conditions, medications and automobile crashes in the elderly: A population-based case-control study. *American Journal of Epidemiology*, 152 (5), 424-431.
- Mullen, N., Charlton, J.L., Devlin, A., Bédard M. (in press). Simulator Validity: Behaviors Observed on the Simulator and on the Road. In *Handbook of Driving Simulation for Engineering, Medicine and Psychology*. D. Fisher, M., Rizzo, J Caird & JD Lee (Eds). Chapter 19.
- NHMRC (1995). Evidence based medicine (EBM). Quality of evidence ratings. Accessed on 4th March 2003 from <http://www.qvhs.com.au/Dict-Ref/ebm.htm>
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J.M. (2002). Evaluating the crash and citations rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, 34, 237-246.
- Zhang, J. & Yu, K. F. (1998) What's the Relative Risk? A Method of Correcting the Odds Ratio in Cohort Studies of Common Outcomes, *JAMA*. 280(19):1690-1691.

CHAPTER 3 REVIEW OF SPECIFIC MEDICAL CONDITIONS: CRASH RISK AND APPROACHES TO MANAGEMENT

In this chapter, specific medical conditions of interest in the context of driving are defined briefly and their prevalence in Western developed countries identified. This section is not intended to provide a detailed description of the aetiology, pathology and medical treatment of the conditions, but rather to provide a brief account of the nature of the problem and the kinds of functional impairments that may impact upon driving. Next, evidence for driver risk, with the major emphasis on motor vehicle crashes is reviewed. Each section concludes with a discussion of management issues including a review of selected guidelines for assessing fitness to drive from six jurisdictions (Canada, Australia, United Kingdom, United States of America, New Zealand and Sweden. More information on the licensing classifications and guidelines for each jurisdiction can be found in Appendix C). Of particular interest is the level of agreement between these guidelines and scientific evidence of driver risk. In addition, issues relating to self-regulation and decisions about limiting or ceasing driving are considered. These should be important considerations for clinicians and licensing authorities when making decisions as to whether particular individuals should be allowed to continue driving, with or without special driving restrictions or conditions placed on them.

In this chapter, Sections 3.2 to 3.13 summarise evidence reviewed in Charlton et al 2004 as well as evidence from studies published post-May 2003. It was not possible to update the review of literature on Section 3.1 on alcohol abuse and dependence within the scope and available resources for this project and hence, this section presents evidence from studies pre-May 2003 only.

3.1 ALCOHOL ABUSE AND ALCOHOL DEPENDENCE

According to the Diagnostic and Statistical Manual of Mental Disorders criteria (DSM-IV), substance abuse disorders are defined as a maladaptive pattern of behaviour leading to clinically significant impairment or distress, as manifested by one (or more) of the following symptoms: recurrent substance use resulting in failure to fulfil role obligations; use in situations in which it is hazardous; substance-related legal problems; and continued use despite recurrent social or occupational problems caused by the substance (American Psychological Association, 1994). Substance abuse disorders can result from the use of alcohol or illicit drugs, or indeed prescribed drugs.

Consumption of alcohol and its effects on central nervous system are widely recognised and the relationship between alcohol use and impaired driving ability has been well documented (Mitchell, 1985). The relationship between raised levels of alcohol in the blood and increased crash risk has been recognised for many years, and it has been estimated that driving whilst intoxicated contributes to 30-50% of fatal crashes, 15-35% of injurious crashes, and 10% of non-injurious crashes (Council For Scientific Affairs, 1986). In the state of Victoria in Australia in 2002, 72 of the 186 drivers killed (39%) died with a blood alcohol content (BAC) over the legal limit for unrestricted drivers of 0.05g/100ml; over half were more than 3 times over the legal limit.

When considering the relationship between alcohol use (and abuse) and driving it is necessary to differentiate between two different ways in which alcohol leads to increased crash risk:

- Reduced capability in the long term, that is alcohol dependency and its long term physical and cognitive effects;
- Reduced capability in the short-term, i.e. alcohol intoxication with or without dependence.

The two are not entirely mutually exclusive; it is possible for a long-term alcohol-dependent person to be involved in a crash purely due to reduced capability from the effects of recent alcohol consumption, over and above any long standing problems.

Short term alcohol use and drink driving, although a serious problem and a major contributor to road crashes, is not a “chronic” illness and is widely discussed elsewhere (see Ferguson, Sheehan, Davey & Watson, 1999; Mitchell, 1985 for a review). For the purposes of the present review, the primary focus will be on long term alcohol use and abuse and the effects of this on driving ability either directly or indirectly. However, as discussed below, methodological limitations in the research in this area make it difficult to make a clear distinction between the long term effects of alcohol and the temporary effects of alcohol consumption in drivers with alcohol abuse disorder.

Definition of alcohol abuse and alcohol dependence

The DSM-IV classifies two types of problem alcohol use: abuse and dependence (APA, 1994). Alcohol *abuse* is characterised by continued use that has a negative effect on a person's life. Alcohol *dependence* includes abuse plus the physiologic properties of tolerance and withdrawal. In order for a DSM-IV diagnosis of alcohol dependence, an individual must demonstrate a maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

- Tolerance, as defined by either of the following: a need for markedly increased amounts of the substance to achieve intoxication or desired effect markedly diminished effect with continued use of the same amount of substance;
- Withdrawal, as manifested by either of the following: the characteristic withdrawal syndrome for the substance the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms;
- The substance is often taken in larger amounts or over a longer period than was intended;
- There is a persistent desire or unsuccessful efforts to cut down or control substance use;
- A great deal of time is spent in activities to obtain the substance, use the substance, or recover from its effects;
- Important social, occupational or recreational activities are given up or reduced because of substance use;

- The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., continued drinking despite recognition that an ulcer was made worse by alcohol consumption).

Similarly, the World Health Organisation (WHO) International Classification of Diseases – Tenth Revision (ICD-10) defines alcohol dependence as an interrelated cluster of psychological symptoms, such as craving; physiological signs, such as tolerance and withdrawal; and behavioural indicators, such as the use of alcohol to relieve withdrawal discomfort (WHO, 2002). However, in a departure from the DSM, ICD-10 includes the concept of *harmful use* rather than alcohol abuse. Harmful use implies alcohol use that causes either physical or mental damage in the absence of dependence.

Prevalence of alcohol abuse and alcohol dependence

The World Health Organization (WHO) estimates that the prevalence of individuals with alcohol use disorders, which covers both harmful use and dependence as defined by ICD-10 (Code F10.1 and 10.2), is approximately 75.4 million worldwide (Mathers, Stein, Ma Fat, Rao, Inoue, Tomijima, Bernard, Lopez, & Murray, 2002). The WHO also estimates that the proportion of men affected by alcohol use disorders is overwhelmingly higher than women. In 2000, the prevalence of the disease in Western European countries (EURO A group, which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated at around 2% of this population (approximately 8.7 million). These figures vary for different countries. For example, for North American countries (AMRO A group, which includes Canada, U.S. and Cuba) estimates are slightly higher at approximately 3% of the population. The United States National Institute on Alcohol Abuse and Alcoholism report on the National Longitudinal Alcohol Epidemiologic Survey (NLAES) estimates that 13,760,000 adults in the U.S.A. (7.41% of persons aged 18 years and older) met standard diagnostic criteria (DSM-IV) for alcohol abuse or alcohol dependence during 1992 (Grant, Hartford & Dawson, 1994). Prevalence of alcohol dependence was 4.38% while alcohol abuse was 3.03%. Most persons with alcohol dependence also met alcohol abuse criteria. Alcohol use disorder rates were higher among males (11.0%) than females (4.08%) and highest amongst the 18-29 year old group (15.94%). It is noted that these figures are considerably higher than WHO estimates. This may be in part explained by differences in inclusion categories (alcohol abuse and dependence versus harmful use and dependence for NLAES and WHO figures, respectively). In addition, the two sets of data are based on different population bases and different diagnostic criteria. As noted by Brinkmann and colleagues, ICD-10, used by WHO, will yield lower rates than DSM-IV which was used for the NLAES data (Brinkmann, Beike, Köhler, Heinecke & Bajanowski 2002).

Functional impairments associated with alcohol abuse relevant to driving

Neurocognitive deficits are a common and potentially severe consequence of long-term, heavy alcohol consumption (see Bates, Bowden & Barry, 2002 for a review; Fox, Coltheart, Solowij, Michie & Fox, 2000; Beatty, Katzung, Moreland & Nixon, 1995).

Research has shown that individuals who abuse alcohol have widespread, multifaceted impairments in many domains of cognitive function, including:

- Short-term memory and learning impairments, which become more evident as the task difficulty increases;
- Impaired perceptual-motor speed;
- Impairments in visual search and scanning strategies;
- Peripheral neuropathies experienced as numbness or paraesthesias of the hands or feet;
- Deficits in executive functions such as mental flexibility, problem solving skills, difficulty in planning, organising and prioritising tasks, difficulty focussing attention, sustaining focus, shifting focus from one task to another, or filtering out distractions, difficulty monitoring and regulating self-action and impulsivity.

Autopsy results show evidence of greater cerebral atrophy and smaller brain volume in individuals who exhibit chronic alcohol abuse compared to non-alcoholic adults of similar age and gender. The findings of brain imaging techniques consistently show an association between heavy drinking and physical brain degeneration, even in the absence of liver disease or dementia. Brain atrophy is especially extensive in the cortex of the frontal lobe, an area responsible for many higher-order cognitive functions (Anstey et al., 2006).

Ratti, Bo, Giardini and Soragna (2002) reported a study that attempted to identify the pattern of executive function impairment in chronic alcoholism. Executive function (or frontal lobe function) is generally accepted to play a role in cognitive flexibility, attention resource allocation, and speed of information processing, planning, perceptual motor speed and suppression of task irrelevant information. All of these abilities have apparent relevance to successful driving, and impairments have the potential to increase crash risk. The sample of 22 male participants with alcoholism and 22 controls matched for age and general ability were administered a battery of neuropsychological tests, aimed at assessing the above abilities. They found that the alcoholic group showed poorer performance in almost every functional ability assessed. Importantly, participants with alcoholism were particularly impaired in tests that assessed both cognitive and motor performance (e.g. digit cancellation and reaction time), impairment being most pronounced in tests of cognitive processing speed. These deficits are important to driving, and must potentially increase crash risk. However the small sample size, and the fact that they were all male, weakens the findings somewhat, however these results do point to an important area of research in relation to driving ability and crash risk.

DeFranco, Tarbox & McLaughlin (1985) examined the relationship between duration of alcohol abuse and cognitive impairment. They tested 125 participants aged 40-50 years at an inpatient alcohol treatment clinic. Participants were assigned to short-term or long-term groups using a median split at 5 years of problem drinking. It should be noted that the median split used here was an arbitrary decision, and does not represent a genuine cut-off point between short and long-term alcohol abuse. Standardised tests were used to measure: global cognitive functioning (e.g., Wechsler Adults Intelligence Scale, WAIS), perceptual motor function (trail making), memory function (Wechsler Memory scale, WMS), and visual function (Benton VRT). The long-term group showed significantly greater deficits, particularly in visuo-spatial ability, psychomotor speed

and general cognitive function. All of these have been implemented in decrements in driving performance (see sections 3.3 and 3.4). Due to the limited age range of participants and the arbitrary criteria for determining long and short term alcohol abuse, these findings should be considered cautiously. Nevertheless, the results are suggestive of increased decrements in performance with problem drinkers of 5 years compared with those with a shorter history of problem drinking.

In addition to cognitive changes associated with alcohol abuse, other factors have been implicated in both the involvement of alcoholics in crashes and their ability to recover from injury. Beirness (1993) proposed that existing personality factors such as hostility and aggression may interact with depressive effects of alcohol and may contribute to the vulnerability of alcoholics to crashes. Waller and colleagues have also noted that there is growing evidence that alcohol increases the level of injury and that long term use of alcohol can result in increased bone fragility and impaired liver function and generally impedes injury recovery following trauma (Waller, Blow, Maio, Singer, Hill & Schaefer, 1995).

Korsakoff's syndrome

The most striking neuropsychological deficit associated with alcoholism is the gross memory impairment of Korsakoff's syndrome (sometimes called Wernicke-Korsakoff's syndrome) (Lezak, 1995). Korsakoff's syndrome (KS) is an organic brain disease (psychosis) brought on by prolonged heavy alcohol use in conjunction with severe thiamine (Vitamin B-1) deficiency. Thiamine is used in maintenance of circulation, neurotransmitter synthesis, and has been implicated in efficiency of memory and learning, with the degree of cognitive impairment related to frequency, quantity and duration of alcohol consumption (Krabbendam, Visser, Derix, Verhey, Hofman, Verhoeven, Tuinier & Jolles, 2000). Individuals with KS typically demonstrate those functional impairments associated with chronic alcohol abuse (see above), as well as:

- Anterograde amnesia (an inability to form new memories);
- Retrograde amnesia (an inability to retrieve long-term memories);
- Plausible confabulations (honest lying).

Krabbendam et al. (2000) described a study designed to contrast cognitive impairment of chronic alcoholics with impairment associated with Korsakoff's Syndrome. Neuropsychological profiles and Magnetic Resonance Imaging (MRI) scans of brain structure were obtained for 14 participants with KS, 15 participants with chronic alcoholism (CA) and 16 control participants. While the CA group showed normal cognitive performance and brain structure volumes, participants with KS showed deficits in visuoperceptual performance, executive function, memory as well as diminished volumes of specific brain structures. The primary specific impairments that are of concern in regard to the ability to drive safely include inattentiveness, disorientation in place situation and time, as well as retrograde amnesia. Added to this, confabulation and inappropriate emotional responses such as cheerfulness may occur. This may pose problems not only in medical history taking, but also in fitness for interview by police and crash investigators. Korsakoff's syndrome is not unique to long term chronic alcoholics. It has been also shown to exist in young heavy drinkers. These findings indicate that chronic alcohol consumption is a contributory factor in the

deficits. Relatively low participant numbers weaken the statistical analysis employed in this study and the use of self report also implies a potential reporting bias. The role of comorbidity should not be overlooked since the study groups were not screened in the same way as controls for the presence of other medical conditions. Further, the classification of KS as an organic psychosis, that is to say a brain damaging illness, may also mean that these individuals may have found compensatory strategies for their cognitive deficits.

Summary

Deficits of memory and executive function appear to be the most prevalent impairments associated with chronic alcohol abuse. These functions are central to many tasks in everyday life and are indeed central to a complex task such as driving and are therefore likely to impact on a person's competence to drive safely. A related problem for research in this area is distinguishing the deficits due to alcohol consumption, from those of true dementia or effects of normal ageing in older people. The alcoholic individual often gives the mistaken impression of being more capable than they are, because verbal abilities are among the few cognitive functions that are relatively spared in chronic alcohol abuse. For these reasons it is important for road safety, to be able to identify people with alcoholism and to be able to evaluate their ability to drive when sober, and take appropriate action against those deemed unfit to drive even when sober.

Elevated BAC and functional impairments

Research examining the effect of elevated BAC on cognitive and motor impairments is also instructive in our understanding of risk associated with chronic alcohol abuse, at least for chronic alcoholics driving under the influence of alcohol. This research is reviewed extensively elsewhere (e.g. Mitchell, 1985; Moskowitz & Fiorentino, 2000; Moskowitz, Burns, Fiorentino, Smiley & Zador, 2000). This section summarises two recent studies describing these effects.

Grant, Millar & Kenny (2000) studied the effects of BAC on psychomotor abilities, which, as discussed elsewhere in this report, have been shown to relate to impaired driving ability (see sections 3.4.1 and 3.4.2 for a review of psychomotor impairments in Alzheimer's disease and traumatic brain injury, respectively). Twelve healthy participants were tested on measures of dual task tracking and choice reaction time. Participants' self-reported alcohol history was moderate (range 3-35 units per week). Following pre-tests at zero BAC, participants were given varying doses of alcohol intravenously to allow for exact measurement of quantity. As BAC increased, choice reaction time increased and dual tracking performance decreased significantly. The maximum BAC level of 80mg/100ml, reduced reaction time by 120ms. This can be translated into 4m extra stopping distance at 70 mph. The participants themselves reported by this stage that they felt too impaired to consider driving. The small sample size comprising healthy adults limits the generalisability of these findings to the population of drivers with chronic alcohol abuse who may respond quite differently to equivalent alcohol doses compared with drivers who are not alcoholics.

Fogarty and Vogel-Sprott (2002) also studied performance of healthy males under conditions of moderate BAC (0.62g/kg of absolute alcohol) (n = 10) and placebo or zero BAC conditions (n = 10). Of particular interest was the comparison of effects of BAC on motor performance and cognitive performance. Performance on the motor skills task reflected changes in BAC, with increased impairment as BAC rose (at 7, 25 and 45

minutes after consumption), poorest performance as BAC peaked (at 60 minutes post-consumption) and lessening impairment as BAC declined (at 95 and 115 minutes post-consumption). The cognitive task, requiring rapid information processing, showed no such relationship, rather a more widespread random pattern of impairment. These results were replicated in a second experiment (n = 14 per group). The authors concluded that this mismatch between motor and cognitive performance under a moderate alcohol dose has important safety implications. Level of intoxication is often judged purely on performance of motor tasks, this may fail to detect cognitive impairment that could contribute to the risk of accidents. It should be noted, however, that this study examined performance with moderate alcohol intake only. This is especially important when considering implications of findings for chronic alcoholics since they generally have developed a tolerance to high levels of BAC and, as noted above, may have quite different responses to moderate alcohol intake. This research could also be advanced by studying a larger sample, including women.

Pre-May 2003: Relationship between alcohol abuse and road safety outcomes

There are few studies reported within the designated review period (1980-2003) that directly examine the effects of long-term alcohol abuse on crash or citation rates and driving ability. In an early review of the epidemiology of alcoholism, Vingilis (1983, cited in Soderstrom, Dischinger, Smith, Hebel, McDuff, Gorelick, Kerns, Ho & Read, 2001, p. 771) reported analyses of studies published between 1950-1981 concerning convicted drink drivers and crash involvement. Vingilis concluded that individuals with alcohol dependence, when compared with controls, seemed to be 'high-risk' drivers. This conclusion was based on their higher representation among alcohol-related violations and collisions, as well as over-representation amongst non-alcohol-related violations and crashes compared with controls. Importantly, the author points out that 'although they are as a group at generally higher risk, this does not mean that all alcoholics are drinking drivers and/or high-risk drivers'.

The following review focuses on those studies that have been conducted since 1980. The major findings of these studies are summarised in Table 2 at the end of this section.

Crashes

Del Rio, Gonzalez-Luque and Alvarez (2001) conducted a study that attempted to relate drinking history to frequency of crashes and violations. This study examined the alcohol consumption patterns of 8043 drivers attending 25 Medical Driving Test Centres in Spain, and classified them according to CAGE (test of drinking prevalence) and the incidence of alcohol related problems (DSM-IV criteria for abuse, disorder and alcohol induced disorder). Information on crashes and violations was obtained by self-report. The authors noted that 60.3% of drivers reported that they drink alcohol on a regular basis, with 2% meeting the DSM-IV criteria for alcohol abuse, dependence or induced disorder. When consumption rate was related to traffic accidents, drivers who met the DSM-IV criteria for alcohol abuse, dependence or induced disorder were significantly more likely to have been involved in a traffic accident over the past three years (23.2%) than drivers who did not meet the criteria for alcohol abuse (12.1%, $p < 0.0001$). The authors cite factors such as reduced reaction time and reduced coordination as being responsible for deficits in driving ability. Overall, the findings of this study suggest a two-fold increase in risk of crashes amongst drivers with a diagnosis of alcohol abuse compared with controls. A limitation of the study, however, was the reliance on self-reports from individuals who were being evaluated for renewing or obtaining (first

issue) of licences, this is likely to make them under-report alcohol use. It is also important to note that alcohol abusers are notoriously unreliable historians in general.

In a recent population-based study, Vernon, Diller, Cook, Reading, Suruda and Dean (2002) compared the relative risk of drivers with medical conditions, including alcohol abuse, and those without a medical condition, during a five-year study period from 1992-1996. A retrospective case-control design was used to examine crash and citation rates per 10,000 licence days (Utah Department of Transport official records) for drivers with various medical conditions and a control group of drivers without medical conditions who were matched by age, sex and place of residence. The study population included all drivers licensed in the state of Utah who reported a medical condition when making application for or renewing a licence. The Utah licensing program requires assessment of drivers' severity of disorder and level of impairment, on a scale of 1 to 12. (Levels 1 and 2 are used for commercial drivers only, Levels 3-5 indicate low severity of impairment/high functional ability with no licence restrictions. Levels 6-11 indicate higher severity of impairment/low functional ability and restrictions of licence privileges. Level 12 signifies no driving privileges). For the purposes of the study, drivers with medical conditions were classified in two groups: unrestricted drivers (impairment Levels 3-5) and restricted drivers (Levels 6-11). Restrictions included speed, area, time of day, accompanied by licensed driver, other special limitations. Drivers with a history of drug use or alcohol abuse totalled 149. The majority of these cases (n = 124) had no licensing restrictions.

Overall, the findings showed that drivers with a history of drug use and/or alcohol abuse who were on restricted licences (highest level of impairment) had significantly higher rates of at-fault crashes (RR: 5.75, 95% CI 2.26-14.61) and all crashes (RR: 4.21, 95%CI 1.80-9.85) than controls. In addition, those without licence restrictions (lowest level of impairment) had significantly elevated at-fault crashes and crash rates (RR: 2.22, 95% CI 1.25-3.94, $p < 0.05$; RR: 1.82, 95%CI 1.18-2.81, $p < 0.05$ respectively). Vernon et al. concluded that both unrestricted and restricted drivers with a history of drug use, including alcohol abuse, posed a significantly higher crash risk than controls. However, one of the main limitations of this study was that the authors did not control for driver exposure, which assumes that drivers with medical conditions, such as alcohol abuse, and matched controls drive similar distances. Other serious methodological limitations of this study include the small sample of cases (n = 149). The findings from this study need to be confirmed with a larger sample size, particularly the group of drivers with licence restrictions. In addition, the lack of precise inclusion criteria for identifying alcohol abuse and the inclusion of drug abusers in the same category makes it difficult to compare the study findings with other research literature.

In a study that focused on older drivers, Koepsell and colleagues examined the influence of medical conditions, including alcohol abuse, on the rates of crashes resulting in injury (Koepsell, Wolf, McCloskey, Buchner, Louie, Wagner & Thompson, 1994). Cases and controls were drawn from members of a health plan in the state of Washington, USA. Cases (n = 234) were drivers aged 65 years and older who were involved in injury crashes (1987-88). Controls (n = 446) were matched by age, gender and place of residence and were randomly selected from the same health plan as cases but were not involved in any injury crashes during the study period. Potentially eligible participants were first identified from police reports and confirmed using health records. A survey was conducted with all participants to ascertain information including driving distances and health. For potential participants who had died or who were unable to

complete the survey, survey responses were obtained from a significant other. (Surrogates for the case's matched control were also used). It is important to note that while this study minimised sample bias through use of a population-based design, there remains some potential bias. For example, while the study group is of adequate size, not all of those who were eligible agreed to participate and there were relatively small numbers of drivers with diabetes. Cases and controls represented 75% and 69% of all eligible participants. In addition, the study only investigated drivers who had not had their licence revoked due to a self-reported medical condition or had not voluntarily given up driving.

Koepsell and colleagues found that approximately 3.4% of those who were involved in injury crashes and 1.8% of controls (no injury crash involvement) had a medical diagnosis of alcohol abuse. Appropriate analyses were conducted to control for age, gender and place of residence as well as other potentially confounding factors. The results that alcohol abuse was associated with an increased risk of collision injury of borderline statistical significance (OR: 2.1, 95%CI 0.8 - 6.0). The authors note that adjustment for race, marital status and exposure (miles driven in previous year) resulted in only slight changes in these ORs, although no data are provided. Notwithstanding the relatively small number of drivers with alcohol abuse amongst cases and control groups for this study, these findings suggest a modest relationship between older drivers and injury crashes.

Soderstrom et al. (1997) reported a study that compared prevalence of alcohol dependence or abuse in people in motor vehicle crashes with others not involved in a crash. All participants were attending a trauma clinic. Alcohol abuse or dependence was diagnosed using an interview based around the Substance Abuse section of the DSM-III-R, a widely used diagnostic procedure. At the time of admission, 38% had a diagnosis of lifetime alcoholism and one quarter of the drivers had a diagnosis of current alcoholism (i.e., within the past 6 months). The authors noted that the prevalence of current alcoholism did not vary significantly among the groups of vehicular crash victims (23.5%), other unintentional injury victims (29.3%) and victims of violence (24.6%). Among injured car, truck and motor cycle drivers, approximately 31% of crash involved drivers were diagnosed as lifetime alcohol dependent and 17.2% were found to meet the criteria for current alcohol dependence, rising to 32.6% for men. The authors noted that this latter figure was nearly twice the level of alcoholics diagnosed in a population of (non-crash) convicted drunk drivers (19%) (Miller, Whitney & Washousky, 1986 cited in Soderstrom et al., 1997). In addition, the authors noted that 62% of these crash-involved drivers with alcoholism tested positive to having alcohol in their blood (BAC+) on admission.

The study reported only on the 629 participants admitted to the trauma clinic that were capable of participating, therefore people with severe cognitive deficits through brain injury were omitted, and no proxy data from family were collected. This limits the ability of the study to generalise to all vehicular accident trauma patients. Notwithstanding these limitations, this study provides important information about the prevalence of alcohol dependency amongst drivers involved in injury-related motor vehicle crashes. However, from the data provided, it is not possible to determine the relative risk of crashes amongst alcohol dependent drivers compared with controls.

In another approach to understanding the question of risk, Stevenson, D'Alessandro, Bourke, Legge and Lee (2003) studied alcohol dependency amongst drivers involved in

alcohol-related crashes. The authors conducted a population-based cohort study of 3,286 drivers who were admitted to hospital following a police-attended motor vehicle crash. Alcohol-related crashes were defined as a crash where the driver had a BAC exceeding 0.05gm/100ml, as determined using a calibrated breath test by a police officer. Seven percent of the cohort crashes were classified as alcohol-related (n = 217). Unlike the studies outlined above, the outcome of interest in this study was any subsequent alcohol-related hospital admission, defined as a medical diagnosis that could only have resulted from excessive alcohol consumption. Consequently, drivers were followed over an eight to 13 year period. The authors reported that if the driver was involved in an alcohol-related motor vehicle crash, they were almost twice as likely to have a future alcohol-related hospital admission compared to drivers who were not involved in an alcohol-related crash (OR: 1.96, 95%CI 1.06-3.61). The authors concluded that drink-driving resulting in a motor-vehicle crash and hospitalisation could be considered an indicator of a less overt problem of alcohol dependency. The authors note that this study is limited by the fact that hospitalisations represent one of the most severe outcomes of alcohol-related disease, and therefore the current results will underestimate the true risk value.

Using a different methodology, Bjerre reported that the accident rate for three groups of DWI offenders in Sweden (total n = 3,303) was 4-5 times higher than for the average driver in that country (Bjerre, 2003). Based on police reports, the annual rates of police-reported accidents involving injury ranged between 20 – 22 per 1,000 drivers for the three DWI groups compared with 4 per 1,000 for the population of Swedish drivers. The study also noted a high prevalence of alcohol dependence or alcohol abuse (60%) (DSM-IV criteria) amongst a group of DWI offenders (n = 311) who were participants in an interlock program. While Bjerre's findings do not provide a direct link between alcohol abuse or dependence and crash risk, the findings of over-representation of DWI offenders in crashes coupled with highly elevated numbers diagnosed with alcohol disorders amongst DWI offenders suggests a significant safety concern associated with these disorders.

Focusing on fatal crashes, Hedlund and Fell (1995) estimated the contribution of persistent drink driving to crash rates in the U.S. The study examined data from the National Highway Traffic Safety Administration's (NHTSA) Fatal Accident Reporting System (FARS). The authors reported that while approximately 4% of all licensed drivers had a prior arrest for driving while intoxicated (DWI) within the past three years, 11% of drivers with a BAC level of 0.01 at the time of the crash had a prior DWI and 13% of drivers with a BAC level of 0.10 at the time of the crash had a prior DWI. Hedlund and Fell noted that these findings are consistent with a previous study conducted by Fell (1992, cited in Hedlund & Fell, 1995) who showed that drivers with at least one prior DWI conviction in the past 3 years were over represented in fatal crashes. For example, Fell observed that persistent drinking-drivers were 4.1 times more likely to be involved in a fatal alcohol related crash than first time offenders. It should be noted that FARS data are limited in several important respects: FARS includes only fatal crashes and only contains information from official sources, such as police reports and driver records, and consequently is silent on many important issues. While FARS does contain information on drivers with prior DWI convictions before they had their fatal crash, this is a narrow definition of the persistent drinking driver: convictions, not arrests, within the past three years only. Furthermore, it is not known whether the repeat DWI offenders in this study met standard diagnostic criteria for alcohol abuse.

This finding is consistent with the study conducted by Brewer, Morris, Cole, Watkins, Patetta and Popking (1994) who also reported strong evidence for an elevated risk of dying in a motor vehicle crash among recidivist drink driving offenders. Brewer and colleagues found that compared with drivers killed in non-alcohol-related crashes, drivers aged 21 to 34 years who died in alcohol-related crashes were 4.3 times more likely to have been arrested on a previous DWI offence and those over aged 35 years were 11.7 times more likely to have a previous DWI offence.

The studies by Hedlund and Fell (1995) and Brewer, Morris, Cole, Watkins, Patetta and Popking (1994) both point to a higher risk among recidivist drink drivers of dying in an alcohol-related crash. What is not reported in these studies, however, is whether the recidivist drink drivers had a chronic alcohol problem. Baker, Braver, Chen and Williams (2002) carried out a retrospective study of the drinking histories of 818 fatally injured drivers in the U.S. The study aimed to address whether drivers with high BAC who are killed in motor vehicle crashes are primarily those with a chronic alcohol problem. They compared official driving records, BAC at time of fatal crash, and familial reports of drinking behaviour. Three groups were identified based on their BAC at time of fatal crash: High-Very High BAC (these drivers are over the limit); Low-Moderate BAC, and Zero BAC. They found that the drivers with a very high BAC level at the time of the crash were more likely to be classified as problem drinkers by familial report (31%) than the low-moderate and zero BAC groups (0% and 1% respectively). Problem drinkers were defined by the authors as “a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work or school because of drinking, problems with money because of drinking, or problems with the police because of drinking, such as drunk driving” (p.222). Compared to drivers with a zero BAC level, drivers with a high BAC were: 2.7 times more likely to have had a conviction for driving under the influence three years before the crash (95% confidence intervals: 2.3-3.2); 3.3 times more likely to be described as a problem drinker in their last month of life (95% confidence intervals: 2.8-3.8); 6.8 times more likely to have driven within 2 hours after having 5 or more drinks at least one month during last year of life (95% confidence intervals: 3.3-5.0); 8.1 times more likely to be classified as heavy or very heavy drinkers during their last year (95% confidence intervals: 5.9-11.1); and 4 times more likely to have five or more drinks at a time at least once a month during their last year (95% confidence intervals: 5.0-9.2).

A limitation of the study by Baker and colleagues is the potential for reporting bias; that is, it is likely that family members may report lower incidence of drinking and or drink driving especially in the groups deemed non-problem drinkers. Notwithstanding this limitation, the authors argued that this research suggests a need for widening prevention strategies, especially targeting repeat offenders (e.g. impounding vehicles). Others, on the other hand, have emphasized the need to take seriously the risk of all DWI offenders, particularly first offenders. Rauch and colleagues (2002) make a strong case that most DWI offenders have an extensive history of alcohol-impaired driving by the time of first arrest. This is particularly so due to the very small likelihood of being arrested for such offences. These authors found that first-time alcohol-related traffic offenders are at a significantly high risk of recidivism. They argue, therefore, that high priority should be placed on early intervention and treatment strategies for first offenders.

Citations

In their study outlined above, Del Rio et al. (2001) also investigated the relationship between alcohol abuse and driving infringements in a sample of over 8,000 drivers. The authors reported that drivers who met DSM-IV criteria for alcohol abuse, dependence or induced disorder were significantly more likely to have incurred a traffic infringement or fine over the past three years (18.7%) than drivers who did not meet the criteria for alcohol abuse (9.3%, $p < 0.0001$). As with many other similar studies in this area, the infringement data were gained from self-reports and is therefore susceptible to reporting bias.

Similarly, Vernon et al. (2002) conducted a retrospective case-control study of crash and citation rates of drivers with medical conditions during 1992 – 1996 (see above for details of the study method). Consistent with the findings for crash rates, unrestricted and restricted drivers with a history of drug use or alcohol abuse had significantly elevated citation rates compared to controls (unrestricted: RR: 2.38, 95%CI 1.82-3.12; restricted: RR: 5.83, 95% CI 3.19-10.66, respectively).

Dawson (1999) examined data from a longitudinal research program concerned with alcohol epidemiology, using a sample of 18,352 current drinkers aged over 18 years in the US. US Census Bureau officials collected data through personal interviews at participant's homes. The survey asked respondents about frequency of drinking. Numbers of incidences of driving while impaired were also reported (participants knowingly driving whilst intoxicated, yet not having a driving incident), as were actual driving incidents due to alcohol impairment. The criteria from DSM-IV (APA, 1994) were used to classify alcohol dependence. One tenth of the overall sample was classified as alcohol dependent ($n = 1,067$). Overall, 11.8% of current drinkers reported one or more incidents of impaired driving in the past year, with the mean annual number of impaired driving incidents reported as 0.54. The prevalence of impaired driving for the lowest volume drinkers was 2.5% of respondents, rising to 39.4 per cent of the highest volume drinkers. Dependant drinkers were six times likely to report any impaired driving (46%) compared to those without alcohol dependence (8%). For actual driving incidents, dependent drinkers were ten times as likely to report an incident in the last year (average 3.1) as opposed to non-dependent drinkers (average 0.26).

In another recent study, Cavaiola, Strohmets, Wolf and Lavender (2003) examined the relationship between recidivist drink-driving and chronic alcohol problems. The authors compared a group of DWI offenders with either one ($n = 77$) or multiple DWI offences or repeat offences ($n = 71$) with a group of non-offenders ($n = 61$). The Minnesota Multiphasic Personality Inventory (MMPI) provided an indirect assessment of alcoholic potentiality and the Michigan Alcoholism Screening Test (MAST) provided a more direct measure of problem drinking and alcoholism symptoms. The MAST has been shown to correlate ($r = 0.6$) with DSM-IV (Conley, 2001). The authors reported that the responses of the repeat offenders were similar ($p < 0.06$) to those of self-admitted alcoholics (on the potentiality scales), and that a larger percentage of the multiple offenders (31%) scored in the alcoholic range of the MAST than first offenders (20%). The authors concluded that individuals with multiple DWI offences might be at risk of becoming alcoholic, potentially raising their crash risk. Notwithstanding the limited sample size and reliance on self-reports, this study points to the usefulness of multiple DWI offences as a potential 'flag' for increased crash risk.

Brinkmann, Beike, Köhler, Heinecke and Bajanowski (2002) conducted a study designed to determine the prevalence of alcoholism amongst drivers with drink driving violations. Biological markers of alcoholism identified from blood tests were used to overcome the unreliability of self-reports. This study sought to determine the prevalence of chronic alcoholism amongst drivers with drink driving violations. Using a random sample of 327 drunk drivers (BAC ranging from 0.03 to 3.74), they found that 48% of the drivers would be classified (by German criteria, a combination of 4 biological markers present in blood samples, known as an Alc-Index) as being alcohol dependent. This indicates that the prevalence of problem drinkers amongst those arrested for drunk driving may be far greater than would be uncovered by self-report or interview. This has implications for road safety, as many of these offenders may be habitual drink drivers, and may also demonstrate the cognitive deficits associated with alcoholism. The authors argue that for drivers with moderate to high BACs, additional biological markers of alcoholism should be tested to confirm the initial BAC reading.

Driving performance

No studies reporting the relationship between chronic alcohol abuse and driving performance were found.

Summary

From the review of research pre-May 2003: Despite the strong evidence linking chronic alcohol abuse and cognitive impairment, there is limited available information on the relationship between chronic alcohol abuse and crash risk. Evidence from the three reviewed studies showed that individuals with alcohol dependency have approximately twice the risk of crash involvement as controls. In general, the quality of evidence linking chronic alcohol abuse and crashes is limited by methodological shortcomings. These include limited use of a population based case-control study design, potential reporting bias in self-reported data (medical and crash involvement), use of small samples and inadequate diagnostic criteria, failure to control for exposure, comorbidity and other variables. Large-scale, population-based case-control studies are needed to address these shortcomings.

An important issue identified in this review is the prevalence of “problem” drinkers or alcohol dependence amongst people who are caught drink driving. Studies examining citations, particularly DWIs, indicate that participants with alcohol dependency are more likely to drive while intoxicated despite prior convictions. This may be a result of cognitive impairment through alcohol related brain damage, or may simply be attributable to greater exposure; that is, they are more likely to have consumed levels of alcohol above the legal limits and are therefore more likely to be drunk when driving. Drink driving offenders are often divided into two categories: first time offenders and the recidivist drink driver. The patterns of behaviour and crash risk are likely to be different in people who repeatedly drive under the influence compared with those who have an isolated incident of drink driving. This is clearly expressed by the following: *“A significant proportion of convicted drink-drivers are at serious risk of developing, or have already developed, alcohol-related and other problems. This is particularly so with recurrent offenders...for whom a drink driving conviction is more often an inevitable outcome of well-established habits rather than an isolated ‘unlucky’ event”* (Victorian Social Development Committee, 1988, p. xii).

Table 2 Summary of studies of risk associated with alcohol abuse (pre-May 2003)

Study: Author/date	Methods	Outcome Measure of Risk	Results
Baker et al. (2002)	Retrospective cohort study of 818 fatally injured drivers	<ul style="list-style-type: none"> - official driving records - Problem drinking indicators - BAC level 	<p>31% had very high BACs Drivers with higher BAC at the time were more likely to be alcoholics, as reported by family history. percentages only reported. Compared to drivers with zero BAC, drivers with high BAC were:</p> <ul style="list-style-type: none"> - 2.7 times more likely to have been convicted of drink driving during past three years (95% CI: 2.3-3.2) 3.3 times more likely to be described as a problem drinker in their last month of life (95% CI: 2.8-3.8) - 6.8 times more likely to have driven within 2 hours after having 5 or more drinks at least one month during last year of life (95% CI: 3.3-5.0) - 8.1 times more likely to be classified as heavy or very heavy drinkers during their last year (95% CI: 5.9-11.1) - 4 times more likely as having five or more drinks at a time at least once a month during their last year (95% CI: 5.0-9.2)
Bjerre (2003)	<p>Cases were three groups of DWI offenders: (i) volunteers for an interlock program (n=311), (ii) abstainers from the interlock program (n=625) and (iii) matched participants from other counties in Sweden where the program was not available (n=2,367). Comparison data were population rates for all of Sweden (n=5.6 million)</p>	<p>Injury-crashes based on police reports in 5 year period prior to DWI offence.</p>	<p>Drivers with DWI offences had a 4-5 times higher crash involvement than the average Swedish driver: Annual crash rates per 1000 drivers for three groups of DWI offenders were 22, 21 and 22. Annual crash rates per 1000 drivers for the population of all Swedish drivers was 4.</p>

Study: Author/date	Methods	Outcome Measure of Risk	Results
Brinkmann et al. (2002)	Using a random sample of 327 drunk drivers (BAC ranging from 0.03 to 3.74),	biological markers in blood tests, they found that	48% of the drivers would be classified (by German criteria, a combination of 4 biological markers present in blood samples, known as an Alc-Index) as being alcohol dependent.
Cavaiola, Strohmetz, Wolf & Lavender (2003)	Group of (DWI) offenders: - 1 DWI (n = 77) - multiple DWI offences or repeat offences (n = 71) - group of non-offenders (n=61)	- Minnesota Multiphasic Personality Inventory (MMPI) Michigan Alcoholism Screening Test (MAST)	- individuals with multiple DWI offences may be at risk of becoming alcoholic, potentially raising their crash risk.
Dawson (1999)	Longitudinal study current drinkers aged over 18 in the US (n=18,352) 10% classified as alcohol dependent (n=1067).	Survey data: - frequency of drinking - Number of incidences of driving while impaired - actual incidents due to alcohol impairment. - Alcohol dependence (DSM-IV criteria).	Prevalence of impaired driving for the lowest volume drinkers was 2.5 per cent of respondents, rising to 39.4 per cent of the highest volume drinkers. For actual incidents, dependent drinkers were ten times as likely to report an incident in the last year (average 3.1) as opposed to non-dependent drinkers (average 0.26). However no distinction is made between incidents which occurred while drink driving and those that occurred whilst sober.
Del-Rio et al. (2001)	8043 drivers attending Medical Driving Test Centres: Drivers with no alcohol-related problems = 7888 Drivers who met the DSM-IV criterion for alcohol related problem = 155	Number of traffic crashes in the past three years Number of traffic infringements in the past three years	Drivers with alcohol-related problems more likely to have had a traffic accident (23.2%) than drivers without alcohol-related problems (12.1%, $p < 0.0001$) Drivers with alcohol-related problems more likely to have had a traffic infringement (18.7%) than drivers without alcohol-related problems (9.3%, $p < 0.0001$)

Study: Author/date	Methods	Outcome Measure of Risk	Results
Hedlund & Fell (1995)	Used FARS data N = 2,252 fatal crash-involved drivers	Prior DWI in past 3 years BAC level	4% of all licensed drivers had a prior DWI 11% of drivers with BAC of 0.01 had prior DWI 13% of drivers with BAC of 0.10 had a prior DWI
Koepsell et al., (1994)	Case-control; n=234 (65yrs+) injury crashes n=446 no injury crashes;	Police-reported injury crashes requiring medical care	Relative risk of motor vehicle collision injury: OR: 2.1 (0.8-6.0)
Stevenson et al. (2003)	Population-based cohort study of 3,286 drivers who were admitted to hospital following a police-attended motor vehicle crash. Cases: drivers involved in an alcohol-related motor vehicle crash (n = 217). Alcohol-related crashes were defined as a crash where the driver had a BAC exceeding 0.05gm/100ml, as determined using a calibrated breath test by a police officer.	Subsequent alcohol-related hospital admission, defined as a medical diagnosis that could only have resulted from excessive alcohol consumption	Drivers involved in an alcohol-related motor vehicle crash, were almost twice likely to have a future alcohol-related hospital admission compared to drivers who were not involved in an alcohol-related crash (OR: 1.96, 95% CI 1.06-3.61)*
Soderstrom et al. (1997)	Examined alcohol abuse amongst 629 patients at a trauma clinic - 51% vehicle trauma - 23% interpersonal violence - 26% non-violent injuries N= 157 current alcoholics (25.0%)	BAC Injury data Psychoactive Substance Use Disorder (PSUD) of the SCID	17.2% of injured drivers met the criteria for alcohol dependence, rising to 32.6 % for men [1.7 times the level of alcoholics diagnosed in a population of (non-crash) convicted drunk drivers (19%)] 54% of current alcoholics were BAC+ at the time of admission

Study: Author/date	Methods	Outcome Measure of Risk	Results
Vernon et al., (2002)	Pop/case-control; Cases (history of drug use and/or alcohol abuse) n=149 (Restricted and unrestricted licence holders) Control (without medical conditions) n= 20,210	(i) All Crash (ii) At-fault crash (iii) Citations Rates per 10,000 lic days	For low impairment cases (unrestricted): RR: 1.82 (1.18-2.81) * (p < .05), all crashes RR: 2.22 (1.25-3.94)* (p < .05, at-fault crash RR: 2.38 (1.82-3.12), p < .05 citations Higher impairment cases (restrictions): RR: 4.21 (1.80-9.85) * (p < .05) all crash RR: 5.75 (2.26-14.61) * (p < .05) at-fault crash RR: 5.83 (3.19-10.66) * (p < .05) citations

Approaches to management

Assessing fitness to drive

As summarised in Table 3, the guidelines for private vehicle licensing vary widely between countries. In the EU, under the Council Directive 91/439/CEE (1991), Annex III specifies that drivers who are alcohol dependent or unable to refrain from drinking and driving shall not be issued a private vehicle driving licence or have their licence renewed. Regulations within the EU vary. Some authorities revoke licences if alcoholism (or alcohol dependency) has been present in the previous year (UK) or in the last 6-24 months (Sweden). After a demonstrated period of abstinence and with medical opinion, a licence may be re-issued with a prior diagnosis of alcohol dependency. Generally, the regulations for commercial vehicle licences do not vary greatly from guidelines for private driver's licences.

In Australia, a person diagnosed with alcoholism may hold a conditional licence, only if rehabilitation is progressing and no long-term damage exists. New Zealand does not restrict driving unless there is evidence of cognitive, perceptual or motor impairment. Similarly, the guidelines for USA specify that driving must be prevented if any motor or intellectual impairment is present.

As noted in the previous section, three of the studies reviewed showed evidence that drivers with alcohol dependency have an elevated risk of crashing. Notwithstanding the fact that the quality of evidence in these studies was compromised by methodological problems, the findings were consistent in demonstrating a risk amongst chronic alcohol abusers that was approximately twice as high as drivers without alcohol problems. The fitness to drive guidelines outlined above appear to be consistent with the limited scientific evidence reviewed here, however more research is needed to address the methodological problems identified.

An issue of particular concern is how to identify the at-risk driver with a chronic alcohol problem. More informative assessments may also be important for targeting interventions that are specific to the needs of drink-driving offenders. Del Rio and colleagues (2001) note that there are no valid tests or standardized criteria for identifying competency of drivers affected by alcohol dependency. Research by these researchers showed that 7 out of 10 drivers in Spain who were diagnosed with alcohol-related problems were deemed fit to drive by the licensing authority's Medical Driving Test Centres. Del Rio et al. also cited problems due to reticence of drivers to report their alcohol problem to authorities and reticence of medical practitioners to intervene in decisions about licensing.

Interventions

A wide range of interventions has been developed to control the problem of drink driving. Ferguson and colleagues (1999) describe two main categories:

- (i) *General interventions*, designed to target the population in which the problem occurs, through community education and deterrence measures. These include such strategies as BAC limits, random breath tests and media campaigns.

- (ii) *Specific interventions*, aimed at convicted offenders to prevent them from further offences. These strategies rely on the assumption that the intervention will elicit a change in the behaviour of the targeted individual. Specific strategies include punitive measures such as licence removal, vehicle controls such as car ignition interlocks, as well as rehabilitation programs including education and/or counselling.

Drink driving treatment programs have been established to reduce the need for purely punitive measures, including expensive and counter-productive prison sentences, in favour of measures that provide rehabilitation and prevent re-offending. Ferguson and colleagues (1999) also propose that the nature of the drink driving offence requires both a traffic and health-related outcome. Thus, a multidisciplinary approach to rehabilitation is required, involving both authorities responsible for health and those responsible for transport. A preferred approach is to use screening methods to match the particular problems of the driver to the type of rehabilitation that is most suitable, eg. driver re-education or counselling or a combination of both. In some countries these programs can be offered to drink drivers at the discretion of the court, and can in some cases be offered with a reduction of the sentence for a drink driving offence (see Table 3).

One example of a rehabilitation intervention is the Victorian Accredited Driver Education Program (VADEP), which operates under the authorisation of the Department of Human Services in Victoria, Australia. The programs include both drink driver education courses and clinical drug assessments offered to certain drivers convicted of drink driving. These programs are paid for fully by the drivers, and are operated by various agencies across the state. Most programs consist of two clinical assessments, one year apart, plus an eight-hour drink driver education program and possible referral for further treatment. On successful completion of a program a licence restoration report is lodged with the court to support the offender's application for licence return.

Several recent reviews and meta-analyses of the benefits of interventions have been conducted that indicate a positive effect on recidivism and alcohol-related crashes amongst targeted drink-drivers (Ferguson et al., 1999; Mann et al., 2001; Shults et al., 2001; Wells-Parker, Bangert-Drowns, McMillen & Williams, 1995). Although frequently subject to methodological problems, there is evidence to show that the impact of rehabilitation programs is more long lasting than deterrence interventions such as licence suspensions (ATSB, CR184). Wells-Parker et al. showed a 7-9% decrease in recidivism and alcohol-related crashes amongst convicted drink drivers, over and above licence suspension approaches. Ferguson and colleagues note the beneficial effects of the combined use of these approaches.

Another recent approach to intervention is the ignition interlock device. The devices work on the basis that the driver must show a zero BAC breath test reading before the vehicle can be started. The objective of such interventions is that they provide convicted drivers with immediate feedback on inappropriate alcohol levels and assist in changing poor drinking and driving habits and prevent an alcohol affected driver from driving. Weinrath (1997) and others (see Ferguson et al., 1999 for a review) have demonstrated a positive effect of interlock systems on recidivism, at least during the intervention period.

Overall, evaluation of drink driving programs has been fraught with methodological problems. There is a lack of randomised case-control studies and many studies have a

self-selection bias (e.g. program costs can often be prohibitive to some offenders) and limitations in evaluation instruments employed (Ferguson et al. 1999). Ferguson and colleagues also make the point that evaluations of the effectiveness of interventions have mainly been conducted in the United States and caution should be exercised in applying the findings in other contexts where laws and enforcement practices may differ. Moreover, much of the research has focused on reasons for non-attendance/drop out, or re-convictions of attendees (Davies & Smith, 2003; Stone, Buttress & Davies, 2003).

It is important to note that the kinds of interventions described here primarily are designed to address the problem of drink driver offenders and are not specific to drivers with chronic alcohol abuse. Hence, research into the effectiveness of these programs does not specifically address the relationship between crashes and interventions for chronic alcohol abuse and alcohol dependency. However, some general interventions such as BAC limits do appear to result in a general deterrence on all drink-drivers. Mann et al. (2001) note that the effects appear to be the strongest at the highest BAC levels and the 'hard core' drink driver. However, the mechanism for these effects is not well understood (Mann et al., 2001). More research is needed to evaluate effectiveness of various interventions on drivers with chronic alcohol abuse.

Table 3 Private licensing guidelines for drivers with alcohol dependency and abuse

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	CCMTA (2009)	Austrroads (2003)	Drivers Medical Group, Swansea (2003)	Utah Driver License Division (1992)**	Land Transport Safety Authority (2002)	Swedish Road Administration (1999)
Alcoholism/ Alcohol Dependency	<p>Diagnosis of Dependency: Desist from driving all vehicles.</p> <p>Driving may resume if following conditions are met:</p> <ol style="list-style-type: none"> 1. Must complete recognised treatment program. 2. Must abstain from alcohol for 1 year. <p>Timeframes may be reduced to 3 months if person is also monitored by an addiction specialist + if risk of drink driving is absent.</p> <p>Repeated reviews</p>	<p>Person may not hold an unconditional license.</p> <p>A conditional licence may be issued if:</p> <ol style="list-style-type: none"> 1. Satisfactory treatment is being undertaken. 2. There are no end-organ effects. 	<p>Diagnosed Alcoholism: Licence denied or refused if alcoholism has been present in the previous year.</p> <p>Licence restoration may occur if the person has:</p> <ol style="list-style-type: none"> 1. Abstained from alcohol + 2. Is free of alcohol-related problems for 1 year + 3. Blood parameters have been normalised, where applicable + 4. “Satisfactory” GP reports have been obtained. <p>May also require independent verification via medical</p>	<p>Chronic Alcohol Use: No driving if there is impairment of motor +/- intellectual functions.</p> <p>Alcohol use causing intermittent functional impairment outside of work + driving hours: A restricted licence may be issued. Speed, area + time of day restrictions apply.</p>	<p>In general, no restrictions on driving.</p> <p>Exceptions: Dependency has affected the person’s cognitive, perceptual + motor skills so that the ability to drive safely is impaired.</p> <p>Therefore, person to desist from driving until “effective treatment has been established” (p141).</p> <p>In addition, care needs to be taken as alcohol may exacerbate other existing medical conditions eg epilepsy.</p>	<p>Diagnosed Dependency: Licence denied or revoked.</p> <p>Licence may be reinstated after a sober lifestyle has been demonstrated for a period of 6 – 24 months + continued sobriety is likely. For institutionalised people, the sobriety period commences after release.</p> <p>Sobriety to be confirmed via regular medical assessment + laboratory tests.</p> <p>Exceptions: Person may retain their licence if there is evidence of other favourable circumstances eg very good progress in a</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	CCMTA (2009)	Austrroads (2003)	Drivers Medical Group, Swansea (2003)	Utah Driver License Division (1992)**	Land Transport Safety Authority (2002)	Swedish Road Administration (1999)
	required to ensure compliance.		+ blood tests organised by DVLA + support/ referral to appropriate consultants.			rehabilitation program. In all cases above, 3 reviews are required – the first at 6 months, then 1 year + finally 2 years.
Alcohol Misuse/ Binge Drinking/ Hazardous Drinking	<p>Drink-driving: If there is evidence that this behaviour will re-occur, person to desist from driving for 1 year.</p> <p>May be reduced if enrolled in a recognised treatment program + monitored by an addition specialist + supported by favourable specialist report.</p>	<p>Binge drinking: Poses a threat to safe driving.</p> <p>GP (if aware of problem) to counsel person as to the safety risks + legal consequences of driving during binges.</p> <p>Hazardous Drinking: GP to advise person of short + long-term consequences of this behaviour on driving.</p>	<p>Persistent Alcohol Misuse: Licence refused or revoked upon medical diagnosis or confirmation via blood markers.</p> <p>May resume driving after person has abstained or controlled his/her drinking for a period of at least 6 months.</p> <p>It is recommended that the person obtain advice/ counselling during the non-driving</p>	<p>Alcohol use without adverse personal or social outcomes in the past 1 to 3 months: A private licence may be held if abstinence is verified via a medical test.</p> <p>Alcohol use without adverse personal or social outcomes in the past 6 months: If alcohol use resulted in illegal outcomes, a private licence may be held if abstinence is verified via a medical</p>	Not specifically addressed.	<p>Gross Drunk Driving Conviction:</p> <ol style="list-style-type: none"> 1. A statement that complies with the Driving Licenses Ordinance is to be obtained two months prior to applying for a license. 2. A medical certificate shall be obtained from a medical specialist + contain pertinent information on person's alcohol habits, laboratory test results + if necessary, psychological test results. 3. The person is subject to a monitoring period of 3 – 6 months, during which time 2 laboratory tests are to be conducted.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	CCMTA (2009)	Austrroads (2003)	Drivers Medical Group, Swansea (2003)	Utah Driver License Division (1992)**	Land Transport Safety Authority (2002)	Swedish Road Administration (1999)
			period.	test.		A review is to undertaken at 6 months and then 12 months. Further reviews may be required on a case-by-case basis.
Alcohol-Related Disorders	<p>Alcohol-induced seizures:</p> <p>If after a thorough neurological examination no neurological abnormality is found the patient should be referred to and assessed by an addictions specialist recognised by the licensing authority.</p>	<p>Epilepsy:</p> <p>Epileptics who are frequently intoxicated are considered unfit to drive.</p> <p>Diabetes:</p> <p>Insulin-dependent diabetics may forget to take medication + maintain food balance whilst intoxicated.</p> <p>It is recommended that they desist from driving.</p> <p>End Organ Effects:</p>	<p>Seizures:</p> <p>Single seizure: Licence denial or revocation for 1 year following the seizure.</p> <p>Multiple seizures: person must comply with the epilepsy licensing requirements.</p> <p>Medical confirmation required that person has been free of alcohol misuse/ dependency for an “appropriate” period.</p> <p>May also require independent</p>	<p>Impairment of motor +/-or intellectual functions.</p> <p>No driving.</p>	<p>Seizures:</p> <p>Care is recommended about the possibility of alcohol exacerbating other existing medical conditions eg epilepsy.</p>	Not specifically addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	CCMTA (2009)	Austroroads (2003)	Drivers Medical Group, Swansea (2003)	Utah Driver License Division (1992)**	Land Transport Safety Authority (2002)	Swedish Road Administration (1999)
		End organ effects that impair driving must not be present. If they are present, the person does not meet the requirements for a conditional license.	verification via medical, blood + consultant reports. Impairment from Alcohol-Induced Cirrhosis/Psychosis Recommendation that licence be revoked or denied until satisfactory recovery has been achieved.			

** No distinction is made in this manual between alcohol use/misuse/abuse. Distinction is made in terms of functional ability.

References

- American Psychiatric Association (APA, 1994). *Diagnostic and statistical manual of mental disorders (DSM-IV-TR)*. Washington, DC: APA.
- Anstey, K. J., Jorm, A. F., Rejlade-Meslin, C., Maller, J., Kumar, R., von Sanden, C., et al. (2006). Weekly Alcohol Consumption, Brain Atrophy, and White Matter Hyperintensities in a Community-Based Sample Aged 60 to 64 Years. *Psychosom Med*, 68(5), 778-785.
- ATSB (2002). *Drink Driving Rehabilitation: The Present Context*. ATSB-Road Safety Consultant Report: CR 184.
- Austroroads. (2003). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. 3rd Ed. Sydney, Australia.
- Baker, S.P., Braver, E.R., Chen, L-H. & Williams, A.F. (2002). Drinking histories of fatally injured drivers. *Injury Prevention*, 8, 221-226.
- Bates, M.E., Bowden, S.C., & Barry, D. (2002). Neurocognitive impairment associated with alcohol use disorders: Implications for treatment. *Experimental and Clinical Psychopharmacology*, 10, 193-212.
- Beatty, W.W., Katzung, V.M., Moreland, V.J. & Nixon, S.J. (1995). Neuropsychological performance of recently abstinent alcoholics and cocaine abusers. *Drug and Alcohol Dependence*, 37, 247-253.
- Beirness, D.J. (1993). Do we really drive as we live? The role of personality factors in road crashes. *Alcohol Drugs and Driving*, 9, 129-143.
- Bjerre, B. (2003). An evaluation of the Swedish Ignition Interlock Program. *Traffic Injury Prevention* 4 (2), 98-104.
- Brewer, R., Morris, P, Cole, T., Watkins, S., Patetta, M. & Popking, C. (1994). The risk of dying in alcohol-related automobile crashes among habitual drunk drivers. *New England Journal of Medicine*, 331, 513-517.
- Brinkmann, B., Beike, J., Köhler, H., Heinecke, A. & Bajanowski, T. (2002). Incidence of alcohol dependence among drunken drivers. *Drug and Alcohol Dependence*, 66, 7-10.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Cavaiola, A.A., Strohmets, D.B., Wolf, J.M., & Lavender, N.J. (2003). Comparison of DWI offenders with non-DWI individuals on the MMPI-2 and the Michigan Alcoholism Screening Test. *Addictive Behaviours* 28, 971-977.
- Council for Scientific Affairs (1986). Alcohol and the driver. *JAMA*, 255 (4), 522-527.
- Davies, G.P. (2003). *Reconvictions of drink/drive course attenders: a six year follow up*. Department of Transport TRL Report TRL 574: Crowthorne, UK.

- Dawson, D.A. (1999). Alternative definitions of high risk for impaired driving: the overlap of high volume, frequent heavy drinking and alcohol dependence. *Drug and Alcohol Dependence*, 54, 219-228.
- DeFranco, C., Tarbox, A.R., & McLaughlin, E.J. (1985). Cognitive Deficits as a Function of Years of Alcohol Abuse. *American Journal of Drug and Alcohol Abuse*, 11(3 & 4), 279-293.
- Del Rio, M.C., Gonzalez-Luque, J.C. & Alvarez, F.J. (2001). Alcohol-Related Problems And Fitness To Drive. *Alcohol and Alcoholism*, 36(3), 256-261.
- Drivers Medical Group DVLA Swansea. (2008). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Ferguson, M., Sheehan, M., Davey, J. & Watson, B. (1999). *Drink Driving Rehabilitation: The Present Context*. Australian Transport Safety Bureau Report CR 184.
- Fogarty, J.N., & Vogel-Sprott, M. (2002). Cognitive Processes and Motor Skills Differ in Sensitivity to Alcohol Impairment. *Journal of Studies on Alcohol*, 63, 404-411.
- Fox, A.M., Coltheart, M., Solowij, N., Michie, P.T. & Fox, G.A. (2000). Dissociable Cognitive Impairments In Problem Drinkers. *Alcohol and Alcoholism*, 35(1), 52-54.
- Grant, B.F., Hartford, T.C., Dawson, D.A., et al. (1994). Prevalence of DSM-IV alcohol abuse and dependence: United States, 1992. *Alcohol Health Res. World*, 18, 243-248.
- Grant, S.A., Millar, K. & Kenny, G.N. (2000). Blood alcohol concentration and psychomotor effects. *British Journal of Anaesthesia*, 85(3): 401-406.
- Hedlund, J. & Fell, J. (1995). Persistent drinking drivers in the U.S. *39th Annual Proceedings, Association for the Advancement of Automotive Medicine*, 39, 1-11.
- Koepsell, T., Wolf, M. & McCloskey, L. (1994). Medical conditions and motor vehicle collision injuries in older adults. *Journal of the American Geriatrics Society*, 42, 695-700.
- Krabbendam, L., Visser, P.J., Derix, M.M., Verhey, F., Hofman, P., Verhoeven, W., Tuinier, S. & Jolles, J. (2000). Normal cognitive performance in patients with chronic alcoholism in contrast to patients with Korsakoff's syndrome. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 12(4), 44-50.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Lezak, M.D. (1995). *Neuropsychological assessment* (3rd ed.). Oxford: Oxford University Press.

- Mann, R.E., Macdonald, S., Stoduto, G., Bondy, S., Jonah, B. & Shaikh, A. (2001). The effects of introducing or lowering legal per se blood alcohol limits for driving: an international review. *Accident Analysis and Prevention*, 33, 569-583.
- Mathers, C.D., Stein, C., Ma Fat, D., Rao, C., Inoue, M., Tomijima, N, Bernard, C., Lopez, A.D. & Murray, C.J.L. (2002) *Global burden of disease 2000: Version 2. Methods and Results*. Global Programme on Evidence for Health Policy Discussion Paper No. 50. World Health Organisation. Available at http://www3.who.ch/whosis/discussion_papers/pdf/paper50.pdf
- Mitchell, M. (1985). Alcohol-Induced Impairment of Central Nervous System Function: Behavioural Skills Involved in Driving. *Journal of Studies on Alcohol, Supplement 10*, 109-116.
- Moskowitz, H. Burns, M., Fiorentino, D., Smiley, A., & Zador, P. (2000). *Driver characteristics and impairment at various BACs*. National Highway Traffic safety Administration (NHTSA), Report No. DOT 809 075, Washington: Department of Transportation.
- Moskowitz, H. & Fiorentino, D.A. (2000). *A review of the literature of the effects of low doses of alcohol on driving-related skills*. National Highway Traffic Safety Administration (NHTSA), Report No. DOT 809 028, Washington: Department of Transportation.
- Ratti, M.T., Bo, P., Giardini, A. & Soragna, D. (2002). Chronic alcoholism and the frontal lobe: which executive functions are impaired? *Acta Neurologica Scandinavia*. 105, 276-281.
- Rauch, W.J., Zador, P.L., Ahlin, E.M., Baum, H., Duncan, D., Raleigh, R., Joyce, J. & Gretsinger, N. (2002). Alcohol-Impaired driving recidivism among first offenders more closely resembles that of multiple offenders. *Proceedings of the 16th International Conference on Alcohol, Drugs and Traffic Safety*, Montreal, 289-294.
- Shults, R.A., Elder, R.W., Sleet, D.A., Nichols, J.L., Alao, M.O., Garande-Kulis, V.G., Zaza, S., Sosin, D.M., Thompson, R.S. and the Task Force on Community Preventive Services. (2001). Reviews of evidence regarding interventions to reduce alcohol-impaired driving. *American Journal of Preventive Medicine*, 21(4S), 66-88
- Soderstrom, C.A., Dischinger, P.C., Smith, G.S., Hebel, J.R., McDuff, J.R., Gorelick, D.A., Kerns, T.J., Ho, S.M., & Read, K.M. (1997). Alcoholism at the time of injury among trauma center patients: Vehicular crash victims compared with other patients. *Accident Analysis and Prevention*, 29(6), 715-721.
- Soderstrom, C.A., Ballesteros, M.A., Dischinger, P.C., Kerns, T.J., Flint, R.D., & Smith, G.S. (2001). Alcohol/drug abuse, driving convictions, and risk taking dispositions among trauma center patients. *Accident Analysis and Prevention*, 33, 771-782.

- Stevenson, M., D'Alessandro, P., Bourke, J., Legge, M. & Lee, A.H. (2003). A cohort study of drink-driving motor vehicle crashes and alcohol-related diseases. *Australian and New Zealand Journal of Public Health*, 27(3), 328-332.
- Stone, J., Buttress, S.C., & Davies, G.P. (2003) *Drink/drive rehabilitation courses: Survey of non-attenders*. Department for Transport, TRL Report TRL 575: Crowthorne, UK.
- Swedish National Road Administration. (1998). *Swedish National Road Administration Statute Book*. Sweden.
- Utah State Driver License Medical Advisory Board. (2006). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J.M. (2002). Evaluating the crash and citations rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, 34, 237-246.
- Victorian Social Development Committee (1998). *Drink-driver education and treatment: Second and final report upon the inquiry into the management of drink-drivers apprehended with high blood alcohol levels*. Melbourne, Australia: Parliament of Victoria.
- Waller, P.F., Blow, F.C., Maio, R.F., Singer, K., Hill, E.M., Schaefer, M. (1995). Crash characteristics and injuries of victims impaired by alcohol versus illicit drugs. *39th Annual Proceedings, Association for the Advancement of Automotive Medicine*, 39, 89-104.
- Weinrath, M. (1997). The ignition interlock program for drunk drivers: A multivariate test. *Crime and Delinquency*, 43(1), 42-59.
- Wells-Parker, E., Bangert-Drowns, R., McMillen, R. & Williams, M. (1995). Final results from a meta-analysis of remedial interventions with drink/drive offenders. *Addiction*, 90, 907-926.
- World Health Organisation (1992). *The ICD-10 Classification of mental and behavioural disorders. Diagnostic Criteria for research*. Geneva: WHO.

3.2 CARDIOVASCULAR DISEASE

Definition of cardiovascular disease

Heart disease, also known as cardiovascular disease (CVD), is a broad term for a group of disorders that affect the heart, arteries, and veins that supply oxygen to vital life-sustaining areas of the body like the brain, the heart itself, and other vital organs. Cardiovascular diseases include coronary heart disease, syncope, cardiac arrhythmias, high blood pressure and cerebrovascular disease (CVA) or stroke (see section 3.3 for a separate review on CVA).

The presenting symptom in over 80% of older people who have heart disease is angina. Angina is described by Wielgosz and Azad (1993) as chest pain that is pressure-like or squeezing in nature.

Syncope

Syncope is the sudden and transient loss of consciousness, with spontaneous recovery (Bonema & Maddens, 1992). It has a variety of causes; cardiac (sudden fall of blood pressure), neurological, psychiatric, and hypoglycaemic (Rehm & Ross, 1995). Syncope does not include seizures, coma or shock (Medscape, 2008). At least three percent of the adult population has experienced one or more syncopal episodes, during which they lost consciousness (Savage, Corwin, McGee, Kannel & Wolf, 1985). For 38 to 47% of people who experience syncope, no cardiac or neurologic abnormality can be found during diagnostic evaluation (Kapoor, Hammill & Gersh, 1989; Kapoor, Karpf, Wieand, Peterson & Levey, 1983; Spudis, Penry & Gibson, 1986).

Cardiac Arrhythmia

Arrhythmia refers to an irregular rhythm of the heart, not occurring in the acute phase of myocardial infarction or as a result of drug toxicity or electrolyte imbalance (Canadian Cardiovascular Society, 1996). Arrhythmias encompass a wide range of conditions, of which the vast majority are not seriously disabling and which are treated with drugs or pacemakers. The main issues with arrhythmias of relevance to driving are the risk of a recurrence causing transient disturbance of consciousness, as well as any side effects or failures of the therapy (rare).

The presence of some types of arrhythmia may pose a problem for safe and efficient driving because of their treatment: implantable cardioverter defibrillators (ICDs). ICDs are used to manage ventricular arrhythmias by delivering a high-energy shock to the heart. This shock can sometimes result in syncope (loss of consciousness) or presyncope that is severe enough to impair or prevent voluntary motor activities (Epstein et al., 1996; Kou et al., 1991). It should be noted, however, that people at risk of Ventricular Fibrillation (VF) who are treated with ICDs are relatively *uncommon* compared to people being treated for less serious arrhythmias. In these cases, it is the VF that causes an instant reduction in cardiac output that leads to syncope. The shock, while unpleasant, hopefully acts to revive the patient quickly by restoring cardiac function. These drivers are also much more likely to be under constant specialist medical supervision than most other drivers with cardiovascular disease.

Prevalence of cardiovascular disease

The WHO estimates that the prevalence of ischaemic heart disease (the class of cardiovascular disease generically caused by poor blood flow to the heart muscle) is just over 39 million worldwide (Mathers et al., 2002). In 2000, the prevalence of the disease in Western European countries (EURO A group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated at 3.4 million or around 0.8% of this population. Similarly, prevalence estimates for the USA and Canada suggest that approximately 3.4 million or 1% of the population have this disease. In Australia in 2004 the prevalence of cardiovascular disease was 17.9% for adults over the age of 18 years (ABS, 2006). Heart disease is mostly prevalent in the older-adult population. Coronary heart disease (CHD) is the leading cause of death among US individuals age 65 and over (Kannel, Gagnon & Cupples, 1990). Fifty-two percent of deaths in the older adult population are due to heart disease (WHO, 1990). Furthermore, the risk of cardiac fatality rises exponentially with age. There is at least a one-hundredfold increase of risk of cardiac death for a 65-year-old man, compared with a 35-year-old man (US Public Health Service, 1990).

Many cardiovascular events are not fatal but may be sufficiently debilitating to seriously affect functional ability. This is hard to assess without reliable morbidity data, but it may well be that 25-30% of the cardiovascular disease burden arises from disabling sequelae of heart disease and stroke.

Functional impairments associated with cardiovascular disease

Cognitive Impairment

Ahlgren and colleagues (2002) have reported that between 1 - 6% of people suffer a stroke after cardiac surgery, and cognitive impairment such as memory dysfunction and concentration disturbances are reported to occur in 33-83% of people. Lack of insight and difficulties with judgement are also implicated with stroke following surgery and have major implications for safe driving. The cognitive impairment is often transient and about 50% of the people have recovered after 6 weeks, to 6 months, but in one-third of the people's symptoms have remained 1 year after surgery (see Ricksten, 2000 for a review).

Syncope

Functional impairments associated with syncope-related driving incidents have been reported to include dizziness, diaphoresis (sweating), weakness, abdominal pain, headache and arm-pain (Huagui, Weitzel, Easley, Barrington, & Windle, 2000). In a study conducted by Dhala et al. (1995) ninety people experienced syncope or near-syncope, described by most people as a grey-out or black-out spell with either total loss of consciousness or a feeling of dimness or unawareness of their surroundings associated with extreme weakness at least once during an episode of supraventricular tachycardia (Dhala et al., 1995). In that same study, 499 people experienced light-headedness, dizziness, shortness of breath, chest discomfort, or palpitations. The authors suggested that physicians encountering people with supraventricular tachycardias and symptoms such as syncope or pre-syncope are encouraged to inquire specifically about

impairment driving abilities and participation in other activities where transient loss of consciousness is likely to result in harm to the person and others.

Finch and colleagues (1993) surveyed motor vehicle departments in the Southeast of the US, to determine driving rules for patients with syncope, loss of consciousness, arrhythmias, and ICDs. While no state in this region specifically monitors the driving practices of patients with arrhythmias, they do consider that arrhythmias would impair a driver's ability to operate a motor vehicle safely. Those applying for or renewing a driver's licence are asked about physical disabilities, such as arrhythmias, that may cause dizziness or syncope. If such a disability is present, the applicant's physician completes a report that is evaluated by the Department of Motor Vehicles.

Pre-May 2003: Relationship between cardiovascular disease and road safety outcomes

Cardiovascular disease (general)

Despite several decades of studies, the association between cardiovascular diseases (considered as a group) and being involved in MVC remains controversial. Some studies have reported an increased risk, whereas others have found no risk or even a negative association for the same medical conditions (refer to Table 4 for a summary of the study findings regarding CVD and crash risk). There is still a limited amount of evidence for a link between CVD and crashes. This is in agreement with other reviews (e.g., Guibert et al., 1998a). Generally, there is a lack of population-based, case-control studies taking into account risk exposure. To estimate risk of an event behind the wheel, the literature was reviewed for reports of the incidence of sudden cardiac death, syncope, arrhythmias, and other general cardiovascular diseases. The relationship between treatments for cardiovascular disease and risk of having a motor vehicle crash (MVC) is also discussed.

Crashes

Salzberg and Moffat (1998) examined the crash and driving citation records of 47 older drivers with cardiovascular disease who were referred to the Washington State Department of Licensing Special Examination Program (see section 3.5 for a more detailed description of the study design). The records of these drivers who passed the exam were examined over a 5-year period (1.75 years prior to the examination and 3.75 years after) and compared to 449 drivers in a control group of older drivers without medical conditions matched on age, gender and city. The control group had a crash rate of 3.82 per 100 licensed drivers prior to the examination period and 1.17 in the post examination period. This compares to a total of approximately 4 million licensed drivers in Washington State that recorded a rate of 3.47 collisions per 100 licensed drivers during 1996. The older drivers with cardiovascular disease that continued to drive had a pre-exam crash rate of 7.29 per 100 licensed drivers. This pre-exam crash risk was almost two times higher than age-matched control participants without medical conditions and the Washington State population. After the special exam, the rate of crashes for drivers with cardiovascular disease decreased substantially to 1.96 per 100 licensed drivers. A critical methodological limitation of this study was the failure to adjust the risk estimates for driver exposure or comorbid conditions. It should also be noted that the sample was restricted to older drivers who were referred to the licensing authority potentially because of concerns for their driving ability. Thus, case

participants are not representative of the population of all drivers with cardiovascular disorders and therefore findings cannot be generalised to the broader population of interest.

In a retrospective case-control study, Vernon et al. (2002) compared the rates of adverse driving events (crash, at-fault crash and citations) experienced by drivers licensed with medical conditions such as cardiovascular disease to those of age-, sex- and location-matched controls in the state of Utah (see section 3.1 for a full description of the study methodology). Vernon et al. (2002) reported that drivers with cardiovascular conditions did not show a significant difference in the rates of adverse driving events compared with controls. Possible under-reporting of medical conditions and accurate assessment of exposure rates are potential weaknesses in the program.

A study by McGwin and colleagues (2000) conducted a case-control study of chronic medical conditions and automobile crashes among older drivers. A total of 901 drivers aged 65 years and older were selected in 1996 from Alabama Department of Public Safety driving records: 249 at-fault drivers involved in crashes; 182 not at-fault drivers involved in MVC; and 475 drivers not involved in MVC were enrolled. Data collection included demographic factors, chronic medical conditions, medications, driving habits, visual function and cognitive status. Collected information on driving habits included self-reported quality of driving, estimated annual mileage, level of comfort with certain driving situations (e.g. at night) and type(s) of vehicle(s) most commonly driven. The authors pointed out although not validated, research on self-reported mileage suggests that this information is accurate compared with actual mileage, even among older drivers (Murakami & Wagner, 1997). The results showed that after adjustment for age, gender, race and annual mileage no differences were noted for at-fault and not at-fault drivers. They also showed that older drivers with heart disease were more likely to be involved in both at-fault and not at-at fault automobile crashes than those without the medical condition (adjusted OR=1.5). The interpretation of the results could be biased on the basis of the method undertaken to collect information. This was based on a self-reporting method, which may be a concern for a number of reasons especially in regards to health status. Subjects may be unwilling to divulge this information or simply misunderstand or forget the diagnosis. However the authors point out that this factor would be consistent across both the cases and controls thus the bias would be null. In summary the study showed a small association between subjects with heart disease and MVC risks. A reason for this weak association may be due to the heterogeneity of medical diagnoses, which makes it difficult to identify older drivers who are at risk of crash.

In another large population-based study military male drivers (aged 18-21 years) were investigated to identify the association between those with valvular heart diseases and involvement in MVC (Lerman, Mutar, Lavie, & Danon, 1995). The study population was divided into two groups according to whether (n = 1,300) or not (n = 4,305) the driver was involved in MVC according to the Military Crash Report for the same time frame. Data collection including health measures, demographic data, sociometric and psychometric data and involvement in MVC were compiled from the Israel Defence Forces computerized personal records. The results showed that subjects with mild -to-moderate valvular heart diseases had a higher risk of involvement in MVC, compared to those without the same health problem. The interpretation of the results of this study may be biased due to restricted sampling of young male professional civilian drivers. A

larger cohort of older drivers should be investigated. In addition, the severity of valvular heart disease and exposure measure were not accounted for in this study.

Contrary to the reports mentioned previously, the following studies demonstrated a negative association between CVD and MVCs. Naughton and colleagues (1982) followed up the driving records of three groups of drivers for a period of 18 months. The cohort of exposed subjects was composed of 975 male and female individuals who were hospitalised for CVD or ischemic heart disease (IHD). The first cohort of unexposed subjects comprised drivers not hospitalised in the same period, and matched on place of residence, age and sex only; while a second cohort of unexposed subjects was matched on place of residence and sex only. In this study, special attention was given to the severity of the disease and the researchers took in to account an estimate of exposure to risk of a crash when computing crash rates. Results showed no increased risk of crashes for people who had been hospitalised for CVD or IHD, whether or not there were adequate controls for exposure to the risk of a crash. Furthermore, there was no significant relationship between the severity of the disease and the risk of a crash. In this study, the medical status of the comparison group was not assessed, it was thus assumed that since they had not been hospitalised for IHD, they were in “much better health” than patients. These are very strong assumptions. Theoretically, the problem of the medical status of comparison groups could be attenuated with reliable reporting of changes in medical status.

A population-based case-control study by Guilbert and colleagues (1998b) examined whether or not male drivers aged 45-70 years suffering from CVD were more likely to be involved in MVCs. Data on drivers ages and medical conditions were compiled from the Societe de L'Assurance Automobile de Quebec's (SAAQ) computerized files. A questionnaire was mailed to all subjects to collect additional information on annual distances driven and various driving behaviours. Participants included 2,504 drivers involved in MVC during a 6-month period, controls were 2,520 drivers not involved in crashes. They showed that drivers with CVD were less likely to be involved in MVC (OR=0.82) than drivers without CVD. Their estimates for risk of involvement in MVCs for those reporting CVD were similar to those in studies that used control groups for comparison (Gresset, 1991; Naughton et al., 1982). The authors commented that their study included only MVCs reported to the police. It is possible that drivers with CVD are at a greater risk of MVCs but because they modify their driving habits after CVD diagnosis, the crashes in which they are involved are less serious and might not be reported to the police. Under-reporting medical conditions to the licensing bureau may have occurred, however the authors point out that if under-reporting occurred this would lead to a more conservative estimate of risk, if risk actually exists. They found no difference in any result when comparing severity of crash. In addition, no exposure measure was accounted for in the study design. The results also do not apply to all CVD patients. They do not apply to patients with severe CVD or to patients who perceive themselves at increased risk for MVC because of their CVD and choose not to renew their driving licences. The study suggests that a longitudinal study, could answer these questions, but logistics, lack of instruments and high costs unfortunately render such a study unrealistic at this point.

Citations

As outlined above, Salzberg and Moffat (1998) examined the violation records of 47 older drivers with cardiovascular disease. State violations records were examined over a

5-year period including 1.75 years prior to the exam and 3.25 years after. Older drivers with cardiovascular disease were found to have a violation rate prior to the exam of 20.67 violations per 100 licensed drivers in a year. This pre-exam violation rate was almost three times higher than that of age-matched control participants without medical conditions (7.51). After the special exam, the rate of violations for drivers with cardiovascular disease dropped to 2.61, which was comparable to the rate of age-matched control participants (2.26).

Driving performance

No studies reporting the relationship between cardiovascular disease and driving performance were found.

Sudden death

Crashes

A number of studies have investigated natural deaths in traffic (Antecol & Roberts, 1990; Ostrom & Eriksson, 1987). The results of these studies suggest that sudden natural deaths play a minor part in traffic crashes, and tend not to result in serious injuries. The contribution of medical impairment in traumatic deaths in traffic has, however, not received much attention, probably because this is a more difficult issue to investigate.

A study by Sjogren and colleagues (1996) attempted to investigate this issue. Their study involved autopsied car drivers (N = 480) aged 18 years and over, who were fatally injured and died within 3 days of the crash in northern Sweden over a 13-year period. Police reports of these victims were also examined for information on crash circumstances. A grading system was developed to assess the probability of contribution of intrinsic medical factors (IMF) to the crash, these included atherosclerosis, coronary thrombosis and myocardial infarction. Since it is difficult to be completely certain that IMF contributed to the crash, the investigators used a scale that gave a measure of probability that IMF were the major pre-crash factors. Almost one quarter of the drivers were found to have IMF that were considered to constitute a risk of sudden incapacitation. Twenty-five percent of these drivers exhibited moderate to severe coronary atherosclerosis and 4% had occlusions. Limitations of this study include lack of a control group, lack of information on certain medical conditions such as dementing illness or vision problems that may have been of relevance to the crash. In addition, the study was limited to the police reports for information on extrinsic contributing factors.

Antecol and Roberts (1990) reported that CVD and coronary artery disease (CAD) are the most common cause of sudden death from natural disease in drivers. However, the only studies they provide in their reports supporting their statement include very early studies prior to the 1980's (Bowen, 1973; Myerburg & Davis, 1964). These early studies may not be sufficient to support the statement as road systems have changed and medical treatment of the conditions has changed in the last 30-40 years, so these early findings may not be valid today. Antecol and Roberts (1990) studied the heart autopsies of 30 persons who died suddenly from natural causes in the driver's seat of an automobile, truck or bus. Available clinical records, autopsy records and police reports were examined in all 30 subjects. Twenty had cardiac arrest while driving, 16 died from

CAD; 12 had minor collisions and 4 did not. This proportion of cases involving collisions is similar to previously published studies (Christian, 1988; Copeland, 1987; Ostrom & Eriksson, 1987). No exposure measure was included in this study.

Copeland (1987) studied 188 natural deaths of drivers during a 5-year period in Florida. CVD was found responsible for 82% of these events, and most of the victims had had previous cardiac symptoms. Thirty-eight percent of these collisions were with fixed objects and therefore, the driver was the only victim.

The previously mentioned studies suggest a high proportion of people with CVD die due to natural causes while driving. However, these events seem to cause very few crashes involving other moving vehicles. This conclusion is further confirmed by the autopsy study of Antecol & Roberts (1990). Furthermore, it is likely that at least some drivers may have the time to pull off the road when they feel the symptoms of a heart attack. The number of drivers found dead in a stopped car is an important indication of this phenomenon. Lastly it seems that, in many cases, the driver was not previously aware of having CVD.

In regards to the aforementioned studies, identifying crashes attributable to illness from examination of medical files and driving records of a sample of passenger car drivers may lead to underestimation of the true proportion of crashes that are due to chronic heart disease. Finally, the absence of any form of control group prevents the estimation of risk.

Citations

No studies reporting the relationship between sudden death and citation rates were found.

Driving performance

No studies reporting the relationship between sudden death and driving performance were found.

Syncope

Crashes

The risk of having a motor vehicle crash due to syncope remains uncertain. Little information is available on the magnitude of the risk for syncope or near-syncope during driving in participants with ventricular tachycardias. If syncope occurs during driving it could have serious consequences for both the driver themselves or others who might be harmed by the vehicle

A study by Dhala and colleagues (Dhala et al., 1995), retrospectively evaluated the impact of incidence of near-syncope or syncope on driving in 90 participants with these symptoms. Of the 90 participants, 2 participants had MVCs precipitated by syncope. An additional 22 participants had, on occasion, stopped driving because of the onset of pre-syncope. All participants were treated by radio-frequency catheter ablation of aberrant conducting pathways. Nine participants with syncope required additional pharmacological therapy for concomitant vasovagal dysfunction. During a mean follow-

up of 21 ± 12 months, no recurrence of syncope was noted. From the findings of this study, the authors suggest that whereas syncope may occur and can result in impairment in driving ability, voluntary restriction is uncommon. The problem with this study is that 20% of participants were self-referred or had chosen ablative therapy as a primary treatment modality and thus do not represent the most severe or recalcitrant cases of supraventricular tachycardias. No clinical or electrophysiological characteristic other than a history of syncope was helpful in identifying patients potentially at risk.

A study based on the responses of physicians to questionnaires indicated that they had cared for patients involved in MVC as a result of presumed vasovagal syncope, before initiation of treatment (Lurie, Iskos, Sakaguchi, Fahy, & Benditt, 1999). However, it was not possible to determine the prevalence precisely. A more accurate estimate could be made of the number of patients involved in MVCs after treatment. Nine of the respondents monitored and reported on at least 1 patient who sustained one or more vehicle crashes due to syncope recurrence after evaluation had begun. In the 11,500 cases studied, there were only 17 instances where MVCs was due to syncope (approximate prevalence among treated patients of 0.1% to 0.2%).

The presence of some cardiac events or symptoms, such as syncope and angina, may be predictive of the future risk of sudden incapacitation due to a life-threatening cardiac event. Any basis for assessing whether an individual is fit to drive must include data on his or her current functional status, and the risk that a cardiac event may occur. Because there is a lack of scientific evidence that estimates driving risk for certain medical conditions, the risk can be calculated with two variables: the probability of an incapacitating event occurring and the time spent driving (Wielgosz & Azad, 1993). The following study used this technique to estimate crash risk in drivers with at least one syncopal episode.

Sheldon and Koshman (1995) conducted a study, between January 1989 and March 1994, of 217 adult participants with at least one syncopal spell and a positive tilt-test result. Vaso-vagal syncope (VVS) generally has its onset while the participant is in the upright position (Sra et al., 1993). For this reason, the head-up tilt test (HUTT) has been used to precipitate its occurrence (Grubb & Kosinski, 1997). Five patients fainted while driving a motor vehicle. They suggested that the risk of having a person with at least one previous episode of syncope subsequently fainting while driving is 0.33% per driver-year, the risk of syncope causing a crash is 0.26% per driver-year, and the risk of injury to the driver is 0.13% per driver-year. The authors reported that the risk reported by them would appear to be unacceptably high according to both English and Canadian standards. However, the risk of syncope after assessment and counselling may decrease by approximately 90% (Sheldon, Rose, Flanagan, Koshman, & Killam, 1994). This suggests that the risk of a crash to drivers after assessment may be as low as 0.026%. These estimates therefore are similar to a previous estimate of acceptable risks documented in the Canadian Cardiovascular Society consensus conference report (Brennan et al., 1992). It must be kept in mind that a weakness of this study may include the estimates based on patients recollections of crashes hence under-reporting is possible.

Using the same approach, Huagui et al. (2000) interrogated the medical records of patients who underwent HUTT for unexplained syncope while driving a motor vehicle during the period from March 1990 to May 1996. They also performed a follow-up analysis on the outcome of patients who had syncope-related driving crashes. The

authors showed that the crashes associated with vasovagal syncope while driving could cause property damage and personal injury, and even death. Of those 245 patients undergoing HUTT, 23 (9%) had at least 1 episode of syncope during driving. They showed that many patients (19 of 23) had syncope before the syncope-related driving incident. In addition, 1 patient had syncope recurrence during driving after a positive HUTT. These results suggest that the probability of having a syncope-related driving incident may increase with the recurrence of VVS. Thus, the authors suggested that it might be wise to advise patients with VVS to withhold driving temporarily if the trigger for syncope cannot be avoided. There are a number of issues relating to this study that suggest the possibility of bias. One important consideration is that only a small proportion of patients with VVS in the general population ever seek medical attention and undergo HUTT. Hence, the incidence of syncope-related driving incidents in this study only represents a sub-group of patients who have had serious consequences from VVS. The incidence of VVS-related driving incidents in the general population remains unknown and needs a community-based study. In addition, because of the nature of the retrospective study, there may be other patients who had syncope-related driving incidents but were not identified due to absence of an available record.

Over a 1-year period, all drivers older than 59 years of age who caused an injury-producing road crash (based on police reports) who were treated at the New Jersey Regional Trauma Center (sic) were reviewed concurrently (Rehm & Ross, 1995). Out of the 79 drivers, thirty-three did not have an apparent aetiological explanation for the crash. Twenty-five of the 33 had syncope. Ten were due to cardiac problems.

A case study by Varga et al. (2002) involved a 60-year-old man who was seriously injured in a MVC that resulted in a crash into a concrete column. The cause of the collision was unknown. Many tests were undertaken by the patient including HUTT which caused him to have a syncope. The authors report the importance of recognition of patients with a high risk for incapacitating symptoms due to VVS, and the use of HUTT to determine the diagnosis and to guide therapy with beta-blocking agents.

Many of the aforementioned studies indeed showed a positive relationship between syncope and the incidence of a MVC however many studies carry important methodological flaws: no population-based sampling frame, or lack of control groups or controls for distance driven or driving habits.

Citations

No studies reporting the relationship between syncope and citation rates were found.

Driving performance

No studies reporting the relationship between syncope and driving performance were found.

Arrhythmias

Crashes

Larsen and colleagues (1990) conducted a follow-up study of 501 drivers who had survived ventricular tachycardia or fibrillation (VT/VF) to assess if they were at risk for

symptom recurrence (defined by the authors as haemodynamically significant rhythm recurrence, HSRR) following hospital discharge. The rate of HSSR was determined from participant interviews and clinical records and included sudden death, VF, syncope, impaired VT or defibrillator discharge. HSSR rates for survivors of VT (n = 290) were: highest in the first few months following hospital discharge (1st month: 4.4 %; 2nd month: 3.2 %; 3-7 months: 2.1 %; 8-12 months: 0.8 %). Similarly, HSSR rates for survivors of VF (n = 211) were: highest in the first few months following hospital discharge (1st month: 3.5 %; 2nd month: 1.1 %; 3-7 months: 1.3 %; 8-12 months: 0.4 %). The authors concluded that HSSR risk in VF/VT survivors is highest in the first two months after hospital discharge and stabilises after seven months. This has significant implications for risk of crashes amongst drivers who have survived VT/VF. Addressing this issue in relation to likelihood of crashes, Beauregard and colleagues (1995) assessed the risk of arrhythmias occurring during driving. A questionnaire was used to gather information about driving habits and opinions about restrictions on drivers with ventricular tachycardia. In addition, the literature was reviewed for approximate incidence of sudden death and syncopal and non-syncopal device therapy, in order to estimate the risk of having a defibrillator discharge while driving.

Based on responses from the questionnaire, on average, mean driving distance was 178 kilometres per week (range = 1.6-960 km/wk). Patients with defibrillators (n = 57) reported driving an average of 196 kilometres per week compared with 161 kilometres per week for those with pacemakers (n = 45), ($p > 0.05$). This group were reviewed for reports of the incidence of sudden cardiac death, syncope prior to device discharge, and device discharge without syncope during follow-up.

In the review of literature, Beauregard and colleagues (1995) reported on a finding by Tchou and colleagues (1988) who found a 1.4% rate of sudden death (n = 1), a 17% incidence of syncopal or pre-syncopal arrhythmia prior to device discharge (n = 12), and a 23% incidence of shocks without symptoms (n = 16) over a mean follow up of 18 months. Adjusted for 1 year of follow-up, Beauregard et al. estimated that the risk of sudden death would be 0.93%; symptomatic shock, 11.3%; and asymptomatic shock, 15.3%. The authors commented that these projections for sudden death were similar to the 1-year sudden death rate found by Winkle and colleagues (1989). Beauregard et al. evaluated the risk of sudden death, based on these projections, at 0.0025% per day and the daily risk of having a symptomatic and asymptomatic shock at 0.031% and 0.042%, respectively. Thus this cohort supports the contention that the risk of patients having a syncopal arrhythmia and receiving a defibrillator discharge while driving is low.

In 1991, Gresset (1991) used a case-control study design to examine the relationship between CVD and crash involvement. In Quebec, all drivers must undergo a medical examination when they are over 70 years old, the results of which are transmitted to the licensing agency. In this study, 1,400 drivers involved in a crash when they were 70 years old were compared with 2,636 controls randomly selected from the 30,000 drivers aged 69 years old who had had no crashes during the same 1-year period. The information on crashes, traffic violations and medical conditions obtained from the licensing agency was supplemented by information on exposure gathered from questionnaire. They found a weak but significant increase in the risk of crashes for drivers with arrhythmias (OR: 1.63). However, the low response rate to the self-report driving questionnaire (40 %) probably does not allow for proper control of the exposure variables.

Citations

No studies reporting the relationship between arrhythmias and citation rates were found.

Driving performance

No studies reporting the relationship between arrhythmias and driving performance were found.

Coronary Heart Disease (CHD)

Crashes

A study by Koepsell and colleagues (1994) employed a matched case-control study design in which cases and controls were drawn from the membership of Group Health Cooperative of Puget Sound (GHC), a consumer-owned Health Maintenance Organisation in Washington State (see section 3.1 for a more detailed description of the study). Cases were defined, as persons aged 65 or older who received medical care within 7 days for injuries sustained in a MVC in which they were driving one of the vehicles involved. Possibly eligible persons were initially identified from police reports of MVC in 1987 and 1988. Controls were matched to cases on age, gender and country of residence but experienced no such injury during the study years. Information about eligible subjects came from GHC medical records and from a questionnaire completed by each subject or by a surrogate for cases who had died or incapacitated. The survey questionnaire included questions about driving habits, number of miles driven per year, health habits and sociodemographic characteristics. The results of the study indicated that those with both diabetes and coronary heart disease and those with CHD had a higher motor vehicle collision injury risk (OR: 8.0 and 1.4 respectively) than healthy controls in the same age.

The study by Koepsell et al. (1994) avoids referral bias, unlike the study by Ahlgren & Rutberg (2002) as it was population based. The authors pointed out that the case-control design employed in their study was efficient for rare outcomes. However, many of the medical conditions investigated affected only a small proportion of cases and controls, thus the confidence limits were quite wide, and it is possible that small to moderate effects escaped detection. In general, the results of this study suggest that many medical conditions do not appear to be associated with large increases in the risk of MVC injuries. However, the authors point out that two mechanisms are at work that may have already eliminated persons with more severe medical impairments from the population of drivers. First, the Washington State department of Licensing requires a medical evaluation as a condition of licensure for people with certain chronic or progressive illnesses or diseases that could result in loss of consciousness or control, including CVD. Second, older people tend to self restrict their driving in amount and type Charlton et al. (2006), and some studies suggest that they often do so because of growing awareness of medical impairments (Friedland, Koss, & Kumar, 1988). Thus, this study investigates only older people who have not been denied driving privileges and who have not self-selected themselves to give up driving.

Citations

No studies reporting the relationship between coronary heart disease and citation rates were found.

Driving performance

No studies reporting the relationship between coronary heart disease and driving performance were found.

Treatment of CVD and road safety outcomes

Implantable cardio-defibrillators (ICD) are now widely used for secondary prevention of sudden cardiac death and are being offered as a primary preventative therapy (Sanjeev & Passaic, 1994). ICDs terminate ventricular tachycardia (VT) and ventricular fibrillation and reduce the rate of sudden cardiac death in patients with otherwise fatal arrhythmia (Bocker, Block, & Isbruch, 1995; Mirowski, Reid, & Mower, 1980; Reid, Mirowski, & Mower, 1983). However, incapacitating symptoms, such as pre-syncope or syncope may still occur (Kou et al., 1991). This may cause harm to patients and others and imply restrictions/banning on driving of these patients. Fatal crashes caused by patients during ICD therapy nevertheless seem to be infrequent (Curtis et al., 1995; Luderitz & Jung, 1996). However, crashes may be under-reported for various reasons.

Crashes

A German group performed a retrospective analysis of data from 421 patients with an ICD over a period of 12-36 months (Bansh et al., 1998). They showed that occurrence of syncope is a frequent clinical problem in patients with an ICD. More than one-third of patients with recurrent VT will have at least one episode of syncope, and almost half of these (44%) will have a second episode during a 3-year follow-up. They showed that the risk of syncope proved to be the highest during the first year of ICD therapy (10%) and decreased in the second year (5%) but remained considerable in the third year (4%). Most syncope episodes occurred shortly after the first ICD intervention. Most incapacitating events occurred in patients with inducible fast VT. Based on the formula suggested by Canadian Cardiovascular Society:

$$TD \times V \times SCI \times Ac = \text{Risk of harm from driving}$$

where TD=time behind wheel [1h/day for private, 6h for commercial driving], V a constant based on the type of vehicle driven [0.28 for private, 1.0 for commercial driving], SCI=the risk of unconsciousness and Ac the risk of producing a fatal or injury-producing accident [Ac =0.02]

Bansh and colleagues (1998) estimated the number of extra crashes/100,000 patient-years based on the risk of syncope for patients driving privately [commercially], if driving were not prohibited until first syncope (CCS, 1996). All patients with an ICD would cause 2.3 [50] crashes/100,000 patients in the first, 1.2 [25] in the second and 0.9 [20] in the third year. Some working groups have suggested (Anderson & Camm, 1994) estimating the risk of fatal crashes on the basis of a “worst case” scenario; that is all VTs in patients with an ICD may compromise consciousness and result in a crash. However, according to Curtis and colleagues (1995), only 10.5% of shocks delivered during driving resulted in a crash. Therefore the risk of any VT or shock may overestimate the risk of a patient with an ICD causing a crash. The reported risk of crashes is ~25/100,000 patient-years with a fatality rate of 7.5/100,000 patient-years (Curtis et al., 1995). However, Bansh et al. reported that for patients with inducible fast

VT, the number of extra crashes would be 3.3 [70] in first, 0.9 [20] in the second and 1.2 [25] in the third year. An estimation much lower than the aforementioned studies.

Research conducted by Anderson and colleagues (1994) suggests that episodes of arrhythmia associated with hemodynamic symptoms such as dizziness, greying of vision, or chest pain result in significant impairment of psychomotor performance (Anderson, Katritis, Gibson, & Ross, 1992). In the absence of clear evidence on the proportion of arrhythmic episodes resulting in impairment that is likely to cause a crash, they assumed that all episodes of ICD therapy delivery are associated with such impairment. In addition, Kou and colleagues (1991) reported that patients who had ICD implanted for VT are at moderate risk for experiencing loss of consciousness during ICD shocks. Thus patients should not assume to be safe whilst driving.

Post-May 2003: Relationship between cardiovascular disease and road safety outcomes

Cardiovascular disease (general)

In the review period post-May 2003, six studies were identified on this topic. The review revealed one study addressing crash risk in cardiovascular disease. No studies were found addressing citations and only one study was found addressing driving performance outcomes. Additionally, two studies addressed sudden death at the wheel attributed to cardiovascular disease and two studies were found relating to driving outcome measures and treatment of cardiovascular disease. Table 4 includes a summary of the findings of studies that have investigated the relationship between cardiovascular disease and road safety outcomes since May 2003.

Crashes

Sagberg (2006) investigated the relative crash involvement risk associated with various diagnosed medical conditions from 4448 *crash-involved* drivers of all ages. Participants were drawn from the files of a Norwegian insurance company and asked to complete questionnaires outlining information about their crash, whether they were at fault or not for the crash, to indicate from a list of 27 medical conditions, 6 categories of medicinal drugs, 21 common symptoms which were applicable to themselves and personal background information. The odds of having a medical condition was conducted separately for at fault and not at fault drivers and the (crude) odds ratio (OR) derived from these values was taken as the measure of relative risk of that condition. This method is described as the ‘induced exposure method’ and is a case-control approach used to estimate relative risk in the absence of exposure data. Sagberg explains that “the crash involvement of not at fault drivers (controls) is directly proportional to their exposure, and the prevalence of a given risk factor among controls is a good proxy for the prevalence in the driving population at large” (p. 29). Logistic regression analyses were also conducted with culpability as the dependent measure to provide odds ratios for each condition adjusted for age and annual driving distance. Of the 4448 participants, 98 had suffered a myocardial infarction; 67 were at fault, and 31 not. The adjusted odds ratio showed a significantly elevated risk (adjusted OR 1.77) for drivers who had a myocardial infarction. The authors also reported a trend in the same direction for drivers with angina but this effect failed to reach significance. Drivers with other cardiovascular disorders and symptoms including hypertension and arrhythmias were not associated with elevated at-fault crash risk. Limitations of the study included self-

reporting of medical conditions and symptoms, low prevalence of the condition, and a likelihood that at-fault drivers were less likely to respond to the survey (suggested by the very low response rate of 30%). However, the author dismisses these concerns, suggesting that relative risk is robust against such bias, and may in fact underestimate the true risk of these conditions.

Citations

No studies focused on the relationship between the number of driving citations and cardiovascular disease since 2003.

Driving Performance

The relationship between cardiovascular disease and fitness to drive in a Spanish population was investigated by Alvarez and colleagues (Alvarez, Fierro, Vicondoa, Ozcoidi & Gómez-Talegón, 2007). Participants were recruited from two driving assessment centres in Spain. The sample included 5234 drivers aged between 14-98 years ($M = 44$, $SD = 16$) who presented at the centre to obtain their licence or to renew it. The majority of the sample was male (71%). Medical conditions were classified according to ICD-10 criteria, and alcohol consumption, and medication dose was recorded. No exclusion criteria were noted. A psychologist, general practitioner and an occupational therapist evaluated participant performance on medical evaluations, a hearing and eye test as well as psychological tests in order to determine fitness to drive. Final classifications resulted in 82.7% of drivers considered fit to drive, 16.65% were fit to drive with restrictions, and 0.65% were unfit to drive. The most common medical condition amongst drivers was cardiovascular problems. The sample consisted of 605 (11.6%) individuals with CVD, of which 10 (1.6%) were found to be unfit to drive. All participants were on medication and 10 of them reported daily alcohol intake. It is of interest to note that none of these people were considered unfit to drive as a result of their cardiovascular disease. In seven of the cases, ophthalmology problems were identified as responsible for the outcome. These results suggest that the diagnosis of cardiovascular disease does not significantly affect fitness to drive. However, a serious limitation of the study was the method of classification of fitness to drive employed by the health specialists. The system was related to the likelihood of suffering sudden loss of consciousness or sudden death at any time, and was not specifically related to driving.

Schanke, Rike, Mølmen and Osten (2008) assessed driving behaviour of CVA and TBI patients' pre and post injury. The researchers recruited 135 patients who had presented at a hospital rehabilitation clinic from 1997-2000. Sixty-five patients had suffered from a brain injury after a CVA, and 28 had experienced a traumatic brain injury. The CVA patient group was significantly older than the TBI patient group and differed according to gender proportions, although both patient groups were similar in terms of the duration of their illness. Upon presentation to the hospital patients were assessed for medical conditions that would impact upon their driving ability, such as seizures, visual conditions and stroke, and the majority also completed an on road driving test. Information relating to pre and post injury was obtained via a questionnaire administered to all the patients in 2006 concerning driving exposure and frequency, driving patterns and self-regulatory practices. The crash rate was determined by the sum of crashes experienced by the group divided by total driving exposure. Family and friends were also invited to respond to questions about the patients driving behaviour. The researchers found that the CVA group significantly reduced their driving post

injury ($M = 162$ km/week, $SD = 125.5$ km/week) compared to before the injury ($M = 289.1$ km/week, $SD = 357.7$ km/week, $p = 0.04$). However, there was no significant change in driving exposure after the injury for the TBI group. A binomial regression was used to investigate contributing factors to crash rates such as gender, driving distance, cause of injury, crash rate pre injury and duration of diagnosis. After adjusting for confounds, there were no significant differences in crash involvement between the groups. The CVA crash rates were found to be comparable to the rates of the general population in Norway, however the TBI crash rates were found to be higher (15.0 vs 6.25 crashes per million km driven). The accident rate of the TBI group post injury was almost two times higher than in the general population. Therefore, the authors concluded that TBI patients are at an increased crash risk after injury compared to patients who drive after acquiring a brain injury as a result of a CVA. The limitations of the study include small sample size, self-reporting of crashes and lack of information regarding cause of injury. It is also acknowledged by the authors that information regarding previous crash history and a longer follow up period (i.e., greater than 6 years) would have enhanced the credibility of the study.

Syncope

There were no studies published on the relationship between syncope and crashes, citations or driving performance in the post-May 2003 review period.

Sudden death

Tervo and colleagues (2008) reported on a study investigating causes of fatal motor vehicle accidents in Finland from 1995 to 2005. The aim was to investigate the association between age and fatal motor vehicle accidents in Finland and to determine the relationship between fatalities that were caused by an immediate medical problem (including cardiovascular disease) and fatalities that were due to a distraction by the driver (OFD). Fatality records were obtained from the Finnish Motor Insurers' Centre.

Five hundred and twenty-two fatal motor vehicle accidents were recorded between the years 2003-2004. A total of 54 of these accidents were due to a medical disease, and in 23 cases, the driver died as a result. The majority of drivers (63%) were aged greater than 65 years. The main cause of death was cardiovascular disease in 38 (70%) of the cases, all of whom had a prior history of heart disease. These crashes typically involved single vehicles (59%), and drivers were travelling at speeds less than 50km/hr (88%). Over half of cardiovascular patients (57%) died as a result of the disease rather than from secondary injuries resulting from the crash. In conclusion, the authors stated that cardiovascular disease was the main cause of accidents in this study (that is, fatality attributed to sudden death at the wheel), particularly for middle aged males, emphasising the importance of monitoring the health of older drivers when determining fitness to drive. It is important to note that a serious limitation of this study is that driving exposure was not accounted for.

Motozawa and colleagues (2008) investigated the characteristics of 34 (M age = 52.1, SD age = 12.7) individuals who experienced sudden death from natural causes while driving a four wheeled motor vehicle. The researchers obtained forensic autopsies for the 32 males and 2 females from the Department of Legal Medicine at the Dokkyo Medical University School. Medical history, drug treatment, and reasons for driving

were obtained from the relatives of the deceased. In order to determine injury severity, the researchers used the injury severity score as well as the abbreviated injury scale. Body mass index and heart weight were also determined from the autopsy. Information regarding the crash was derived from police records and included; type of vehicle, type of collision and the driver's operational behaviour before the crash such as avoidance manoeuvres.

The majority of participants suffered from cardiac problems (67%), which concurs with the finding that ischemic heart disease was the most common cause of death for 22 of the 34 participants. Five people died from cerebrovascular disease, five from aortic disease, one from liver cirrhosis, and one from lung tuberculosis. Very few individuals (20%) initiated an avoidance manoeuvre before time of death, and the researchers found no relationship between performing an avoidance manoeuvre and cause of death. The authors noted that 78.1% of the sample had a heart that was heavier than a normal heart weight. Specifically, the heart had expanded by more than 20% above its normal size in eleven cases. It was concluded that in this study individuals with ischaemic heart disease were more susceptible to onset of sudden death while driving. In addition, a large proportion of individuals who experienced an ischaemic heart attack had an increased heart weight. The authors suggest that investigators consider the heart weight in order to correctly identify sudden death cases.

Arrhythmias

There were no studies published in order to investigate the relationship between arrhythmias and driving crashes, citations or driving performance since 2003.

Coronary Heart Disease (CHD)

There were no studies published on the relationship between CHD and crashes, citations or driving performance since 2003.

Treatment of CVD and road safety outcomes

Crashes

Warfarin (Coumadin) is one of the most common oral anticoagulant medications for treating heart problems (Medscape, 2008). Delaney et al. (2005) employed a case-control method in order to investigate the effects of Warfarin exposure in a cohort of older Canadian drivers. Participant information, including frequency of exposure, was obtained from medical records of 5 579 cases and 12 911 controls (aged between 67-84 years) from the Quebec Automobile Insurance Agency. Information regarding prescription medication was obtained from the Quebec health insurance agency. In addition, information about whether the participant suffered from a stroke or cardiac disease in the previous year was collected. Cases were individuals who were prescribed warfarin and had been involved in a crash resulting in injury during 1990 – 1993, controls on the other hand represented drivers who were prescribed warfarin and had not been involved in an accident during the study time period. The cases and controls were similar in age, gender, residency, and chronic disease score. The exclusion criteria for all participants consisted of living in a long term care institution, and the occurrence of a hospital admission. The index date for the cases was defined as the date of the first

accident in the study time period, and the index date for the controls was a random day selected by the researchers in the study period. Participants were defined as being exposed to Warfarin if they had been prescribed the medication either within 30 days or one year of the index date. The researchers found that people who had been prescribed Warfarin within thirty days of the index date were not at any increased risk of experiencing an MVA (RR: 0.58, 95%CI 0.36 - 0.93), nor were participants who were prescribed Warfarin within the past year (RR: 0.74, 95%CI 0.55 - 1.05). The researchers concluded that the results from their study suggested that Warfarin does not increase the risk of having a motor vehicle accident in a sample of older drivers.

The primary methodological limitation in the study by Delaney and colleagues for the purpose of this literature review is the absence of clearly defined illness amongst the warfarin users. While warfarin is a commonly used anticoagulant for people with cardiovascular disease, the authors provide no information describing the participants' medical condition(s) or method of diagnosis. The authors also fail to mention the issue of medication compliance and dosage. A common methodological problem with compliance studies is that it is not possible to determine whether or not participants took the medication. In response to the study by Delaney et al. (2005), Alvarez (2006) stated that although no relationship between warfarin and driving performance was found, the cases and controls were frequently taking sedating drugs which could have contributed to the number of crashes experienced by controls. Additionally, as is the case with many studies of this kind, driving exposure was not accounted for in this study. Finally, alcohol consumption was not controlled for in the study by Delany et al. (2005) and could be a confounding factor.

Kobza and colleagues (2008) assessed life activities (including driving behaviour) of patients with ICD's who had a range of cardiac pathologies. The sample consisted of 276 patients aged between 25-86 years ($M = 65$ years) who had an ICD at some point in time. Participants completed a survey about leisure activity, sport, occurrence at high altitudes, and driving. The majority of participants were male (81%) and 53% reported experiencing an ICD shock. A large number of participants drove before the implantation of the ICD (85%), compared to 79% who drove after the operation. Very few drivers ($n = 5$, 2%) experienced an ICD shock while driving, and none of these people were involved in a traffic accident as a result. A larger proportion of drivers (18%) reported signs of arrhythmic symptoms while driving. From these findings the researchers calculated that there was a 0.4% annual probability of experiencing an ICD shock while driving. A limitation of this study is that the drivers who did not resume driving after experiencing an ICD shock were not taken in to account when the probability estimate of experiencing a shock was calculated. Furthermore, the data is self-report and therefore the participants were more likely to underestimate the number of shocks experienced while driving for fear of their driving privileges being removed. In summary, the researchers concluded that the probability of experiencing an ICD shock while driving was very low.

Driving Performance

The association between cognitive impairment after coronary bypass surgery and driving behaviour is relatively unknown. Ahlgren et al. (2003) investigated the effects of coronary artery bypass surgery on cognitive ability and its relationship with driving performance. The researchers employed a case-control study design. The cases comprised 27 people with stable angina pectoris who were scheduled for a coronary

artery bypass grafting (CABG). The control group were matched for age, gender, education and driving experience and consisted of 20 patients who were scheduled for a percutaneous coronary intervention (PCI). All participants had been driving for more than thirty years. Participants were excluded if they had a history of alcohol abuse, psychiatric history or cerebral lesion that could have interfered with the recovery process.

One to three days prior to surgery, and four to six weeks after, participants underwent neuropsychological testing, and completed a test drive in a simulator as well as an on-road drive. The neuropsychology test battery assessed visual memory, psychomotor speed, attention and concentration. Specifically, the tests included; Trails A and B, Rey Complex Figure Test, Rey Auditory Verbal Learning Test, K-test of focused attention and computer based reaction time tests. A certified driving instructor blind to the patient group assessed on-road driving in terms of speed, manoeuvring, lane position, traffic behaviour and attention. Each of the driving behaviours was rated from a scale of 1-5, and participants also rated their own driving performance. Following this, participants completed challenging drives in the Swedish Road and Transport Institute Driving Simulator (VTI) advanced driving simulator. A distraction mobile phone task was also administered during one of the drives. The dependent variables were; speed, lateral position, reaction time and time to collision. Both patient groups were comparable on the neuropsychology measures prior to surgery, and both groups performance improved after surgery. A greater cognitive decline after surgery was evident in the CABG treatment group ($n = 11$) compared to the PCI group ($n = 2$), notably on the following tests; TMTA, TMTB, Rey AVLT, K-test and simple reaction time. In regards to driving performance, the CABG group were worse at traffic behaviour and attention after surgery, compared with the PCI group who were worse at manoeuvring. A significantly relationship between those with cognitive decline after surgery and poorer driving performance was observed for speed ($M = 11, SD = 85$ vs $M = 9, SD = 41, p = .0001$), lateral position ($M = 11, SD = 85$ vs $M = 11, SD = 38, p = .010$) and traffic behaviour ($M = 11, SD = 85$ vs $M = 14, SD = 48, p = .032$). Driving simulator results showed that controls drove faster than cases before the surgery, and no differences in speed were found after surgery.

It was concluded that the CABG patients experienced greater cognitive decline after surgery than the PCI patients, and driving performance was worse after surgery for both groups. The authors proposed that CABG patients had difficulty with more cognitively demanding driving behaviours such as attention and traffic behaviour, in contrast to PCI patients who had difficulty with tactical behaviours such as manoeuvring. This is the first study of its kind to assess cognitive changes after coronary artery bypass surgery, and the associated effects on driving performance. One major limitation of the study acknowledged by the researchers is the contribution of practice effects to the neuropsychology test performance after the surgery. Another limitation is the lack of random assignment of individuals to participant groups.

Summary

This review highlights the complexity involved in identifying the association between chronic medical conditions and the risk of crashes. Four basic methodological problems are at issue: 1) the relatively low occurrence of crashes; 2) the difficulty in defining a suitable comparison group; 3) the classification difficulties of exposure to CVD categories; and 4) the control for exposure to the risk of crashes. Crashes are relatively

rare events, and this has important consequences on study designs. Prospective cohort studies would require either very large cohorts or a very long follow-up period such as Sjogren and colleagues (1996) study, who followed up their patients for 13 years.

The absence of any control group is indicated in most of the published studies (Antecol & Roberts, 1990; Bansh et al., 1998; Finch et al., 1993; Huagui et al., 2000; Rehm & Ross, 1995; Sheldon & Koshman, 1995). This precludes any capacity to estimate a risk. In the few studies where a control group was used, there is a lack of details concerning the source population and the sampling frame. An alternative approach used by Sagberg (2006), called the ‘induced exposure’ approach, estimated relative risk based on crash culpability. Sagberg explains that “the crash involvement of not at fault drivers (controls) is directly proportional to their exposure, and the prevalence of a given risk factor among controls is a good proxy for the prevalence in the driving population at large” (p. 29).

Another problem concerns the diagnosis criteria for correctly classifying drivers as exposed or not exposed to a CVD. In some studies this was taken directly from the records of the local licensing agency (Gresset, 1991) others relied on participants’ self-report of their medical condition (Vernon et al., 2002; Sagberg, 2006).

The assessment and control of what is called “exposure to the risk” is another problem (Waller, 1985). This concept is an attempt to translate the probabilistic notion of trials in the denominator for the risk of crashes. For instance, holding a drivers licence does not mean that one is effectively driving. Ideally, a good assessment of this variable would allow verification that, given equivalent driving habits and exposure. The modification of exposure could explain some contradictory results (Guibert et al., 1998b compared with Vernon et al., 2002).

Very few studies have investigated the impact of treatment options for cardiovascular disease on driving performance. In 2003 Algrehn et al. (2003) investigated the impact of cardiovascular surgery on driving performance and found that individuals who underwent coronary artery bypass grafting (CABG) experienced greater cognitive decline after surgery than individuals who received surgery for a percutaneous coronary intervention (PCI). Similarly, Delaney et al. (2005) found no relationship between individuals taking warfarin and driving performance. Evidence from the studies by Tervo et al. (2008) and Motozawa and colleagues (2008) implies an association between cardiovascular disease and ischemic heart attack and sudden death while driving. However, these studies involve small samples with relatively small numbers of driver with cardiovascular disease and further research is warranted.

Overall the studies reviewed here fail to show a consistent and clinically convincing association between CVD and the risk of a crash. It is our opinion that there is very little scientific evidence to sustain this association. Carefully designed studies taking into account mileage, usual driving habits, age, sex, and mileage drive in the statistical analysis should be undertaken to obtain more valid evidence.

Table 4 Summary of studies of risk associated with cardiovascular disease

Study: Author/Date	Method	Outcome Measure	Crash Risk/ Main Findings
Algrehn et al. (2003)	<p>Case-control</p> <p>Cases (n = 27) people with stable angina pectoris who were scheduled for a coronary artery bypass grafting (CABG).</p> <p>Controls (n = 20) were scheduled for a percutaneous coronary intervention (PCI). Matched for age, gender, education and driving experience.</p>	<p>Pre and Post surgery:</p> <ul style="list-style-type: none"> - Neuropsychology test battery - Sim. drive - On-road drive 	<p>CABG treatment group performed worse after surgery compared to controls on; TMTA, TMTB, Rey AVLT, K-test and simple reaction time.</p> <p>CABG group were worse at traffic behaviour and attention after surgery, compared with the PCI group who were worse at manouvering.</p> <p>Cog. decline after surgery was related to speed (M = 11, SD = 85 vs M = 9, SD = 41, p = .0001), lateral position (M = 11, SD = 85 vs M = 11, SD = 38, p = .010) and traffic behaviour (M = 11, SD = 85 vs M = 14, SD = 48, p = .032).</p>
Alvarez et al. (2007)	<p>Cohort</p> <p>N = 5234 drivers (14-98 years) (M = 44, SD = 16) who presented at the centre to obtain their licence or to renew it. 71% were male.</p>	<p>Fitness to drive: Medical evaluations, a hearing and eye test as well as psychological tests in order to formulate their decision</p> <p>Health conditions (ICD-10 codes)</p>	<p>The sample consisted of 605 (11.6%) individuals with CVD, of which 10 (1.6%) were unfit to drive. None of these people were considered unfit to drive as a result of CVD.</p>
Antecol & Roberts (1990)	<p>Cohort study</p> <p>N = 30 with atherosclerotic coronary artery disease (CAD)</p> <p>Mean age CAD victims = 54 ± 7</p>	<p>(1) Sudden death while driving due to CAD, n=16</p> <p>(2) Sudden death while driving not due to CAD, n=4</p> <p>(3) Sudden death behind wheel vehicle parked, n=10</p>	<p>75% of gp1 had minor collisions</p> <p>25% of gp2 had collision involved non-vehicle property damage</p>

Study: Author/Date	Method	Outcome Measure	Crash Risk/ Main Findings
Bansch et al. (1998) (18)	Patients, n=421 with ICD Retrospective analysis of data B/w July 1988 and Jan 1995	Estimated the no.of extra accidents/100,000 patient-years based on the risk of syncope for patients driving privately [commercially], if driving were not prohibited until first syncope (31).	All patients with ICD=2.3[50] accidents/100,000 patients in the first yr, 2 nd yr=1.2[25] and 0.9 [20] in 3 rd yr. 100,000 patients with no risk factor (no 1 statrial fibrillation,>40%left ventricular ejection fraction (LVEF), no inducible fast ventricular tachycardia)=~0.9% [20] accidents in 3 rd yr.
Beauregard et al., (1995) (16)	Patients with VT, n= 122 Defibrillators, n=57 Pacemakers, n=45 Based on driving questionnaire	(i) sudden death (ii) syncopal defibrillator discharge (symptomatic shock) (iii) nonsyncopal defibrillator discharge (asymptomatic shock)	Patients with defibrillators, who drove an average of 196 km/wk, risk of sudden death and syncopal and nonsyncopal defibrillator discharge were estimated at 0.0009%, 0.0011% and 0.0015% per km driven, respectively.
Curtis et al. (1995)	Cohort Study N= 286 with ICD, Period=1980-1992 i) MVC-involved -9 fatal crashes and 21 nonfatal crashes (iii) 256 non MVC-involved	Based on questionnaire	Estimated fatality rate for patients with ICD = 7.5/100,000 patient-years significantly lower than general pop= 17.6/100,000 patient-years (p < 0.05) Estimated injury rate 17.6/100,000 patient-years significantly lower than general public= 2,224/100,000 patient-years, p < 0.05)
Delaney et al. (2005)	Unmatched case-control (67-84 years) Cases n = 5579 – involved in an accident in the past 3 yrs, prescribed warfarin Controls n = 12 911 – no accident in past 3 yrs, prescribed warfarin	Warfarin exposure Chronic disease, cardiac disease, motor vehicle crash	People who were exposed to warfarin within 30 days of the crash were not at an increased risk of experiencing an MVA (RR 0.58, 95% CI 0.36, 0.93)

Study: Author/Date	Method	Outcome Measure	Crash Risk/ Main Findings
Dhala et al. (1995)	<p>Patients, n=589</p> <p>Magnitude of the risk for syncope or near-syncope during driving in patients with supraventricular tachycardias.</p> <p>Evaluated the impact of symptoms of presyncope or syncope on driving. Group 1- syncope, n= 90 (age= 46 ± 22) Group 2- no syncope, n= 499 (age= 41 ± 19)</p> <p>worst symptom=light-headedness, dizziness, shortness of breath, chest discomfort, palpitations</p>	Self-reported	<p>2 patients had MVC precipitated by syncope</p> <p>22 patients stopped driving, on occasion because onset of presyncope,</p> <p>15 % incidence of syncope or near-syncope was seen in this study.</p>
Finch et al. (1993) (140)	<p>Patients , n=40</p> <p>With automatic implantable cardioverter defibrillators (AICD) type of therapy</p> <p>Based on questionnaire</p>		<p>AICD discharge=65% patients</p> <p>AICD discharge=7% while driving</p>
Guibert et al. (1998) (33)	<p>Case Control</p> <p>Cases n = 2504 MVC- involved</p> <p>Controls n = 2520</p> <p>Age groups= 45-70</p>	MVC- involved	<p>OR: 0.82, CI 0.67 - 0.99</p> <p>controlled for age and still no difference OR: 0.82, CI 0.67-1.00)</p>
Gresset (1991)	<p>Case Control</p> <p>Cases n = 1400 MVC-involved</p> <p>Controls n = 2636</p> <p>Drivers with arrhythmias</p>		OR: 1.63*

Study: Author/Date	Method	Outcome Measure	Crash Risk/ Main Findings
Huagui, et al. (2000) (13)	Patients, n = 245 Medical records of patients who underwent HUTT for evaluation of unexplained syncope from March 1990 to May 1996 were reviewed to identify cases of syncope during driving of a motor vehicle,	Reported on the occurrence of syncope during driving among patients undergoing Head-up tilt test (HUTT) in center. Follow-up analysis on the outcome of patients who had syncope-related crashes	Syncope-related driving incident occurred on the first episode of syncope in 3 patients Other 16 patients had prior syncope (1-9) episodes not associated with driving Seven Gp A patients had 2 syncope-related driving incidents and remaining patients only had 1 syncope-related driving incident Group B, HUTT was negative, n=4
Kobza et al. (2008)	Patients n = 276 Carried an ICD	Driving exposure Experienced an ICD shock while driving Accidents related to ICD shocks	Very few drivers (n = 5, 2%) experienced an ICD shock while driving, and none of these people were involved in a traffic accident as a result.
Koepsell et al. (1994)	Pop/case-control Cases n = 234, injury MVC involv Control n = 446, no injury MVC Rates in 3 yrs		Coronary heart disease only OR: 1.2, CI=0.8-1.9 Both diabetes and coronary heart disease OR: 8.0, CI 1.7-37.7
Kou et al. (1991)	Cohort study N=180 with ICD Mean age=60 ± 11 Follow-up=16 ± 12 months		59% experienced ICD shocks during follow-up 9% experienced loss of consciousness (7% had syncope and 2% died suddenly)

Study: Author/Date	Method	Outcome Measure	Crash Risk/ Main Findings
Lerman et al. (1995) (15)	<p>Study population n=5,605 male drivers (military) Age= 18-21yrs</p> <p>Health parameter= mild -to- moderate valvular heart diseases</p> <p>N=1,300 drivers MVC-involved</p> <p>N=4,305 Drivers not non-MVC involved</p>	Self-reported crashes (Military crash report) in data base	<p>Predetermined p value (0.01) sigt assoc between involvement in MVCs = heart disease, $p=0.0002,^2=13.89$</p> <p>The association between the cumulative probability of involvement in MVCs and time since onset of military service for professional drivers with valvular heart disease demonstrated a significant difference in the likelihood to be involved in MVCs compared with those who did not.</p>
Lurie et al. (1999)	Physicians in 9 countries answered questionnaire in regard to method by which they specialize in the treatment of cardiac rhythm disturbances arrived at recommendations regarding resumption of driving for patients with vasovagal syncope.	<p>>11,500 patients with syncope</p> <p>77% physicians used follow-up tilt-Table testing to assess treatment efficacy.</p> <p>92% used b-blockers as 1st or 2nd line of TM</p> <p>54% used disopyramideas second –line therapy.</p>	<p>A more accurate estimate cld be made of the no. of patients involved in MVC after TM.</p> <p>9 of respondents followed at least 1 patient who sustained \geq to 1 MVC due to syncope recurrence after evaluation had begun.</p> <p>In only 17 instances were MVC due to syncope noted after therapy in the 11,500 patients reported by respondents (~ prevalence among treated patients of 0.1% to 0.2%).</p>
McGwin et al. (2000)	Pop/case-control Cases n =249 MVC-involved at fault Control n =198 MVC-involved not at-fault n = 454 not MVC- involved	(i) At-fault MVC (ii) Not at-fault MVC	<p>MVC-involved OR:1.5</p> <p>Non MVC involved OR: 1.0</p>
Motozawa et al. (2008)	Retrospective Study Cases = 34 autopsies	Medical history Collision type Injury severity BMI, and heart weight Avoidance manoeuvre	<p>The majority of individuals died from ischemic heart disease (64.7%).</p> <p>Very few people (20%) performed an avoidance manoeuvre before death.</p>
Sagberg (2006)	Case-Control	Self-reported medical	Adjusted (age and driving annual driving

Study: Author/Date	Method	Outcome Measure	Crash Risk/ Main Findings
	n=4448 crash-involved drivers;98 with myocardial infarction: n=67 at-fault; n=31 not at fault	condition amongst at fault (cases) and not at fault (controls)	distance) OR = 1.77 (p=0.03)
Salzberg et al. (1998)	Case-control; Cases n = 47 with cardiovascular disease; passed Washington state special exam in 1994 Controls n= 449 drivers not in special exam program in 1994; age, gender, city of residence matched	(i) Crashes per 100 drivers per year (ii) Violations per 100 drivers per year	Pre-exam crash rate: Case:Control 7.29%:3.8% Post exam crash rate: Case:Control 1.96%:1.2% Pre-exam violations: Case:Control 7.51%:7.5% Post-exam violations: Case:Control 2.61%:2.3%
Schanke et al. (2008)	Cases: n = 35 patients with TBI n = 65 brain injury after a CVA Controls: Norwegian driving population	Driving exposure Post injury crashes (carer report)	CVA group driving ($M = 162$ km/week, $SD = 125.5$ km/week) < TBI group post injury. ($M = 289.1$ km/week, $SD = 357.7$ km/week, $p = 0.04$). TBI crash rates were found to be higher than the general population (15.0 vs 6.25 accidents per million km driven). No significant risk after injury for crashes for CVA patient group.
Stewart et al. (1993)	Participants, n=1,431 Females, n=77.8, Age= sd=4.6 Males, n=596, Age= sd=4.5	Self-reported crashes	
Rehm & Ross (1995)	Cohort Study N=79 unexplained MVC-involved Collected from police reports Age=60-98 31.65% had a positive syncope	.	12.66% due to cardiac problems -arrhythmia= 10.13% -angina= 1.27% -acute myocardial infarction= 1.27%
Sheldon & Koshman (1995)	Cohort N=217 8 excluded	(i) Syncope while driving (ii) Syncope with MVC	(i) 0.33% driver/yr (ii) 0.26% driver/yr

Study: Author/Date	Method	Outcome Measure	Crash Risk/ Main Findings
	thus n =209 men, n=92 women, n=117	(iii) Risk of harm	(iii) 0.13% driver/yr
Tervo et al. (2008)	Retrospective Study N = 522 motor vehicle accidents, 362 were fatalities between 1995-2005 N = 991 fatal motor vehicle accidents due to observational failures between 2003-2004	Disease as the cause of death including cardiovascular disease Age, gender, crash type, medical history	54/522 fatalities were due to a medical condition 63% were aged > 65 yrs. Main cause of death was CVD in 38 (70%) of the cases, all of whom had a prior history of heart disease.
Vernon et al. (2002)	Pop/case-control; Cases n= 19,039 Control n= 20,210 'Cases'= heart disease, rhythm disturbances, or history of myocardial infarctions, heart surgery or hypertension	(i) Crash-all (ii) At-fault crash (iii) Citation Rates per 10,000 lic days	Not restricted (n=18,865) 1.05, all crashes 1.00, at-fault 0.76, citations

Approaches to management

Assessing fitness to drive

A comparison of the six licensing jurisdiction guidelines (Table 5) shows a number of differences in the issuing of licences for drivers with various cardiovascular disorders. The main differentiating factor is the duration of the symptom free period required before relicensing is permitted. This raises an interesting point in relation to the studies reviewed in the previous section because there is little or no reference to symptom-free duration in any of the studies reviewed. This begs the question of whether the licensing guidelines are grounded on evidence. Alternatively, it is possible that duration of symptoms is indeed an important factor in determining risk and that a failure to control for this has created potential bias in studies to date.

The regulations for licensing reviewed here include a vast array of CVD conditions. However, for the purpose of this review, discussion is limited to the key conditions of syncope, arrhythmia, CVD and ICD.

The recommendations made in reference to drivers with various CVD operating a private vehicle are shown in Table 5. Regulations with regard to syncope vary widely across licensing authorities. For instance, the Canadian licensing authority recommendations appear to be very lenient, that is for drivers with single syncope episode there is no restriction whereas in the UK the driver may only resume driving after 4 weeks. For those with a history of syncope, driving cessation is recommended until symptoms are controlled, however the type of licence issued (restricted vs. conditional) and symptom free period is highly variable across the licensing authorities in the different countries. Similarly, the greatest discrepancy observed in relicensing of patients with arrhythmia is the type of licence issued and symptom-free period of issuing a licence. On the other hand, guidelines for CAD are similar across Canada, Australia, UK, NZ, and USA where recommendations generally indicate that driving should cease for a minimum of 4-6 weeks.

It is important to highlight that no recommendations were made for drivers with ICD, although multiple studies have been carried out to assess the association of ICD and risk of MVC (refer to previous section). However the studies showed contradictory outcomes. Hence, the absence of guidelines may simply be a reflection of the relative lack of clarity on crash risk and ICD. It is also important to bear in mind that ICD is relatively rare, making up a very small proportion of all CHD. It is likely that they have been studied more, because they are so easily identifiable.

In the case of syncope, several jurisdictions have common licensing guidelines for drivers with different CVD driving commercial vehicles (Appendix D) including Australia, UK, and NZ. These guidelines state that drivers with syncope should be restrained from driving for 3 months and relicensing may occur after medical analysis. However the regulations in the USA and Sweden are much more stringent. With regards to the arrhythmia disorder, the regulations seem to be highly variable across the countries. For instance in the UK, driving is not permitted if the arrhythmia has caused or is likely to cause syncope. Once the arrhythmia has been controlled for a minimum of 3 weeks, relicensing may be permitted provided that left ventricular ejection fraction is > 0.40 . In contrast, in the USA and NZ, relicensing may occur once the arrhythmia has been controlled for a minimum of 3 and 6 months respectively. Drivers with CAD are

not permitted to drive for a minimum period of 3 months in Australia and NZ, however, in the UK and USA the minimum period is 6 weeks.

Self-Regulation

A number of studies have illustrated a tendency for drivers with a CVD condition to self-regulate their driving habits. After cardiac surgery, 21% of patients reduced their driving activity due to the cognitive impairment they experienced (Ahlgren & Rutberg, 2002).

Cognitive impairments after cardiac surgery include memory, attention, and concentration disturbances and impairment in visual-spatial skills, information processing and problem solving. The reported rate of postoperative cognitive dysfunction has varied widely (33-83 %) depending on study design, differences in participant selection and the cognitive test battery used (see Arrowsmith, Grocott, Reves, & Newman, 2000 for a review). In a study by Ahlgren & Rutberg (2002), 97 participants who had undergone cardiac surgery were interviewed about their driving habits before and 12 weeks after surgery. The mean age of the sample was 66 years. Before the operation, 78% were active car drivers. They drove several times a week including longer than 100 km distances. After the operation, 64% continued to drive and most of them commenced driving within 6 weeks. Interestingly, 13 patients described symptoms of cognitive dysfunction after the operation which made them feel not fit to drive, drive less and for shorter distances. Extrapolation of the postoperative driving activity found in their study and the expected incidence of postoperative cognitive impairment found in other studies that they described, estimated that 1,150 to 2,900 people a year will suffer cognitive impairment 6 weeks to 6 months after heart surgery and that 700 to 2,000 of these people will be active car drivers thus this may have a great impact on MVC. The limitations of this study are the small number of patients and the fact that only one centre was included. The number of dropouts, however, was low.

In 2003, Maas, Ventura, Kretzschmar, Aydin, and Schuchert (2003) administered an anonymous survey to 108 participants who had experienced syncope and who held a valid driver licence. The survey was based on self-reporting and consisted of two short structured interviews about history and recurrence of syncope, driving, and road crashes. Three (2.9%) of the 104 participants reported that they had experienced syncope while driving. After the first syncope, only seven (6.7%) of the 104 drivers had immediately stopped driving by themselves and two (1.9%) participants had stopped driving because of recommendations by the referring physician. When contacted for the second interview after three to six months, 82 (78.8%) participants remembered the advice on driving. However, all 95 drivers (100%, 96.1% to 100%) continued to drive irrespective of any recommendations. The authors concluded that current driving recommendations for drivers with syncope might have only limited practical consequences as drivers do not adhere to them. The authors note that participants in the current study could have been a rather selected group as they were attending a referral centre.

A study by Finch and colleagues (1993) determined the driving behaviour of participants following the placement of an ICD. Their results indicated that 65% of the drivers who were physically able to drive did so, in spite of advice from the physicians. Only 7% of drivers experienced ICD discharge while driving and these drivers reported that they continued to drive after the discharge. These drivers denied dizziness, syncope, or loss of consciousness. Three hundred and sixty patients followed up for 9 years

experienced numerous ICD discharges while driving, but only one had a minor crash (Luceri L, MD oral communication, July 1993, cited in Finch et al., 1993).

In 1995, Beauregard et al. (1995) conducted a retrospective survey of attitudes about driving and driving restrictions for people with an ICD. Specifically, participants with an ICD were asked about whether physicians should impose restrictions or whether the drivers should regulate themselves. Many participants reported that restrictions on distance or time of day would be adequate to protect the patients and the public. One participant felt quite strongly that, given his history of cardiac arrest and ICD, he knew about his condition and could monitor himself, however the authors pointed out that this was not true of many drivers on the road.

Table 5 Private licensing guidelines for drivers with a cardiovascular disease

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Acute Myocardial Infarct (AMI)	<p>Desist from driving for minimum of 1 month after discharge</p> <p>Angiographic demonstration of 70% greater reduction in the diameter of the left main coronary artery should disqualify the patient from private driving unless treated with revascularization.</p>	<p><i>Uncomplicated:</i> Desist from driving for minimum of 2 weeks after AMI.</p> <p>Resume driving after sufficient general convalescence, OR if more than 1 AMI, cardiologist approval is required. Periodic review required.</p>	<p>Desist from driving for minimum of 4 weeks.</p> <p>Resume driving if no other disqualifying condition present.</p> <p>No notification to DVLA required.</p>	<p>Desist from driving for 6 weeks or until the condition has stabilised.</p> <p>No licence restrictions if the condition was unusually mild. A treadmill stress test should be repeated after 6 months.</p>	<p><i>Uncomplicated:</i> Desist from driving for minimum of 2 weeks.</p> <p>Resume driving only on specialist's advice.</p>	<p>Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p>
Angina Pectoris	<p><i>Stable angina:</i> No restrictions and no waiting period</p> <p><i>Unstable angina:</i> If a percutaneous coronary intervention is performed during initial hospital stay the patient should wait 48hrs before driving.</p> <p>If a percutaneous coronary intervention is not performed during initial hospital stay the patient should wait 7 days</p>	<p>No licence restriction if symptoms are absent with mild exertion and person complies with treatment. Periodic review required. DVLA notification not required.</p> <p><i>Unstable angina:</i> If angina is unstable or symptoms occur at rest or with minimal exertion, a conditional licence may be granted,</p>	<p>Desist from driving if symptoms occur whilst at rest or driving.</p> <p>Resume driving when symptoms are satisfactorily controlled. DVLA notification not required.</p>	<p><i>For any diagnosis of heart disease:</i></p> <p>No licence restrictions if:</p> <ol style="list-style-type: none"> 1. Complete recovery. 2. Symptom-free or no undue symptoms with normal activity. 3. Slight physical limitations with mild exertion. <p>A medical report may be required as well as periodic review.</p> <p>A restricted licence</p>	<p>Desist from driving if symptoms occur at rest or with minimal exertion</p>	<p>Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	after discharge.	subject to medical opinion. Periodic review required.		may be issued if person experiences marked physical limitations with mild exertion. Speed, area & time of day restrictions may apply & 6-monthly review required.		
Heart Failure	<p>No licence restrictions.</p> <p>Disqualified from driving only when given diagnosis of NYHA Class IV.</p> <p>Definition of NYHA Class IV given as cardiac disease resulting in inability to carry out physical activity without discomfort. Symptoms of heart failure or anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort increases</p>	<p>May not hold an unconditional licence if person experiences symptoms with moderate exertion.</p> <p>A conditional licence may be issued if response to treatment is satisfactory.</p>	<p>May continue to drive if there are no symptoms that cause driver distraction.</p> <p>No need to notify DVLA.</p>	<p><i>For any diagnosis of heart disease:</i></p> <p>No licence restrictions if:</p> <ol style="list-style-type: none"> 1. Complete recovery. 2. Symptom-free or no undue symptoms with normal activity. 3. Slight physical limitations with mild exertion. <p>Periodic review required.</p> <p>A restricted licence may be issued if person experiences marked physical limitations with mild exertion. Speed, area & time of day restrictions may apply & 6-monthly review required.</p>	<p>People with recent or uncontrolled heart failure are unfit to drive or if dyspnoea occurs with mild exertion.</p> <p>May resume driving on specialist medical advice if:</p> <ol style="list-style-type: none"> 1. Dyspnoea does not occur with mild exertion. 2. There are no ECG changes, poorly controlled anticoagulant treatment, severe hypertension or other conditions that may impair driving. 	<p>Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Heart Transplant	Desist from driving for 6 weeks after discharge if NYHA Class I or II and on stable immunotherapy.	Desist from driving for 6 weeks. Not eligible to hold an unconditional licence. A conditional licence may be issued if response to treatment is satisfactory. Subject to periodic review.	No restrictions. May continue to drive as long as there are no other conditions present that would make the person unfit to drive. No notification to DLVA required.	Not specifically addressed.	Desist from driving for a minimum of 6 weeks after successful transplant. May resume driving with specialist's approval & if there are no ongoing symptoms eg electrocardiographic changes, severe hypertension, cardiac failure, arrhythmias etc. Licence may be conditional on periodic medical assessments.	Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving. Assessments are to take account of the causes, development & treatment of the disease.
Pacemaker	Desist from driving for 1 week after implant. Conditions: 1. No impaired level of consciousness after implant 2. Normal sensing and capture on ECG 3. No evidence of pacemaker malfunction at regular pacemaker clinic checks	Desist from driving for minimum of 2 weeks. Not eligible to hold an unconditional licence. Conditional licence may be issued if there are no other conditions present that may preclude driving. Periodic review required.	Desist from driving for 1 week. May resume driving if there are no other conditions present that would make the person unfit to drive.	Not specifically addressed.	Desist from driving for 1 week after successful insertion of pacemaker. May resume driving upon specialist advice if there are no other conditions present that would make the person unfit to drive.	Not specifically addressed.
Hypertension	Hypertension that is continually above	No driving restrictions on people	Person may continue to drive provided	No driving restrictions if	<i>Severe hypertension:</i> Person should not drive	Licence denial for any CVA disease that results in

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austrroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>170/110 may pose a traffic safety risk & must be carefully assessed.</p> <p>Stricter standards are required of commercial drivers than private drivers.</p>	<p>with hypertension that is less than 200/110, whether treated or untreated.</p> <p>No notification to DLA is required.</p> <p>Periodic medical review required to monitor the condition.</p> <p>An unconditional licence may NOT be held by those with hypertension that is continually above 200/110 or there is end organ damage that interferes with driving.</p> <p>A conditional licence may be issued if blood pressure is controlled and medication does not have any significant side-effects.</p> <p>Periodic review required.</p>	<p>there are no unacceptable side effects from medication.</p> <p>No notification to DVLA is required.</p>	<p>hypertension is controlled by medication, or is partially controlled by medication & diastolic is less than 120 mm.Hg.</p> <p>Periodic reviews may be required.</p> <p>A restricted licence may be issued if diastolic is continually higher than 120 mm.Hg &/or systolic is higher than 200 mm.Hg.</p> <p>Speed, area & time of day driving restrictions apply.</p> <p>6-monthly review required.</p>	<p>if medication impairs alertness or results in significant postural hypotension.</p> <p>Driving may resume if side effects of medication have been adequately remedied & there are no other conditions present that may preclude driving.</p>	<p>acute impairment of the cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p>
Dysrhythmia/ Arrhythmia	<i>Ventricular fibrillation or sustained ventricular</i>	<i>Atrial fibrillation:</i> Person may not hold an unconditional	Desist from driving if any incapacity results or may result from	No licence restrictions for arrhythmias that	If dizziness or syncope are present, or there is a history of collapse,	Licence denial for any CVA disease that results in acute impairment of the

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p><i>tachycardia:</i> Desist from driving for 3-6 months, depending on treatment type.</p> <p><i>Chronic atrial fibrillation:</i> No restrictions if without impaired level of consciousness. Otherwise, symptoms must be satisfactorily controlled.</p> <p><i>Paroxysmal atrial fibrillation, or non-sustained paroxysmal ventricular fibrillation, or paroxysmal supraventricular tachycardia:</i> No restrictions if there is no impaired level of consciousness and adequate ventricular rate control. The driver must be on chronic anticoagulation, if indicated.</p>	<p>licence if dizziness or syncope result.</p> <p>A conditional licence may be issued if the condition is stabilised for 1 week. Periodic review required.</p> <p><i>Paroxysmal Arrhythmias:</i> An unconditional licence may not be held if the person collapsed or nearly did so.</p> <p>A conditional licence may be issued if treatment results are satisfactory & “there are no haemodynamic disturbances” (p42). Periodic review required.</p>	<p>the condition.</p> <p>Driving may resume when the cause of the condition has been controlled for a minimum of 4 weeks.</p> <p>No need to notify DVLA except if the symptoms are distracting or disabling.</p>	<p>occurred</p> <ol style="list-style-type: none"> 1. In childhood. 2. Transient isolated arrhythmias occurring over 5 years ago. 3. Arrhythmias that have been controlled or stable for 3 months minimum. <p>5-yearly review required for 1. Yearly review required for 2 & 3.</p> <p>Restricted licence may be issued if the person has an unstable rhythm profile. Speed, area and time of day restrictions apply. 6-monthly review & medical recommendation required.</p>	<p>desist from driving until condition has been stabilised with treatment. For some arrhythmias a 6 week to 3 month-period free of symptoms may also be required.</p> <p>Yearly assessment by cardiologist may be required.</p> <p>Licence denial for arrhythmias that may lead to syncope or death.</p>	<p>cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p>
Angioplasty	A waiting period of 48 hours is required.	Desist from driving for 2 days minimum.	May resume driving after 1 week	<i>Requirements following any heart</i>	Desist from driving for 2 days minimum.	Licence denial for any CVA disease that results in

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		<p>An unconditional licence may not be held if angioplasty has been performed.</p> <p>A conditional licence may be issued if:</p> <ol style="list-style-type: none"> 1. An AMI did not occur after the angioplasty, and 2. There is no angina after mild exertion, and 3. There is no hypertension, no arrhythmias, no ECG changes or any other condition that would impair driving. <p>Periodic review.</p>	<p>minimum provided</p> <ul style="list-style-type: none"> - no other revascularisation is planned - LVEF is at least 40% at discharge <p>May resume driving if there is no other underlying condition that may impair driving.</p> <p>DVLA notification not required.</p>	<p><i>surgery:</i></p> <p>Desist from driving for 6 weeks or until the condition has stabilised.</p> <p>No licence restrictions if the condition was unusually mild, the person is symptom-free upon strenuous exercise 1 year following surgery, or symptom-free whilst resting 3 months post-surgery.</p>	<p>If complications occur that may interfere with driving ability (eg AMI), driving may not resume until medical clearance is obtained.</p>	<p>acute impairment of the cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p>
Coronary Artery bypass	Desist from driving for 1 month after hospital discharge.	<p>Desist from driving for 4 weeks minimum.</p> <p>Person may not hold an unconditional licence.</p> <p>A conditional licence may be issued if</p> <ol style="list-style-type: none"> 1. No angina or dyspnoea upon mild exertion, and 2. Minimal musculo-skeletal pain, and 	<p>Desist from driving for minimum of 4 weeks.</p> <p>Resume driving if no other disqualifying condition present.</p> <p>No notification to DVLA required.</p>	<p><i>Requirements following any heart surgery:</i></p> <p>Desist from driving for 6 weeks or until the condition has stabilised.</p> <p>No licence restrictions if the condition was unusually mild, the person is symptom-free upon strenuous</p>	<p>Desist from driving for 4 weeks.</p> <p>Driving may resume following specialist approval and if there are:</p> <ol style="list-style-type: none"> 1. No angina or dyspnoea upon mild exertion, and 2. No musculo-skeletal or other pain that may interfere with driving, and 3. No ECG changes, 	<p>Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		3. No other heart condition that impairs driving. Periodic review required.		exercise 1 year following surgery, or symptom-free whilst resting 3 months post-surgery.	arrhythmias, severe hypertension, poorly controlled anticoagulant treatment or any other condition that impairs driving.	
Cardiac Arrest	Desist from driving for 6 months after the event.	Desist from driving for 6 months. May not hold an unconditional licence. A conditional licence may be issued if there is no other heart condition that would cause the person to be unfit to drive. A reduction in the period before resumption of driving may be considered upon specialist advice & if the cardiac arrest occurred within 2 days of an AMI or if the arrhythmia that cause the cardiac arrest has been treated with a pacemaker or radio frequency ablation surgery.	<i>For any acute coronary syndrome:</i> Desist from driving for minimum of 4 weeks. Resume driving if no other disqualifying condition present. No notification to DVLA required.	<i>For any diagnosis of heart disease:</i> No licence restrictions if: 1. Complete recovery. 2. Symptom-free or no undue symptoms with normal activity. 3. Slight physical limitations with mild exertion. Periodic review required. A restricted licence may be issued if person experiences marked physical limitations with mild exertion. Speed restrictions apply & 3-monthly review required.	Desist from driving for 2 months minimum. Driving may resume with specialist approval & if there is no other condition that would impair driving.	Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving. Assessments are to take account of the causes, development & treatment of the disease.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		Periodic review required.				
Syncope	<p><i>Single episode:</i> No licence restriction. Period of observation recommended.</p> <p><i>Diagnosed and treated cause of syncope:</i> Desist from driving for 1 week.</p> <p><i>Reversible cause of syncope:</i> Desist from driving until successful treatment of underlying condition.</p> <p><i>Single episode of unexplained syncope:</i> Desist from driving for 1 week.</p> <p><i>Recurrent episode of unexplained syncope within 12 months:</i> Desist from driving for 3 months.</p>	<p>Desist from driving for 3 months.</p> <p>An unconditional licence may not be held if the person periodically loses consciousness without warning.</p> <p>A conditional licence may be issued if the cause of syncope has been determined & satisfactorily treated.</p> <p>Periodic review required.</p>	<p><i>Syncope with low recurrence risk:</i> May resume driving after 4 weeks after the event.</p> <p><i>Syncope with high recurrence risk:</i> May resume driving after 4 weeks if the cause of syncope has been determined & treated.</p> <p>If the cause cannot be identified, desist from driving for 6 months.</p>	<p>Guidelines for syncope are the same as for seizures and other episodic conditions.</p> <p>An unrestricted licence may be issued if seizure or episode-free for a suitable period with or without medication upon approval from a health professional. Each case will be considered individually One or two-yearly review required.</p> <p>A restricted licence may be issued if seizure or episode-free for 3 to 5 months, without medication or with medication but no side effects. Speed, area & time of day restriction apply, depending on the length of time without seizures. Six-monthly review</p>	<p>Desist from driving for a minimum of symptom-free period of 2 months OR Until the cause of syncope is identified & successfully treated, with the person remaining symptom-free for “an adequate period” (p63).</p>	<p>The risk of recurrence is to form the basis of assessment for licensing.</p> <p>Reviews are to be conducted after one, two and five years.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				required.		

References

- Ahlgren, E., Lundqvist, A., Nordlund, A., Aren, C., & Rutberg, H. (2003). Neurocognitive impairment and driving performance after coronary artery bypass surgery. *European Journal of Cardio-Thoracic Surgery*, 23(3), 334-340.
- Ahlgren, E. & Rutberg, H. (2002). Patients with coronary artery disease are active car drivers both before and soon after heart surgery. *Traffic Injury Prevention*, 3, 205-208.
- Alvarez, F. J., Fierro, I., Vicondoa, A., Ozcoidi, M., & Gómez-Talegón, T. (2007). Assessment of fitness to drive and cardiovascular diseases at the Spanish medical traffic centres. *Circulation Journal: Official Journal of the Japanese Circulation Society*, 71(11), 1800-1804.
- Anderson, M. & Camm, A. (1994). Legal and ethical aspects of driving and working in patients with an implantable cardioverter defibrillator. *American Heart Journal*, 127, 1185-1193.
- Anderson, M., Katritis, D., Gibson, S. & Ross, D. (1992). Automobile driving and ventricular arrhythmias: implications of a study of motor performance during rapid ventricular pacing [abstr]. *Circulation*, 86, 1313.
- Antecol, D. & Roberts, W. (1990). Sudden death behind the wheel from natural disease in drivers of four-wheeled motorized vehicles. *The American Journal of Cardiology*, 66, 1339-1335.
- Arrowsmith, J., Grocott, H., Reves, J., & Newman, M. (2000). Central nervous system complications of cardiac surgery. *British Journal of Anaesthesia*, 84, 378-393.
- Australian Bureau of Statistics (2006), National Health Survey: Summary of Results, Australia, 2004-05, cat. no.4364.0, ABS, Canberra.
- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia.*
- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia.* Sydney, Australia.
- Bansh, D., Brunn, J., Castrucci, M., Weber, M., Gietzen, F., Borggreffe, M., Breithardt, G. & Block, M. (1998). Syncope in patients with an implantable cardio-defibrillator: incidence, prediction and implications for driving restrictions. *Journal of the American College of Cardiology*, 31, 608-615.
- Beauregard, L.-A., Barnard, P., Russo, A., & Waxman, H. (1995). Perceived and actual risks of driving in patients with arrhythmia control devices. *Archives of Internal Medicine*, 155, 609-613.
- Bocker, D., Block, M. & Isbruch, F. (1995). Benefits of treatment with implantable cardioverter-defibrillators in patients with stable ventricular tachycardia without cardiac arrest. *British Heart Journal*, 73, 158-163.

- Bonema, J. & Maddens, M. (1992). Syncope in elderly patients: Why their risk is higher. *Postgraduate Medicine*, 91, 129-144.
- Bowen, D. (1973). Deaths of drivers of automobiles due to trauma and ischaemic heart disease: a survey and assessment. *Forensic Science*, 2, 285-290.
- Brennan, J., Davies, R., MacDonald, R., MacKenzie, B., Mitchell, L., Mizgala, H., Swirsky, S., Symes, J., Tremblay, J. & Webb, G. (1992). Assessment of the cardiac patient for fitness to drive. *Canadian Journal of Cardiology*, 8, 406-411.
- Brignole, M., Allboni, P., Benditt, D., Bergfeldt, L., Blanc, J., Thomsen, P. B., Dijk, J. v., Fitzpatrick, A., Hohnloser, S., Janousek, J., Kapoor, W., Kenny, R., Kulakowski, P., Moya, A., Raviele, A., Sutton, R., Theodorakis, G., & Wieling, W. (2001). Task force on Syncope European Society of Cardiology: guidelines on management (diagnosis and treatment) of syncope. *European Heart Journal*, 22, 1256-1306.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- CCS (1996). CCS Consensus Conference Assessment of the cardiac patient for fitness to drive. *Canadian Journal of Cardiology*, 12, 1164-1170.
- Charlton, J., Oxley, J., Fildes, B., Oxley, P., Newstead, S., Koppel, S., & O'Hare, M. (2006). Characteristics of older drivers who adopt self-regulatory driving behaviours. *Transportation Research Part F*, 9, 363-373.
- Christian, M. (1988). Incidence and implications of natural deaths of road users. *British Medical Journal*, 297, 1021-1024.
- Copeland, A. (1987). Sudden natural death 'at the wheel'-revisited. *Medicine, Science & the Law*, 27, 106-113.
- Curtis, A., Canti, J., Tucker, K., Kubilis, P., Reilly, R. & Woodard, D. (1995). Motor vehicle accidents in patients with an implantable cardioverter-defibrillator. *Journal of the American College of Cardiology*, 26, 180-184.
- Delany, J.A., Opatrny, L., & Suissa, S. (2005). Warfarin use and risk of motor vehicle crash in older drivers. *British Journal of Clinical Pharmacology*, 61(2), 229-232.
- Dhala, A., Bremner, S., Blanck, Z., Sra, J., Deshpande, S., Jazayeri, M., & Akhtar, M. (1995). Impairment of driving abilities in patients with supraventricular tachycardias. *The American Journal Of Cardiology*, 75(5), 516-518.
- Drivers Medical Group DVLA Swansea. (2008). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Epstein, A., Miles, W., Benditt, D., Camm, A., Darling, E., Friedman, P., Garson, A., Harvey, J., Kidwell, G., Klien, G., Levine, P., Marchlinski, F., Prysiosky, E. & Wilkoff, B. (1996). Personal and public safety issues related to arrhythmias that may affect consciousness: Implications for regulation and physician

- recommendations. A Medical/Scientific Statement from the American Heart Association and the North American Society of Pacing Electrophysiology. *Circulation*, 94, 1147-1166.
- Finch, N., Leman, R., Kratz, J. & Gillette, P. (1993). Driving safety among patients with automatic implantable cardioverter defibrillators. *The Journal of the American Medical Association*, 270(13), 1587-1588.
- Friedland, R., Koss, E. & Kumar, A. (1988). Motor vehicle crashes in dementia of the Alzheimer type. *Annals of Neurology*, 24, 782-786.
- Gresset, J. (1991). *Etude du risque d'accident de la route en presence d'une vision minimale ou de maladies chroniques chez les conducteurs ages*. Unpublished Doctor of Philosophy, Laval University, Quebec.
- Grubb, B., & Kosinski, D. (1997). Tilt table testing: concepts and limitations. *Pacing & Clinical Electrophysiology*, 20, 781-787.
- Guibert, R., Ciampi, A., Potvin, L., Loisele, J., Philibert, L., & France, E. (1998a). Association between cardiovascular diseases and motor vehicle crashes in men. *Canadian Family Physician*, 44, 770-776.
- Guibert, R., Potvin, L., Ciampi, A., Loisele, J., Philibert, L., & Franco, E. (1998b). Are drivers with CVD more at risk for motor vehicle crashes? *Canadian Family Physician*, 44, 770-776.
- Huagui, L., Weitzel, M., Easley, A., Barrington, W. & Windle, J. (2000). Potential risk of vasovagal syncope for motor vehicle driving. *The American Journal Of Cardiology*, 85(2), 184-186.
- Janke, M., (1994). *Age-related disabilities that may impair driving and their assessment. A literature review*. Sacramento: California Department of Motor vehicles. Report RSS-94-156.
- Kapoor, W., Hammill, S. & Gersh, B. (1989). Diagnosis and natural history of syncope and the role of invasive electrophysiologic testing. *American Journal of Cardiology*, 83, 730-734.
- Kapoor, W., Karpf, M., Wieand, S., Peterson, J. & Levey, G. (1983). A prospective evaluation and follow-up of patients with syncope. *New England Journal of Medicine*, 309, 197-204.
- Kobza, R., Duru, F., & Erne, P. (2008). Leisure-time activities of patients with ICDs: findings of a survey with respect to sports activity, high altitude stays, and driving patterns. *Pacing and clinical electrophysiology*, 31(7), 845-849.
- Koepsell, T., Wolf, M. & McCloskey, L. (1994). Medical conditions and motor vehicle collision injuries in older adults. *Journal of the American Geriatrics Society*, 42, 695-700.
- Kou, W., Calkins, H., Lewis, R., Bolling, S., Kirrsch, M., & Langberg, J. (1991). Incidence of loss of consciousness during automatic implantable cardioverter-defibrillator shocks. *Annals of Internal Medicine*, 86, 363-374.

- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Larsen, G., Stupey, M., & Walance, C. (1990). When should survivors of ventricular tachycardia/fibrillation resume driving (abstr). *Circulation*, 82(suppl), III-83.
- Lerman, Y., Mutar, M., Lavie, B., & Danon, Y. (1995). Effect of valvular heart diseases, migraine headaches and perianal diseases on the risk of involvement in motor vehicle crashes. *The Journal of Trauma*, 39(6), 1058-1062.
- Luderitz, B., & Jung, W. (1996). Driving behaviour after cardiovertr-defibrillator implantation in Europe Society of Cardiology. *New Trends in Arrhythmia Management*, 11, 9-12.
- Lurie, K., Iskos, D., Sakaguchi, S., Fahy, G., & Benditt, D. (1999). Resumption of motor vehicle operation in vasovagal fainters. *The American Journal of Cardiology*, 83(4), 604-606.
- Maas, R., Ventura, R., Kretzschmar, C., Aydin, A. & Schuchert, A. (2003). Syncope, driving recommendations, and clinical reality: survey of patients. *British Medical Journal*, 326(7379), p 21.
- Medscape (2008). *Syncope*, Retrieved 8th June 2009 from <http://emedicine.medscape.com/article/811669-overview>
- Medscape (2008). *Toxicity, warfarin and superwarfarins*. Retrieved 11th June 2009 from <http://emedicine.medscape.com/article/821038-overview>
- McGwin, G. Jr., Sims, R. V., Pulley, L. & Roseman, J. M. (2000). Relations among chronic medical conditions, medications, and automobile crashes in the elderly: A population-based case-control study. *American Journal of Epidemiology*, 152(5), 424-431.
- Mirowski, M., Reid, P., & Mower, M. (1980). Termination of malignant ventricular arrhythmias with an implantable automatic defibrillator in human beings. *New England Journal of Medicine*, 303, 322-324.
- Murakami, E. & Wagner, D. (1997). *Comparison between computer-assisted self-interviewing using GPS with retrospective trip reporting using telephone interviews*. Washington DC: Federal Highway Administration, US Department of Transportation.
- Motozawa, Y., Hitosugi, M., Kido, M., Kurosu, A., Nagai, T., & Tokudome, S. (2008). Sudden death while driving a four-wheeled vehicle: an autopsy analysis. *Medicine, Science, and the Law*, 48(1), 64-68.
- Myerburg, R., & Davis, J. (1964). The medical ecology of public safety: Sudden death due to coronary heart disease. *American Heart Journal*, 68, 586-595.
- Naughton, T., Pepler, R. & Waller, J. (1982). *Investigate road accident risk levels for heart attack (myocardial infarction) victims*. Washington, DC: National Highway Traffic Safety Administration. Report No HS-806 383.

- Ostrom, M. & Eriksson, A. (1987). Natural death while driving. *Journal of Forensic Science*, 32, 988-998.
- Rehm, C. & Ross, S. (1995). Syncope as etiology of road crashes involving elderly drivers. *The American Surgeon*, 61, 1006-1008.
- Reid, P., Mirowski, M. & Mower, M. (1983). Clinical evaluation of the internal automatic cardioverter-defibrillator in survivors of sudden cardiac death. *Am J Cardiol*, 51, 1608-1613.
- Ricksten, S. (2000). Cerebral Dysfunction after cardiac surgery-Are we moving forward? *Curr Opin Anesth*, 13, 15-19.
- Sagberg, F. (2006). Driver health and crash involvement: A case-control study. *Accident Analysis and Prevention*, 38, 28-34.
- Salzberg, P. & Moffat, J. (1998). *The Washington State Department of Licensing Special Exam Program: An Evaluation*. Washington Traffic Safety Commission. Olympia, WA.
- Sanjeev, S. & Passaic, N. (1994). The impact of implantable cardioverter defibrillator therapy on health care systems. *American Heart Journal*, 127, 1193-1200.
- Savage, D., Corwin, L., McGee, D., Kannel, W. & Wolf, P. (1985). Epidemiological features of isolated syncope: The Framingham study. *Stroke*, 16, 626-629.
- Sheldon, R. & Koshman, M. L. (1995). Can patients with neuromediated syncope safely drive motor vehicles? *The American Journal Of Cardiology*, 75, 955-956.
- Sheldon, R., Rose, S., Flanagan, P., Koshman, M. & Killam, S. (1994). Multivariate predictors of syncope recurrence in drug-free patients following a positive tilt table test (abstr). *Circulation*, 90, I-55.
- Sjogren, H., Eriksson, A. & Ostrom, M. (1996). Role of disease in initiating the crashes of fatally injured drivers. *Accident Analysis and Prevention*, 28(3), 307-314.
- Spudis, E., Penry, J. & Gibson, P. (1986). Driving impairment caused by episodic brain dysfunction: Restrictions for epilepsy and syncope. *Archives of Neurology*, 43, 558-564.
- Sra, J., Jazayeri, M., Dhala, A., Deshpande, S., Blank, Z. & Akhtar, M. (1993). Neurocardiogenic syncope. Diagnosis, mechanisms, and treatment. *Cardiology Clinics*, 11, 183-191.
- Swedish National Road Administration. (1998). *Swedish National Road Administration Statute Book*. Sweden.
- Tervo, T. M. T., Neira, W., Kivioja, A., Sulander, P., Parkkari, K., & Holopainen, J. M. (2008). Observational failures/distraction and disease attack/incapacity as cause(s) of fatal road crashes in Finland. *Traffic injury prevention*, 9(3), 211-216.

- Tchou, P., Kadri, N., Anderson, J., Caceres, J., Jazayeri, M., & Akhtar, M. (1988). Automatic implantable cardioverter defibrillators and survival patients with left ventricular dysfunction and malignant ventricular arrhythmias. *Annals of Internal Medicine*, *109*, 529-534.
- Utah State Driver License Medical Advisory Board. (2006). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Varga, E., Worum, F., Szabo, Z., Varga, M., & Lorincz, I. (2002). Motor Vehicle accident with complete loss of consciousness due to vasovagal syncope. *Forensic Science International*, *130*, 156-159.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J.M. (2002). Evaluating the crash and citations rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, *34*, 237-246.
- Waller, J.A. (1985). *Injury Control: A Guide to the Causes and Prevention of Trauma*. Lexington, MA: Lexington Books.
- Wielgosz, A. T., & Azad, N. (1993). Effects of cardiovascular disease on driving tasks. *Clinics in Geriatric Medicine*, *9*(2), 341-348.
- Winkle, R., Mead, R., & Ruder, M. (1989). Long-term outcome with the automatic implantable cardioverter-defibrillator. *Journal of the American College of Cardiology*, *13*, 1353-1361

3.3 CEREBROVASCULAR ACCIDENT (STROKE)

Definition of cerebrovascular accident

Cerebrovascular disease are disorders of the supplying blood vessels to the brain or its covering membranes. A cerebrovascular accident (CVA) or stroke occurs when the blood supply to an area of the brain is unexpectedly is unexpectedly blocked or bleeds. This interruption can either interrupt the flow of oxygen to the brain (ischaemic CVA) or allow blood into the areas surrounding the brain cells (haemorrhagic CVA) respectively, applying harmful pressure on the brain. Brain cells will die if they do not receive adequate oxygen and nutrients from blood or if bleeding within or around the brain damages them. Typical symptoms of stroke include: a range of cognitive impairments; loss of strength (paralysis) or feeling of limbs, particularly on one side of the body (contralateral to the site of the stroke); loss of balance; confusion or difficulty in generating and comprehending speech and visual disturbances. Damaged cells can in some cases be treated and functionality can be maintained. A variety of methods and tools are used for diagnosing stroke including neurological examination, CT or MRI scans, ultrasound and arteriography. The major risk factors for stroke are high blood pressure, heart disease, diabetes and tobacco smoking. Lesser risks include high cholesterol, physical inactivity, excess weight, poor diet and excessive alcohol use. There is also some evidence to suggest that family members may have a genetic tendency for stroke or may share a lifestyle conducive to stroke.

Transient Ischaemic Attack

A transient ischaemic attack (TIA) is a transient stroke, which, by definition, lasts less than 24 hours. It occurs when the blood supply to part of the brain is briefly interrupted. TIA clinical symptoms, which usually occur suddenly, are similar to those of stroke and typically last less than one hour but may persist for longer. As with CVAs, symptoms of TIAs vary depending on the area of the brain affected and can include: numbness or weakness in the face, arm or leg – especially on one side of the body; confusion or difficulty in speaking or understanding speech; vision disturbances in one or both eyes; difficult with walking; and dizziness, or loss of balance and coordination (NINDS, 2009). TIAs have great significance as indicators of an incipient stroke especially if their frequency is increasing.

Prevalence of CVA

In 2004 World Health Organisation (WHO) estimates place the worldwide prevalence of CVA at approximately 30.7 million (WHO, 2008). In 2003, the prevalence of CVA in the United States of America was estimated at almost 5.5 million, or approximately 2.6% of the population (Thom et al., 2006). The estimated cost to the country is approximately \$57.9 billion. In 2000, the prevalence of CVA in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 4.5 million or around 1% of the total population.

Lings & Jensen (1991) estimated prevalence of stroke at 2 per 1,000 head of population with a 40% fatality rate. These figures are likely to increase in the absence of lifestyle changes that can prevent stroke. Bonita (1992) reports that between 15 and 25% of people who have experienced a stroke will remain permanently incapacitated in

someway. About 75% of strokes occur in people aged 65 and over. Around 35% of participants die within the first 3 weeks following stroke. In the UK estimates of prevalence of stroke amongst community dwelling individuals suggests a frequency of 831 cases per 100,000 people, which equates to approximately half a million people (Clark & Opit, 1994). A similar rate of 833/100,000 was reported in New Zealand (Bonita, Solomon & Broad, 1997). In Australia, 44,000 stroke events occur each year; approximately 1.8% of Australians have had a stroke (AIHW, 2006).

Functional impairments associated with CVA relevant to driving

Functional impairments associated with stroke and TIA vary depending on the location and severity of damage to the brain. Impairments may affect a range of neuropsychological and motor abilities including:

- memory;
- cognition (e.g. decision-making, executive functions);
- attention (a specific condition worthy of note here is *hemineglect*, e.g. visual neglect, which results in lack of awareness of or failure to attend to one side of space);
- visuospatial perception;
- speech and language comprehension;
- vision (e.g. visual field disturbances such as hemianopia; refer to section 3.13);
- sensory and motor functions (e.g. hemiparesis, which may result in paralysis or partial paralysis as well as loss of sensation in limbs).

It is important to note that these higher order cognitive impairments associated with stroke and TIA may continue even after the recovery of visual perception and motor strength (Lundberg, Caneman, Samuelsson, Hakamies-Blomqvist & Almkvist, 2003). The consequences of cognitive impairments on safe driving are described in more detail in other sections (see 3.4 and 3.13).

There are two main consequences of stroke, 1) physical (refer to section 3.7) and 2) cognitive. Many people affected by stroke also have physical impairments that result in a reduction in mobility. This increases the need to return to driving successfully. For this reason it is important to develop an understanding of the relationship between dysfunction (both physical and cognitive) caused by stroke and the subsequent impact on driving ability. This will allow development of screening procedures to assess fitness to drive in people who have experienced a stroke. Some of the difficulties associated with stroke that affect physical mobility may be addressed by technological adaptations to the motor vehicle. However, the extent to which individuals can benefit from compensatory strategies depends greatly on the extent to which cognitive abilities are compromised, existence of visual field loss or hemineglect and, importantly, on level of insight into their impairments.

Other medical complications

The risk of a further stroke and seizure increases following the occurrence of a primary stroke. This has important implications for guidelines for assessing fitness to drive following an initial stroke.

Pre-May 2003: Relationship between CVA and road safety outcomes

A number of studies have investigated the relationship between CVA and road safety outcomes including crashes, citations and driving performance. Seven of these were reviewed in the 2004 version of the report. The current review of literature published since May 2003 revealed two studies related to stroke and road safety outcomes. A summary of the findings from these studies is shown in Table 6.

Crashes

A population based case-control study, carried out by McGwin, Sims, Pulley and Roseman (2000), estimated the association between chronic illness and at-fault involvement in crashes among older drivers, after adjustment for driving exposure and demographic variables. They conducted a telephone survey of a random sample of older drivers who had been involved in a crash in 1996, and a matched sample of controls who had not. The study is described in more detail in section 3.2. For the stroke group, the authors reported that individuals were twice as likely to have been involved in a crash than controls. McGwin et al. added a cautionary note that the participants affected by stroke may have been suffering from age-related cognitive problems as well as those resulting from their stroke. However, this was not controlled for by appropriate matching and statistical procedures. Also data obtained from self-reported telephone surveys can often fall prey to inaccuracy and reporting bias (Parker, McDonald, Rabbitt & Sutcliffe, 2000).

Koepsell et al. (1994) conducted a case-control study to determine whether medical conditions, including CVA, increased the risk of injury due to motor vehicle collisions in older drivers (see section 3.1 for a more detailed description of the study). Drivers (n = 234) aged 65 years and older who were injured in a crash during 1987 or 1988 were compared with 446 drivers, not involved in injury crashes, and matched by age, gender and county of residence. A more detailed description of this study method can be found in section 3.5. Amongst cases, the prevalence of stroke was 1.7% and 2.2% amongst controls. The odds ratio, adjusted for age, sex and place of residence only (i.e. not corrected for exposure) showed that prevalence of stroke amongst those who were injury crash-involved was 0.8 times that of the control group who had not been involved in an injury crash (CI: 0.2-2.5). For TIAs, the odds ratio was 1.6 (CI: 0.5-4.8). Hence, the authors reported that there was no clear tendency towards elevated risk among older drivers who had experienced a stroke or a TIA. The study should be replicated with a larger sample and with appropriate adjustments for driving exposure.

Salzberg and Moffat (1998) examined the crash and driving citation records of 21 older drivers who had experienced a stroke or CVA who were referred to the Washington State Department of Licensing Special Examination Program (see section 3.13 for a more detailed description of the study design). The records of drivers who passed the exam were examined over a 5-year period (1.75 years prior to the examination and 3.75 years after) and compared to 449 drivers in a control group of older drivers without medical conditions matched on age, gender and city. The control group had a crash rate

of 3.82 per 100 licensed drivers prior to the examination period and 1.17 in the post examination period. This compares to a total of approximately 4 million licensed drivers in Washington State that recorded a rate of 3.47 collisions per 100 licensed drivers during 1996. The older drivers who had experienced a stroke or CVA who continued to drive had a pre-exam crash rate of 5.44 per 100 licensed drivers. This pre-exam crash risk was slightly higher than age-matched control participants without medical conditions and the Washington State population. After the special exam, the rate of crashes for drivers who had experienced a CVA decreased slightly to 4.40 per 100 licensed drivers, which was still significantly higher than controls. Methodological limitations of this study include a lack of information regarding exposure rates and possible comorbid conditions. The study was also restricted to a small sample of older drivers who were referred to the licensing authority by family, police physicians and others, presumably because of concerns for their driving ability. Thus, case participants are not representative of the population of all drivers with CVA and therefore findings cannot be generalised to the broader population of interest.

Citations

In the study outlined above, Salzberg and Moffat (1998) examined the citation records, as well as crash involvement, of 21 older drivers who have experienced a stroke or CVA. State citations records were examined over a 5-year period including 1.75 years prior to the exam and 3.25 years after. Older drivers who have experienced a stroke or CVA were found to have a citation rate prior to the exam of 8.16 citations per 100 licensed drivers in a year. This pre-exam citation rate was slightly higher than that of age-matched control participants without medical conditions (7.51). After the special exam, the rate of citations for drivers who have experienced a stroke or CVA dropped to 7.32, which was still 3.2 times higher than age-matched control participants.

Driving Performance

Nouri, Tinson and Lincoln (1987) investigated the relationship between cognitive ability and driving after stroke. Forty participants who had experienced a stroke completed a cognitive test battery including tests of reaction time, attention, spatial ability and reasoning. The participants then took part in an on-road driving evaluation carried out by an independent qualified driving instructor. Analysis showed that 94% of the driving evaluation outcomes were predicted by performance on the cognitive tests. The lack of a control group precludes generalisation of the findings, and the relatively small participant numbers may weaken the statistics used, as there were a large number of predictor variables involved. In light of this, the authors moderated their conclusions, suggesting that their battery may be useful for identifying drivers clearly able to drive, and those who are clearly unsuitable.

Lings and Jensen (1991) carried out a study comparing the performance of 111 participants who had experienced a stroke, with 109 healthy controls. Using a mock car they compared reaction times to a variety of stimuli encountered on the road, and found that the stroke participants performed far worse than the control group. Reaction time when braking was particularly impaired in the stroke group, regardless of which hemisphere of the brain had been injured.

Heikkila, Korpelainen, Turkka, Kallanranta and Summala (1999) reported on a case-control study, examining differences in cognitive and psychomotor skills between 20 male stroke participants and 20 male controls (matched for age and driving experience).

A neurologist, using clinical examination, neuropsychological evaluation and observation of behaviour as to their suitability to drive, evaluated participants. A traffic psychologist then administered tests of driving related cognitive and psychomotor performance. The cases performed significantly worse on these than the controls, with 60% being found unfit to drive. Agreement between the traffic psychologist and neurologist was 75%. This indicates that there may be an important role for multi-disciplinary testing to evaluate fitness to drive in situations where real driving tests are unavailable, but the small sample size and inclusion of no females reduce the representativeness of this study.

Akinwuntan, Feys, DeWeerd, Pauwels, Baten and Strypstein (2002) reported a study which examined factors involved in deciding whether stroke participants should be licensed or not, in Belgium. Forty-one participants took part in the study and were administered a neuropsychological test battery, and an on-road test. They found that the best predictors of the final decision to allow driving or not were kinetic vision, scanning, and road test performance ($r^2 = .51$). Again as in previously reviewed studies, this study lacks statistical power due to small sample size, and there is also no control group. The authors noted some of these shortcomings and concluded that more real road evaluation is necessary to increase predictive power of evaluations.

Post-May 2003: Relationship between CVA and road safety outcomes

In the review period post-May 2003, a total of two studies were identified on this topic. The review revealed one study addressing crash risk in stroke. No studies were found addressing citations and only one study was found addressing driving performance outcomes. Table 6 includes a summary of the findings of studies that have investigated the relationship between cerebrovascular disease and road safety outcomes post-May 2003.

Crashes

Sagberg (2006) investigated the relative crash involvement risk associated with various diagnosed medical conditions from 4448 crash-involved drivers. Participants were drawn from the files of a Norwegian insurance company and asked to complete questionnaires outlining information about their crash, whether they were at fault or not for the crash, to indicate from a list of 27 medical conditions, 6 categories of medicinal drugs, 21 common symptoms which were applicable to themselves and personal background information. Details of the study method and limitations of the study are described in section 3.2. Of the 4448 participants, 49 were stroke involved: 36 were at fault, and 13 not. A crude odds ratio of 2.31 was found for drivers who had had a stroke – the highest across all conditions. However, when adjusted across age and driving distance, the adjusted odds ratio (1.93) was not significant, suggesting that the risk could be partly explained by the aging process.

Driving Performance

Kotterba, Widdig, Brylak and Orth (2005) conducted a case-control study assessing driving skills in patients at an early stage of their recovery after stroke. Thirty-two patients with acute ischaemia in the middle cerebral artery (MCA; $n = 24$) and in the vertebral artery (VA; $n = 8$) were tested 7-14 days of hospital admission. Twelve of the patients fully recovered after 24 hours and showed no cerebral infarction on a CT scan, indicating a transient cerebral ischaemia (TIA). Inclusion criteria of the study

maintained that patients were only little-to-mildly impaired post-stroke/TIA. 12 healthy volunteers also took part in the study. All participants were administered a neuropsychological test of attention and tested in a driving simulator. They found that 'complete' stroke patients had significantly more crashes on the simulator than controls. No difference was found between controls and TIA patients. Performance in the simulator (crash rate and rate of faults) was significantly worse for patients of MCA infarction than VA. MCA patients were also significantly worse than controls. Limitations of the study include a very small sample size and limited information regarding the differing characteristics of the control and clinical group, questioning the representativeness of the study. The authors did not note any shortcomings in their paper, but argue that their paper shows an elevated crash risk for mildly impaired patients who believe that they are fit to drive.

Summary

In conclusion, the evidence reviewed above suggests that generally, stroke appears to lead to impairment that may affect driving ability to some degree. However, the evidence is considerably limited in assessing the relationship between stroke and crash risk. Only four studies, including one post-May 2004, were found that addressed crash risk following stroke and of these, two reported increased crash risk (one based on self-reported crashes) and two showed no elevated risk. However, no detail of the severity or nature of the impairments in these studies was available. More research on risk of crash following stroke is needed. The wide variety of assessment for measuring impairment following stroke makes firm conclusions difficult to support satisfactorily. Most authors noted the need for a standardised neuropsychological test battery designed to best predict the driving performance of people affected by stroke. Some authors also suggest that extensive evaluation of participants including an on-road driving test where possible should be a requirement of returning to drive after stroke. However, as discussed earlier in relation to dementia, the wisdom of routinely (i.e. without prior medical or neuropsychological clearance) conducting on-road assessments for drivers with known cognitive impairments must be questioned as it potentially place both driver and assessor at unnecessary risk. Nevertheless, a standardised and validated procedure for assessing risk is needed to allow clinicians to better inform participants and their families of a person's driving capabilities, putting them in a better position either to limit driving or to decide to stop altogether. This in turn would allow the independence (and attendant self-esteem) of participants to be maintained as fully and as long as possible, and would in some cases protect the participants and the general public from unsafe driving behaviours.

Table 6 Summary of studies of risk associated with CVA

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Akinwuntan et al. (2002)		Neuropsych tests & on-road test	Found 3 predictors of permission to drive Vision, scanning, driving performance
Fisk et al. (1997)		Looked at prevalence of evaluations post stroke.	98 participants who returned to driving, 48% had no advice at all, and 87% were not evaluated
Heikkila et al. (1999)	Case control study 20 CVA participants: 20 age matched	Neurologist evaluation Traffic psychologist evaluation	60% of participants unfit to drive. 75% agreement between evaluators
Kotterba et al. (2005)	Case-control study 32 CVA (mildly impaired) 12 healthy controls	Neuropsychological test Driving simulator evaluation	Complete stroke patients had sig more accidents than controls (2.5 ± 3.2 vs. 1.3 ± 1.4). TIA patients did not differ significantly from controls. Accident rate sig higher in MCA infraction than controls (2.88 ± 3.6 vs 1.25 ± 1.36 , $p < .05$). MCA patients caused more accidents and had a higher rate of faults than VA (2.88 ± 3.6 vs. 1.5 ± 8.63 , $p < .05$). Neuropsych data showed no difference across patient groups, and no correlation between results and driving simulator performance.
Lings & Jensen (1991)		Mock car study	Cases sig. poorer than controls, $p < .01$
McGwin et al. (2000)	Case-control study Telephone interview about crash history	Involvement in crash	2:1 ratio for crashes in CVA: control OR :1.9, 95% CI: 0.9, 3.9
Nouri et al. (1987)		Cognitive battery & on-road evaluation	94% of evaluation outcomes predicted by cognitive battery

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Sagberg (2006)	Case-control study 15,000 drivers drawn from a Norwegian insurance company's files (drivers who had reported a crash in the last 6 months) 4448 drivers responded (30% response rate) of whom, 49 had a stroke: At fault n = 36, Not at fault n = 13.	Self-reported presence of medical condition (stroke);	Crude odds ratio for at-fault crash: 2.31 – highest across all medical conditions Age and distance-adjusted odds ratio (p value): 1.930 not sig. (0.07) – Suggests risk partly explained by aging.
Salzberg & Moffat (1998)	Case-control; Cases n=21 with stroke or CVA; passed Washington state special exam in 1994 Controls n= 449 drivers not in special exam program in 1994; age, gender, city of residence matched	(i) Crashes per 100 drivers per year (ii) Citations per 100 drivers per year	Pre-exam crash rate: Case:Control 5.4:3.8 Post exam crash rate: Case:Control 4.4:1.2 Pre-exam citations: Case:Control 8.2:7.5 Post-exam citations: Case:Control 7.3:2.3

Approaches to management:

Assessing Fitness to Drive.

After a CVA, private licences are revoked for a one-month period in Canada, UK, and New Zealand and are reissued subject to neurological assessment and periodic reviews (see Table 7). In the US, Sweden and Australia licences are permitted subject to thorough medical and neurological evaluation and regular reviews. Guidelines regarding a one-month non-driving period and regular review of fitness to drive following stroke seems prudent given the increased risk of subsequent stroke and seizure following an initial stroke.

Commercial licences (see Appendix D) are generally issued if recovery from CVA is deemed sufficient to meet with medical approval. Typically periodic reviews and monitoring are required, with the exception of New Zealand where stroke participants are considered unfit to drive, unless there are ‘sound reasons’ to the contrary.

Training and Rehabilitation

CVA or stroke can cause both cognitive and physical impairment, both of which have the potential to impose serious constraints on driving ability. The site and degree of brain damage determines the functions and abilities that are affected. For example, damage to the occipital cortex can result in visual field and visual attention deficits (Mazer et al. 2003), while damage to the frontal lobe can result in impaired higher order executive functions such as planning (Sims, 1992). As described previously, all areas of cognitive ability may be affected and wide individual differences are observed not only in the extent to which brain damage is manifest, but in the degree of compensatory behaviour in which individuals are able to engage. Furthermore, it is important to note that these higher order cognitive impairments may continue even after the recovery of visual perception and motor strength (Lundberg et al., 2003). Lundberg et al. caution that these long-term impairments should be taken into consideration when determining a person’s rehabilitation potential – including his or her fitness to return to driving.

Fisk, Owsley and Pulley (1997) reported a study, which investigated the advice and evaluations participants with stroke received concerning returning to driving. Thirty percent of their sample of 290 participants resumed driving after their stroke, with 48% of these reporting no advice from health care professionals, and 87% receiving no evaluation. This indicates that there is a serious need for research in the area of returning to driving post-stroke, to better inform professionals and to provide participants with better evaluations and knowledge on which to base their decisions.

Post-CVA rehabilitation strategies tend to focus mainly on physical problems and attempt to maximise the amount of motor recovery. Adaptive equipment is frequently used for physical problems. A spinner knob can be attached to the steering wheel to allow controlled steering with the use of one hand. Pedals may be relocated or reassigned depending on degree of use of the feet, electronic control touch pads, and brake extension levers are also available. It must be noted that individual assessment should be sought to ensure that each specific case can be referred to the most appropriate modifications (if necessary) and most suitable retraining program. Following Traumatic Brain Injury (TBI), the deficits are not generally physical in nature, so vehicle modification is not an issue here. Rehabilitation is likely to focus on relearning driving skills in the face of any cognitive deficits due to injury. One study

that looked at this type of issue was Mazer et al. (2003). They looked at the outcome of either UFOV (useful field of view) training of visual processing speed, divided attention, and selective attention or traditional computerized visuoperception retraining. Outcome was measured for an on-road driving test. No differences between the two types of training were found overall, but participants with right-sided lesions were twice as likely to pass the on-road test (52.4% compared to 28.6%). This indicates that training programs should be targeted on an individual basis. Particularly when the differences in individuals with TBI and individuals with stroke for example, are considered. People who have experienced a stroke in general are likely to be older and therefore more experienced drivers than individuals with TBI. Retraining program differences should reflect this. People with TBI are also less likely to have physical deficits, so vehicular modifications will not be required.

A major drawback (particularly for the elderly) with vehicle adaptation and driver rehabilitation/retraining programs is cost. The majority of freely available retraining courses are refresher courses, targeting the general elderly population, and not tailored to the specific needs of stroke or TBI patients.

Table 7 Private licensing guidelines for drivers with CVA

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austrroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Stroke	<p>Desist from driving for 1 month minimum.</p> <p>Driving may resume if:</p> <ol style="list-style-type: none"> 1. Person has functional ability to drive a vehicle (no clinically significant motor, cognitive, perceptual or visual deficits); 2. No risk of recurrence found in neurological assessment and post stroke seizure has not occurred in interim; 3. Any underlying cause has been treated. <p>Person may be required to undergo a road test if there is any “residual loss of motor power” (p43).</p> <p>Any changes in personality, alertness or decision-making</p>	<p>An unconditional licence may not be held if the person has had a stroke.</p> <p>A conditional licence may be issued upon medical advice taking into consideration completeness of recovery, visual field impairments, risk of recurrence & subject to a driving assessment.</p> <p>Periodic review required.</p>	<p>Desist from driving for 1 month.</p> <p>Driving may resume if there is a satisfactory recovery.</p> <p>DVLA notification required if residual neurological impairment remains 1 month after the stroke, especially visual field & cognitive defects & limb disabilities.</p> <p>Car modifications may be required for severe physical impairments.</p> <p>A driver experiencing multiple TIAs may require at least a period of 3 months without attacks before driving.</p> <p>Epileptic seizures that occur within 24 hours of a stroke are</p>	<p>An unrestricted licence may be issued if the person is able to control equipment & has no, minimal or slight neurological impairment.</p> <p>A medical report and regular review is required for minimal to slight impairment.</p> <p>If the person has moderate impairment of dexterity, a road and driving skills test must first be passed before licensing can occur. Annual review is required.</p> <p>Greater restrictions (speed/area/time of day/must be accompanied by licensed driver) are imposed if there is <i>temporary</i> significant neurological impairment.</p>	<p>Desist from driving for 1 month minimum.</p> <p>Licence denial for any of the following sequelae of stroke:</p> <p>Homonymous hemianopia, ataxia, vertigo, diplopia, epilepsy, recurrent ischaemic attacks & significant CVA disorders.</p> <p>Resume driving only when recovery is complete & there is no significant disability that will impair safe driving.</p> <p>Car modifications for any residual limb disability may be</p>	<p>Fitness to drive is assessed using the same criteria as that set down for CVA disease i.e. licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Stroke assessment is also to make particular note of any transient ischaemic attacks or other risk factors eg high blood pressure, high cholesterol, atrial fibrillation or vascular deformity.</p> <p>Other after effects of stroke such as paralysis, visual problems, or cognitive & consciousness disturbances are to be assessed using the standards set down under the appropriate disorder.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
	<p>ability to be taken into consideration by GP.</p> <p>Patients with visual field deficits require examination by an optometrist or ophthalmologist.</p> <p>Regular review required.</p>		<p>to be treated as provoked if the person has not had a seizure before.</p>	<p>Six monthly review is required.</p>	<p>required.</p> <p>Only in exceptional circumstances will people with ischaemic attacks & significant CVA disorders be granted a conditional licence & only 1 year after stroke occurrence.</p>	<p>Assessments are also to take account of the causes, development & treatment of the disease.</p>

References

- Akinwuntan, A.E., Feys, H., DeWeerd, W., Pauwels, J., Baten, G. & Strypstein, E. (2002). Determinants of Driving After Stroke. *Archives of Physical Medicine and Rehabilitation*, (3) 334-341.
- Australian Institute of Health and Welfare [AIHW]. (2006). *Chronic disease and associated risk factors in Australia, 2006* (Cat. no. PHE 81). Canberra: Australia. AIHW.
- Austrroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Bonita, R. (1992). Epidemiology of stroke. *The Lancet*, 339, 342-344.
- Bonita, R., Solomon, N. & Broad, J.B. (1997). Prevalence of stroke and stroke-related disability. Estimates from the Auckland stroke studies. *Stroke*, 28, (10), 1898-902.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Clark, I.D. & Opat, L.J. (1994). The prevalence of stroke in those at home and the need for care. *Journal of Public Health Medicine*, 16(1), 93-6.
- Drivers Medical Group DVLA Swansea. (2008). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Fisk, G.D., Owsley, C. & Pulley, L.V. (1997). Driving after stroke: Driving exposure, advice, and evaluations. *Archives of Physical Medicine and Rehabilitation*, 78, (12), 1338-45.
- Heikkila, V.M., Korpelainen, J., Turkka, J., Kallanranta, T. & Summala, H. (1999). Clinical evaluation of the driving ability in stroke participants. *Acta Neurologica Scandinavica*, 99, (6), 349-55.
- Koepsell, T., Wolf, M. & McCloskey, L. (1994). Medical conditions and motor vehicle collision injuries in older adults. *Journal of the American Geriatrics Society*, 42, 695-700.
- Kotterba, S., Widdig, W., Brylak, S., & Orth, M. (2005). Driving after cerebral ischaemia – a driving simulator investigation. *Wiener Medizinische Wochenschrift*, 155, 348-353.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Lings, S. & Jensen, P.B. (1991). Driving after stroke: a controlled laboratory investigation. *International Disability Studies*, 13, 74-82.
- Lundberg, C., Caneman, G., Samuelsson, S., Hakamies-Blomqvist, L. & Almkvist, O. (2003). The assessment of fitness to drive after a stroke: The Nordic Stroke Driver Screening Assessment. *Scandinavian Journal of Psychology*, 44, 23-30.

- McGwin, G. Jr., Sims, R. V., Pulley, L. & Roseman, J. M. (1999). Diabetes and automobile crashes in the elderly. *Diabetes Care*, 22(2), 220-227.
- McGwin, G. Jr., Sims, R. V., Pulley, L. & Roseman, J. M. (2000). Relations among chronic medical conditions, medications, and automobile crashes in the elderly: A population-based case-control study. *American Journal of Epidemiology*, 152(5), 424-431.
- Mazer, B.L., Sofer, S., Korner-Bitensky, N., Gelinas, I., Hanley, J., & Wood-Dauphinee, S. (2003). Effectiveness of a visual attention retraining program on the driving performance of clients with stroke. *Archives of Physical Medicine and Rehabilitation*, 84(4), 541-50.
- National Institute of Neurological Disorders and Stroke (NINDS, 2009). Transient Ischemic Attack (TIA) Information Page. Retrieved May 11th, 2009 from <http://www.ninds.nih.gov/disorders/tia/tia.htm>
- Nouri, F.M., Tinson, D.J. & Lincoln, N.B. (1987). Cognitive ability and driving after stroke. *International Disability Studies*, 9, 110-115.
- Parker, D., McDonald, L., Rabbitt, P. & Sutcliffe, P. (2000). Elderly drivers and their accidents: the Aging Driver Questionnaire. *Accident Analysis and Prevention*, 32, 751-759.
- Sagberg, F. (2006). Driver health and crash involvement: A case-control study. *Accident Analysis and Prevention*, 38, 28-34.
- Salzberg, P. & Moffat, J. (1998). *The Washington State Department of Licensing Special Exam Program: An Evaluation*. Washington Traffic Safety Commission. Olympia, WA.
- Sims, B. (1992). Private Transport for elderly and disabled people. Report 308 Transport and Road Research Laboratory. Department of Transport: Crowthorne.
- Swedish Road Administration. (1998). *Swedish Road Administration Statute Book*. Sweden.
- Thom, T., Hasse, N., Rosamond, W., Howard, V. J., Rumsfeld, J., Manolio, T., et al. (2006). Heart Disease and Stroke Statistics—2006 Update: A Report From the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*, 113, e85-e151.
- Truelsen, T., Bonita, R. & Jamrozik, K. (2001). Surveillance of stroke: a global perspective. *International Journal of Epidemiology*, 30, S11-S16.
- Utah State Driver License Medical Advisory Board. (2006). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- World Health Organisation [WHO]. 2008. *The global burden of disease: 2004 update*. Geneva: Switzerland. World Health Organisation Press.

3.4 COGNITIVE IMPAIRMENT

Cognitive impairment is a broad term given to a wide variety of dysfunctions resulting from an enormous number of potential causes. These range from organic diseases such as the dementias, to physical injury and also conditions such as pre-operative hypoxia. For the purposes of the present review it is necessary to limit the discussion to the major causes of cognitive impairment namely dementia, traumatic brain injuries (TBI, see this section) and cerebrovascular accident (stroke) (see section 3.3).

There are many issues involved in assessing these cognitive abilities, notably the wide variety in assessment methods of cognitive and or motor ability. The relative merits of the myriad of performance tests provide an extremely large and complex debate within modern cognitive psychology and an account of this is beyond the scope of the present review. It is necessary though to point out that there is much disagreement among psychologists as to which tests of ability are reliable and valid for which facets of cognition. This is likely to have an important bearing on the analysis of evidence on driving and crash risk amongst drivers with cognitive impairment. To give an example of one important feature of the debate: many authors arbitrarily label tests as “cognitive” or “motor” tests, when many tests involve more than one area of ability. Motor skills by definition must involve some degree of cognitive processing, therefore it will be near impossible to develop a task of “pure” motor function, conversely many cognitive tasks involve learned motor skills. In reviewing the literature here, only brief discussion of the merits of particular cognitive tests employed will be given where the tests are either unusual or idiosyncratic.

Further when discussing cognitive impairment there are other problems that must be considered, notably those concerning clinical issues. In particular, there are variations in clinical judgement concerning diagnosis of disease. This is especially salient if the research reported is based on participants who have been assessed on criteria established (as is frequently the case) without the input of suitably qualified medical professionals (British Psychological Society, 1999). Related to this is the possibility that (especially where older drivers are concerned) there may be the presence of non-diagnosed conditions present within control samples, another possible confounding factor will be cognitive changes associated with normal ageing (Stutts, Stewart & Martell, 1998).

Comorbidity may also be a contributory factor in diminished driving ability due to cognitive impairment, especially in older drivers. That is to say one or more other (non-cognitive) conditions may compound cognitive impairment, increasing the risk of crashing. Also this may give rise to the need for medication, which in itself may cause a degree of cognitive impairment. Although these issues appear somewhat tautological it is necessary to bear them in mind when critically assessing research in the area of cognitive impairment and crash risk.

3.4.1 DEMENTIA

Definition of dementia

Dementia refers to a global deterioration of cognitive function due to atrophy of the central nervous system. The level of deterioration in a range of areas of cognitive function varies widely between individuals. Diagnostic criteria in common use include

those specified by National Institute of Neurological and Communicative Disorders and Stroke (NINCDS), the National Institute of Neurological Disorders and Stroke (NINDS) and the American Psychiatric Association DSM-IV (APA, 1994).

DSM-IV criteria specify as necessary components for a diagnosis of dementia: loss of function in multiple cognitive domains such as memory impairment and at least one of the following: aphasia, apraxia, agnosia, or disturbances in executive functioning.

It is also important to note that there exists a state of “pre-clinical dementia” wherein the brain is affected by the disease with some level of impairment experienced by the individual for many years prior to diagnosis. In some individuals this may be assumed to be simply a corollary of normal ageing. For example, individuals can experience mild cognitive impairment (MCI) which is a diagnosis used to describe individuals with memory impairment who do not have dementia. Mild cognitive impairment can be progressive, remain stable or even improve. In some cases MCI can predispose dementia. However unlike dementia MCI does not interfere with the ability to partake in daily activities (Gauthier et al. 2006). This has important implications for driving. This has important implications for driving risk.

The most common form of dementia, *Alzheimer’s disease (AD)*, accounts for 50-75% of all cases of dementia. Another 10-20% of dementia cases are attributed to blood vessel disease or diffuse ischaemia. This form of dementia is called *vascular dementia*. The remaining cases of dementia result from a variety of less common disorders. Other types of dementia have been classified including *fronto-temporal dementia* (1 in 5000 people), which is more common at younger ages (onset around 45-50 years) and *dementia with Lewy Bodies* (up to 10% of dementia cases).

Vascular dementia: In vascular dementia, ischaemia or blockage in cerebral blood vessels leads to damage to or death of brain tissue (see also section 3.3 for discussion of stroke). The location and severity of the interruption of blood flow in the brain determines the severity of the cognitive deficits and the resulting problems. Speedy onset of dementia-like symptoms may be an indicator of this type of dementia (see Roman, Tatemichi, Erkinjuntti, et al., 1993 for criteria for probably vascular dementia). Individuals with vascular dementia may possibly remain at a stable level of functioning or indeed even show slight improvements in cognitive capabilities, before quickly displaying further symptoms if successive infarcts occur (see Schneider, Wilson, Cochran, Bienias, Arnold, Evans, & Bennett, 2003). High blood pressure plays a crucial role in the onset of many cases of vascular dementia.

Dementia of the Alzheimer’s type (AD): AD is a progressive degenerative brain disorder that seriously impacts upon a person’s ability to carry out tasks involved in daily living. AD damages many parts of the brain including those that control planning, attention, memory, and language (see Morris, 1996 for review). Symptoms may include asking the same questions repeatedly, getting lost in familiar surroundings, being unable to form plans and follow directions, becoming confused about time, people, and locations, and failing to monitor personal safety. Although these general problems will be evident in most people with dementia, the progression of the disease varies from person to person. In its early stages, the symptoms of AD may be difficult to separate from declines in cognitive performance experienced by normal healthy elderly people (see Rabbitt, 1993).

AD is the most commonly occurring of the dementias, encompassing approximately 50 – 70% of all presentations of dementia (Eby et al., 1994; Cohen & Dunner, 1980). Prevalence estimates for AD increase dramatically with age, as specified above. It is also important to note that many more potential cases of AD and other dementias go undiagnosed because individuals generally accept early symptoms of ageing.

Criteria for probable Alzheimer's disease NINCDS-ADRDA (McKhann, Drachman, Folstein, Katzman, Price & Stadlan, 1984) are specified as follows:

Dementia established by clinical examination and documented by the Mini Mental State Examination (MMSE) (Folstein, Folstein & McHugh, 1975); Blessed Dementia Scale or some similar examination, and

- confirmed by neuropsychological tests;
- deficits in two or more areas of cognition;
- progressive worsening of memory and other cognitive functions;
- no disturbance of consciousness;
- onset between ages 40 and 90, most often after age 65; and
- absence of systemic disorders or other brain diseases that in and of themselves could account for the progressive deficits in memory and cognition.

It is common for individuals to show symptoms of both AD and vascular dementia at the same time, their severe symptoms being an interaction of the two (Alafuzoff, Iqbal, Freiden & Winblad, 1989). It is important to note that many clinicians fail to distinguish between AD and vascular dementia. This diagnostic debate (and whether it is truly a concern for experimental studies) is beyond the scope of the current review, but nevertheless may appear as a caveat in some of the reviewed papers. Many indeed do not mention the selection criterion of the included participants with AD.

For the purposes of the present review the terms dementia or Alzheimer's disease will be used, consistent with their use in the reviewed papers.

Assessment of cognitive dysfunction

General level of cognitive dysfunction is commonly assessed using the MMSE or an equivalent instrument. This comprises a set of general memory questions, where regardless of intellectual ability, it is unlikely that a normally functioning individual would make many, if any, errors. For example, individuals are asked: "What day is it today?" Another test used for assessment of cognitive function in AD is the Clinical Dementia Rating (CDR) scale. This classifies people with dementia into "no dementia", "mild", "moderate" or "severe" (Berg, 1988). These tests are not equally sensitive in assessing all types of dementia, and particularly may lack sensitivity in detecting cognitive problems in frontal lobe dementia. Indeed, establishing cut-off scores on different tests for diagnostic purposes has been problematic, not least because no two people with exactly the same brain damage perform in the same way.

There are of course many other (and more sensitive) diagnostic tools for assessing level of cognitive function in dementia that aim to determine the extent of the impairment of the cognitive symptoms. Detailed descriptions of these can be found elsewhere (see Alberta Medical Association, 2002). Importantly it must be remembered that the only fully accurate method of diagnosing AD (in particular) is at autopsy.

Generally younger people with dementia will approach medical help in the earlier stages of the disease, as their symptoms are likely to appear unusual to them. However, in the case of frontal lobe dementia, which generally has a younger age of onset than other dementias, lack of insight into declining abilities may also contribute to delays in seeking medical advice. Similarly, older people may not present until the disease is relatively more advanced as they may have accepted the earlier symptoms as a natural corollary of growing old. The influence of relatives and spouses should not be overlooked either. If they accept the symptoms as natural ageing they are likely to delay seeking help, yet if they are worried then help may be sought sooner.

Prevalence of dementia

In 2004 the WHO estimated that 24.2 million people suffered from dementia worldwide (WHO, 2004). The majority of people with dementia live in developed countries in Europe and the Western Pacific. In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at almost 6.8 million or around 2% of the total population. Similarly, the prevalence of this disease in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 10.8 million or around 3% of the total population. Recent estimates for Australia show that there were 190 000 cases of dementia (approximately 0.95%) in 2006 (AIHW, 2006).

Dementia can occur at any stage of adulthood however the risk of developing dementia increases markedly with age (see Table 8). With the ageing of the population in most Western countries, this means that the number of cases of dementia is also on the increase. Recent estimates suggest that there will be four times as many people with dementia in developed countries by the year 2025 compared with the year 2000 (Access Economics, 2003).

Table 8 Prevalence of dementia by age and continent

Age Group	Europe	North America	Australia (2002)
60-64	0.4-1.0	0.2-0.3	
65-69	0.9-1.4	0.8-0.9	
70-74	2.1-4.1	1.3-2.0	1.5
75-79	4.6-14.6	3.6-6.3	
80-84	9.6-27	8.9-12.7	6.3
85-89	20.4-38.3	16.3-29.7	
90-94	28.3-57.3	40.4-74.3	30.2
95+	42.3-55.8	58.6	

(Sources: Wimo, Jonsson, Karlsson & Winblad, 1998, p. 14; and Australian Bureau of Statistics; cited in Access Economics, 2003, p. 31.)

Functional impairments associated with AD relevant to driving

Although AD affects every aspect of behaviour, the cognitive impairments (and particularly the memory deficits) are the most obvious early symptoms and have attracted the most research attention (Lezak, 1995). Wide variations exist in the nature and level of these impairments between individuals.

Detailed discussion of diagnostic issues and underlying mechanisms is beyond the scope of the present review. These issues are widely discussed elsewhere (e.g. see Alberta Medical Association (AMA), 2002 for discussion of diagnostic issues).

Symptoms of dementia and AD in particular have been identified in the above sections. In addition, the cognitive impairments most relevant to driving are outlined below. While these cognitive impairments are presented here as separate constructs, it is not intended to imply that treatment can be approached in a mechanistic way. Nor is it likely that any one area of difficulty will explain fully the difficulties experienced by drivers with dementia. Rather, these constructs may be helpful in understanding the wide range of areas of impairment that might impact on driving.

Memory

Memory is perhaps the most notably affected cognitive function in dementia. The most severe problems occur in the areas of procedural memory (Morris, 1996), semantic memory (Hodges, Salmon & Butters, 1992) and prospective memory (Smith, Della Sala, Logie & Maylor, 2000). Deficits in these areas may all have a detrimental effect on driving ability and may consequently increase crash risk (Duchek, Hunt, Ball, Buckles & Morris, 1998). The histopathological indicators of AD, amyloid plaques and neurofibrillary tangles, are generally found in large numbers in the hippocampus, causing widespread atrophy (see Squire, 1992 for full discussion). The hippocampus is a brain structure generally considered to be involved in memory functioning. That is not to say that this is the sole location of pathology and other areas such as the frontal lobes can also be seriously affected, with consequent impairments in executive functions (see below).

The importance of memory to all facets of daily life is so great that memory deficits can preclude individuals from performing even the most fundamental of everyday tasks successfully. In summary, people with dementia have been shown to have significantly reduced short-term memory spans, impaired working memory performance and long term and prospective memory deficits (see Smith et al., 2000; Della Sala & Spinnler, 1999 Spinnler et al., 1988 for further discussion).

Psychomotor abilities

People with dementia also generally exhibit decreased psychomotor abilities; they are not as effective at controlling their own movements as age matched healthy elderly people. Gilleard (1984) reported younger people with dementia were markedly slower than their healthy peers whereas with advancing age it was accuracy of movement, which was more adversely effected. This is intuitive as dementia, by definition, is a global reduction in cognitive performance. Also movement difficulties may emerge, such as apraxia and Parkinson-like symptoms (see section 3.8.1 for a more detailed description of Parkinson's Disease).

Attention

Parasuraman and Nestor (1991) carried out an extensive review of the role of attention in driving skills, both in normal ageing and Alzheimer's disease. Attention is an important component of driving as a cognitive task, and declines in attentional abilities are known to occur very early in the onset of AD. This is important, as it is likely that those in the early stages of the disease will be the largest group with the disorder who are continuing to drive.

As attention is widely discussed in many studies, many of which include attentional measures a discussion of attention and its' relation to dementia is necessary. Cognitive psychology generally distinguishes three forms of attention: sustained attention or vigilance, selective attention, and divided attention. All three are important in driving and all three are diminished in dementia (Spinnler et al., 1988).

Driving requires on-going monitoring of both environmental factors outside the vehicle and internal controls of the vehicle. This requires continual attentional shifting (selective attention) between the two and is generally measured using tasks such as the Stroop and Trail Making tests, or tests of dichotic listening. Poor performance in these types of tasks in individuals with dementia has been widely reported (see Grady, Haxby, Horwitz, Sundaram, Berg, Schapiro, Friedland, & Rapoport 1989 for discussion).

Many studies have also shown that dementia impairs performance on divided attention tasks (see Baddeley, Logie, Bressi, Della Sala and Spinnler, 1986). Dementia studies show that for tests of divided attention, observed dual task decrements (that is to say the reduction in simultaneous performance on both parts of a dual task compared with performance on the separate components) follow a similar pattern to that of normal ageing (McDowd & Craik, 1989). However the extent of the deficits is greater in dementia than in normal ageing (Logie, Maylor, Della Sala & Smith; 2003, in Press).

Dementia has also been shown to impair sustained attentional performance or vigilance, again following a similar pattern as in normal ageing but the magnitude of the deficits being greater in dementia (Salthouse, 1985). Impaired sustained attention is an

important issue for driving ability, and may also be an underlying factor in deficits in performance in other general cognitive tasks (Rabbitt, 1990).

Visuospatial functions

Driving requires the ability to process visual information, and to interpret the location of object with respect to the drivers environment. Visuo-spatial skills and visual perception ability are also severely impaired in people with dementia, which has been largely attributed to diminished attentional abilities. Impairments in these areas have significant consequence for driving safely. Again, many tasks used to assess visuo-spatial abilities have been devised and a comprehensive review is not feasible here (see Moss & Albert, 1988 for discussion). Specific tasks used in reviewed papers will be described where appropriate.

Executive functions

The term executive function generally refers to a grouping of high-level cognitive processes underlying everyday abilities such as planning, anticipation, mental flexibility, problem solving and feedback utilisation. Within the executive system it is proposed that there are the processes themselves and an overall processing capacity (Spinnler, Della Sala, Bandera & Baddeley, 1988). Declines in performance in normal ageing are due to reductions in overall capacity. In contrast, in people with AD, there is thought to be a disturbance in both the functionality of the individual processes as well as a decline in overall capacity (Baddeley, 1999). The damaging neuropathology in AD disrupts the running of these processes with the utility of dependent everyday abilities seriously compromised. In normal ageing these abilities are only slowed or are unable to cope with as much information, whereas in AD it is the individuals ability to cope that is lost. Also people suffering the effects of normal ageing are able to compensate for any dysfunction here in other ways, AD patients cannot. Loss of abilities such as planning, adaptivity to circumstance and feedback utilisation will have serious implications for driving.

Summary

Numerous studies have considered the impact of cognitive decline in older drivers, including risk associated with impairment in specific domains such as attention, visual search and visual attention and executive functions (e.g. Ball et al, 1998; Marottoli, Cooney, Wagner, Doucette & Tinetti, 1994; Owsley et al., 1991; 1998; Stutts, 1998; Stutts et al., 1998; see also section 3.13 for a review of vision-related impairments, and also Staplin, Lococo, Stewart, & Decina, 1999 for an annotated compendium of assessment methods for age-related cognitive impairments and findings relevant to driving).

Hunt, Morris, Edwards and Wilson (1993) outline the situations that arise whilst driving where people with dementia may experience difficulties:

- forgetting of familiar routes and getting lost;
- confusion between pedals in a stressful situation;
- situations requiring complex or fast cognitive processing may cause a person with dementia to stop in traffic, when there is no need to stop;

- at intersections people with dementia may fail to yield right-of-way appropriately;
- verbal suggestions from passengers e.g. directions may not be interpreted quickly enough or appropriately for timely action to be taken.

Safe driving places demands on memory, attention (both selective and divided), decision-making, planning, reactions, vision and other sensory processing. It is likely, then, that diminished capability in any of these facets of cognition has the potential to compromise driving performance and lead to an increase crash risk.

Pre-May 2003: Relationship between dementia and road safety outcomes

Previous reviews of the literature concerning driving and dementia appear to agree in general terms about the major issues involved in this subject area. They concur that the issue of driving with dementia is important in regard to personal independence and mobility, and is also important with regard to personal and public safety (see Adler, Rottunda & Dysken, 1996; Dubinsky, Stein & Lyons, 2000; Donnelly & Karlinsky, 1990; Lloyd, Cormack, Blais, Messeri, McCallum, Spicer & Morgan, 2001; Withaar, Brouwer & van Zomeren, 2000). Early studies published around the mid-eighties, of driving and dementia in general do not appear to show differences between safe and unsafe drivers based on their performance on cognitive tests (Lucas-Blaustein, Filipp, Dungan and Tune, 1988). Some predictive studies have shown that persons displaying cognitive deficits do perform significantly worse on neuropsychological test batteries and tests of driving ability. In general, only moderate correlations between cognitive performance and driving ability have been shown. This makes it very difficult to differentiate between people with forms of cognitive impairment who are competent to continue driving, and those who are not. There is of course wide debate concerning representativeness and selection of participant samples, selection of cognitive/neuropsychological measures, absence of reporting cut off scores and the measuring of driver performance (Molnar et al. 2006).

These previous reviews reach a consensus stating in general that decisions to remove licences from people with dementia is a complex issue, and clinicians, general practitioners, licensing authorities and other health professionals should work in conjunction to develop the best possible practice for assessing driving ability in such cases. Yet little consensus as to what this will be or indeed how this will be achieved is apparent. Understandably, the many studies that have been conducted in this area have contributed to the understanding of driving in relation to cognitive performance and the abilities required for successful driving, and subsequently this work has given rise to new questions and research avenues. It is to be hoped that with recent developments in technology, particularly in the areas of computer software and driving simulators, that many of these research issues may be satisfactorily addressed. Table 9 shows a summary of the findings of studies that have investigated dementia and road safety outcomes including crashes, citations and driving performance.

Crashes

Lucas-Blaustein, Filipp, Dungan and Tune (1988) reported pilot questionnaire data concerning involvement in crashes of drivers who continued to drive after a diagnosis of dementia of varying types. Criteria specified by NINCDS and DSM-III were used for diagnosis. The authors found that 33% of the 53 participants with dementia had at least

one crash since onset of symptoms, and further 11% had “caused” crashes according to the reports from carers. They found no differences on clinical or cognitive test parameters between those who had crashes since onset and those who had not. This would suggest that the cognitive tests employed were either not sensitive enough or not specific enough. The accuracy of carer reports on crashes and ‘fault’ also limits the reliability of these findings. Moreover, the relatively small sample size and lack of a control sample raises further doubts about the strength of their recommendations to stop people with dementia from driving.

Drachman and Swearer (1993) also report a questionnaire study, administered to the carers of 130 participants with AD and 112 controls, to investigate the frequency and severity of crashes. The participants with AD were reported as having 0.091 crashes each year compared with 0.040 for controls. The authors further analysed the crashes of participants with AD by year since onset of dementia, showing a steady increase in crashes as the disease progressed. This procedure indicated that in the early stages of the disease, the frequency of crashes involving participants with AD was no different from the controls. The point should be raised as to why there were fewer controls than cases involved in this study.

Dubinsky, Williamson, Gray and Glatt (1992) conducted an interview study of 67 family members of participants with AD and compared them with a sample of 100 control participants. They report that 68.7% of the participants with AD had stopped driving either voluntarily or through the insistence of their families. These participants were significantly more cognitively impaired than the remainder who continued to drive. The measure of cognitive impairment in this study was the MMSE (a useful if not overly sensitive tool). The participants with AD who continued to drive had significantly more crashes ($M = 26.3$, averaged per million miles driven) than the controls ($M = 14.3$ per million miles driven). Another point to be noted is that the age of the participants with AD ($M = 71.3$) was significantly higher than that of the controls ($M = 64.6$). This is highly likely to contribute to a degree of bias in the results, as it is well known in the ageing literature that there are significant differences in general cognitive and motor performance across an age range as wide as this (e.g. Rabbitt, 1993; Salthouse, 2003; Salthouse, 2000).

In an Argentinean study, Zuin, Ortiz and Lopez (2002) examined driving behaviours in 56 people with dementia using DSM-IV criteria and 31 normal elderly controls, comprehensively acknowledging the various types of dementia within their sample. Caregivers were interviewed concerning the driving behaviour and frequency of collisions exhibited by the participants they cared for. The people with dementia displayed significantly more frequent crashes ($\chi^2 = 2.73, p = 0.012$), and more multiple crashes ($\chi^2 = 3.68, p = 0.05$). They concluded that the presence of dementia is a strong indicator of crashes and abnormal driving behaviour. Interestingly, they also found that being male was a strong predictor of crashes in the dementia group. This may be explained by the common trend in current cohort of older drivers for males who do more driving than females. In an attempt to overcome this, they collapsed the dementia types to give a more acceptable number of participants, which unfortunately reduces the power and generalisability of their findings. A significant limitation of this study is the spouses and carers of people with dementia as controls.

Tuokko, Tallman, Beattie, Cooper and Weir (1995) carried out a retrospective review of the driving records of 249 participants (with age matched controls) referred to a

dementia clinic. Using the NINCDS-ADRDA criteria for dementia, 165 met the criteria and 84 did not. The participants with dementia were found to be 2.5 times as likely than the controls to have been involved in a crash. Even the 84 people who did not meet the criteria were 2.2 times more likely than the controls to have been involved in a crash. Due to varying times since onset of dementia these authors were unable to standardise a time period equating driving exposure for cases and controls.

Waller, Trobe and Olson (1993) reported findings that are contrary to the apparent trend in the literature. They reported no differences in crash rate between participants with AD and normal elderly participants, and no differences in the characteristics of reported crashes. The sample consisted of 99 participants with AD and 495 age and gender matched comparison participants. Structured interviews with the primary caregivers of the participants with AD were carried out and State driver records were accessed for crash information. Standardisation of crashes per driver year was analysed, giving 6.8 crashes for participants with AD and 6.2 crashes for controls per hundred driver years. These authors also looked at the types of crashes experienced by the two groups. There were no differences in type of crashes between the groups, neither were there differences in crash severity.

A later study which goes against the general trend of increased crash risk in AD, is that reported by Trobe, Waller, Cook-Flannagan, Teshima and Bieliauskas (1996). This study compared 143 participants with AD with a 5:1 (715) ratio of age-matched controls. It must be pointed out though that whether or not the controls were still driving was not verified in all cases, and the controls were not screened for possible early stage dementia. The crash and citation history of the participants with AD was obtained from the State Authority. All participants completed a comprehensive neuropsychological test battery including sub-tests from the Weschler Adult Intelligence Scale-Revised (WAIS-R) and the MMSE. After standardising the data to generate an overall annual crash rate they found that the participants with AD had a crash rate of 0.05 pre diagnosis and 0.08 post diagnosis. These rates did not differ from those of the controls (0.05 and 0.08), although significantly higher than the crash rate (0.03) for all drivers (i.e. all U.S. licensed drivers aged 55 years in 1999). Curiously, the participants with AD who crashed scored significantly better on the neuropsychological tests than those who did not crash, with the exception of the MMSE where there was no difference. However there is a strong possibility that the crash rate of the better test scorers may have been inflated as they were driving more and had more exposure to risk. Also there is the possibility of restrictions being placed on driving by the participants themselves, family or physicians' recommendations.

Salzberg and Moffat (1998) examined the crash and driving citation records of 46 drivers with dementia and psychiatric illnesses (i.e., Alzheimer, bipolar disorders, dementia, and confusion/memory loss) who were referred to the Washington State Department of Licensing Special Examination Program (see next section for more information regarding citations rates). As outlined in more detail in section 3.13, this special exam program included an in-depth interview and an extended on-road driving test typically within a limited range of travel near the driver's residence and routes used by the driver. The most common outcome of the examination process was to restrict the driver's travel to within specific areas and times of day, and requires the driver to use corrective lenses or particular vehicle controls (e.g., power steering). However, drivers who failed the exam had their licences cancelled. The records of drivers with dementia/psychiatric illness who passed the exam were examined over a 5-year period

(1.75 years prior to the examination and 3.75 years after) and compared to 449 drivers in a control group of older drivers without medical conditions matched on age, gender and city. The control group had a crash rate of 3.82 per 100 licensed drivers prior to the examination period and 1.17 in the post examination period. This compares to a total of approximately 4 million licensed drivers in Washington State that recorded a rate of 3.47 collisions per 100 licensed drivers during 1996. The older drivers with dementia/psychiatric illness that continued to drive had a pre-exam crash rate of 12.42 per 100 licensed drivers. This pre-exam crash risk was 3.3 times higher than age-matched control participants without medical conditions, and 3.58 times higher than the Washington State population. After the special exam, the rate of crashes in the dementia/psychiatric illness group dropped to 4.68 per 100 licensed drivers. While the crash rate reduced substantially in the period after the special exam, drivers with dementia/psychiatric illness still had a crash risk approximately four times higher than age-matched controls. However, this study could be criticised because of its use of an aggregate crash outcome measure, which tends to mask the influence of one or two high-risk participants having multiple crashes. In addition, a critical methodological limitation of this study was the failure to adjust the risk estimates for driver exposure or comorbid conditions. It should also be noted that the sample was restricted to a relatively small number of older drivers who were referred to the licensing authority, presumably because of concerns for their driving ability. Thus, case participants are not representative of the population of all drivers with cardiovascular disorders and therefore findings cannot be generalised to the broader population of interest.

In a case-control study with a different methodological approach to those reviewed above, Koepsell, Wolf, McCloskey, Bucher, Louie, Wagner and Thompson (1994) examined whether specific medical conditions, including dementia, increased the risk of injury due to motor vehicle collisions in older drivers (for a more detailed description of this study method see section 3.5). Drivers ($n = 234$) aged 65 years and older who were injured in a crash during 1987 or 1988 were compared with 446 drivers, not involved in injury crashes, and matched by age, gender and county of residence (see section 3.1 for a more detailed description of the study). Amongst cases, the prevalence of dementia was 1.3% whilst only 0.4% of controls had dementia. The odds ratio, adjusted for age, sex and place of residence only (i.e. not corrected for exposure) showed that prevalence of dementia amongst those who were injury crash-involved was 2.8 times that of the control group who had not been involved in an injury crash. However, the rate of dementia was quite rare and confidence limits around the risk estimate were wide (CI: 0.4-17.0). Hence, the reliability of the risk estimate is questionable. The study should be replicated with a larger sample and with appropriate adjustments for driving exposure.

The same kind of approach was adopted by Johansson, Bronge, Lundberg, Persson, Seideman and Viitanen (1996) who examined the incidence of dementia amongst older drivers (65 years and older, in this case, with and without licence suspensions). Dementia was found in 49% of cases (drivers with licence suspensions due to crashes or moving violations during the previous five-year period) and in 11% of controls (drivers with no licence suspensions in the past five years). The authors also reported a significantly higher incidence of dementia (Clinical Dementia Rating greater than 0) amongst those suspended drivers who were crash involved ($n = 23$) compared with controls who had no involvement in crashes in the previous five years.

Using another approach to understanding the role of AD in fatal crashes, Johansson, Bogdanovic, Kalimo, Winblad and Viitanen (1997) carried out autopsy studies of 98

older drivers who died in crashes. The authors reported that 53% of cases showed sufficient neuritic plaques to satisfy a full diagnosis of AD. This would indicate that drivers with AD might face an increased risk of fatalities in motor vehicle crashes amongst older drivers. Interestingly in commentary on the Johansson et al. study, Rizzo, McGehee, Dawson and Anderson (2001) claimed that none of these cases had previously been diagnosed with AD and that their relatives were unaware of any problem, although Johansson et al. makes no explicit mention of these points. Nevertheless, this raises an important issue for clinicians and the need for better screening tools for early detection.

Following the Johansson et al. (1997) study, Viitanen et al. (1998) reported a study of the neuropathology in drivers aged over 65 who were killed in car crashes in Sweden and Finland between 1992 and 1995. The authors classified crashes as single vehicle, multi-vehicle at intersections and multi-vehicle elsewhere. They found frequencies of pathology within groups were 50%, 47% and 44% respectively. Only 98 out of 188 (52%) of deaths underwent neuropathological study at autopsy. The authors do usefully mention the debate around the classification of AD, and differences with and difficulties within histological procedures used in various centres. They do not account for comorbidity that is an important oversight, as drivers of this age group are more likely to be suffering from non-related yet risk increasing factors.

A recent study, which addressed two key areas of attention and executive disorder, was conducted by Daigneault, Joly and Frignon (2002). The authors conducted two studies looking at relationships between attitude, aptitude and driving behaviour in older people. Although this study did not include people with a diagnosis of dementia, performance measures of attention and executive function were used to assess cognitive functioning of older drivers participants in order to explore associations with crashes. The first study compared self-reports of driving habits between two groups: those having had motor vehicle crashes (n=89) and those who had not (n=90). All drivers were males aged over 65 years. Analyses of variance showed that with age drivers reduced their exposure to risky situations. Yet there were no major differences between crash groups. This may indicate that the relationships may be more complex than first indicated. There was a significant difference between crash groups in the numbers of errors made on the questionnaire. These authors argue that this may reflect general underlying cognitive deficits that could impact on driving. However this is a strong conclusion to draw from a study of this nature, self-reports and no use of multivariate statistics may weaken their position. The same authors then carried out a study aimed at investigating their findings further. Two groups of 30 as in their previous study participated, however when they were separated by age, there was a ratio of 46:14 young to old. Four neuropsychological tests were used: Wisconsin card sort (attention), Colour trails (visual search), Stroop Colour Word (controlled responses) and The Tower of London (planning). Demographics and self-reports of risky behaviours were also collected. Using MANCOVA, the authors were able to conclude that drivers in the motor vehicle crash group showed more cognitive deficits than controls, but a causal relationship is unknown. Drivers having crashes showed more deficits that reflected mental rigidity and poor planning ability. The crash group reported significantly higher scores on the intention to drive carefully measures. This may be due to the fact that they have realised that they have problems in other areas of life. Methodological concerns with this study were the use of self-reports, imbalance in the group sizes for MANCOVA and wide differences in duration of testing sessions (2-4 hours). Any of these could weaken the findings.

Citations

As outlined above, Salzberg and Moffat (1998) specifically examined the citation records of 46 older drivers with dementia/psychiatric illness who were referred to the Washington State Special Examination Program and passed (although most had restrictions imposed on their driving). State citations records were examined over a 5-year period including 1.75 years prior to the exam and 3.25 years after. Older drivers with dementia/psychiatric illness were found to have a citation rate prior to the exam of 23.60 citations per 100 licensed drivers in a year. This pre-exam citation rate was approximately three times higher than that of age-matched control participants without medical conditions (7.51). After the special exam, the rate of citations in the dementia/psychiatric illness group dropped to about one third of the pre-exam rate (8.03), which remained 3.5 times higher than age-matched control participants.

Driving Performance

Fitten, Perryman, Wilkinson et al. (1995) carried out an ambitious and informative study of driving abilities in AD and multi-infarct dementia (MID), using healthy elderly and young groups as controls. Participants who met the stringent diagnosis criteria (NINCDS-ADRDA) undertook an extensive cognitive battery. This battery included well-validated tests of memory, visual tracking, vigilance, divided attention and the MMSE. Performance on these was compared with ratings of on-road driving capability carried out by qualified driving instructors who were blinded to the group membership of the participants. The findings indicated that participants with AD in general drove more slowly than the control groups and committed more driving errors. The participants with AD also performed significantly poorer on the cognitive tests than either control group. These authors also related actual collisions (as recorded by the authorities) to the participants' scores, and adjusted these per thousand miles driven. This analysis indicated that the participants with AD had significantly more collisions and moving citations than the control groups (including participants in the MID group). Their main objective was to contrast actual driving performance between participants with brain disease and healthy individuals. Although the participant numbers were relatively low, (participants with AD =17, participants with MID = 14) the stringent statistical controls allow for some of the former conclusions to be supported. The participants in the study were referred from clinicians, so had already sought help whereas the controls were volunteers, which introduces the issue of strict comparability of the groups and may have implications for the generalisability of the findings. Yet this study does show a clear relationship between dementia, drive score and frequency of movement citations and collisions, and suggests that brain health is more critical to safe driving than age.

Fox, Bowden, Bashford and Smith (1997) reported on a study that attempted to predict the on-road competence of drivers diagnosed with AD. Nineteen probable participants with AD underwent a standardised medical examination (including MMSE) and a neuropsychological assessment. They were then assessed for driving performance on the open road by independent judges. They found that MMSE was a significant predictor of on-road performance. The prediction of the medical examination and the neuropsychological tests were non-significant. Importantly, 63.2% of participants failed the on-road evaluation, yet all participants indicated they wished to continue driving. The authors concluded that AD diagnosis alone may not be a good enough reason for stopping people driving and that an on-road test must be carried out. Once again it must

be noted that the small sample size and lack of age-matched controls severely weakens the study.

Duchek, Hunt, Ball, Buckles and Morris (1998) investigated the role of visual attention in driving performance in participants diagnosed with varying stages of AD and normal elderly individuals. The attentional tasks involved selecting targets from distractors, detecting changes in a continuous visual display, and a useful-field-of-view task (pointing to a presented target in varying positions in the field of view, see section 3.13 for more information regarding field of view). Degree of dementia was assessed using the Washington University Clinical Dementia Rating scale (CDR). The on-road driving test lasted 45 minutes along a pre-determined route, in traffic. A psychometric battery including subsets of the WAIS was also administered. Regression analyses revealed that the visual attention tasks were affected by degree of dementia and that this predicted on-road driving performance. More specifically, error rate in the visual search task was the best predictor of driving ability. The authors concluded that poor ability to discriminate target information from distracting information was a good predictor of driving ability. Given the relatively small sample size and the large number of predictor variables (and their degree of inter-correlation) the overall power of the analysis may be somewhat diminished. But as the authors point out, they have shown that attentional performance is a useful predictor of driving ability, and may go some way to allow identification of drivers who may be “at risk” of crashing, allowing interventions when and if appropriate.

Rebok, Bylsma and Keyl (1990) report a study of 12 participants with AD compared with 18 age-matched controls. Participants viewed films and were asked to respond to incidents in the same manner as they would in a real-life driving scenario. The participants with AD performed significantly worse on all measures than the controls. These authors fail to indicate whether the controls were matched for gender, a pertinent issue particularly in elderly samples, neither did they attempt to standardise for driving exposure.

Rizzo et al. (2001) undertook a study of crashes involving participants with AD in a driving simulator, using 18 (probable) participants with AD and 12 controls. All participants undertook extensive neuro-cognitive test batteries, the main differences between participants with AD and controls were on the Useful Field of View test (visual processing speed and attention skills), and on overall cognitive score. During the simulator testing the critical event was an illegal incursion by another car at an intersection and safe/unsafe avoidance or crash was recorded. The participants with AD crashed 33.3% of the time compared with 0% in the control group, and were able to avoid the crash safely only half as frequently as the controls. The authors also found that a composite measure of the neuro-cognitive battery successfully predicted the likelihood of crashing. Importantly, it was noted that none of the participants with AD committed a safety error whilst driving on the uneventful section of the simulator course prior to the critical incursion. The authors do report a large number of predictor variables within this composite measure that may bias the statistical procedures used given the relatively small sample size.

This study extended the findings of Rizzo, Reinach, McGehee and Dawson (1997), who used a sample of 21 participants with AD and 18 controls also on a driving simulator. This study showed that 29% of the participants with AD experienced “rear-end” crashes compared with 0 controls, and that the participants with AD were twice as likely to

experience a close call than the controls. A limitation of this study and indeed all studies using only driving simulator performance, is that it is difficult to make any meaningful interpretation about participants' real world crash risk. This is discussed in more detail in Chapter 2.

Carr, LaBarge, Dunnigan and Storandt (1998) attempted to differentiate between drivers with dementia and control participants using a traffic-sign naming task. They compared 38 participants with very mild dementia, 30 participants with mild dementia, and 12 participants with moderate dementia with 66 control participants. All participants completed a cognitive battery including WAIS subtests and tests of visuo-spatial ability. The intention was to develop an instrument to screen for drivers with dementia. The total score on the traffic sign-naming task was monotonically related to severity of dementia. It also related significantly to many of the cognitive tests, scores on which also declined with dementia severity. The authors failed to measure years of driving, and did not differentiate between participants who were still driving or those who had given up, this may lead to a biased sample. They also failed to take account of any comorbidity issues, which may have impacted on performance on any of the given tasks. Small sample sizes in the dementia groups may also be problematic given the number of variables included in parts of the analysis. Carr et al. do point out that an important next step in their research would be to validate whether their test identifies drivers at increased risk of crashes.

In the study by Zuin, et al. (2002), described above, driving behaviours of 56 people with dementia and 31 normal elderly controls were compared in addition to their crash involvement. Carers were asked about evidence of abnormal driving by the AD participant, including (i) driving the middle of the road; ii) driving on one side of the road (iii) no recognizing traffic lights; (iv) slow or high speed. The people with dementia displayed significantly more abnormal driving behaviour than controls ($\chi^2 = 1.83$, $p = 0.017$). They concluded that the presence of dementia is not only a strong indicator of crashes and multiple crashes as described in the previous section, but also a strong indicator of abnormal driving behaviour. As noted in the previous description of the study, use of the spouses and carers of people with dementia as controls is problematic. In addition, studies have shown that the relationship between the carer and the individual with AD can lead the carer to hold different beliefs about their own abilities compared with those who do not have an individual with AD in their life to use as a reference point (Smith, Della Sala, Logie & Maylor, 2000).

Post-May 2003: Relationship between dementia and road safety outcomes

Since May 2003, just two studies were identified which address the risk of crashes associated with dementia. In contrast, there has been a large number of studies (Table 9) which have investigated the association between the driving performance of individuals with dementia. The studies evaluating crash risk are reviewed below and a summary of all studies addressing crashes and other risk outcome measures is provided in Table 9.

Crashes

Gorrie et al. (2007) investigated the presence of Alzheimer's disease characteristics in the autopsies of older individuals who were involved in a fatal motor vehicle accident. Participant autopsy reports were obtained from the Department of Forensic Medicine in Sydney and were conducted from 1997 to 2003. The 27 cases were aged greater than 65 years and were in the driver seat at the time of death. An age-matched control group

comprised 28 drivers who died from other causes, or people whose death related to heart attacks while driving. Participants were not included in the study if they died as a result of homicide or suicide. The post mortem revealed that 11 cases and 11 controls showed signs of cardiovascular disease. None of the drivers showed any signs of vascular dementia. The authors found that a greater number of drivers displayed sparse neuritic plaques than controls (OR: 3.4, 95%CI 1.03 - 11.26), $p = 0.04$, and had a more severe age-related plaque score than controls (OR: 8.5, 95%CI 1.54 - 46.87), $p = 0.01$. Both symptoms are indicative of mild AD pathology. The number of people with moderate AD pathology did not differ between cases and controls (OR: 1.7, 95%CI 0.28 - 10.08), $p = .55$. The authors claimed that their findings relating to the presence of mild AD pathology were higher than expected for the general population. The strengths of the study included the ability to determine the cause of death and neuropathology at the time of death. However, the researchers were unable to determine the extent of the cognitive decline and its impact upon driver behaviour. Furthermore, due to the small sample size, the evidence while supporting the case for an increased risk associated with mild dementia, is relatively weak.

The relationship between car crashes and dementia was explored as part of a large cohort study conducted by Lafont Laumon, Helmer, Dartigues and Fabrigoule (2008). The sample was a subset of the larger Three Cities (3C) and comprised community-dwelling residents aged 65 years and older from Bordeaux. A total of 1051 drivers completed a driving questionnaire (including self-reported crashes) and an assessment by a psychologist and a neurologist. Participants were asked questions relating to driving status and crashes in the past five years, and were assessed for visual ability, hearing problems, cognitive ability as determined by the MMSE, visual working memory as measured by Benton Visual Retention Test and verbal semantic fluency measured by Issacs Set Test (IST), executive and information processing assessed by TMT-A and B performance. In addition, health-related information was collected regarding medical conditions and drugs. The assessments for dementia were conducted at the initial stage of the study and at a two-year follow up. Clinical diagnosis of dementia was made using DSM IV criteria. The follow up diagnosis enabled the researchers to retrospectively determine the drivers who were probable cases of future dementia at baseline. The researchers took into account the participants education level, driving exposure, age, living status and driving status.

Lafont et al. (2008) reported that 16 of the 1051 drivers were diagnosed with dementia at baseline, and this number increased to 17 drivers two years later. A total of 240 out of 986 active drivers (24%) reported being involved in a crash in the past five years. Typically, these people were significantly older ($M = 72.6$, $SD = 5.3$, vs $M = 73.4$, $SD = 4.7$, $p < .05$), received fewer years of education ($M = 22.4$, vs $M = 34.6$, $p < .05$), and had a higher driving frequency compared to those who were not involved in a crash. The researchers also found that participants who displayed signs of dementia were involved in more crashes. Regression modelling was conducted to examine factors associated with crash involvement for a subset of 986 participants who were current drivers throughout the previous 5 years. Participants who had ceased driving during the previous 5 years (6.2%) were included in separate analyses. Results from a multivariate logistic regression analysis indicated that in addition to age, poor education and high driving frequency, poor performance on TMT-B was significantly associated with crash history. A diagnosis of future dementia was associated with self-reported crashes (OR: 3.4, 95%CI 1.0-11.4), $p < .05$ while a diagnosis of Parkinson's disease, head trauma, and stroke were not. The researchers concluded that the detection of executive and

attention deficits is important in determining those individuals who are fit to drive. The limitations of the study include the reliance on self-report data for crashes which arguably should be verified for those with dementia who may have poor insight. The sample was drawn from one single city centre and therefore may not be representative of all older drivers. Only people who were driving during the previous 5 years were included in the main analysis of crash risk. Furthermore, the small number of participants diagnosed with a neurological disease (1.5% with dementia at initial assessment) may limit the statistical power of the analysis and the generalisability of findings.

Summary

From the studies reviewed above, four main general methodological problems emerge:

- Most of the studies attempted to relate previous driving performance to present cognitive status. As dementia is a progressive illness, present level of cognitive functioning should not be used to explain events up to five years previously. It is likely that prospective studies will provide a more satisfactory methodology for studying these issues.
- Many of the studies rely on reports from relatives and friends or caregivers that may provide incomplete or inaccurate data; this is also true of self-ratings, especially retrospectively for people with dementia. Also state authority or insurance company databases may be incomplete, as not all crashes are reported. It may be of great interest to be able to investigate 'near misses' also as they may reflect poor driving skills more accurately. For this reason, on-road evaluations, or to a lesser extent simulator studies, may provide additional insight on driver risk.
- More effort should be put into standardising driving exposure. Certain drivers may limit themselves to short trips only or avoid particular conditions such as wet weather driving or night driving. Although some studies do attempt to standardise for kilometres driven, this may not be sensitive enough to yield accurate results.
- Many studies rely on too narrow a range of cognitive/neuropsychological measures. For example the MMSE is widely used in the literature as a measure of cognitive status. One consideration here is that the MMSE places emphasis on orientation and memory, when clearly driving as a skill involves perceptuomotor abilities, complex decision-making, executive functions, attention, and ability to integrate these capabilities effectively. More comprehensive cognitive/neuropsychological should be included where possible. This is discussed further in the following section.

Notwithstanding the above-mentioned methodological limitations, overall, the evidence reviewed from 15 studies relating to crash risk does indicate that drivers with dementia have a higher risk of deficits in driving skill and crashes compared with normal healthy age-matched controls. This concurs with a recent review by Marshall 2008, who concluded that dementia is associated with a moderately high risk of collision compared to controls. Nevertheless, the evidence is not strong enough (and there is some to the contrary) to suggest that all people with dementia should have their licences revoked or restricted. There is enough evidence though to recommend that once symptoms of

dementia are detected, however mild, close on going monitoring of the individual's driving abilities and cognitive state should be undertaken by family/friends and clinicians. This should assist in making the decisions primarily to restrict the individual's driving exposure and ultimately when driving should cease.

Table 9 Summary of studies of risk associated with dementia

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Anderson et al. (2007)	n = 70 participants with mild dementia n = 132 participants without a neurological disease	- Driving sim. performance - Neuropsychological test battery performance	Overall simulator score correlated with neuropsych. performance score ($r = 0.34, p < 0.001$). Poor performance on verbal memory (Rey AVLT, $p = 0.004$) and visual memory (CFR recall, $p = 0.036$) were associated with crash history. Poor performance on visuomotor abilities (CFT copy, $p = 0.002$, and WAIS-III block design, $p = 0.003$) were associated with simulator crashes.
Akamatsu et al. (2006)	N = 202 drivers aged 60 years and above	<u>Survey:</u> - Physical function - Cognitive function - Driving exposure - Awareness of change in driving skills	Participants who drove more aware of their ability to change in functional ability. Low mileage drivers were more aware of their ability to change in functional ability than high mileage drivers.
Allahyari et al. (2008)	Questionnaire/case study, n=160 male taxi drivers 18 to 65 years ($M = 35, SD = 11.1$)	Driver Error Questionnaire Self reported crash history and citations Cognitive Failures Questionnaire	CFQ total score significantly correlated with DEQ total score ($r = .51, p < .001$), and the CFQ total score predicted driving errors ($F(1, 146) = 48.42, p < .001$). 36% of drivers had been involved in three or more crashes in the past 3 years.
Berndt et al. (2007)	n = 117 participants with cognitive impairment (87 male, 30 female) (48 -88 years)	On-road driving task (pass/fail)	Failing the on-road test was significantly associated with age ($p = 0.0043$), and dementia severity score (MMSE average = 22, $p = 0.0001$).
Berndt et al. (2008)	N = 115 participants with dementia	On-road assessment (pass/fail) MMSE scores were converted to CDR scores	Participants who failed were sig. older ($M = 77.2$ yrs, $SD = 5.6$ yrs, $p = 0.0042$), and had a lower MMSE score ($p = 0.0001$).

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Bhalla et al. (2007)	n = 84 patients with AD n = 44 healthy adults matched on age and education	- On-road driving test - Fear and anxiety ratings pre and post test	Pre-test AD group tension score was correlated with on road score ($r(80) = .24, p < .05$). Post test AD group tension ($r(80) = .2, p < .07$), and fear ($r(80) = .24, p = .05$) was correlated with on road score. Control pre or post test fear and tension scores were not correlated with on-road test score.
Brown et al. (2005a)	n = 31 participants with mild dementia n = 24 healthy older adults	On-road driving test NAB driving scenes	Healthy older adults performed better on the driving test ($M = 4.92, SD = 5.06$), than dementia participants ($M = 14.03, SD = 8.34, p < .01$). Healthy older adults performed better on the NAB driving scenes test ($M = 49.13, SD = 11.87$) than dementia participants ($M = 26.61, SD = 8.58, p < .01$).
Brown et al. (2005b)	n = 17 participants with mild AD n = 33 participants with very mild AD n = 25 healthy older adults	On road driving test Neurologist rated driving ability Participant rated their own driving ability	Healthy adults performed better ($M = 5.2, SD = 5.2$) on the on road test than the very mild AD group ($M = 13.8, SD = 8.4$), and the mild AD group ($M = 13.1, SD = 9.7$), $p < .01$. Neurologist rating of driving ability from a clinical interview was more accurate than the participant and driving informant ratings.
Carr et al. (2005)	n = 142 participants with dementia who had ceased driving n = 58 current drivers with dementia	Cognitive assessment Questionnaire pertaining to reasons for driving cessation	There were no differences in cognitive performance between drivers with AD who had ceased driving and those who were currently driving.

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Clark et al. (2000)	N = 55 participants with mild dementia	Neuropsychology evaluation On-road driving assessment	TMT A was a sig. predictor of failing the driving test (OR = 10.97, 95%CI (5.83-18.22), $p < .01$), as was an MMSE score < 24 (OR = 6.20, 95%CI (2.37-14.38), $p < .05$) and block design performance (OR = 10.86, 95%CI (4.81-22.70), $p < .05$).
Cushman et al. (2008)	n = 35 young healthy drivers n = 26 older healthy controls n = 12 older adults with mild cognitive impairment n = 14 older adults with early AD	Real world navigation test Virtual environment navigation test	Real world navigation performance correlated with virtual environment navigation for all groups irrespective of AD diagnosis or age. Mean scores of navigation ability were sig. different between all groups and decreased in performance from young, older healthy, mild cog, and early AD.
Dawson et al. (2009)	n = 40 participants with mild AD n = 115 controls matched on education level	- On-road test - Neuropsychology evaluation - Visual tests - Motor tests	AD participants $>$ safety errors than C, $p < 0.0001$. Poor performance on-road test for AD group was ass. with poor performance on BVRT ($p = .012$), Complex figure copy ($p < .05$), TMT-A ($p = .0513$) and Functional Reach test ($p < .05$).
De Simone et al. (2006).	n = 15 with frontotemporal dementia (FTD) n = 15 healthy controls	Neuropsychology evaluation Driving simulator task	FTD recorded more collisions and accidents in the sim. than controls. FTD $>$ speed variability (M = 14.2, SD = 2) than C (M = 8, SD = 1), $p < 0.0001$. FTD $>$ speed (M = 31, SD = 9), than controls (M = 25.6, SD = 3).

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Duchek et al. (2003)	n = 21 participants with very mild dementia of the Alzheimer's type (DAT) n = 29 participants with mild DAT n = 58 healthy controls	On-road driving assessment	The mild DAT group displayed the greatest decline in driving performance compared to very mild dementia patients and controls. Age at baseline was sig. related to a "not safe" driving rating over time (HR = 1.06, 95% CI = 1.02-1.09, $p = .002$).
Frittelli et al. (2009)	n = 20 with probable AD n = 20 mild cognitive impairment n = 19 healthy age matched controls	Driving simulator task Simple visual reaction time test	AD group had a shorter time to collision ($M = 0.5$, $SD = 0.9$), than MCI group ($M = 1.7$, $SD = 1.3$) and controls ($M = 2.7$, $SD = 0.8$, $p < 0.0001$). AD group were sig. slower on visual reaction time task ($M = 511$, $SD = 63.2$), than controls ($M = 390$, $SD = 29.5$) and the MCI group ($M = 384$, $SD = 31.8$), $p < 0.001$.
Gorrie et al. (2007)	Autopsies Cases n = 27 (died while driving) Controls n = 28 (died in other circumstances)	AD pathology CVA pathology	Mild AD pathology was present in 52% of cases and 25% of controls Drivers displayed sparse neuritic plaques than controls (OR 3.4, 95%CI 1.03-11.26), $p = 0.04$), and had a more severe age-related plaque score than controls (OR 8.5, 95%CI 1.54-46.87), $p = 0.01$) The number of people with moderate AD pathology did not differ between cases and controls (OR 1.7, 95%CI 0.28-10.08), $p = .55$.
Innes et al. (2007)	n = 35 participants with stroke, 4 with TBI, 4 with AD, and 7 other. n = 12 healthy controls	On-road driving test Computerised sensory-motor and cognitive tests (SMC)	Five of the SMC test measures predicted on-road driving performance with 94% accuracy (planning, complex attention, divided attention, tracking, & ballistic movement).

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Lafont et al. (2008)	N = 1051 participants with dementia	Self report crashes Neuropsychology evaluation Health evaluation	Poor performance on TMT-B was significantly associated with crash history (OR 7.7, 95% CI (2.5-24.0), $p < .001$. A diagnosis of future dementia was associated with self-reported crashes (OR: 3.4, (1.0-11.4), $p < .05$)
Lincoln et al. (2006)	n = 42 with dementia n = 33 healthy older adults	On-road assessment Cognitive test battery	10 out of 27 dementia patients were safe to drive. Safe and unsafe drivers did not differ in any cognitive test performance except for Test of Everyday Attention telephone search subtest ($p = 0.008$).
McKenna et al. (2004)	n = 98 participants with a CVA, n = 18 with head injury, n = 17 with dementia, subcortical CVA, n = 9 with PD/HD, n = 6 with cerebral infections, n = 10 with mixed pathologies and n = 16 as other. n = 200 healthy controls	On-road driving test Cognitive battery; visual perception, executive function and praxis skills.	The cognitive test battery was found have 100% accuracy for predicting drivers who failed the on-road test who were aged greater than 70 years old regardless of pathology.
Ott et al. (2003)	Study 1 n = 27 participants with dementia Study 2 n = 6 healthy adults, n = 21 with probable AD, n = 11 mild cognitive impairment, n = 1 frontotemporal dementia, n = 1 mixed degenerative and vascular dementia.	Driving ability rating by family member Self-reported crash history Neuropsychology evaluation (Porteus maze errors, Controlled oral word association, TMT-B).	Porteus maze drawing time was a sig. predictor of driver ratings by care-givers (chi sq = 9.14, $p = .003$). Performance on 10 computerised maze tasks predicted driver ratings by care-givers.

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Ott et al. (2008)	n = 66 drivers with probable, very mild AD n = 23 drivers with possible AD n = 45 controls without any cognitive impairment	Clinical assessment On-road test Crash history previous 3 years	Driver age ($p = 0.016$) and education level ($p = 0.027$) were sig. predictors of failure on the driving test. Drivers with very mild AD had a greater decline in driving performance over time (median time to failure = 324 days) compared to drivers with possible AD (median time to failure = 605 days). Hazard of failure was 3.5 times higher for the mild AD group (HR = 3.51, 95% CI = 1.09, 11.32).
Ott et al. (2005)	N = 50 participants with very mild to mild dementia	Clinical assessment by six clinicians On-road test Crash history previous 3 years	Clinician ratings were 62% to 78% accurate in predicting on road driving performance. Professionals who specialised in dementia assessments were the most accurate at classifying fitness to drive.
Rizzo et al. (2005)	n = 48 participants with a diagnosis of AD n = 101 healthy controls	Visual tests Cognitive tests Driving simulator task	AD participants were worse at all cog. and vision tests compared to controls. AD participants did not reduce their speed as much as controls ($p = 0.003$), had a greater no. of inappropriate reactions ($p = 0.0146$), and took more time to respond to an emergency vehicle ($p = 0.0091$).
Rosenbaum et al. (2005)	n = 1 taxi driver with probable AD n = 1 taxi driver with encephalitis n = 9 healthy controls (including 1 taxi driver)	Battery of neuropsychology tests Remote memory test for landmark location and spatial navigation Recognition and identification of landmarks	Participants with occipitotemporal damage have a loss of memory for landmarks. Allocentric spatial memories are retained in patients with hippocampal loss in AD, however the ability to form new layouts is compromised.

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Snellgrove (2005)	N = 115 drivers with MCI or early dementia	On-road driving assessment Cognitive assessment	70% drivers failed the on-road driving assessment Performance on the maze task accurately predicted the on-road pass/fail classification of 79% of drivers.
Uc et al. (2004)	n = 32 participants with mild AD n = 136 participants without any cognitive impairment	Cognitive tasks Vision tasks On-road drive in an instrumented vehicle	AD group performed worse than controls on all vision and cognitive tests
Uc et al. (2005)	n = 33 participants with mild AD n = 137 participants without any cognitive impairment	Cognitive tasks Vision tasks On-road drive in an instrumented vehicle	Drivers with AD identified fewer landmarks and traffic signs ($M = 28.9$, $SD = 15.5$, $M = 44.4$, $SD = 22.9$ respectively) than controls ($M = 45.4$, $SD = 15.7$, $M = 72.0$, $SD = 17.1$, $p < 0.0001$). AD safety errors ($M = 1.8$, $SD = 1.7$) > C ($M = 0.5$, $SD = 1.0$, $p = 0.0009$).
Uc et al. (2006)	n = 61 participants with mild AD n = 115 participants without any cognitive impairment	Cognitive tasks Vision tasks Motor tasks Driving simulator task	AD performed sig. worse than C on all motor, cognitive and visual tasks except for 3-D structure of-form motion test ($p = 0.1933$). AD were more likely to slow abruptly (70%) than C (37%), $p < 0.0001$.

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Whelihan et al. (2005)	n = 23 participants with mild dementia n = 23 healthy age matched controls	Neuropsychology evaluation On-road driving test	AD on-road performance was sig. corr w Trails B time ($r = .46, p < .05$), maze navigation time ($r = .52, p < .01$), UFOV I ($r = .61, p < .01$), UFOV II ($r = .46, p < .05$), & UFOV III ($r = .46, p < .05$). Controls on-road performance was sig. corr. w age ($r = .45, p < .05$). Maze navigation performance was sig. corr. with on-road driving performance for both cases and controls.

Approaches to management

Assessing Fitness to Drive.

As shown in Table 10 current recommendations with regard to licensing drivers with dementia vary across different countries. Private licences require periodic reviews and assessment until dementia-related impairment reaches levels of severity that give medical staff reason to recommend ceasing driving. Commercial licences are revoked in Canada, UK, New Zealand and Sweden, and issued conditionally subject to reviews and recommendations of medical staff, in Australia and the US.

Fairly arbitrary criteria for determination of risk, are suggested by all but one of the six jurisdictions and complicates the task of the clinician. Furthermore, most of the guidelines considered here, offer little in the way of specific tools or methods for the practitioner to assist in making judgements about disease severity and driving risk. Generally, this reflects the diversity of evidence from the medical literature. The new Canadian (2006) guidelines do, however, provide a tool (MMSE) and a suggested cut-off score of 24 points, so that drivers with a score less than 24 should not be permitted to continue driving. This raises the question of whether there is sufficient evidence for the effectiveness of this tool and indeed, the validity of this cut-off score, in establishing driver risk. This is a difficult question, not least because the nature of the disease means the participant will vary with time and regular review would be required. The studies reviewed above concerning driver crash risk do not address this issue directly and provide no consensus on this question. A score of 24 indeed may be erring on the overly cautious side. For example, others have used 19 as a cut-off for mild dementia, albeit not related to driving, based on psychiatric recommendations (Smith et al., 2000). A more extreme position put forward by the American Academy of Neurology is that all people with a diagnosis of dementia, even if only mild, should cease driving (Dubinsky et al., 2001).

However, as noted by Hecker (2000) - “Compulsory suspension of a driving licence for individuals with Alzheimer’s disease raises the issue of how to deal with other dementias. Patients with primary degenerative frontal dementia or vascular pathology affecting frontal connection fibres are more likely to be unsafe behind the wheel, particularly at equivalent MMSE performance. In this group, judgement, impulse control and insight is often impaired at an early stage of dementia, despite high scores on screening tests, which do not assess frontal functions. In general, large variations in cognitive deficits occur between types of dementia and between individuals with the same diagnosis. Judgements about driving safety based on global dementia severity scores are unlikely to reflect performance in specific tasks involved. Individual assessment of the relevant functional skills would appear the fairest way to determine capacity” (pages 158-159).

In 1997, a position statement was put forward by 22 prominent researchers in this area (Lundberg et al., 1997). The position statement (determined by the majority, although a consensus was not reached) is worth considering in some detail here (cited in Staplin et al., 1999, p. 169):

- “Cut-off scores must be considered as being relative, forming a small part of the basis of making decisions about driving, and secondary to a clinical evaluation;

- MMSE scores of ≤ 10 , accompanied by a diagnosis of dementia, indicate a sufficiently low level of cognitive functioning to justify recommending immediate cessation of driving;
- MMSE scores of 11-17, accompanied by a diagnosis of dementia, suggest severe cognitive impairment; the patient should be referred for specialised assessment unless the clinician feels that it is unnecessary;
- MMSE scores of 18-23 indicate mild impairment; decisions concerning possible assessment should be based on the functional level of the patient. If the functional level is stable, then a periodic follow-up is recommended;
- For patients without diagnosis of dementia, scores of 17 or less and scores of 18-23 with accompanying signs of neurological deterioration should be indications for specialised assessment” (cited in Staplin et al., 1999, p. 169);

Worthy of note, however, are the reasons for non-acceptance of the use of MMSE and proposed cut-off scores, put forward by some of the researchers:

- “Risk of designating false positives; low scores are related to illiteracy, aphasia, depression, and resistive behaviour; may not correctly assess mental status of patient;
- MMSE does not assess poor judgement and impulse control; persons with scores above the cut-off may be inappropriately viewed as safe drivers;
- Use (of the MMSE) may be wasteful adding nothing more to evaluation of competence than clinical observation of general cognitive functioning” (p. 169).

Hence, while the MMSE is undoubtedly one of the mostly widely used tools for assessment of dementia, its use in decision-making about driving is not without debate. There is also debate as to whether general practitioners can accurately recognise drivers with increased crash risk. Moreover, there is evidence that a clinical examination alone is not sufficient to predict increased crash potential (Johansson, Bronge, Lundberg, Persson, Seidman & Viitanen, 1996). Indeed, as already discussed, the problem of pre-clinical dementia, where cognitive decline may exist prior to diagnosis, further complicates the decision-making process for clinicians. The issues raised here highlight the need for a simple and valid assessment tool for clinicians to identify drivers who may be potentially at risk so that they may be referred for more detailed assessment. In addition, as we have argued elsewhere, there is a need for safe and valid methods for accurately assessing risk following preliminary screening prior to on-road assessments that potentially place both driver and assessor at unnecessary risk (Fildes, Pronk, Langford, Hull, Frith, & Anderson, 2000).

Self Regulation

As can be seen from the research reviewed above, there is much debate about when people with dementia should either give up driving voluntarily or on the advice of others, or if the licensing authorities can/should intervene (Dobbs, 2001). Some people with dementia (particularly in the early stages) may be able to drive safely, yet others will present a significant danger to themselves and other road users (Cable, Reisner, Gerges & Thirumavalavan, 1999). The progression of the disease in many cases may be

so gradual that the participants and their carers are unaware of the implications for driving. Indeed, even if those involved are aware of progressing cognitive deficits, there may be reluctance on behalf of the participant to give up, or of the family to persuade them to do so. Driving plays a large part in the social independence of older people, and cessation may not only be of detriment to convenience (for them and spouses etc.) but also it can be a blow to self-esteem and may lead to feelings marginalisation (Coni, 1996).

Cotrell and Wild (1999) studied participants with AD who had recently given up driving and reported that the decision was made by the driver and or their primary caregiver in the majority of cases. Worthy of concern though is their finding that the delay between the caregivers concluding that driving should stop and actual cessation varied between 0.5 months and 48 months. In many cases the caregivers were unable to identify indicators, which flagged the need for the participants with AD to stop driving. This supports the idea that given the absence of formal guidelines and regulations, decisions about when to give up driving due to dementia are not being taken by those best qualified to make them (see also Zanetti, Geroldi, Frisoni, Bianchetti & Trabucchi, 1999). It is likely that issues of reliance on the participant for transport and avoidance of conflict and upsetting the participant may play a role in these inappropriate judgements.

The role of the general practitioner in providing advice about limiting or stopping driving may be crucial in many cases, but this issue has considerable legal, ethical and social considerations. It should be noted that the legal requirements with regard to reporting diagnoses and or symptoms of dementia to the licensing authorities vary widely from country to country and between states/provinces within countries.

Wild and Cotrell (2003) conducted a study to investigate the relationship between driving ability and awareness of deficits in participants with AD and their carers, and the differences in this awareness contrasted with that of normal elderly drivers using a questionnaire survey and a standardised road test. Their study contrasted 15 participants with AD and 15 controls that were driving a minimum of once per week, the diagnosis of AD was based on the NINCDS-ADRDA guidelines. They found that healthy elderly participants tended to be overly critical of their own driving ability, in contrast with the participants with AD who rated themselves more highly than their performance merited. The caregivers were more accurate in their ratings of the participants with AD's abilities, but tended to miss some potentially dangerous behaviour. Again this supports the argument that the participants and carers may not be the most appropriate groups to make decisions concerning cessation of driving.

In 1999, Adler, Rottunda and Kuskowski studied 75 participants aged 60 years and older, who met the DSM-IV dementia criteria to investigate driving habits and perceptions. They validated these judgements with those of a healthy person ('collateral') who was able to corroborate the participants with AD's responses. Most continued to drive for five days per week, and in widely varying conditions such as night and bad weather. There was on average 60% agreement between the driver and their collateral. Further there was equal agreement that the driver would continue to drive throughout the course of the disease and that the best judge of when to stop driving would be the drivers themselves. Although this was a worrying conclusion, given the potential for danger, there was no reliable measure of driving ability, neither was there a group of age matched controls. Following on from this study Adler, Rottunda, Bauer and Kukowski (2000) reported research into the effects of giving up

driving on participants with AD and their families. Their sample consisted of 54 participants with AD (measured using MMSE), and a group of collaterals for the participants, and 170 controls. The possibility of mild dementia in the collaterals or control group was not addressed as no report of MMSE score is given. It should also be noted that only 84.9% of the collateral group could drive. They found that the participants with AD were significantly more likely to have had a crash than controls, and got lost more frequently. There was no difference in the likelihood of having made plans to give up driving between the two groups, and the participants with AD reported that giving up driving would cause less inconvenience to them and their families than the controls ($p < 0.0005$). Almost 50% of the collaterals reported being concerned about the capabilities of the AD drivers in their care. There are some problems of methodology in this study. Aside from the small number of cases, all of the cases were male whereas the controls were both male and female. Also the controls were recruited from an older driver improvement course, and for this reason may not be representative of the population of older drivers. Also, the variables age, residence and education were statistically controlled where a genuine matching procedure would be preferable. Still, the authors pointed out the need for ongoing monitoring of the participants with AD condition and capabilities by carers and clinicians especially in cases where the participants continue to drive in the face of advice to the contrary.

A further issue that arises following cessation of driving is that of alternative means of accessing necessary destinations. Impairments in memory, visuospatial abilities and attention may preclude those diagnosed with the disorder from travelling on public transport (at least by themselves) for fear of getting lost. This may lead to increased dependence on family members with all the attendant difficulties that may involve, especially if the person in question is an adult child living away from the person with dementia. It is likely that in this situation, social and recreational trips will be curtailed, with essential trips becoming the focus of those involved for example visits to doctors or shopping. There is also the concern that caregivers may find themselves missing work to assist with transportation. This issue would suggest that as procedures for assessing people with dementia with a view to restricting or cancelling licences are developed, strategies for providing suitable and appropriate alternative means of travel must be considered in parallel (see Taylor & Tripodes, 2001).

Table 10 Private licensing guidelines for drivers with dementia

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Dementia	<p><i>Memory impairment or mild dementia:</i> Eligible for licence subject to a satisfactory driving assessment. Annual reassessment is recommended.</p> <p><i>Moderate or severe dementia:</i> Ineligible for any class of licence.</p>	<p>May not hold an unconditional licence if cognitive functioning is significantly impaired.</p> <p>A conditional licence may be issued subject to medical advice, driving assessment & treatment response.</p> <p>Subject to periodic review.</p>	<p><i>Early dementia:</i> Driving may continue if sufficient skills to do so & if the disease progresses slowly. Annual medical review required.</p> <p>Practical road tests may be required.</p> <p><i>Other dementia stages:</i> Persons with cognitive functioning that is more than mildly impaired eg poor short-term memory, judgement or insight are not fit to drive. However, the guidelines acknowledge that there are variable presentations and rates of progression for dementia, so the decision is usually based on medical report.</p>	<p>Frequent review of driving abilities may be required.</p> <p>Special restrictions apply as recommended by medical staff.</p> <p>DLD must be notified.</p> <p><i>Moderate, severe or profound cognitive impairment:</i> No driving.</p>	<p><i>Early dementia:</i> Driving may be permitted if sufficient skills to do so. Formal cognitive testing to be done by medical staff. Regular medical assessment may be required.</p> <p>Desist from driving if impaired cognitive functioning represents a road safety risk.</p>	<p>Licence denied or revoked.</p> <p><i>Mild dementia:</i> A licence may be issued if the person is assessed as having sufficient judgment skills & is able to live an independent life.</p>

3.4.2 *TRAUMATIC BRAIN INJURY*

Definition of Traumatic Brain Injury (TBI)

Traumatic brain injury (TBI) is generally defined as a non-degenerative, non-congenital insult to the brain acquired from an external force, possibly leading to permanent or temporary impairments of cognitive, physical and psychosocial functions with a possible associated diminished or altered state of consciousness. As with dementia, there are many different diagnostic tools and classification criteria (particularly with regard to severity), which can often make interpreting data from clinical studies concerning TBI difficult.

In recent times, improved medical technology has meant that there is a greatly improved survival rate for brain-injured participants. This, along with enhanced rehabilitation techniques coupled with developments in adaptations to motor vehicles to overcome deficits, has led to a situation where the demand to differentiate between safe and unsafe drivers has been increased.

Prevalence of TBI

There are relatively few existing estimates of the prevalence of long-term functional impairments attributable to TBI. The prevalence of TBI differs widely around the world (China, .05%, Spain, .09% and .25% USA)(Fortune & Wen, 1999).

In Australia, a recent report notes that accurate prevalence data are not available (Access Economics, 2009). The same report indicates that for the year 2008 in Australia there were an estimated 1,493 new cases of moderate TBI and 1,000 new cases of severe TBI. In the US approximately 1.4 million people sustain a TBI each year, and of these 235,000 cases require hospitalisation, and 1.1 million individuals are treated and released from an emergency department (Langlois et al. 2006). In addition, TBI is estimated to account for at least one million hospitalisations per year in the European Union (The International Brain Injury Association, IBIA, 2003). Furthermore, the IBIA reports that the highest rate of TBI occurs in individuals aged between 15 and 24 years, with individuals under the age of 5 and over the age of 75 also at a higher risk.

Functional Impairments associated with TBI relevant to driving

People who have experienced a TBI can exhibit deficits in a variety of cognitive and physical domains that are likely to impact on driving including:

- general cognitive function (ability to make judgements, decision-making);
- memory;
- attention;
- executive functions;
- vision and visuo-spatial abilities;
- speech and language;
- emotional control;
- sensation of limb position and movement;
- muscle function, and balance.

Wide individual differences exist in the type and severity of impairments experienced, depending on the location and severity of the brain injury. Worthy of particular note amongst potential deficits following TBI is impairment in higher order executive functioning, which commonly occurs from frontal lobe-damage. This is characterised by difficulties with problem solving, decision making, anticipating consequences of future events and monitoring errors. There is also likely to be associated problems involving insight and awareness of deficits. It is clear that these can present as significant problems in the context of driving (Galski, Bruno & Ehle, 1992; Marshall, Molnar, Man-Son-Hing, Blair, Brosseau, Finestone, Lamothe & Korner-Bitensky, 2007).

The issue of whether or not to start driving again following a head injury is a complex and potentially highly emotive one for the injured, families and health care professionals, as it can often be seen as a landmark in rehabilitation.

Pre-May 2003: Relationship between TBI and road safety outcomes

Several studies were identified in the review period between 1980 and May 2003 which attempted to determine the risk associated with drivers who have sustained a TBI: two pertaining to crashes, one addressed risk of citations and seven reported on performance outcomes. Table 12 provides a summary of findings these studies that have investigated TBI and road safety outcomes including crashes, citations and driving performance.

Crashes

In 2002, Schultheis, Matheis, Nead and DeLuca conducted a study using both subjective (telephone interview) and objective measures (driving records) to evaluate driving behaviours following TBI. Forty-seven participants with TBI and 22 healthy controls were recruited. The authors reported that there were no differences between groups in reported crashes, although the TBI group were more likely to have been involved in unreported incidents. Based on the official driving records there were no significant differences in reported crashes between the two groups. A serious problem with this study is that the participants with TBI were recruited from a group of people who had successfully completed an extensive driver re-evaluation program in the previous 5 years. This suggests that the TBI group may be a specific subset of more motivated drivers who may not reflect the population of people who have experienced TBI; also they do not report severity of injury. This problem biases their overall conclusion that TBI drivers who undergo a comprehensive multi-level evaluation can return to the driving community with few difficulties and in relative safety. This tells us little about the relationship between TBI participants and post injury driving abilities; it does however indicate that extensive driver evaluation is useful.

As outlined in the previous section, Koepsell et al. (1994) conducted a case-control study to determine whether medical conditions increase the risk of injury due to motor vehicle collisions in older drivers. Prevalence of head injury was rare in both groups, although higher amongst cases (0.9%) than controls (0.2%). This yielded extremely wide confidence limits around the estimated relative risk. The authors reported that there was no clear tendency towards elevated risk among older drivers (65 years and older) with head injury (OR: 4.0, 95%CI 0.4 - 44.1) (for a more detailed description of this study method see section 3.13).

Citations

In the study described above by Schultheis et al. (2002), official driving records of the participants with TBI (n = 45) were compared with those of 22 healthy controls. No significant differences were found between the two groups in rates of citations.

Driving Performance

Schanke and Sundet (2002) conducted a study that investigated the relationship between neuropsychological function and on-road driving performance. Their sample comprised 55 participants with varying CT scan verified brain damage (including CVA). The neuropsychological test battery included tests of reaction time, visuo-spatial ability, psychomotor speed and subtests of the WAIS. The on-road test involved an independent instructor, who observed driving behaviour for 1-2 hours in regular traffic. It must be noted that no details on what constituted regular traffic were reported, also the fact that the on-road tests varied in duration may make comparison of results unsatisfactory. The participants with TBI were classified as 'normal', minor impairment, mild impairment and moderate impairment according to their neuropsychological test performance. The authors reported that acceptability to drive from the on-road evaluation decreased with reduced scores on the test battery. However there were exceptions, and the authors argued that these must be judged on a case-by-case basis. Provision of age-matched controls would have improved the study allowing a baseline comparison with normal age related variance in performance. The authors concluded that future work should attempt to cross validate studies of this nature to attempt to reach a consensus on assessment procedures and cut-off points on measures of impairment to provide more stringent guidelines for clinicians and licensing authorities. Importantly, the authors pointed out that it may be critical to reach a suitable level of consistency and sensitivity in a neuropsychological test battery to make decisions about driving based on the tests alone, as many clinicians may not have the availability of on-road testing.

Hawley (2001) reported on an interview study conducted a few months after individuals had sustained TBIs of varying severity, and who had recently returned to driving. At the time of interview, 139 individuals with TBI had returned to driving and 231 had not. The interview involved questions about self-perceived cognitive and behavioural impairments. In general those who had returned to driving reported fewer problems, with less severity than those who had not. The authors also administered, as a more objective measure of driving related problems, the functional independence/functional assessment measure (FIM+FAM, Hall, Hamilton & Gordon, 1993), which rates the participants on items concerning:

- attention - concentration/distractibility;
- orientation;
- emotional status – agitation/responsibility for behaviour/general life functioning;
- safety Judgement – awareness of deficits/planning/risk identification/danger avoidance.

The driving group again scored significantly higher than the non-driving group on all measures of the FIM+FAM. Also overall the driving group had less severe head injuries than the non-drivers. This finding is likely to be biased as the testing and interviews were carried out only a short time after the TBI incidents and intuitively, milder injuries are likely to reach a stage of recovery sooner than more severe injuries. Thus these

participants are more likely to return to driving sooner. The authors concluded that for participants who did not seem fit to drive by various indices, careful monitoring and regular assessment will allow a speedy and safe transition back into driving when possible. A potential problem with the FIM+FAM measure does exist, as it has been shown to have ceiling effects when used with people with TBI, which will reduce its sensitivity to detect change and may miss higher order emotional or cognitive dysfunction (Hall, Mann & High, 1996).

Galski, Ehle and Williams (1996) reported findings from a study of participants with TBI ($n = 63$) and CVA (stroke) ($n = 43$) examining performance on a battery of psychometric tests and in a driving simulator. The cognitive battery used included standard tests of visual scanning, attention, processing speed, perception and planning. During the simulator evaluation, participants were scored for distractibility, inattention, mental slowness, and ability to follow directions. Principal components analysis gave 5 factors, which accounted for 66.14% of the variance in “comprehensive off-road evaluations.” These were:

- higher order visuo-spatial abilities;
- visual recognition and responding;
- anticipatory braking;
- defensive steering;
- behavioural manifestations of complex attention.

It must be noted though that for a number of participants ($n = 106$), the number of variables entered in the analysis may have been too large (> 20) to ensure stringent use of statistics. The factors reported are also very broadly defined yet at least one of them was defined by only one variable. This can be problematic for deriving models based on this type of analysis (Hair, Anderson, Tatham & Black, 1998). Further, the use of a control group to allow comparison of the factors from a control sample would have been helpful, as would an attempt to explain the 34% of variance not accounted for by their factors. The authors’ conclusion that the five factors provided a basis for understanding what is measured in off-road evaluations and for determining a person’s fitness to drive following TBI may not be justifiable.

A study of 39 participants with TBI was conducted by Christie et al. (2001). The study aimed to investigate whether clinicians’ judgement of fitness to drive predicted outcome of a driving assessment, and if neuropsychological tests could discriminate between those deemed fit or unfit. The driving assessment was an on-road standardised assessment carried out by an independent ‘blind’ driving adviser. The clinicians’ judgement was based on medical information about the patients and correlated strongly ($r = 0.8$, $p < 0.015$) with the driving assessment. The neuropsychological battery included the WAIS-R, memory and information processing tests, and attentional and spatial tasks. Visual selective attention was the strongest predictor of driving outcome ($p < 0.008$) although others such as planning and monitoring behaviour ($p < 0.03$) and abstract thinking ($p < 0.04$) tasks also contributed. The authors argued that strategic allocation of attention plays a crucial role in driving and was impaired in the ‘unfit’ group (23%) of the sample. Importantly they reported that few participants regarded themselves as unfit to drive, potentially flagging the role of insight into their deficits as an important avenue for further investigation. However, as the authors pointed out, the

small sample size, and sampling in only one clinic limited the generalisability of the findings to the population of drivers with TBI.

Coleman, Rapport, Ergh, Hanks, Ricker, and Millis (2002) reported on a study aimed at describing predictors of driving outcome following TBI. They included 71 participants who had experienced a TBI and a 'significant other' who knew the participant sufficiently well to obtain ratings of the drivers' ability, and compared these with official driving records and a battery of neuropsychological tests. Those who returned to driving had significantly better scores on the neuropsychological battery than those who did not return to driving. The relationship between caregivers' ratings of driving ability and actual driving incidents was modest. As with other studies in this area, there was no age matched control group to relate the performance of the sample to a normal population. Also the regression analyses (number of predictor variables was large) may not have been appropriate given the number of participants in the driving group ($n = 33$). The authors concluded that there is a need to identify day to day behavioural indices of cognitive functioning which may provide caregivers with more robust information on whether an individual should be driving or not, and if there is a need to refer the individual to medical professionals or licensing authorities. Again this may contribute to the goals of public safety and maintaining independence of TBI participants when appropriate. However, the lack of consensus on measurement of cognitive indicators and ability indices continues to make this a problematic issue (Molnar et al. 2006).

In an effort to address shortcomings of previous studies, Schultheis et al. (2002) (reviewed above) reported on a study using both subjective (telephone interview) and objective measures (driving records) to evaluate driving behaviours following TBI. The authors concluded that significantly more of the participants with TBI restricted their driving (for example avoiding night driving or bad weather driving) compared with controls. Results suggested that drivers with TBI did not have reduced awareness since they demonstrated appropriate use of compensatory strategies. However, it should be noted that the drivers with TBI were a self-selected sample that went along for an extensive driving evaluation. Hence, selection bias may have implications for the generalisability of findings to the wider population of drivers with TBI.

Lundqvist (2001) reported on a case study of 4 participants with brain injuries or lesions in an attempt to demonstrate the complementary value of neuropsychological testing and a driving test. The study aimed to show that a driving test could pick up on compensatory mechanisms that are not evidenced via cognitive and neuropsychological tests. The test battery included tests of reaction time, divided attention, visuo-spatial ability and focussed attention. The driving test was a standardised on-road test evaluated by an independent driving inspector. Table 11 shows a brief summary of the findings for the 4 cases.

All four participants showed impaired reaction time when inhibition of distractors was required, indicating impaired attentional performance under time pressure. The authors claimed that the deficits in driving behaviour are accurately reflected in the test battery performance. Case 4 appeared to use slowing down as a compensatory measure for her attentional deficits, whereas the others drove too fast for their impaired ability suggesting no adaptive strategies.

Table 11 Brief Summary of the findings from Lundqvist (2001)

Case	Injury/Lesion	Neuropsychological Test Performance	Driving Performance	Pass/Fail
1	Sub-dural haemorrhage right hemisphere	Slow/impulsive lack of ability to attend to stimuli accurately	Impaired attention Very slow and careful	Pass*
2	Cerebral infarct-left side of body impaired **	Left Hemi-neglect Very inaccurate on RT tasks†	Too fast/crossed into opposing lane frequently Dangerous/unaware of errors	Fail
3	Infarction and aneurysm	Very slow and inaccurate Impaired divided attention	Inattentive/too fast impulsive Little planning or consistency	Fail
4	Right Ventricular infarction	Very slow. Poor divided attention poor verbal learning and memory. Visuo-spatial dysfunction	Appropriate attention displayed, slow careful and considerate	Pass

* The inspector stated case 1 would have failed if this was a test for a first licence.

† Case 2 was recommended not to drive after the test battery alone but insisted on a driving test.

** See also section 3.3 on stroke.

The authors concluded that their study indicates that it is helpful to look at real driving problems in the context of neurological impairment as measured in tests, to allow for the use of adaptive strategies to compensate for impairments. It does appear particularly in this study that if medical assessment alone is not sufficient to decide on driving suitability, then collaboration between neuropsychology and testing authorities may give a more accurate evaluation, and help to develop a better understanding of specific driving problems. Given the limited selection of participants (n = 4) it is important that the procedure be extended to see if the results hold up for further individuals with these kinds of neurological deficits.

Post-May 2003: Relationship between TBI and road safety outcomes

Two studies addressing crash risk associated with TBI were identified in the review period post-May 2003. The studies evaluating crash risk are reviewed below and a summary of all studies addressing crashes and other risk outcome measures is provided in Table 12.

Crashes

Schanke, Rike, Mølmen and Osten (2008) assessed driving behaviour of CVA and TBI patients' pre and post injury. The researchers recruited 135 patients who had presented at a hospital rehabilitation clinic from 1997-2000. Sixty-five patients had suffered from a brain injury after a CVA, and 28 had experienced a traumatic brain injury. The CVA patient group was significantly older than the TBI patient group and differed according to gender proportions, although both patient groups were similar in terms of the duration of their illness. Upon presentation to the hospital patients were assessed for medical conditions that would impact upon their driving ability, such as seizures, visual conditions and stroke, and the majority also completed an on road driving test. Information relating to pre and post injury was obtained via a questionnaire administered to all the patients in 2006 concerning driving exposure and frequency, driving patterns and self-regulatory practices. The crash rate was determined by the sum of crashes experienced by the group divided by total driving exposure. Family and friends were also invited to respond to questions about the patients driving behaviour.

The researchers found that the CVA group significantly reduced their driving post injury ($M = 162$ km/week, $SD = 125.5$ km/week) compared to before the injury ($M = 289.1$ km/week, $SD = 357.7$ km/week, $p = 0.04$). However, there was no significant change in driving exposure after the injury for the TBI group. A binomial regression was used to investigate contributing factors to crash rates such as gender, driving distance, cause of injury, crash rate pre injury and duration of diagnosis. After adjusting for confounds, there were no significant differences in crash involvement between the groups. The CVA crash rates were found to be comparable to the rates of the general population in Norway, however the TBI crash rates were found to be higher (15.0 vs 6.25 crashes per million km driven). The accident rate of the TBI group post injury was almost two times higher than in the general population. Therefore the authors concluded that TBI patients are at an increased crash risk after injury compared to patients who drive after acquiring a brain injury as a result of a CVA. The limitations of the study include small sample size, self-reporting of crashes and lack of information regarding cause of injury. It is also acknowledged by the authors that information regarding previous crash history and a longer follow up period (i.e., greater than 6 years) would have enhanced the credibility of the study.

Formisano and colleagues (2005) investigated crashes amongst drivers following severe traumatic brain injury. The researchers conducted interviews with 90 carers of patients who were admitted to a rehabilitation hospital in the years 1993 to 1995 who had suffered from a traumatic brain injury with or without coma as measured by the Glasgow Coma Scale duration of less than 48 hours. The average number of years post injury was 4.67 ($SD = 2.35$), and the mean age of the sample was 33 years. The cause of injury varied from TBI (80%), ischemic or haemorrhagic stroke (7%), subarachnoid haemorrhage (6%) and other causes (5%). The researchers found that 29 patients (32%) had resumed driving after the incident, and 11 of these had subsequently been involved in a crash. Those involved in a crash were comparable to the remaining patients by age, gender, and coma duration however the crash involved group had been driving for longer (5.3 years) after the injury compared to the non crash involved patients (3.6 years). A comparison of these results with normalised data for equivalent young male drivers in the general Italian population revealed that the number of expected cases (4.7) was significantly less than the number observed (11), $p = 0.009$ $RR = 2.3$. Limitations of the study include the small sample size, short follow up period, the omission of driving exposure, and driving behaviour information. For these reasons, the findings need to be interpreted with caution. Crash rates were obtained from reports by the carer, which may be underestimated due to a social desirability bias or fear of licence penalties.

Summary

Overall, limited evidence exists on risk of crashes following TBI. Only four studies were identified which provided information on crash rates, including two new studies in the post-May 2003 review period. The evidence was inconsistent across the four studies: two (including one specific to older drivers with TBI) reported no increased risk associated with TBI and two reported increased risk compared with population rates. However, given the serious limitations associated with sample selection bias, no conclusive statements on risk status can be made.

Importantly it must also be pointed out that several of the studies reviewed in this section include CVA/stroke participants in their sample of participants with TBI. While

the cognitive outcomes associated with the most common strokes such as middle cerebral artery stroke are similar to TBI, it is important to bear in mind that physical outcomes and the risk factors for the condition (i.e., driver age, gender, driving experience) can differ. The studies that include stroke and TBI participants also fail to provide separated data for the stroke category. Studies that deal purely with stroke are reviewed in section 3.3.

Table 12 Summary of studies of risk associated with TBI

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Coleman et al. (2002)		Ratings of driver behaviour by significant other, test battery and collection of official records.	Those who returned to driving scored better on tests than those who had not. No relation between ratings and records.
Fisk et al. (1998)		Mail survey of driving habits of TBI participants, examining where advice on driving came from	Primarily families, only 20% did a driving evaluation.
Formisano et al. (2005)	Cases: N = 90 carers of patients with severe TBI all who were drivers pre-injury Controls: Italian driving population	Self-reported crashes after injury	RR for accidents for TBI patients relative to Italian population crash figures was 2.3 $p = 0.009$.
Galski et al. (1996)	N = 63 with TBI N = 43 CVA	Cognitive battery and driving simulator evaluation	5 factors explained 66% of variance in driving performance ($r > .30$)
Hawley (2001)	139 drivers with TBI	Interview about driving, and cognitive performance. FIM+FAM test.	TBI return to driving group scored higher on all measures than non-returned
Koepsell et al (1994)	Case-control		No elevated risk for older drivers with head injury (OR: 4.0, CI 0.4-44.1)
Lundqvist (2001)	4 participant case study	Test battery and on-road evaluation	2:2 pass/fail Tests fail to pick up compensatory strategies evidenced in driving
Schanke & Sundet (2000)	55 (TBI) Participants	Neuropsych. Test battery/on road evaluation	Driving performance and test score correlated – $r = 0.56$, $p < .001$
Schultheis et al. (2002)	Cases n=45 TBI	Official records crashes Telephone interview self-reported driving	No difference in reported crashes between participants and controls
Schanke et al. (2008)	Cases: n = 35 patients with TBI n = 65 brain injury after a CVA Controls: Norwegian driving population	Driving exposure Post injury crashes (carer report)	CVA group driving ($M = 162$ km/week, $SD = 125.5$ km/week) < TBI group post injury. ($M = 289.1$ km/week, $SD = 357.7$ km/week, $p = 0.04$). TBI crash rates were found to be higher than the general population (15.0 vs 6.25 accidents per million km driven). No significant risk after injury for crashes for CVA patient group.

* signif diff from control, $p < 0.05$

Approaches to management

Assessing Fitness to Drive.

As summarised in Table 13 following minor TBI, drivers with private and commercial licences are generally permitted to continue driving (with conditions if required) (see Appendix D for details of commercial licences for TBI). However, Australia and New Zealand require further evaluations if more severe functional impairment is evident. The UK and Sweden do not specifically address minor head injuries. For more serious head injuries, the general consensus across the six jurisdictions is to recommend a period of not driving directly after the incident, with a return to driving based on evaluations of specialists, particularly if any post-traumatic seizures occur.

Christie et al. (2001) (reviewed above) indicated a deal of diverging practice in giving advice to people who have experienced a TBI with regard to resuming driving. Nearly one third of the clinicians that they surveyed (n= 92 clinical psychologists) reported that they were never asked for advice. Three quarters of the clinicians surveyed reported that it is generally the families of patients rather than the individuals with TBI who seek their advice about resuming driving. This highlights another particularly important consideration in determining fitness to drive and the capacity for rehabilitation following TBI (and also other conditions affecting cognitive functions) and that is the role of insight. Many individuals with TBI have limited awareness of their impairments and/or how these might impact on driving performance and therefore see no need to seek advice about continuing to drive.

Interestingly, most of the clinicians surveyed by Christie et al. reported that their units had no policy or guidelines on offering advice and one quarter were unaware of the legal requirements of the DVLA with regard to reporting and assessing abilities both practically and psychometrically. Several authors have argued (e.g. Christie et al., 2001; Fisk et al., 1998) that there should be more research into developing guidelines and procedures must be undertaken at a multidisciplinary level, and that suitable policy and dissemination of these must also be developed.

Training and Rehabilitation

Rehabilitation professionals are frequently required to assess and make recommendations as to whether or not a person who has experienced a TBI is fit to return to driving. A number of methods to assist in this have been developed, taking into account the balance between the individual privilege to drive and the problems that refusal of a licence could present, and the need to maintain public safety. Yet the diversity of opinion and research methods used in this field may lead to reliance upon non-optimal criteria, which may lead to inappropriate decisions, which will have both personal and potentially legal consequences.

Clinicians have failed to reach consensus over what a standardised assessment of driving ability should comprise. Tests used and cut-off levels for adequate function vary widely. However the outcome measures in the various existing reports do appear to be converging (Sundet, Goffing & Hoft, 1995). Incidence of motor vehicle crashes in brain injured people who have not been specifically assessed for driving ability are reported to be higher than in the normal population (Friedland, Koss, Kumar, Gaine, Metzler, Haxby & Moore, 1988) but participants recommended to be allowed to drive following

detailed assessment fall within population crash rates (Haselkorn, Mueller & Rivera, 1998).

The Glasgow Coma Scale is one of the most frequently used scales for describing severity of TBI in the acute phase, and to a limited extent, the subsequent likelihood of recovery. This scale rates injury severity based on the individual's ability to open and close their eyes, movement and speech, the lower the score the greater the severity of the injury (Teasdale & Jennett, 1974). Although this scale is useful for predicting early outcome following a head injury, it was not intended to have predictive ability as to how a person will function in daily life or how independent they will become in the future. The Ranchos Los Amigos Scale of Cognitive Function (Rappaport, Hall, Hopkins, Belleza & Cope, 1982) provides a better predictive instrument. This scale allows progress to be rated from coma to appropriate behaviour and cognitive function and is useful in determining when a participant can begin rehabilitation. Nevertheless this scale also does not detect some changes in cognitive, memory and motor functions indicative of whether a person should re-commence driving or return to work. Further, more detailed assessments by neuropsychologists and other specialists are required in most cases.

Galski, Ehle, McDonald and Mackevich (2000) reviewed many of the considerations and problems in developing criteria for allowing individuals with brain injury to drive. They attempted to address not only the cognitive issues, but also the legal issues. Importantly, they pointed out that research as yet has failed to describe a consistent pattern of neuropsychological, motor, perceptual and cognitive deficits that makes any given person unfit to drive. They explained: "This failure is probably due to the fact that there is no single constellation but, instead, an array of patterns characterised by individual differences in areas of asset and deficit"(p. 899).

In the US, a crucial legal point in licensing is whether or not the presence of one or more deficits is enough to impede driving ability, and how much of a deficit is required before driving should be prevented. This is clearly an area where multidisciplinary teams including medical, neuropsychology and rehabilitation specialists should be involved. Galski and colleagues (2000) concluded that further standardisation of findings from controlled studies of driving ability and a wide range of cognitive and neuropsychological testing is required to arrive at a more effective set of guidelines to assist those charged with making decisions as to the safety or not of a particular driver.

Self-regulation

As outlined above, people who have experienced a TBI can exhibit deficits in a variety of cognitive domains that are likely to impact on driving. Consequently, self-regulatory practices following TBI are particularly important because the cognitive impairments are likely to affect judgements regarding driving. Individuals with cognitive impairment can develop coping strategies including avoiding difficult driving environments such as heavy fog and driving more cautiously (Lundqvist, Alinder & Ronnberg, 2008).

In 1998, Fisk, Schneider and Novack conducted a study to gather information on driving prevalence, exposure time, and details of what advice and evaluations participants receive to help them make the decision to return to driving post TBI. Participants were surveyed via mail and 83 people responded. Approximately 60% of respondents were driving post TBI, and 64% of these were driving 7 days a week, with the majority driving over 50 miles per week, indeed 25% reported driving over 200

miles per week. Primary sources of advice were family members and physicians, but 18% reported no advice or discussion at all. Only 20.5% were recommended to take a driver evaluation. When drivers and non-drivers post TBI were compared, the driving group had significantly higher FIM (measure of impairment in daily living) scores than non-drivers on discharge from hospital following the TBI. These authors concluded (in line with the converging opinion) that a consensual evaluation of driving ability needs to be developed to better inform people with TBI, families and health care professionals about who can be considered safe to drive and who can not. As with some of the other studies discussed in this review, use of self-reporting of abilities is particularly problematic, given the likelihood of memory deficits and lack of insight and awareness of deficit following TBI (Lundqvist & Alinder, 2007). Furthermore, there were no measures of actual driving performance, crashes or other road safety outcomes that could be used to substantiate the self-reported driving performance measures.

Table 13 Private licensing guidelines for drivers with TBI

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Head Injuries	<p>The individual should be evaluated and the degree and severity of disability determines the individual's eligibility to operate any motor vehicle.</p> <p>Cognitive and motor functions to be determined when considering any class of licence.</p>	<p>Desist from driving immediately following the injury.</p> <p>If loss of consciousness does not last more than 24 hours & there are no complications, the person is not viewed as posing a road safety risk.</p> <p>An unconditional licence may not be held if the person sustains chronic functional impairments.</p> <p>A conditional licence may be issued subject to medical & neuropsychological assessments & practical driver assessment, and if there are no other disabilities that may interfere with driving ability.</p> <p>Subject to periodic review.</p>	<p>Not specifically addressed.</p>	<p>Special restrictions apply for cognitive & communication impairment resulting from closed head injury as recommended by medical staff.</p> <p>Regular reviews required.</p> <p>DLD must be notified.</p>	<p>If no loss of consciousness, or other complications, desist from driving for a minimum of 3 hours.</p> <p>If loss of consciousness occurs, desist from driving for 24 hours & obtain medical assessment.</p> <p>Longer stand-down periods may be required if the person displays any of the following:</p> <ol style="list-style-type: none"> 1. Impaired judgment, vision or intellectual capacity. 2. Loss of motor skills. 3. Seizures. <p>Person must obtain GP clearance before driving is resumed.</p>	<p>Not specifically addressed.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Serious Head Injuries	<p>If concussion, post-traumatic amnesia or any residual brain damage results, a full medical evaluation is required prior to resumption of driving. Patients with moderate to severe TBI (Glasgow coma scale <13 or requiring hospital admission) will need comprehensive assessment</p> <p><i>Seizures associated with trauma or intracranial lesions:</i> Once the underlying cause has been resolved, neurological assessment of functional or cognitive sequellae will determine fitness to drive.</p>	<p>An unconditional licence may not be held if the person sustains chronic functional impairments.</p> <p>A conditional licence may be issued subject to medical & neuropsychological assessments & practical driver assessment, and if there are no other disabilities that may interfere with driving ability.</p>	<p>Desist from driving for 6-12 months depending on recovery and clinical features, such as post traumatic amnesia, dural tear, focal signs, seizures).</p> <p>Drivers must desist from driving for 6 months for a significant head injury (contusion not requiring surgery).</p>	<p>Evaluation by a State driver licence examiner required.</p> <p>No driving If there is moderate, severe or profound cognitive impairment.</p>	<p>Desist from driving for a minimum of 6 months.</p> <p>If post-traumatic seizures occur (except those that occur in the first 24 hours after the event), the same guidelines required for tonic clonic epilepsy apply.</p> <p>Driving may resume subject to a full neurological assessment. Depending on the symptoms, neuropsychological, visual & occupational therapist assessments, as well as on-road tests may also be required. Vehicle modifications or other driving aids may be required, as well as periodic medical reviews.</p>	<p>Licence denial or revocation if serious cognitive disturbances result from injury.</p> <p>Medical assessment will take into account disturbances in judgement, memory, vision, psychomotor & emotional functioning.</p>

References

- Access Economics (2009). *The Economic Cost of Spinal Cord Injury and Traumatic Brain Injury in Australia*. Report prepared for The Victorian Neurotrauma Initiative.
- Access Economics (2003). *The Dementia Epidemic: Economic Impact And Positive Solutions For Australia*. Report prepared for Alzheimer's Australia. Canberra.
- Adler, G., Rottunda, S., Bauer, M. & Kuskowski, M. (2000). Driving cessation and AD: Issues confronting participants and family. *American Journal of Alzheimer's Disease*, 15(4), 212-216.
- Adler, G., Rottunda, S. & Dysken, M.W. (1996). The driver with dementia: A review of the literature. *The American Journal of Geriatric Psychiatry*, 4(2), 111-120.
- Adler, G., Rottunda, S. & Kuskowski, M. (1999). Dementia and driving: Perceptions and changing habits. *Clinical Gerontologist*, 20(2), 23-34.
- Akamatsu, M., Hayama, K., Iwasaki, A., Takahashi, J., & Daigo, H. (2006). Cognitive and physical factors in changes to the automobile driving ability of elderly people and their mobility life. *IATSS Research*, 30(1), 38-51.
- Alberta Medical Association (2002). Guideline for Cognitive Impairment: Is This Dementia? Symptoms to Diagnosis. (Alberta Clinical Practice Guidelines Program). Dementia Part 1.
- Alafuzoff, I., Iqbal, K., Frieden, H. & Winblad, B. (1989). Histopathological classification of dementias by multivariate data analysis. In K. Iqbal, H.M. Wisniewski & B. Winblad (eds). *Alzheimer's Disease and Related Disorders*. New York: Alan R. Liss.
- Allahyari, T., Nasi Saraji, G., Ali, J., Hoosseini, M., Iravani, M., Younesian, M., & Kass, S.J. (2008). Cognitive failures, driving errors and driving accidents. *International Journal of Occupational Safety and Ergonomics*, 14(2), 149-158.
- American Psychiatric Association (APA, 2000). *Diagnostic and statistical manual of mental disorders (DSM-IV-TR)*. Washington, DC: APA.
- Anderson, S.W., Rizzo, M., Shi, Q., Uc, E., & Dawson, J. (2005) *Cognitive abilities related to driving performance in a simulator and crashing on the road*. Proceedings of the Third International Driving Symposium on Human Factors in Driver Assessment, Training and Vehicle Design, Rockport, Maine.
- Australian Institute of Health and Welfare [AIHW]. (2006). *Chronic disease and associated risk factors in Australia, 2006* (Cat. no. PHE 81). Canberra: Australia. AIHW.
- Austroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Baddeley, A. (1988). *Working memory*. Oxford, U.K.: OUP.

- Baddeley, A. (1999). *Human memory theory and practice*. (Revised Edition). East Sussex, UK: Psychology Press Ltd.
- Ball, K., Owsley, C., Stalvey, B., Roenker, D. L., Sloane, M. E. & Graves, M. (1998). Driving avoidance and functional impairment in older drivers. *Accident Analysis & Prevention*, 30(3), 313-322.
- Berndt, A., May, E., & Clark, M. (2007). *Drivers with dementia: Environment, errors and performance outcomes*. Proceedings of the fourth International Driving Symposium on Human Factors in Driver Assessment, Training and Vehicle Design, Washington.
- Berndt, A., Clark, M., & May, E. (2008). Dementia severity and on-road assessment: Briefly revisited. *Australasian Journal on Ageing*, 27(3), 157-160.
- Bhalla, R. K., Papandonatos, G. D., Stern, R. A., & Ott, B. R. (2007). Anxiety of Alzheimer's disease patients before and after a standardized on-road driving test. *Alzheimer's and Dementia*, 3(1), 33-39.
- Brain Injury Association of America (2003). What is brain injury? Retrieved 26th June 2003- www.biausa.org/Pages/what_is_brain_injury.html
- British Psychological Society (BPS, 1999). Fitness to Drive and Cognition. A document of the Multi-Disciplinary Working Party on Acquired Neuropsychological Deficits and Fitness to Drive. Leicester: The British Psychological Society.
- Berg, L. (1988). Mild senile dementia of the Alzheimer type: Diagnostic criteria and natural history. *Mount Sinai Journal of Medicine*, 55, 87-96.
- Brown, L. B., Stern, R. A., Cahn-Weiner, D. A., Rogers, B., Messer, M. A., Lannon, M. C., Maxwell, C., Souza, T., White, T., & Ott, B. R. (2005a). Driving Scenes test of the Neuropsychological Assessment Battery (NAB) and on-road driving performance in aging and very mild dementia. *Arch Clin Neuropsychol*, 20(2), 209-215.
- Brown, L., Ott, B., Papandonatos, P., Sui, Y., Ready, R., & Morris, J. C. (2005b). Prediction of on-road driving performance in patients with early Alzheimer's Disease. *Journal of the American Geriatrics Society*, 53(1), 94-98.
- Cable, G., Reisner, M., Gerges, S. & Thirumavalavan, V. (1999). Dementia participants should they be driving? *New Jersey Medicine*, 96(7), 43-46.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Carr, D.B., LaBarge, E., Dunnigan, K. & Storandt, M. (1998). Differentiating drivers with dementia of the Alzheimer type from healthy older persons with a traffic sign-naming test. *The Journals of Gerontology: Biological Sciences and Medical Sciences*, 53A(2), M135-M139.
- Carr, D. B., Shead, V., & Storandt, M. (2005). Driving cessation in older adults with dementia of the Alzheimer's Type. *Gerontologist*, 45(6), 824-827.

- Chaumet, G., Quera-Salva, M. A., Macleod, A., Hartley, S., Taillard, J., Sagaspe, P., Mazaux, J. M., Azouvi, P., Joseph, P. A., Guilleminault, C., Bioulac, B., Léger, D., & Philip, P. (2008). Is there a link between alertness and fatigue in patients with traumatic brain injury? *Neurology*, *71*(20), 1609-1613.
- Christie, N., Savill, T., Buttress, S., Newby, G., & Tyerman, A. (2001). Assessing fitness to drive after head injury: A survey of clinical psychologists. *Neuropsychological Rehabilitation*, *11*, (1), 45-55.
- Christie, N., Savill, T., Grayson, G., Ellison, B., Newby, G., & Tyerman, A. (2001). *The assessment of fitness to drive after brain injury or illness*. TRL Report 485: Crowthorne, TRL Ltd.
- Clark, M., Hecker, J., Cleland, E., Field, C., Berndt, A., Crotty, M., & Snellgrove, C. (2000). *Dementia and driving*. Australian Transport Safety Bureau, Canberra.
- Cohen, D. & Dunner, D. (1980). The assessment of cognitive dysfunction in dementing illness. In J.O. Cole & J. E. Barrett (eds) *Psychopathology in the Aged*. New York: Raven.
- Coleman, R.D., Rapport, L.J., Ergh, T.C., Hanks, R.A., Ricker, J.H. & Millis, S.R. (2002). Predictors of driving outcome after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *83*, 1415-1422.
- Coni, N.K. (1996). Dementia and driving. *The Lancet*, *348* (9041), 1591-1592.
- Cottrell, V. & Wild, K.V. (1999). Longitudinal study of self imposed driving restrictions and deficit awareness in participants with Alzheimer disease. *Alzheimer Disease and Associated Disorders*, *13*, 151-6.
- Cushman, L. A., Stein, K., & Duffy, C. J. (2008). Detecting navigational deficits in cognitive aging and Alzheimer disease using virtual reality. *Neurology*, *71*(12), 888-895.
- Daigneault, G., Joly, P. & Frigon, J-F. (2002). Executive functions in the evaluation of accident risk of older drivers. *Journal of Clinical and Experimental Neuropsychology*, *24*(2), 221-238.
- Dawson, J. D., Anderson, S. W., Uc, E. Y., Dastrup, E., & Rizzo, M. (2009). Predictors of driving safety in early Alzheimer disease. *Neurology*, *72*(6), 521-527.
- Della Sala, S. & Spinnler, H. (1999). Slowly progressive isolated cognitive deficits in degenerative diseases of the central nervous system. In F. Denes and L. Pizzamiglio (Eds.) *Handbook of Neuropsychology*. Hove: Psychology Press, pp. 775-808.
- de Simone, V., Kaplan, L., Patronas, N., Wassermann, E., & Grafman, J. (2007). Driving abilities in frontotemporal dementia patients. *Dementia and geriatric cognitive disorders*, *23*, 1-7.
- Dobbs, B. M. (2001). *Medical conditions and driving: current knowledge*. Technical Report, National Highway Transportation Safety Administration and the Association for the Advancement of Automotive Medicine Project.

- Drachman, D.A. & Swearer, J. (1993). Driving and Alzheimer's disease: the risk of crashes. *Neurology*, 43(12), 2448-2456.
- Drivers Medical Group DVLA Swansea. (2008). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Dubinsky, R.M., Stein, A.C. & Lyons, K. (2000). Practice parameter: Risk of driving and Alzheimer's Disease (an evidence based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*, 54, 2205-2211.
- Dubinsky, R.M., Williamson, A., Gray, C.S. & Glatt, S.L. (1992). Driving in Alzheimer's disease. *Journal of the American Geriatrics Society*, 41(8), 889-891.
- Duchek, J.M., Hunt, L., Ball, K. & Morris, J.C. (1998) Attention and driving performance in Alzheimer's disease. *The Journal of Gerontology; Psychological Sciences and Social Sciences*, 53B(2), 130-141.
- Duchek, J. M., Carr, D., Hunt, L., Roe, C., Xiong, C., Shah, K., & Morris, J. C. (2003). Longitudinal driving performance in early-stage dementia of the Alzheimer Type. *Journal of the American Geriatrics Society*, 51(10), 1342-1347.
- Eby, E.M., Parhad, I.M., Hogan, D.B. & Fung, T.S. (1994). Prevalence and types of dementia in the very old: results from the Canadian Study of Health and Aging. *Neurology*, 44(9), 1593-600.
- Fildes, B., Pronk, N., Langford, J., Hull, M., Frith, W., & Anderson, R. (2000). *Model Licence Re-Assessment Procedure for Older and Disabled Drivers*. Austroads Report No. AP – R176/00.
- Fisk, G.D., Schneider, J.J. & Novack, T. (1998). Driving following traumatic brain injury: prevalence, exposure, advice and evaluations. *Brain Injury*, 12(8), 683-695.
- Fitten, L.J., Perryman, K.M., Wilkinson, C.J., Little, R.J., Burns, M.M., Pachana, N., Mervis, R.J., Malmgren, R., Siembieda, D.W. & Ganzell, S. (1995). Alzheimer and vascular dementias and driving: a prospective road and laboratory study. *Journal of the American Medical Association*, 273(17), 1360-6.
- Folstein, M.F, Folstein, S.E. & McHugh, P.R. (1975). Mini-mental state: A practical method grading the cognitive state of participants for the clinician. *Journal of Psychiatric Research*, 12, 189-198.
- Formisano, R., Bivona, U., Brunelli, S., Giustini, M., Longo, E., & Taggi, F. (2005). A preliminary investigation of road traffic accident rate after severe brain injury. *Brain injury*, 19(3), 159-163.
- Fox, G.K., Bowden, S.C., Bashford, G.M. & Smith, D.S. (1997). Alzheimer's disease and driving: prediction and assessment of driving performance. *Journal of American Geriatric Society*, 45(8), 949-53.

- Friedland, R.P., Koss, E., Kumar, A., Gaine, S., Metzler, D., Haxby, J.V. & Moore, A. (1988). Motor vehicle crashes in dementia of the Alzheimer type. *Annals of Neurology*, 24(6), 782-786.
- Frittelli, C., Borghetti, D., Iudice, G., Bonanni, E., Maestri, M., Tognoni, G., Pasquali, L., & Iudice, A. (2009). Effects of Alzheimer's disease and mild cognitive impairment on driving ability: A controlled clinical study by simulated driving test. *International Journal of Geriatric Psychiatry*, 24(3), 232-238.
- Galski, T., Bruno, R.L. & Ehle, H.T. (1992). Driving after cerebral damage: A model with implications for evaluation. *American Journal of Occupational Therapy*, 46(4), 324-332.
- Galski, T., Ehle, H.T., McDonald, M. & Mackevich, J. (2000). Evaluating fitness to drive after cerebral injury: Basic issues and recommendations for medical and legal communities. *Journal of Head Trauma Rehabilitation*, 15(3), 895-908
- Gauthier, S., Reisberg, B., Zaudig, M., Petersen, R., Ritchie, K., Broich, K. et al. (2006). Mild cognitive impairment. *The Lancet*, 367(9518), 1262-1270.
- Galski, T., Ehle, H.T. & Williams, J.B. (1996). Off-road driving evaluations for persons with cerebral injury: A factor analytic study of predriver and simulator testing. *American Journal of Occupational Therapy*, 51(5), 352-359.
- Gilleard, C.J. (1984). Ageing, dementia and Gibson Spiral Maze performance: a brief note. *Perception and Motor Skills*, 58, 889-90.
- Gorrie, C. A., Rodriguez, M., Sachdev, P., Duflou, J., & Waite, P. M. E. (2007). Mild neuritic changes are increased in the brains of fatally injured older motor vehicle drivers. *Accident Analysis & Prevention*, 39(6), 1114-1120.
- Grady, C.L., Haxby, J.V., Horwitz, B., Sundaram, G., Berg, M., Schapiro, M., Friedland, R.P. & Rapoport, S.I. (1988). A longitudinal study of the early neuropsychological and cerebral metabolic changes in dementia of the Alzheimer's type. *Journal of Clinical and Experimental Neuropsychology*, 10, 576-596.
- Hair, J.F., Anderson, R.E., Tatham, R.L. & Black, W.C. (1998). *Multivariate Data Analysis* (5th Edition). Prentice Hall: New Jersey.
- Hall, K.M., Hamilton, B.B. & Gordon, W.A. (1993). Characteristics and comparisons of functional assessment indices: disability rating scale, functional independence measure, and functional assessment measure. *Journal of Head Trauma Rehabilitation*, 8, 60-74.
- Hall, K.M., Mann, N. & High, W.M. (1996). Functional measures after traumatic brain injury: ceiling effects of FIM+FAM, DRS and CIQ. *Journal of Head Trauma Rehabilitation*, 11, 27-39.
- Haselkorn, J.K., Mueller, B.A. & Rivara, F.A. (1998). Characteristics of drivers and driving record after traumatic and non-traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 79(7), 738-42.

- Hawley, C.A. (2001). Return to driving after head injury. *Journal of Neurological and Neurosurgical Psychiatry*, 70, 761-766.
- Hecker, J. (2000). Driving into danger? Compulsory driving licence cancellation in Alzheimer's disease. *Australian Journal of Ageing*, 19(4), 158-159.
- Hodges, J.R., Salmon, D.P. & Butters, N. (1992). Semantic memory impairment in Alzheimer's disease: Failure of access or degraded knowledge. *Neuropsychologia*, 30, 301-314.
- Hunt, L., Morris, J.C., Edwards, D., & Wilson, B.S. (1993). Driving in persons with mild senile dementia of the Alzheimer's type. *Journal of the American Geriatrics Society*, 41, 747-753.
- Innes, C. R. H., Jones, R. D., Dalrymple-Alford, J. C., Hayes, S., Hollobon, S., Severinsen, J., Smith, G., Nicholls, A., & Anderson, T. J. (2007). Sensory-motor and cognitive tests predict driving ability of persons with brain disorders. *Journal of the Neurological Sciences*, 260(1-2), 188-198.
- Johansson, K., Bogdanovic, N., Kalimo, H., Winblad, B. & Viitanen, M. (1997). Alzheimer's disease and apolipoprotein E ϵ 4 allele in older drivers who died in automobile accidents. *The Lancet*, 349, 1143-44.
- Johansson, K., Bronge, L., Lundberg, C., Person, A., Seidman, M. & Viitanen, M. (1996). Can a physician recognise an older driver with increased crash risk potential? *Journal of the American Geriatrics Society*, 44, 1198-1203.
- Koepsell, T., Wolf, M. & McCloskey, L. (1994). Medical conditions and motor vehicle collision injuries in older adults. *Journal of the American Geriatrics Society*, 42, 695-700.
- Lafont, S., Laumon, B., Helmer, C., Dartigues, J.-F., & Fabrigoule, C. (2008). Driving Cessation and Self-Reported Car Crashes in Older Drivers: The Impact of Cognitive Impairment and Dementia in a Population-Based Study. *J Geriatr Psychiatry Neurol*, 21(3), 171-182.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Langlois, J.A., Rutland-Brown, W., & Thomas, K.E. (2006). *Traumatic brain injury in the United States: Emergency department visits, hospitalisations, and deaths*. Atlanta: Centre for Disease Control and Prevention.
- Lew, H. L., Poole, J. H., Lee, E. H., Jaffe, D. L., Huang, H.-C., & Brodd, E. (2005). Predictive validity of driving-simulator assessments following traumatic brain injury: a preliminary study. *Brain injury*, 19(3), 177 - 188.
- Lezak, M.D. (1995). *Neuropsychological assessment* (3rd ed.). Oxford: Oxford University Press. Lloyd, S., Cormack, C.N., Blais, K., Messeri, G., McCallum, M., Spicer, K., & Morgan, S. (2001). Driving and dementia: A review of the literature. *Canadian Journal of Occupational Therapy*, 68(3), 149-56.

- Lincoln, N., Radford, K., Lee, E., & Reay, A. (2006). The assessment of fitness to drive in people with dementia. *International Journal of Geriatric Psychiatry*, *21*(11), 1044-1051.
- Logie, R. H., Maylor, E. A., Della Sala, S. & Smith, G. (2004). Working memory in event and time based prospective memory tasks: Effects of secondary demand and age. *European Journal of Cognitive Psychology*, *16*(3), 441-456.
- Lucas-Blaustein, M.J., Filipp, C.L., Dungan, C. & Tune, L. (1988). Driving in participants with dementia. *Journal of the American Geriatrics Society*, *36*, 1087-91.
- Lundberg, C., Johansson, K., Ball, K., Bjerre, B., Blomqvist, C., Braehkhus, A., Brouer, W.H., Bylsma, F.W., Carr, D.B., Englund, L., Friedland, R.P., Hakemies-Blomqvist, L., Klemetz, G., O'Neill, D., Odenheimer, G., Rizzo, M., Schelin, M., Seideman, M., Tallman, K., Viitanen, M., Waller, P. & Winblad, B. (1997). Dementia and driving: An attempt at consensus. *Alzheimer Disease and Associated Disorders*, *11*(1), 28-37.
- Lundqvist, A., & Alinder, J. (2007). Driving after brain injury: Self-awareness and coping at the tactical level of control. *Brain injury*, *21*(11), 1109 – 1117
- Lundqvist, A., Alinder, J., & Rönnerberg, J. (2008). Factors influencing driving 10 years after brain injury. *Brain injury*, *22*(4), 295-304.
- Lundqvist, A. (2001). Neuropsychological aspects of driving characteristics. *Brain Injury*, *15*(11), 981-994.
- Marshall SC, Molnar FM, Man-Son-Hing M, Blair R, Brosseau L, Finestone HM, Lamothe C, Korner-Bitensky N. (2007) Predictors of Driving Ability Following Stroke: A Systematic Review, *Topics in Stroke Rehabilitation*, *14*, 98–114
- Marshall, Shawn C. (2008). The Role of Reduced Fitness to Drive Due to Medical Impairments in Explaining Crashes Involving Older Drivers. *Traffic Injury Prevention*, *9* (4), 291-298.
- Marottoli, R.A., Cooney, L.M., Wagner, D.R., Doucette, J., & Tinetti, M.E. (1994). Predictors of automobile crashes and moving violations among elderly drivers. *Annals of Internal Medicine*, *121*, 842-846.
- McDowd, J.M. & Craik, F.I.M. (1988). Effects of aging and task difficulty on divided attention performance. *Journal of Experimental Psychology: Human Perception and Performance* *14*, 267-280.
- McKenna, P. L., Jefferies, Adrian, D., & Neil, F. (2004). The use of a cognitive battery to predict who will fail an on-road driving test. *British Journal of Clinical Psychology*, *43*, 325-336.
- Molnar, F., Patel, A., Marshall, S., Man-Son-Hing, M., & Wilson, K. (2006). Clinical utility of office-based cognitive predictors of fitness to drive in persons with dementia: A systematic review. *Journal of the American Geriatrics Society*, *54*, 1809-1824.

- Morris, R. (1996) *The cognitive neuropsychology of Alzheimer's disease*. Oxford: Oxford University Press.
- Moss, M.B., & Albert, M.S. (1988). Alzheimer's disease and other dementing disorders. In M.S. Albert & M.B. Moss (eds) *Geriatric Neuropsychology*. New York: Guildford.
- Novack, T. A., Baos, J. H., Alderson, A. L., Schneider, J. J., Weed, W., Blankenship, J., & Salisbury, D. (2006). UFOV performance and driving ability following traumatic brain injury. *Brain injury*, 20(5), 455 - 461.
- Ott, B. R., Heindel, W. C., Papandonatos, G. D., Festa, E. K., Davis, J. D., Daiello, L. A., & Morris, J. C. (2008). A longitudinal study of drivers with Alzheimer disease. *Neurology*, 70(14), 1171-1178.
- Ott, B.R., Heindel, W., Whelihan, W., Caron, M., Piatt, A., & DiCarlo, M. (2003) Maze test performance and reported driving ability in early dementia. *Journal of Geriatric Psychiatry and Neurology*, 16(3), 151-155.
- Ott, D., Papandonatos, G. D., D'Abreu, A., Burock, J., Curtin, A., Wu, C., & Morris, J. C. (2005). Clinician Assessment of the Driving Competence of Patients with Dementia. *Journal of the American Geriatrics Society*, 53(5), 829-833.
- Owsley, C. Ball, K., Sloane, M. E., Roenker, D. L. & Bruni, J. R. (1991). Visual/cognitive correlates of vehicle accidents in older drivers. *Psychology & Aging*, 6(3), 403-415.
- Owsley, C., McGwin, G. Jr. & Ball, K. (1998). Vision impairment, eye disease, and injurious motor vehicle crashes in the elderly. *Ophthalmic Epidemiology*, 5(2), 101-113.
- Parasuraman, R. & Nestor, P.G. (1991). Attention and driving skills in aging and Alzheimer disease. *Human Factors*, 33(5), 539-557.
- Pietrapiana, P., Tamietto, M., Torrini, G., Mezzanato, T., Rago, R., & Perino, C. (2005). Role of premorbid factors in predicting safe return to driving after severe TBI. *Brain injury*, 19(3), 197 - 211.
- Rabbitt, P.M.A. (1990). Applied cognitive gerontology: Some problems, methodologies and data. *Applied Cognitive Psychology*, 4, 225-246.
- Rabbitt, P.M.A. (1993). Does it all go together when it goes? *Quarterly Journal of Experimental Psychology*, 46A, 385-434.
- Radford, K. A., Lincoln, N. B., & Murray-Leslie, C. (2004). Validation of the stroke drivers screening assessment for people with traumatic brain injury. *Brain injury*, 18(8), 775-786.
- Rappaport, M., Hall, K.M., Hopkins, K., Belleza, T. & Cope, D.N. (1982). Disability rating scale for severe head trauma: Coma to community. *Archives Physical Medicine and Rehabilitation*, 63(3), 118-23.

- Rapport, L. J., Bryer, R. C., & Hanks, R. A. (2008). Driving and community integration after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(5), 922-930.
- Rebok, G.W., Bylsma, F.W. & Keyl, P.M. (1990). *The effects of Alzheimer's disease on elderly drivers*. Paper presented at the Gerontological Society of America, Boston, MA. November 18.
- Rizzo, M., McGehee, D.V. & Dawson, J.D. (1997). Simulated car crashes and crash predictors in drivers with Alzheimer disease. *Archives of Neurology*, 54(5) 545-51.
- Rizzo, M., McGehee, D.V., Dawson, J.D. & Anderson, S.N. (2001). Simulated car crashes at intersections in drivers with Alzheimer disease. *Alzheimer Disease and Associated Disorders*, 15(1), 10-20.
- Rizzo, M., Shi, Q., Dawson, J., Anderson, S. W., Kellison, I., & Pietras, T. (2005). Stops for cops, impaired response implementation for older drivers with cognitive decline. *Transportation Research Record: Journal of the Transportation Research Board*, 1922, 1-8.
- Rosenbaum, R. S., Gao, F., Richards, B., Black, S. E., & Moscovitch, M. (2005). "Where to?" Remote memory for spatial relations and landmark identity in former taxi drivers with Alzheimer's Disease and Encephalitis. *Journal of Cognitive Neuroscience*, 17(3), 446-462.
- Salthouse, T.A. (1985). *A Theory of Cognitive Aging*. Amsterdam: North Holland.
- Salthouse, T.A. (2000). Aging and measures of processing speed. *Biological Psychology*, 54(1-3), 35-54.
- Salthouse, T.A. (2003). What needs to be explained to account for age-related effects on multiple cognitive variables? *Psychology and Aging*, 18 (1), 91-110.
- Salzberg, P. & Moffat, J. (1998). *The Washington State Department of Licensing Special Exam Program: An Evaluation*. Washington Traffic Safety Commission. Olympia, WA.
- Schanke, A.-K., Rike, P.-O., Mølmen, A., & Osten, P. E. (2008). Driving behaviour after brain injury: a follow-up of accident rate and driving patterns 6-9 years post-injury. *Journal of rehabilitation medicine. Supplement*, 40(9), 733-736.
- Schanke, A. & Sundet, K. (2000). Comprehensive driving assessment: Neuropsychological testing and on-road evaluation of brain injured participants. *Scandinavian Journal of Psychology*, 41, 113-121.
- Schneider, J. J., & Gouvier, W. D. (2005). Utility of the UFOV Test with mild traumatic brain injury. *Applied Neuropsychology*, 12(3), 138 - 142.
- Schneider, J.A., Wilson, R.S., Cochran, E.J., Bienias, J.L., Arnold, S.E., Evans, D.A. & Bennett, D.A. (2003). Relation of cerebral infarctions to dementia and cognitive function in older persons. *Neurology*, 60(7), 1082-8.

- Scultheis, M.T., Matheis, R.J., Nead, R., & DeLuca, J. (2002). Driving behaviors following brain injury: self-report and motor vehicle records. *Journal of Head Trauma and Rehabilitation, 17*, (1): 38-47.
- Smith, G., Della Sala, S., Logie, R.H. & Maylor, E.A. (2000). Prospective and retrospective memory in normal aging and dementia: A questionnaire study. *Memory, 8*, 311-321
- Snellgrove, C, A. (2005). *Cognitive screening for the safe driving competence of older people with mild cognitive impairment or early dementia*. Australian Transportation Bureau, Canberra, ACT.
- Spaeth, D. M., Mahajan, H., Karmarkar, A., Collins, D., Cooper, R. A., & Boninger, M. L. (2008). Development of a wheelchair virtual driving environment: Trials with subjects with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 89*(5), 996-1003.
- Spinnler, H., Della Sala, S., Bandera, R. & Baddeley, A.D. (1988). Dementia, ageing and the structure of human memory. *Cognitive Neuropsychology, 5*, 193-211.
- Squire, L.R. (1992) Memory and the hippocampus: A synthesis from findings with rats, monkeys and humans. *Psychological Review, 99*, 195-231.
- Staplin, L., Loccoco, K.H., Stewart, J., & Decina, L.E. (1999). *Safe Mobility for Older People Notebook*. National Highway Traffic Safety Administration Model Driver Screening and Evaluation program. DOT HS 808 853.
- Stutts, J. C. (1998). Do older drivers with visual and cognitive impairments drive less? *Journal of the American Geriatrics Society, 46*, 854-861.
- Stutts, J.C., Stewart, J.R. & Martell, C. (1998). Cognitive test performance and crash risk in an older driver population. *Accident Analysis and Prevention, 30*(3), 337-346.
- Sundet, K., Goffeng, L. & Hoft, E. (1995). To drive or not to drive: Neuropsychological assessment for driver's license among stroke participants. *Scandinavian Journal of Psychology, 36*, 47-58.
- Swedish National Road Administration. (1998). *Swedish National Road Administration Statute Book*. Sweden.
- Taylor, B.D. & Tripodes, S. (2001). The effects of driving cessation on the elderly with dementia and their caregivers. *Accident Analysis and Prevention, 33*, 519-528.
- Teasdale, G. & Jennett, B. (1974). Assessment of coma and impaired consciousness: a practical scale, *Lancet 2*, 81-84.
- The International Brain Injury Association (IBIA) (2003). Brain Injury Facts. Retrieved 26th June 2003. <http://www.internationalbrain.org/media/mediaFacts.html>
- Trobe, J.D., Waller, P.F., Cook-Flannagan, C.A., Teshima, S.M. & Bieliauskas, L.A. (1996). Crashes and citations among drivers with Alzheimer disease. *Archives of Neurology, 53*, (5), 411-416.

- Tuokko, H., Tallman, K., Beattie, L.B., Cooper, P. & Weir, J. (1995). An examination of driving records in a dementia clinic. *The Journals of Gerontology: Psychological Sciences and Social Sciences, B, 50*(3), S173-S186.
- Uc, E. Y., Rizzo, M., Anderson, S. W., Shi, Q., & Dawson, J. D. (2004). Driver route-following and safety errors in early Alzheimer disease. *Neurology, 63*(5), 832-837.
- Uc, E. Y., Rizzo, M., Anderson, S. W., Shi, Q., & Dawson, J. D. (2005). Driver landmark and traffic sign identification in early Alzheimer's disease. *J Neurol Neurosurg Psychiatry, 76*(6), 764-768.
- Uc, E. Y., Rizzo, M., Anderson, S. W., Shi, Q., & Dawson, J. D. (2006). Unsafe rear-end collision avoidance in Alzheimer's disease. *Journal of the Neurological Sciences, 251*(1-2), 35-43.
- Utah State Driver License Medical Advisory Board. (2006). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Viitanen, M., Johansson, K., Bogdanovic, N., Berkowicz, A., Druid, H., Eriksson, A., Krantz, P., Laaksonen, H., Sandler, H., Saukko, P., Thiblin, I., Winblad, B., & Kalimo, H. (1998). *Forensic Science International, 96*, 115-127.
- Waller, P.F., Trobe, J.D. & Olson, P.L. (1993). Crash Characteristics Associated With Early Alzheimer's Disease. *37th Annual Proceedings of The Association For The Advancement of Automotive Medicine*, pp93-307, November 4-6, San Antonio, Texas.
- Whelihan, W. M., DiCarlo, M. A., & Paul, R. H. (2005). The relationship of neuropsychological functioning to driving competence in older persons with early cognitive decline. *Archives of Clinical Neuropsychology, 20*(2), 217-228.
- Wild, K. & Cotrell, V. (2003). Identifying driving impairment in Alzheimer disease: A comparison of self and observer reports versus driving evaluation. *Alzheimer Disease and Associated Disorders, 17*(1), 27-34.
- Withaar, F.K., Brouwer, W.H. & van Zomeren, A.H. (2000). Fitness to drive in older drivers with cognitive impairment. *Journal of the International Neuropsychological Society, 6*(4), 480-90.
- World Health Organisation (WHO, 2001). *Mental health: new understanding – new hope*. Retrieved on June 26, 2003 from http://www.euro.who.int/mediacentre/PressBackgrounders/2001/20011128_1
- World Health Organisation (WHO, 2004). Disease incidence, prevalence and disability. Retrieved on 22nd June 2009 from http://www.who.int/healthinfo/global_burden_disease/GBD_report_2004update_part3.pdf
- Zanetti, O., Geroldi, C., Frisoni, G.B., Bianchetti, A. & Trabucchi, M. (1999). Contrasting results between caregiver's report and direct assessment of activities

of daily living in participants affected by mild and very mild dementia: the contribution of the caregiver's personal characteristics. *Journal of the American Geriatrics Society*, 47(2), 196-202.

Zuin, D., Ortiz, H., Boromei, D. & Lopez, O.L. (2002). Motor vehicle crashes and abnormal driving behaviours in participants with dementia in Mendoza, Argentina. *European Journal of Neurology*, 9, 29-34.

3.5 DIABETES MELLITUS

Definition of diabetes mellitus

Diabetes mellitus is a chronic illness characterised by high blood glucose (BG) levels and is caused by an inherited and/or an acquired deficiency in production of insulin by the pancreas or ineffective use of insulin, or both. The World Health Organisation (WHO) specifies the following diagnostic criteria for diabetes mellitus: (i) for oral glucose tolerance test: fasting venous plasma glucose concentration of 7.0 mmol/l or greater; or (ii) 2-hour post oral glucose: venous plasma glucose of 11.1 mmol/l or greater (revised 1999; see ICD_10-AM, p.75). There are two main types of diabetes: Type 1, (formerly referred to as insulin dependent diabetes mellitus (IDDM) and Type 2 (formerly called non-insulin dependent diabetes mellitus (NIDDM).

Type 1

Type 1 diabetes is a condition in which pancreatic beta cells are destroyed, resulting in a failure of the pancreas to produce insulin. Risk factors include autoimmune, genetic and environmental factors. This form of diabetes usually develops during childhood and adolescence, but adult onset may occur (American Diabetes Association, 2003). Type 1 diabetes is treated by insulin therapy, delivered by continuous subcutaneous infusion (pump) or intermittent subcutaneous injection.

Type 2 diabetes

Type 2 diabetes arises when the pancreas is unable to produce sufficient insulin and there is inefficient use of insulin. In the early stages, the condition is commonly characterised by insulin resistance in which body cells are unable to use insulin effectively. Loss of ability to produce insulin generally follows this. This type of diabetes is associated with older age although is increasingly being diagnosed in children and adolescents (Diabetes Australia, 2002; American Diabetes Association, 2003). Other risk factors include genetic predisposition, and obesity and other lifestyle factors. Type 2 diabetes represents around 90% of all cases (WHO, 2002). Type 2 diabetes may be controlled by diet and exercise and/or oral medications.

Medical complications

Acute metabolic disturbances associated with diabetes are hyperglycaemia and hypoglycaemia. In addition, increased concentration of glucose in the blood associated with diabetes has a detrimental effect on body systems including the vascular, ocular and nervous systems. Acute manifestations and common medical complications of diabetes are described below.

Hypoglycaemia: refers to *low* blood glucose concentrations. A hypoglycaemic reaction may result when there is “an imbalance between carbohydrate intake, administered exogenous or augmented endogenous (drug therapy) insulin and exercise” (MacLeod, 1999, p. 284). All people with Type 1 diabetes will suffer hypoglycaemia at some time in the course of their illness. The manifestations of the reaction vary widely between individuals and within individuals across time and can impact on visual functions, cognitive functions and general orientation as described below.

Hyperglycaemia: refers to *high* blood glucose concentration, which most commonly is associated with uncontrolled diabetes. Severe hyperglycaemia may lead to biochemical imbalances that can cause acute life-threatening events such as ketoacidosis or hyperosmolar (nonketotic) coma, usually only in Type 1 diabetics (American Diabetes Association, 2003). McGwin and colleagues (1999) also note that hyperglycaemia may result in visual impairment, disorientation and decreased mental processing capacity, which may in turn affect driving performance.

Diabetic retinopathy (DR) refers to eye disease resulting from damage to small blood vessels in the retina. DR is a leading cause of blindness and vision impairment. Abnormalities of the blood vessels caused by diabetes include weakening of blood vessel walls and leakage from blood vessels. DR is strongly associated with time since onset of diabetes and level of blood glucose control. It is common amongst those with Type 1 diabetes and it is estimated that after about 20 years post-onset, almost all those with Type 1 diabetes will have some degree of DR. It is also estimated that about 21% of those with Type 2 diabetes have retinopathy at diagnosis of their condition and most develop DR eventually (American Diabetes Association, 2003). Studies have found that “after 15 years of diabetes, approximately 2% of people become blind, while about 10% develop severe visual handicap”. Other visual conditions such as “glaucoma and cataract may be more common in people with diabetes than in those without the disease” (WHO, 2002, p.3). For a more detailed description of the vision conditions and impairment associated with diabetes, see section 3.13.

Cardiovascular disease, stroke and high blood pressure: Diabetes is frequently associated with high blood pressure and high blood cholesterol and triglycerides, which increase the risk of heart disease and stroke (Diabetes Australia, 2002). Recent studies in Australia have shown that people with diabetes are two to five times more likely to have heart disease or stroke (American Diabetes Association, 2003; Diabetes Australia, 2002) than those without diabetes. In addition, 73% of adults with diabetes have high blood pressure (BP \geq 130/80) or are treated for hypertension and (American Diabetes Association, 2003).

Nephropathy: Nephropathy or kidney disease is associated with both types of diabetes. Nephropathy affects 10-21% of people with diabetes (American Diabetes Association, 2003). Good glucose and blood pressure control is important in prevention of nephropathy. The condition is progressive and takes several years to develop. Damage to blood vessels in the kidney associated with nephropathy results in impaired filtration of wastes, chemicals and excess water from the blood. Eventually the entire filtration system may break down, leading to end-stage renal disease (ESRD) or kidney failure, requiring kidney transplant or dialysis for survival. The risk of ESRD is 12 times higher in those with Type 1 diabetes compared with Type 2 diabetes. However, this higher rate in type 1 diabetes may represent a survival bias as most people with Type 2 diabetes die from cardiovascular disease before developing ESRD.

Neuropathy: Neuropathy or peripheral nerve disease is the most common complication of diabetes, affecting up to 50% of people with both types of diabetes. The condition may result in sensory loss and damage to the limbs (WHO, 2002). ‘Diabetic Foot’ is an example of the complications of peripheral neuropathy, characterised by chronic or recurring diabetic foot ulcers (Mathers et al., 2002). Peripheral vascular disease and

peripheral neuropathy can lead to ulceration, weakness and amputation, which may impair the ability of some people with diabetes to drive safely (MacLeod, 1999).

Prevalence of diabetes mellitus

The Australian Bureau of Statistics (ABS) estimated that the prevalence of diabetes in Australia in 2005 was $699600/19681500 = 3.5\%$ (ABS, 2006). There is an age related increase in the prevalence of diabetes with rates as high as 13.7% amongst those over 65 years (see Table 14).

Table 14 Number of people ('000) suffering from Diabetes Mellitus in Australia 2004-2005 (ABS National Health Survey)

	Age Group				
	0-14	15-17	18-64	65+	Total
Diabetes Mellitus	0.1	0.4	2.9	13.7	699.6
Total no. of people ('000)	3920.6	797.9	12523.0	2440.1	19681.5

According to the AusDiab longitudinal study, the prevalence in Australia from a sample of 11,247 participants was 8.0% for males and 6.8% for females (Dunstan et al. 2002). Approximately 3.8% of participants were newly diagnosed at the time of the study.

Prevalence estimates vary for different countries. The WHO estimates that the prevalence of diabetes in 2004 was just over 220 million worldwide (WHO, 2008). In 2003, the prevalence of the disease in Western European countries (EURO A group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated at 17.8 million or around 4.3% of this population. Recent estimates for the USA and Canada suggest that approximately 6.3% of the population have diabetes and about one third of these are unaware that they have the disease (WHO, 2002; American Diabetes Association, 2003).

The majority of people with Type 2 diabetes in developed countries are aged 65 years or older. For example, in the US, the prevalence of diabetes in those 20 years and older is estimated to be around 10.7% but estimates are much higher (20.1%) among people age 60 years and older (23.1%) (American Diabetes Association, 2009). Recent estimates suggest that, due to the ageing population, the number of people with diabetes worldwide may double by the year 2025 (WHO, 2002).

Functional impairments associated with diabetes mellitus relevant to driving

A number of impairments have been noted amongst people with diabetes (see Frier, 1992; Lindgren, Eckert, Sterberg & Agardh, 1996; MacLeod, 1999; Piotrowski, 1997). The chronic effects of recurrent hypoglycaemic events remains controversial. Warren and Frier (2004) note that structural and functional changes in the brain are associated with severe, recurring hypoglycaemia as well as hyperglycaemia and early disease onset.

A summary of the key impairments identified in the literature is presented below.

Loss of Consciousness

A serious functional impairment associated with diabetes mellitus results from the consequences of acute hypoglycaemia (described above). Hypoglycaemia is a common side effect of insulin therapy and therefore is most likely to occur in Type I diabetes (IDDM). It can also occur in people with Type 2 diabetes who take oral agents or in those with Type 2 diabetes who take insulin and who are also obese. Acute effects of severe hypoglycaemia may result in loss of consciousness which has an obvious and devastating impact on driving performance, especially since the loss of consciousness may be sudden. This is particularly the case for sudden loss of consciousness (syncope). Contrary to popular belief, most hypoglycaemic reactions are mild and do not lead to sudden loss of consciousness. This is discussed further in relation to interventions following early warning symptoms (see below).

Unawareness of Hypoglycaemia

Awareness of hypoglycaemia (hypoglycaemic awareness) is triggered by activation of the autonomic nervous system that gives rise to early warning of onset of a hypoglycaemic reaction. Warning symptoms (neurogenic symptoms) include tremor, palpitations and sweating. With appropriate intervention (food or drink high in carbohydrates) these symptoms can be relieved and the development of neuroglycopenic symptoms affecting cognitive and motor performance (e.g. difficulty concentrating, lack of coordination, visual disturbances, dizziness or light-headedness) may be averted. In some individuals, however, there is no warning or no recognition of an impending hypoglycaemic reaction. This is referred to as hypoglycaemic unawareness.

Impaired awareness of hypoglycaemia is associated with higher levels of temporary cognitive impairment and longer recovery following a hypoglycaemic event (Frier, 2000). Hypoglycaemic awareness is considered important to the level of impairment and crash risk associated with this condition (Cox et al., 1993; Eadington & Frier, 1988; Frier, 2000; Lindgren, Eckert, Sterberg & Agardh, 1996; MacLeod, 1999; McGwin et al., 1999).

Analyses of collapse-at-wheel events recorded by the Driver and Vehicle Licensing Authority (UK), showed that of 2000 cases, around 17% were caused by drivers with diabetes becoming hypoglycaemic (Taylor, 1985; Macleod, 1999). Research findings relating to hypoglycaemia and risk of crashes are discussed in the following section.

A number of functional abilities are thought to be affected during hypoglycaemia (see Cox et al., 1993; Deary, 1999; McGwin et al., 1999; Piotrowski, 1997; Ratner & Whitehouse, 1989). Impairments reported to be associated with the condition include:

- slower reaction time;
- slowed speed of performance in complex tasks;
- slowed speed of visual information processing;
- difficulty in rapid decision making;

- difficulty with sustained attention;
- difficulty with analysis of complex visual stimuli;
- impaired hand-eye coordination;
- impaired visual contrast sensitivity;
- difficulty with control of anger and irritability;
- decreased cognitive functions, mental confusion.

Vision Impairment

A number of visual impairments have been noted amongst diabetics with retinopathy, including:

- impaired acuity and blindness (see diabetes retinopathy, above);
- loss of peripheral field of view associated with retinopathy (including treatment effects of pan-retinal laser photocoagulation);
- poor dark adaptation (resulting in difficulty adjusting to glare when driving at night).

Physical Impairment

Impairments in physical abilities associated with peripheral neuropathy (particularly diabetic foot) include:

- loss of sensation (particularly in the extremities);
- weakness; and
- amputation.

Pre-May 2003: Relationship between diabetes mellitus and road safety outcomes

A number of authors have reviewed early studies on diabetes and road safety outcomes, dating back from 1960 through to the early 1980s (see MacLeod, 1999 and Veneman, 1996 for reviews). These reviews have identified inconsistencies in the findings due to methodological differences. Importantly, too, there is a general consensus that these findings are no longer relevant to current risk estimates because treatment of diabetes has changed so significantly in the last two decades, particularly through improved medications and routine monitoring by individuals with diabetes of their own blood glucose levels (BGL). A case in point here is the recent emphasis by medical practitioners to ensure that individuals achieve near normal BGL in order to reduce long-term medical complications. This change in treatment emphasis has led to a substantial increase in severe hypoglycaemic reactions (Cox et al., 2001; Diabetes Control and Complications Trial, 1993; MacLeod, 1999; Ratner & Whitehouse, 1989). Hence a review of risk based on more recent evidence is essential. The following review focuses on studies conducted since 1980 with a particular emphasis on those that

address crash risk directly. The major findings of these studies are summarised in Table at the end of this section. A brief review of studies that have addressed driving impairments rather than crash risk per se is also provided.

Crashes

In a population-based study, Vernon, Diller, Cook, Reading, Suruda and Dean (2002) compared the relative risk of drivers with diabetes and other metabolic conditions and those without, during a five-year study period from 1992-1996 (see section 3.1 for a more detailed description of the study methodology). Drivers with diabetes and other metabolic conditions (thyroid, parathyroid, pituitary and other metabolic conditions) totalled 10,105. The majority of these cases ($n = 9,731$) had no licensing restrictions. Separate analyses were conducted for those drivers with diabetes who also had other medical conditions (see following section).

Overall, the findings showed that for drivers with diabetes who were on restricted licences according to speed, area and/or time of day (highest level of impairment) rates of crashes and at-fault crashes were elevated but did not differ significantly from controls (RR: 1.38, 95% CI 0.75-2.54; RR: 1.77, CI 0.87-3.61, p 's > 0.05 , respectively). However, those without licence restrictions (lowest level of impairment) had significantly elevated crash rates and at-fault crashes (RR: 1.30, 95% CI 1.23-1.38, $p < 0.05$ and RR: 1.46, 95% CI 1.36-1.58, $p < 0.05$, respectively).

Vernon and colleagues proposed that their findings provided evidence of the effectiveness of the licence restriction program in reducing risk in this population of drivers since the crash risk of those most severely impaired and under some level of licence restriction, appeared to be relatively well controlled. However, another interpretation of the findings is that those who are more severely impaired regulate their amount of driving more than those who are less impaired. Indeed, those who are restricted in area of driving may be expected to drive shorter distances and others may do this by choice. If this were true, then exposure rates of restricted drivers will be lower and there will be less likelihood of crash involvement. Hence, a limitation of this study is that there is no control for exposure rates. While the authors assumed that the matched controls would drive similar distances, it is also plausible that the presence of a medical condition may influence driving distances and in particular, may result in self-limitation of the amount of driving. It is not possible to ascertain the extent to which potential differences in exposure might confound the analyses. Another shortcoming of this study is that not all drivers report medical conditions to the licensing authority since there is a possibility that their licence may be restricted or revoked. The authors of this study also indicate that the number of drivers who reported their diabetic status was less than half of the total population of diabetics in the USA state of Utah where the study was conducted. A third point of caution in relation to potential confounds is that cases included drivers with different types of metabolic conditions, including diabetes. Hence, elevated risk of crashes and at-fault crashes associated with the 'diabetic group' is confounded by metabolic conditions other than diabetes. Very little information is available to demonstrate the risk associated with these other illnesses, however, given the relatively low prevalence of thyroid, parathyroid, pituitary conditions, it is unlikely that the estimated risks for the 'diabetic group' in this study would be greatly affected by their inclusion. Moreover, during uncontrolled thyroid, parathyroid and pituitary episodes people are too ill to drive and if they are controlled, there is unlikely to be any

effect upon driving. Therefore the net effect upon inclusion of these conditions in the metabolic conditions group is negligible. .

Hansotia and Broste (1991) studied rates of crashes and citations ('mishap ratios' (MRs) per 1000 person-years of licensed driving standardised for age) during the four-year period from 1985 to 1988, amongst 30,420 drivers (see next section for the results regarding citations). Participants were drivers from the city and surrounding areas of Marshfield, Wisconsin USA and were aged 16-90 years. Cases ($n = 484$) were identified from medical records and included a random sample (50%) of the population of all diagnosed diabetics (ICD-9-CD diagnostic codes). Controls were active drivers who had no diagnostic code suggestive of diabetes. Cases included 10% with Type 1 and 90% Type 2 diabetes. Around 38% were insulin-treated, of these around 95% did blood glucose self-testing and just fewer than 10% had at least one reported severe (hypoglycaemic) reaction. Presence of comorbid conditions was also recorded (cardiovascular disease: 36%, neuropathy: 20%, retinopathy: 16%; alcohol abuse: 3%). Overall, the study found significantly higher mishap ratios for participants with diabetes for crashes (1.32, $p = 0.01$). Also, of interest was the finding that risk of injury crashes amongst drivers with diabetes, was higher than the non-diabetic cohort (Standardised Mishap Ratio: 1.57, 95% CI: 1.04-2.29), $p < 0.05$. However, there was no significant difference between those with diabetes and those without in risk for crashes involving property-damage only. The reason for this is difficult to ascertain. One explanation for this finding is that people with diabetes may be more vulnerable to injury in the event of a crash. Alternatively, it is possible that differences in crash severity may account for the higher rates of injury amongst diabetic drivers. It is important to note that no adjustments in statistical measures were made for potential confounders such as duration of diabetes, comorbid conditions, diabetes type and the use of different glucose lowering therapies and no consideration was given to disease severity. Furthermore, no adjustments were made for exposure. There are also several potential sampling biases in this study. First, the sample were recruited from the population of drivers in a limited geographical area in Wisconsin, and it is not clear whether the sample is adequately representative of the population of all drivers in the USA (or elsewhere). Second, the medical status of the control group, other than non-diabetes status (absence of diagnostic code suggestive of diabetes) was not recorded. Thus, a limitation of the study is that the control participants may include people with other medical conditions and/or undiagnosed diabetes. Notwithstanding these limitations, it is interesting to note that overall, the findings of this study were similar to those reported for the Utah study (Vernon et al., 2002). On the basis of their findings, Hansotia and Broste concluded that drivers with diabetes have slightly higher risk of crash compared with drivers unaffected by the condition. However, they suggest that when taken in the context of the relatively small size of the population at risk, there was insufficient evidence to warrant further restrictions to driving privileges.

In a study that focused on older drivers, Koepsell and colleagues examined the influence of medical conditions, including diabetes, on the rates of crashes resulting in injury (Koepsell, Wolf, McCloskey, Buchner, Louie, Wagner & Thompson, 1994) (see section 3.1 for a more detailed description of the study). Koepsell and colleagues found that approximately 11% of those who were involved in injury crashes and 4.5% of controls (no injury crash involvement) were affected by diabetes mellitus. Just under half of the cases with diabetes were treated with OHA while the remainder were treated with either insulin or diet. Appropriate analyses were conducted to control for age, gender and place of residence as well as other potentially confounding factors. The

results showed a significant increased odds ratio for diabetes (OR: 2.6, 95% CI 1.4-4.7). In addition, the odds ratio for drivers receiving insulin treatment (IDDM) was also found to be significant (OR: 5.8, 95% CI 1.2-28.7). Treatment with diet alone showed no relationship with crashes (OR 0.9, 95% CI 0.4-2.4). Similarly, while the odds ratio for those treated with oral hypoglycaemic agents (OHA) was elevated, the difference between cases and controls was not significant (OR: 3.1, 95% CI 0.9-11.0). Time since diagnosis was an important factor with drivers who had a diagnosis of diabetes for over 5 years more prevalent amongst injury crash-involved cases compared with non injury crash-involved controls (OR: 3.9, 95% CI 1.7-8.7). A co-existing condition of coronary heart disease in drivers with diabetes also resulted a significant association with crashes (OR: 8.0, 95% CI 1.7-37.7). The authors note that adjustment for race, marital status and exposure (miles driven in previous year) resulted in only slight changes in these ORs, although no data are provided. Notwithstanding the relatively small number of drivers with diabetes amongst cases and control groups for this study, these findings suggest a significant relationship between older drivers with and injury crashes. This was particularly apparent for those receiving insulin, those who have had the condition for more than 5 years and those who have coexisting heart disease. It is also difficult to separate the issues deriving from the aging process and those deriving from the disease.

The significant relationship between crashes and presence of diabetes in older drivers reported by Koepsell et al. is consistent with the findings of Hansotia and Broste (1991) for drivers of all ages with diabetes. Staplin et al. (1999) also reported preliminary evidence from a study of older drivers in Maryland, USA, showing a slightly increased risk in a sample of 363 older drivers (68-89 years) with diabetes (type unspecified) (OR: 1.34). This risk was elevated for females (n = 163) with diabetes (OR: 2.13).

McGwin, Pulley, Sims and Roseman conducted a population-based case control study to examine the association between diabetes and its complications and at-fault crashes (1999; 2000). As with the study by Koepsell et al., the population of interest for this study was restricted to older drivers, aged 65 years and older, who were residents of Mobile County, Alabama USA and who were licence holders (excluding those who retained their licence for identification purposes only). Cases were at fault crash-involved drivers (n = 249). Controls included a sample of (i) crash-involved drivers who were not at fault (n = 198); and (ii) non-crash-involved drivers (n = 454). One limitation of both this study and the study conducted by Koepsell and colleagues (1994) was that drivers under 65 years were excluded from this study. Hence, the findings may not be generalisable to age groups other than those over 65 years. Although diabetes is more prevalent in older people, the condition, particularly Type 1 diabetes, also affects younger drivers. Secondly, the study used self-reporting (telephone interview) techniques to identify presence of diabetes as well as other medical conditions. This is likely to lead to biases in identification of cases as discussed in Chapter 2. Notwithstanding these methodological constraints, the study is one of the few that attempts to control for potential confounds such as age, gender, annual mileage (self-reported), chronic medical conditions and visual function. Overall, the study found no evidence for an association between diabetes and at-fault crash involvement amongst drivers aged 65 years and older. Adjustment for the above-mentioned factors did not greatly influence the risk estimates. The adjusted ORs for diabetes were 0.7 (95% CI 0.4-1.3) and 1.1 (95% CI 0.7-1.9) when cases were compared with the not-at-fault and non-crash-involved control groups, respectively. In contrast to findings of other studies of older drivers (Koepsell et al., 1994) and drivers of all ages with diabetes (Hansotia and Broste 1991), there was no evidence for an association between crashes resulting in

injury and diabetes. Also, contrary to findings of others Koepsell et al., (1994), this study found that treatment modality for diabetes cases (pharmacological control, diet control only, OHAs, insulin treatment) did not significantly influence risk. However, as was the case in the study by Koepsell et al., the study methodological did not allow for a dissociation between the treatment effect and the effect of the diabetes condition per se.

McGwin et al. also considered the effect of medical complications. Their results showed that there was no relationship between diabetic retinopathy and crashes (OR: 1.3, CI: 0.3-5.2) and similarly, although there was an indication of an elevated risk, the odds ratio for neuropathy also failed to reach significance (OR: 2.2, CI: 0.4-11.2). However, as noted by the authors, the ORs should be interpreted with caution because of low numbers of participants with these complications. Interestingly, prior crash involvement (in the preceding 4-year period) significantly influenced the relationship between diabetes and at-fault crash involvement. Diabetes was over-represented amongst those with a prior crash history. The adjusted OR for diabetes was 2.5 (95% CI: 0.9-7.2) amongst cases who had prior crash involvement. In contrast, the OR for diabetes was only 0.9 (95% CI: 0.5-1.7) for those who had no previous crash involvement.

Of further interest in the study conducted by McGwin and colleagues (1999) described above, is their evaluation of crash type. To our knowledge, this is the only study reported in the literature that provides such an analysis. The authors reported that drivers with diabetes were over-represented amongst those who had crashes that were thought to be precipitated by drivers travelling too closely, compared with non-crash involved drivers. Drivers with diabetes who also had neuropathy were also more likely to be involved in travelling-too-closely crashes than those without neuropathy. However, these results need to be interpreted with caution as the sample size in this study was small. In contrast, for crashes due to other causes (i.e. “failure to yield, lack of vehicle control, unseen objects, misjudged stopping distance and failure to heed traffic signs or signals” (p. 244)) there was an over-representation of participants with diabetes. The authors suggest that their finding may indicate poor reaction time. The assumption appears to be based on an over-representation of drivers with diabetes in crashes that may have been caused by failing to notice that the vehicle ahead had stopped.

Salzberg and Moffat (1998) evaluated the effectiveness of a special exam program operated by the state of Washington Department of Licensing in identifying drivers with impairments and in reducing their crash risk. The program targets drivers with medical, vision and physical impairments and the special exam includes an in-depth interview and a drive test. Outcomes of the exam include licence cancellation, licence restrictions (including area/time and equipment restrictions such as outside vehicle mirrors or corrective lenses) and continuation of unrestricted licence status. Cases were all drivers who had special exams during 1994 (n = 449). Controls (n = 449) were randomly selected from the pool of potential drivers who had not had a special exam during the year 1994 and who were matched to each case by age, sex and city of residence. The average age of all participants was 76 years with the majority of participants (87%) over age 60 years. Five-year driving records, including crashes and violations, were obtained for all participants from official records and covered approximately 3.25 years following the exam and 1.75 years prior to the exam. Included in the study were 27 drivers with diabetes and 14 drivers who passed the special exam (also see section 3.12 for a review of the findings for Diabetic Retinopathy). No description of diabetes type, severity or time since onset was given.

Salzberg and Moffat reported that pre-exam crash risk (expressed as a rate per 100 drivers per year) for drivers with diabetes was 1.67 times higher than the control group but crash rates were comparable for the two groups in the post-exam period. Driving violation rates of drivers with diabetes and controls were similar both pre- and post-exam period (pre-exam rates were 8.5 and 7.5; post-exam rates were 2.3 and 2.3, for those with diabetes and controls, respectively). Interestingly, there was a notable reduction in crash rate for the control group as well as amongst drivers with diabetes, although the improvement was less marked for controls. The authors noted that this may be due to a general reduction in driving amount with increased age (across the 5-year pre-post exam period) in both groups. However, as noted by others (see Staplin et al., 1999) since measures of driving distance were not available and estimates of disease severity were not reported it is difficult to estimate the impact of exposure on crash rates. While the findings suggested a higher crash rate amongst drivers with diabetes, a number of methodological shortcomings have led to a significant bias in the conclusions. The limitations included a lack of control for comorbid conditions amongst drivers with diabetes, a small sample of drivers with diabetes, lack of exposure measures, sampling bias of older drivers who were referred for poor driving.

In a study limited to crash risk of drivers with Type 1 diabetes only (n = 166), Eadington and Frier (1988) surveyed driving habits including licence status, self-regulation; crashes and hypoglycaemia-related crashes. Results showed that crash rates were 4.9 and 6.3 per million miles driven, for males and females with diabetes respectively and 10 crashes per million miles driven for the general population. No statistical analyses of these data were reported. About 16% of crashes amongst cases were attributed to hypoglycaemic reactions while driving. More crash-involved male drivers had experienced hypoglycaemia while driving than those who had not ($p < 0.01$). The prevalence of hypoglycaemia was not different between male drivers who did or did not have a crash, although the number of subjects in the sample was small. The authors concluded that there was no 'important change' in crash risk in drivers reviewed 8 years after a previous assessment, supporting the concept of a 'prophylactic effect' of Type 1 diabetes on driving habits. The study also provides some useful information on other aspects of management of drivers with diabetes. Interestingly, 34% of drivers with diabetes, identified by a medical assessment 8 years earlier, still held an unrestricted licence. That is, they had not declared their condition to licensing authorities. Approximately 14% had ceased driving since the previous assessment and all but two drivers had done so voluntarily.

Several limitations of this study need to be taken into consideration when interpreting the findings. First, the crash rate of the control group is based on population data (UK), which, the authors note, includes drivers with diabetes, and presumably, other medical conditions, although this is not clearly stated. Moreover, it is not clear whether the population crash data is for the entire UK population or a subset of these such as Scotland. Second, the cases were described as a diabetic cohort from Edinburgh, who may or may not be representative of the wider driving population of the UK. Self-reported exposure data for 140 cases were used to derive average annual mileage figure to compute mileage-adjusted crash rates for cases. It is not clear how this exposure measure was determined for controls. Lastly, the reliance on self-reporting of driving habits is dependent on participants' memory and willingness to disclose information.

Songer and colleagues (1988) also examined motor vehicle crash involvement of people with Type 1 diabetes in a case-control study of 158 insulin-dependent diabetes cases

and 158 non-diabetic siblings. Cases were drawn from a cohort of children diagnosed with IDDM who were registered at Children's Hospital at Pittsburgh during 1950-54. Eligible cases were aged 21 years or older and had a living non-diabetic sibling of the same sex and approximately the same age (≤ 5 years difference and at least 21 years old). Crash involvement was determined from self-reported responses on a questionnaire. The rate of crashes per 100 drivers was slightly higher amongst IDDM cases than non-diabetic controls (14.2 vs. 7.1 crashes), however the difference was not significant. When the data were adjusted for distance travelled (collision rate per 1,000,000 miles travelled), IDDM cases again had a higher crash rate but this was not significant (10.4 vs. 3.9 crashes/100 drivers per 1,000,000 miles). Crash rates were also significantly influenced by age and gender. Those aged 21-29 had a higher crash rate than 30-39 year olds and 40-49 year olds. Women with IDDM were also found to be involved in around 5 times more crashes than non-diabetic women (32.4 vs. 6.6 crashes per 100 drivers/1,000,000 miles). This elevated crash risk is consistent with results presented by Staplin et al. (1999) for older women with diabetes. Differences between crash rates for male cases and controls were not significant. Multivariate modelling was used to evaluate independent contributions of diabetes, age, sex, marital status and mileage driven. The adjusted OR (and 95% confidence interval) generated from the analysis for diabetic status was 0.99 (0.28-3.50), $p = 0.98$. The adjusted odds ratio for female diabetics was 5.73, $p < 0.05$, confirming that even after controlling for age, marital status and exposure, females with diabetes were at considerably higher risk of crashes than their non-diabetic siblings. Overall, the authors concluded that the crash risk of those with IDDM did not differ from the non-diabetic population. However, females with IDDM were much more likely to be crash-involved than non-diabetic females.

In the same study, Songer et al. (1988) also investigated hypoglycaemic episodes amongst IDDM cases. Eleven IDDM cases (7%) reported that a health-related problem had caused them to be involved in a crash while only 1 control (<1%) of the non-diabetic siblings indicated that a health problem caused them to crash. In 9 of the 11 IDDM cases who reported that a health problem caused a crash, this was attributed to hypoglycaemia while 2 cases were attributed to visual problems. It is important to note that crash attributions in this study were based on drivers' perceptions and therefore subject to biases inherent in self-report. It is possible that drivers with medical conditions are more likely to attribute crash causation to their disease than non-diabetic drivers.

In another study of drivers with Type 1 diabetes, Stevens and colleagues (1989) conducted a retrospective, five-year survey of crash risk in 596 people with insulin-treated diabetes aged 18-65 years (354 were drivers) and 476 non-diabetic control subjects (302 drivers). Cases were volunteers from two diabetic clinics in Belfast who had been taking insulin for at least one year. They represented 92% of the eligible population. Controls were volunteers from gastroenterology and dermatology clinics who did not have diabetes. They represented 100% of the population of eligible patients who attended these clinics over a 4-month recruitment period. Participants completed a questionnaire including questions on driving experience, crashes, driving convictions and alcohol consumption. Visual acuity was tested using a Snellen chart. In addition, diabetic cases were asked about clinical details (clinical details for cases were recorded by one of the authors), home monitoring of BGC, experience of hypoglycaemia while driving, knowledge of relevant legislation on diabetes and driving and recommendations of the British Diabetic Association, and whether or not they had declared their condition

to the licensing authority and insurance company. Crashes and driving convictions were recorded for the period since starting insulin treatment (for cases), and becoming a motorist, or during the past five years, whichever was the shortest. Crashes were defined as any incidents where the participant was the driver, regardless of fault, which resulted in an injury, repair of vehicle in a garage, or an insurance claim, or any combination of these. Rates of crashes in the previous five-year period for those with diabetes (23.2%) did not differ significantly from those without diabetes (24.8%), $\chi^2=0.25$, $p = 0.62$. This was consistent with findings reported by Songer et al. (1988). Rates did not differ when analyses were conducted after stratification for age, sex, duration of licence and alcohol consumption. Similarly, crash rates for those with and without diabetes did not differ per kilometre drives (7.9 vs. 7.8), per driving years (7.1 vs. 7.1), nor per 100 drivers (30.1 vs. 30.8). No differences were observed in rates of driving convictions over the five-year period (4% for the diabetic group and 7% for controls). Further analysis revealed that crash rates of drivers with diabetes who also had other medical conditions were similar to diabetic drivers without other medical conditions (23% and 23%, respectively). Due to small numbers of drivers with comorbid conditions such as heart disease and visual impairment, a meaningful interpretation of these findings is difficult. Approximately 29% of the drivers with diabetes reported experiencing hypoglycaemia while driving in the previous year. The number of hypoglycaemic reactions whilst driving was related to the total number of crashes in the previous five-year period (rates were 19%, 28% and 35% for 0, 1 or 2+ crashes), $\chi^2=7.07$, $p = 0.03$.

In a more recent study, Songer (2002) investigated 428 persons with Type 1 diabetes from the Pittsburgh region of USA. Health status of participants, including the presence of diabetes complications was assessed by clinical examination and frequency of hypoglycaemia in the previous year was provided by self-report. Medical complications included retinopathy (62%) and 42% had hypoglycaemic unawareness, kidney disease (27%), heart disease (23%) and autonomic neuropathy (17%). The average age of participants was 37.2 years and the average duration of the disease was 29 years. No comparison group was studied. Crash data, derived from participant self-report, showed that 11% had been involved in a crash in the previous year. Crash frequency was not influenced by gender, marital status, alcohol intake, glycaemic control, use of insulin treatment, hypoglycaemic unawareness or neuropathy. Crashes were associated with younger age and greater exposure (miles driven). Furthermore, severity of hypoglycaemia was an important factor in crash involvement. Severe hypoglycaemia (resulting in loss of consciousness) was more frequent among those involved in crashes compared with non crash-involved (32.6% vs. 16.9%, $p < 0.02$). However, there was no relationship between crash frequency and episodes of mild hypoglycaemia (symptoms of shakiness, trembling, sweating). In addition, episodes of hypoglycaemia without warning were more frequent amongst those who crashed (54.3% vs. 36.2%), $p < 0.02$. Both factors were significantly associated with crashes after adjustments were made for age, gender, glycemic control, exposure (mileage) and neuropathy (adjusted ORs for severe hypoglycaemia: 3.62, 95% CI: 1.64-7.98, $p < 0.05$; and hypoglycaemia without warning: 2.34, 95%CI, 1.13-4.83, $p < 0.05$). The author notes that because of the cross-sectional design used in the study it is not possible to conclude that low blood sugar caused these crashes. However, on the basis of the evidence presented, it is concluded that severe hypoglycaemia and hypoglycaemia without warning may be important indicators for an elevated (2-4 times higher) risk of crashes.

Cox, Clarke, Gonder-Frederick and Kovatchev (2001) compared self-reported crash rates for people with Type 1 and Type 2 and their spouses in a survey of drivers (n = 1036) from 11 cities in the USA and Europe. Drivers with Type 1 diabetes reported twice as many crashes as their spouses who did not have diabetes, $p = 0.001$. Drivers with Type 1 diabetes also reported significantly more episodes of hypoglycaemia while driving compared with both their spouses who did not have hypoglycaemia and with those with Type 2 diabetes, $p = 0.001$. Crash rates for drivers with Type 2 diabetes were found to be slightly elevated but not significantly different to their spouses without diabetes. Few details of the characteristics of the sample population, such as diabetes severity or duration or details of survey methods were reported; hence these findings need to be interpreted with some caution.

Citations

As outlined above, Vernon et al. (2002) compared the relative risk of driving citations for drivers with diabetes with and without licensing restrictions and compared them to drivers without a medical condition. Overall, the rate of citations amongst those with diabetes did not differ from controls (RR for unrestricted drivers with diabetes: 1.02, 95% CI 0.98-1.07; RR for unrestricted drivers 1.39, 95% CI 0.92-2.09).

Salzberg and Moffat (1998) investigated the citation rates for 27 drivers with diabetes with 449 control participants. Driving citation rates of drivers with diabetes and controls were similar both pre- and post-exam period (pre-exam rates were 8.5 and 7.5; post-exam rates were 2.3 and 2.3, for those with diabetes and controls, respectively).

Hansotia and Broste (1991) reported no evidence of greater rates of violations (speeding, careless driving or alcohol and drug violations) amongst drivers with diabetes were not different compared to control participants (Standardised Mishap Ratio: 1.14, 95% CI: 0.92-1.39, $p = 0.23$).

Driving performance

A number of studies have been conducted by Cox and colleagues using a driving simulator to examine driving abilities in people with Type 1 diabetes. The particular focus of this work has been on the effect of hypoglycaemia on driving performance and drivers' awareness of driving impairments. In 1993, Cox, Gonder-Frederick and Clarke studied 25 drivers with Type 1 diabetes. BG levels were manipulated using intravenous insulin administration and participants were unaware of their actual BG levels. Driving performance was not impaired during mild hypoglycaemia (mean BG 3.6 ± 0.33 mM). However, moderate hypoglycaemia (mean BG 2.6 ± 0.28 mM) resulted in disruptions to steering with significantly more swerving, spinning, time crossing over lanes and driving off the road. Compensatory slowing of driving speed was also observed under moderate hypoglycaemia. These driving impairments were observed in 35% of the sample and 44% of these drivers also indicated that they would drive under such conditions. Driving impairment was not associated with age, sex, disease duration, average miles driven in the past year, driving experience and self-reported crashes. This is an important study since it is the first to demonstrate that hypoglycaemia can have a direct effect upon driving performance. However, it should be noted that people with diabetes are routinely trained in how to react when faced with hypoglycemic symptoms and, therefore it might be reasonable to expect that most diabetics would choose not to drive when experiencing the degrees of hypoglycaemia examined in this study.

In a subsequent driving simulation study by the same authors (Cox, Gonder-Frederick, Kovatchev, Julian & Clarke, 2000; Cox, Gonder-Frederick, Kovatchev & Clarke, 2001), drivers with Type 1 diabetes were found to be similarly impaired at three low BGL levels (<2.8; 2.8-3.3; and 3.4-4.0 mmol/l). Hence, in this study, driving decrements were observed even at mild hypoglycaemic levels. Drivers did not employ corrective measures until BG fell below 2.8 mmol/l. Only about one-third of drivers engaged in self-treatment (drank a glucose drink or pulled off the road to correct their hypoglycaemia). Those who self-treated experienced less neuroglycopenia (using EEG measures) during driving, compared with those who did not self-treat during the drive. The authors concluded that there is a narrow window of time between drivers' detection of hypoglycaemic symptoms which require self-treatment and the onset of neuroglycopenia, which may negatively affect the ability to make judgements about the need to self-manage. The authors suggest that due to the relatively small sample size and the use of a simulator to measure driving performance, these results might not necessarily generalise to actual driving risk for drivers with Type 1 diabetes.

Co-morbidity and road safety outcomes

McGwin et al. (1999) reported that retinopathy and neuropathy were over-represented amongst cases compared with both control groups (1.3-2.2 times greater) but differences were not significant. However, the ORs should be interpreted with caution because of low numbers of participants with these complications. In addition, McGwin and colleagues reported that adjustment for comorbidity of other medical conditions (high blood pressure, stroke, heart disease, cataracts, glaucoma, kidney disease, near and far vision and peripheral vision problems) had little impact on risk ratios.

In a further analyses of drivers licensed with medical conditions in Utah, Vernon et al. studied crash risk of drivers with diabetes and co-existing conditions (data for two-way combinations of conditions only) (2001). Table 15 shows odds ratios for the most common co-existing conditions.

For unrestricted drivers (least level of impairment) with both diabetes and cardiovascular conditions, crash rates and at-fault crash rates were significantly higher than controls but significantly lower for citations compared with controls. A similar pattern was evident for restricted drivers (higher level of impairment). Unrestricted drivers with diabetes and vision conditions (i.e. with a "history of vision conditions affecting driving"), neurological and psychiatric conditions also showed higher crash rates and at-fault crash rates and similarly, drivers with restrictions who had vision conditions also showed higher at-fault crash rates.

Treatments of diabetes mellitus and road safety outcomes

As discussed, the detection and treatment of hypoglycaemia prior to or during driving is clearly a critical concern, primarily for drivers with Type 1, or insulin-treated Type 2 diabetes. Recent emphasis maintaining good glycaemic controls in people with Type 1 diabetes has led to a substantial increase in severe hypoglycaemic reactions (Cox et al., 2001; Diabetes Control and Complications Trial, 1993; MacLeod, 1999; Ratner & Whitehouse, 1989). As noted by Distiller and Kramer (1996), this has led to a paradox such that those with poorly controlled diabetes who require insulin may well be at a lower risk than those with well-controlled diabetes because they are at lower risk of a severe hypoglycaemic episode. Self-treatment using high carbohydrate food or drink upon immediate detection of onset of symptoms is an effective treatment. However,

while the effect of hypoglycaemia has received considerable attention, relatively few of the studies reviewed above have considered the effect of different forms of treatment on crash risk in any systematic way. Only two studies were found which addressed this issue specifically and their findings are contradictory. In the study by McGwin et al. (1999), described above, the effect of treatment modality (including pharmacological control: oral hypoglycaemic agents (OHAs), insulin treatment; or diet control only) was considered. The study found no significant effect of treatment modality on at-fault crash risk. In contrast, Koepsell et al. (1994) found significantly higher crash rates amongst insulin treated and OHA-treated drivers (ORs: 3.1 and 5.8, respectively). McGwin et al. note that the differences in findings of the two studies may be due to different methods of identifying crashes; that is, by crash reports (McGwin et al., 1999) and medical records (Koepsell et al., 1994).

Table 15 Relative risk for citations, all crashes, at-fault crashes for drivers with two medical conditions and corresponding comparison groups (from Vernon et al., 2001)

Condition	LICENCE STATUS	OR	95% CI
Diabetes & cardiovascular (n=5518)	Un-restricted (n=5149)	0.81** Citations 1.17* Crashes 1.41* At-fault crashes	(0.73-0.90) (1.05-1.30) (1.23-1.61)
	Restricted (n=369)	0.64 NS Citations 1.86* Crashes 3.09* At-fault crashes	(0.26-1.55) (1.01-3.41) (1.64-5.83)
Diabetes & vision	Un-restricted (n=456)	0.80 NS Citations 2.27 * Crashes 2.34* At-fault crashes	(0.50-1.28) (1.59-3.24) (1.48-3.68)
	Restricted (n=136)	1.33 NS Citations 1.77 NS Crashes 2.54* At-fault crashes	(0.53-3.35) (0.81-3.90) (1.15-5.62)
Diabetes & neurological	Un-restricted (n=521)	0.99 NS Citations 1.49* Crashes 1.65* At-fault crashes	(0.73-1.34) (1.10-2.01) (1.12-2.44)
	Restricted (n=too small/no analyses)		
Diabetes & pulmonary	Un-restricted (n=653)	0.81 NS Citations 1.16 NS Crashes 1.30 NS At-fault crashes	(0.73-0.90) (0.86-1.57) (0.87-1.93)
	Restricted (n=too small/no analyses)		
Diabetes & psychiatric	Un-restricted (n=434)	1.01 NS Citations 1.51* Crashes 1.92* At-fault crashes	(0.76-1.33) (1.11-2.06) (1.28-2.88)
	Restricted (small n/no analyses)		

* medical conditions group statistically higher rate

** medical conditions group statistically lower rate

In addition to the effect of different glucose lowering therapies used for the control of BG in diabetes, the treatment and assessment of visual conditions has also raised some important issues for safe driving. Assessment of vision is an important component of assessment of medical fitness to drive and a number of licensing authorities advise regular fundoscopic examination of drivers with diabetes. This procedure requires that

the pupils are dilated. Jude, Ryan, O'Leary, Gibson and Dodson (1998) assessed 61 drivers (18 IDDM and 43 NIDDM). Of interest was the effect of pupillary dilatation on binocular visual acuity and contrast sensitivity under different conditions of glare. The results showed a significant reduction in acuity post-dilatation ($p < 0.005$) and this was reduced more under conditions of glare. Contrast sensitivity was not affected by dilatation. The authors concluded that individuals who undergo such treatment should be advised not to drive for at least 2 hours after pupillary dilatation.

Several studies have also considered the effects of treatment of diabetic retinopathy, although no direct measures of driving risk have been reported. For example Hulbert and Vernon (1992) and Pearson and colleagues (Pearson, Tanner, Keightly & Caswell, 1998) report visual field loss in people treated with bilateral retinal pan-photocoagulation (PRP) to improve visual acuity. Field loss was particularly high in participants with Type 2 diabetes. The authors present guidelines for treating clinicians to minimise field loss in patients who wish to continue driving.

Post-May 2003: Relationship between diabetes and road safety outcomes

Eleven papers pertaining to diabetes and driving risk were identified in the review period from May 2003 to mid-2009. A total of 6 studies were identified in the review period post-May 2003 addressing crash risk of drivers with diabetes, one of these also addressed citations. A number of the crash-related studies identified in the post-May 2003 literature search describe specific case studies of drivers with diabetes rather than addressing the question of relative risk of drivers with diabetes (Cox, Kovatchev, Vandecar, Gonder-Frederick, Ritterand & Clarke, 2006; Diamond, Collins & Rohl, 2005; Chlupp & Neoral, 2004; Soule & Egede, 2007). These studies are identified for readers' interest but are not reviewed in detail in this report. Additionally, two papers addressing driving performance and three studies dealing with self-regulation and driving in people with diabetes were identified in the post-May 2003 period. The studies are reviewed below and summarised in Table 16 at the end of this section.

Crashes

In one of the few population based prospective case-control studies of its kind, Skurveit and colleagues (Skurveit et al., 2009), examined the crash risk of Norwegian drivers with diabetes receiving blood glucose-lowering medication. Medication use also served as a proxy for a diagnosis of diabetes. All Norwegian drivers aged 18-69 years (3.1 million) were studied from April 2004 to September 2006. Information on crashes (with injuries) and prescription medications was obtained from population-based databases. The exposure period was designated as the time from first prescription of insulin or oral glucose lowering agents. The incidence of crashes was compared in the exposed and unexposed person time periods using standardised incidence ratios (SIR). A total of 20 494 crashes were reported during the study period, including 183 for registered insulin users and 219 on oral glucose lowering agents without insulin. The results showed a slight increase in risk for drivers prescribed insulin (SIR: 1.4, 95%CI 1.2-1.6). In contrast, no increase in risk was found for drivers prescribed oral glucose lowering agents (SIR: 1.2, 95%CI 1.0-1.3). One limitation of the study is that the non diabetic group almost certainly includes people with diabetes who are not treated by insulin or oral glucose lowering agents, since there was no reliable way to identify this group. However, the authors state that these patients would represent a small part of the non diabetic group and therefore would have little effect on the results.

In 2008, Lonnen and colleagues compared crash rates of insulin treated drivers and non-diabetic drivers using a population register-based study (Lonnen, Powell, Taylor, Shore & Macleod, 2008). The study merged crash data from the police crash database from two regions of the United Kingdom (Devon and Cornwall) with a clinical database for diabetic retinal screening (97% of the known diabetic population in the regions) comprising 12,175 patients aged 15 years and older (2697 required insulin). Analysis of the crash database revealed 29477 crashes reported over the 5-year study period (1998-2002). Of these, 521 occurred in people with diabetes (129 in drivers treated with insulin). The overall crash rates for the non-diabetic population was 1,469 per 100 000, compared with 856 for all drivers with diabetes ($p < 0.001$). RR for crashes for all people with diabetes was 0.58 (CI 0.54-0.65). Gender differences were observed such that females posed the lowest risk (Males RR: 0.59, 95%CI 0.54-0.65; females RR: 0.42, 95%CI 0.35-0.52). The authors concluded that the crash rate in patients with diabetes, including insulin-dependent diabetics, is significantly lower than that of the general non-diabetic population of drivers. As noted by the authors a limitation of the study is the absence of adjustments for potential confounds such as exposure. The authors did adjust for age within the insulin-treated group but crash numbers were small and the authors reported no significant differences in annual crash rates for the insulin-treated group compared with the non-diabetic population (with RR between 0.51, 95%CI 0.25-1.05 and 1.13, 95%CI 0.88-1.46).

Hemmelgarn, Levesque and Suissa (2006) examined crash rates of older drivers with diabetes who were exposed to various medications (anti-diabetic drugs). A cohort of 224,734 drivers in the province of Quebec, Canada, aged 67-84 years, were identified using linked insurance and health databases. Cases were 5,579 crash involved drivers (1990-1993) and a random sample of 13,300 non crash involved control drivers. Of interest was the relative risk of exposure to anti-diabetic medication in the year preceding the index date (i.e., the date of the crash for cases and a randomly selected date during follow-up for controls). After adjusting for age, sex, previous crash and urban/rural place of residence, the rate ratio for current users of insulin monotherapy compared with non-users was 1.4 (95%CI 1.0-2.0) and 1.3 (95%CI 1.0-1.7) for a combined sulfonylurea and metformin treatment. A dose-response effect for the combined treatment group was also identified (RR: 1.4, 95%CI 1.0-2.0). The increase in crash rate corresponded to an excess rate of 32 crashes per 10,000 older drivers, annually. Consistent with the more recent findings of Skurveit et al. (2009), drivers with diabetes using only oral treatment were not found to have an elevated risk of crashes. A limitation of this study is that the sample of drivers with diabetes was aged over 67 years and therefore may not be representative of the population of all age groups of drivers with diabetes in that jurisdiction. Moreover, it is unclear whether the insurance databases used in the study included relevant medication history for all drivers with diabetes in Quebec, since the drug insurance programme in that province does not provide universal coverage for all drivers. Specifically, the insurance database would not contain data for those drivers who have private insurance coverage (likely to be those of lower socio economic status). Hence, the classification of drivers into the treatment (anti-diabetic medication groups) and control groups may be problematic.

Sagberg (2006) investigated the relative crash involvement risk associated with various diagnosed medical conditions from 4,448 crash-involved drivers. Participants were drawn from the files of a Norwegian insurance company and asked to complete questionnaires outlining information about their crash, whether they were at fault or not for the crash, to indicate from a list of 27 medical conditions, 6 categories of medicinal

drugs, 21 common symptoms which were applicable to themselves and personal background information. Details of the study method and limitations of the study are described in section 3.2. Of the 4,448 participants, 24 had non-medicated diabetes (assumed to include only non-insulin dependent, type II diabetics): 16 were at fault, and 8 were not at fault. A crude odds ratio of 1.66 was found for these drivers. The age and driving distance-adjusted odds ratio (3.1) was also significant, suggesting an elevated risk of being at-fault amongst non-medicated drivers with diabetes. The authors stated that the OR for drivers with diabetes who were on medication was not significant.

Using survey methods, Cox et al. (2003) investigated crash involvement of drivers with type 1 (n = 341) and type 2 diabetes (n = 332) and non diabetic spouse controls (363). Participants were recruited consecutively from 11 specialty outpatient clinics, including 7 clinics in the United States, 4 in Europe. Participants completed a survey which included questions on crash involvement and citations in the previous 2 years, driving with hypoglycaemia, annual miles driven, testing of blood glucose prior to driving and self-regulation of driving under different levels of blood glucose. Results showed that drivers with type 1 diabetes reported significantly more crashes (19%) compared with those with type 2 diabetes (12%) and controls (8%), $\chi^2 = 17.0$, $p < 0.01 - 0.001$. Also, no differences were observed between those with type 2 diabetes and controls. No differences in crash involvement were found between drivers with type 2 diabetes who were (n = 159; 11%) or were not using insulin (n = 109; 15%). Crashes involving drivers with Type 1 diabetes were associated with more hypoglycaemic episodes while driving and less frequent blood glucose monitoring. Interestingly, half of the drivers with type 1 diabetes and around three-quarters of those with type 2 diabetes had never discussed hypoglycaemia and driving with their doctors.

Using a different methodological approach, Leproust and colleagues (Leproust, Lagarde, Suissa & Salmi, 2007) investigated the association between consultation with a physician and crashes. Participants were older drivers, aged 65 years and older from the province of Quebec, Canada, and involved in a crash (all crashes, severe crashes and severe and property damage) between 1988 and 2000. Eligible drivers were identified from the provincial insurance and health databases. Four diagnostic groups were identified amongst the sample including diabetes (n=10,663). No detail of the diabetes diagnosis was provided. A case cross-over design was used so that cases were their own controls. The risk period was defined as the first 1-month preceding a crash and control periods were the four 1-month period preceding the risk period. Of interest was the exposure to consultation with a physician in the risk period (immediately preceding the crash) and the control periods. The total number of collisions included in the analysis was 111,699 (3,318 severe). Fifty four percent of drivers had had at least one contact with a physician in the one month preceding the crash compared with 52% in the control period. The frequency of visits was higher amongst drivers with diabetes (60%). Not surprisingly, the frequency of physician visits by the drivers with diabetes was the same in both the immediate pre-crash period (60%) and control periods (60%). For all drivers and for drivers with diabetes, there was a slightly increased risk of crashes associated with a medical contact within one month before the collision (classified as all collisions), [OR: 1.10 (95%CI 1.08 – 1.11) for all drivers, $p < .001$; OR: 1.07 (95%CI 1.03-1.11), $p < 0.01$ for drivers with diabetes]. The authors concluded that any contact with medical practitioners could present an opportunity to detect conditions that put older drivers at risk. Currently, however, more appropriate assessments are required to enable practitioners to successfully evaluate driving competency during the medical consultation.

Citations

In the study described above by Cox et al (2003), drivers with Type 1 diabetes (15%) reported significantly more citations compared with drivers with Type 2 diabetes (8%) and controls (10%), $\chi^2 = 0.8$, $p < 0.01 - 0.05$.

Driving Performance

Cheyne et al. (2004) examined the effects of mild hypoglycaemia (< 2.8 mmol/l with modest alcohol intoxication (levels below UK legal driving limits) on intellectual performance and driving performance. A sample of 17 participants with Type 1 diabetes with mean age 33 years (range 21-46) participated in the study with mean disease duration of 19 years (SD 12 years). All were licensed drivers and reported drinking alcohol (1-4 units per week). Participants were tested on 5 separate sessions, one familiarisation session and four test sessions. On two occasions, hypoglycaemia was initiated and on the other two occasions participants were euglycaemic (normal level of glycaemia). Additionally, participants were administered either a placebo drink or an alcohol drink (0.35mg/kg). Cognitive performance was assessed using the Digit Symbol test from the Weschler Adult Intelligence Scale-Revised, The Trail Making Test (B), a four-choice reaction time test and a test of change detection. Driving performance was assessed using a hazard perception task with a non-interactive driving simulation used to measure response time to hazards and identification and awareness of hazards. The combination of alcohol and hypoglycaemia resulted in performance declines on all cognitive tasks but performance on the driving-related hazard perception task was not affected by alcohol, hypoglycaemia or a combination of both. The authors remarked that it is possible that since the hazard perception task was the final one in the sessions, it is possible that alcohol levels were already beginning to fall. Given the effects of alcohol and hypoglycaemia demonstrated in their study, the authors highlighted the importance of awareness of these effects on their cognitive functions and concluded that clinicians should emphasize the need for complete avoidance of alcohol while driving. .

In a driving simulator study, Rehnova and colleagues (Rehnova, Weinberger and Kotal, 2005) examine driving performance among 32 drivers with diabetes (81% male) and 49 controls without diabetes. The study was part of a large scale European Commission project. This involved completing driving-related questionnaires, a large battery of psychological tests and a driving simulation task. Four categories of driver behaviour were evaluated using a 9-point scale: general level of driving skills, ability to avoid risks, ability to act in standard risk and critical situations and behaviour following simulated 'attacks' by other drivers. The results showed no differences between driving performance of the drivers with diabetes compared with controls on three of the driving measures. Differences were observed between the groups on behaviours following simulated attacks. The authors noted that the tolerance of drivers with diabetes to physical and psychological stress was lower and that towards the end of the drive, fatigue and lack of concentration was evident, however, there was no indication of how these conclusions were reached. The findings of the study are therefore difficult to interpret due to the limited descriptions of performance measures.

Summary

There is little concurrence amongst findings of studies investigating a possible link between crash risk and diabetes. As discussed above, this may be at least in part attributed to differences in methodologies, including differences in criteria for inclusion

(e.g. age group and type of diabetes), measures of risk, control for exposure and other confounding variables. Several studies have demonstrated an elevated risk amongst drivers with diabetes (not distinguished by type), including those with lowest impairment (but not those who were more impaired drivers and with licensing restrictions) (Vernon et al., 2001) amongst older drivers (Koepsell et al., 1994; Staplin et al., 1999) and in both all crashes (e.g. Salzberg & Moffat, 1998) and injury-crashes (Hansotia and Broste, 1991; Salzberg & Moffat, 1998). However, the estimates of risk presented in these studies are relatively modest (range 1.3 to 2.6) and, as noted by Hansotia and Broste (1991), not sufficient to warrant further restrictions to driving privileges. Indeed, others (e.g. McGwin et al., (1999) found no evidence for increased crash risk (both at fault and not at fault) in older drivers with diabetes. While there is some evidence that drivers with Type 1 diabetes or those classified as insulin-treated diabetes pose a higher risk compared with Type 2 diabetes, the evidence for a significant risk has not been consistently found in all studies. For example elevated crash rates have been reported by Cox et al. (2001) for all age groups and also for injury crashes amongst older drivers with Type 1 diabetes (Koepsell et al., 1994) and amongst females with IDDM (Songer et al., 1988), with risk elevated between 2 and 5 times that of control groups. In contrast, others have found no evidence for an increased risk with insulin-treated diabetes (Stevens et al., 1989) including studies in which rates are adjusted for exposure in drivers (Eadington & Frier, 1988; McGwin et al., 1999; Songer et al., 1988). The reader is also referred to the numerous reviews on diabetes and driving published since May 2003, including Frier (2007), Heller (2006), Stork, van Haeften and Veneman (2006) and a systematic evidence-based review by Tregear et al. (2007). In particular, based on their meta-analyses of 16 studies (published between 1965 and 2003), Tregear et al. (2007) found that drivers with diabetes have a 19% increased risk of crashes compared with controls without diabetes and they reported that there was no compelling evidence that those treated with insulin were at higher risk than those not treated with insulin. Of the studies addressing crash risk examined in the post-May 2003 review period, one large population-based prospective study identified a slightly elevated risk (Skurveit et al., 2009), another study also reported elevated risks of the order of 1.3-1.4 (Hemmelgarn et al, 2008), while one large study identified a significantly lower risk for drivers with diabetes compared with controls (Lonnen et al., 2008).

Undoubtedly, the potential effects of unrecognised hypoglycaemia pose the greatest concern about diabetes crash risk (MacLeod, 1999). As discussed above, this view is a reasonable one, given the associated effects of hypoglycaemia on cognition, attention, vision and motor control. It should be noted, however, that none of the studies examining crash risk and hypoglycaemia reviewed here actually measured blood sugar immediately after the crash. Therefore, it is difficult to draw conclusions about the contributory role of hypoglycaemia in crashes. While few studies have addressed this directly, evidence from two studies, both adjusting for confounds including exposure, showed elevated risk (2-4 times) amongst those with hypoglycaemia and hypoglycaemia without warning (Songer 2002) and amongst males with hypoglycaemia (Eadington & Frier, 1989). As noted by Songer, the findings are indicative that severe hypoglycaemia and hypoglycaemia without warning may be important risk markers for higher crash risk. Despite the obvious potential for devastating effects of hypoglycaemia whilst driving suitable guidelines to assist clinicians in making assessments about drivers' degree of unawareness are generally lacking. It is also important to note that risks associated with hypoglycaemia can be moderated or reduced by appropriate self-regulatory and self-treatment strategies. More education is needed to

alert drivers of the importance of these management strategies. Interestingly, to date, studies investigating hypoglycaemia and crash risk have restricted their cases to drivers with Type 1 diabetes. Although one study reported elevated but not significantly higher risk amongst drivers taking OHA (Koepsell et al., 1994), Veneman (1996) cautions that very little is known about hypoglycaemic awareness in drivers with non-insulin dependent diabetes. Recent research by Stork et al (2007) suggest that decisions about driving when hypoglycaemic may be problematic in as many as one-quarter of this group. These are important areas for further research, given that this forms a very large proportion of those with diabetes.

Table 16 Summary of studies of risk associated with diabetes mellitus

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Cox et al, (2001)	Cases n=25 Type 1 diabetes n=25 Type 2 diabetes Controls Spouses without diabetes Total n=1036	Crashes in previous 2 years	Type 1 diabetes twice no. crashes as spouses* (p=0.001) Type 2 diabetes not different to spouses
Cox et al, (2003)	Case-control: Cases: n=341 type 1 diabetes n=332 type 2 diabetes Controls: n=363 spouses without diabetes	Survey: crashes and citations in previous 2 years	Crashes: type 1 (19%) > type 2 diabetes (12%) and controls (8%), $\chi^2 = 17.0$, p<0.01 – 0.001.
Eadington, & Frier, (1989)	Cases n=166 IDDM Controls N=(general population statistics, DOT, London, 1986)	Crashes in previous 8 years expressed as rates per million miles driven	Number crashes per million miles driven: Cases: 5.4 Females: 6.3 Males: 4.4 Controls: 10 Males with/without hypoglycaemia: hypogl > non-hypogl, p < 0.01
Hansotia & Broste (1991)	Pop/retrospective cohort study Cases n= 484 drivers with diabetes (approx 10% type 1) Controls n=30,420 drivers	(i) mishap ratios all crashes and viol (MR) (ii) MR for moving violations (iii) MR for injury crashes (iv) MR for property damage crashes	MR: 1.32 (1.06-1.63)* (p=0.01) MR Moving Viol: 1.14 (0.92-1.39) * (p=0.23) MR Injury Crash: 1.57 (1.04-2.29)* p < 0.05 MR Property Damage Crash: 1.24 (0.95-1.59)
Hemmelgarn et al (2006)	Divers aged 67-84 years Cases: n= 5 579 crash involved drivers (1990-1993) Controls: random sample of 13 300 non crash involved control drivers	Provincial insurance databases used to identify crashes Health database identified medication use	RR: 1.4 (95%CI 1.0-2.0) current users of insulin monotherapy compared with non-users RR: 1.3 (95%CI 1.0-1.7) for a combined sulfonylurea and metformin treatment. RR: 1.4 (95%CI 1.0-2.0) dose-response effect for the combined treatment group was also identified

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Koepsell et al., (1994)	Case-control; n=234 (65yrs+) injury crashes n=446 no injury crashes;	Police-reported injury crashes requiring medical care	OR: 2.6 (1.4-4.7)* for diab OR: 5.8 (1.2-28.7)* for insulin-treated OR: 3.1 (0.9-11.0) for OHA treated OR: 0.9 (0.4-2.4) for diet only OR: 3.9 (1.7-8.7)* for >5yr diag OR: 1.4 (0.5-3.7) for ≤ 5 yr diag OR: 8.0 (1.7-37.7)* for diab & CHD vs neither diab nor CHD
Leproust et al, 2009	Case-crossover: All drivers 65+ who crashed 1988-2000 (n=111 699) (incl drivers with diabetes n=10663) Risk period: 1-month pre crash Control period: 4x 1-month periods prior to risk period	Provincial insurance and health databases used to identify crashes and diagnosis of condition Risk of having a collision (all*; severe; property damage >\$500CAD) while exposed/not exposed to a medical contact within 1 month before collision	OR*: 1.07 (1.03-1.11), p<0.01 drivers with diabetes OR*: 1.10 (1.08-1.11), p<0.001 all drivers
Lonnen et al. (2008)	12 175 patients aged 15+ years (2697 required insulin).	Police-reported crashes Clinical diagnosis of diabetes Comparisons with crashes amongst non-diabetic population	RR 0.58 (CI 0.54-0.65) for all people with diabetes

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
McGwin et al., (1999; 2000)	Pop/rdmzd, case-control; Cases n=198 at-fault crash involved drivers (65+yrs) Controls (i) n=198 not at-fault crash-involved (ii) n=454 non-crash involved drivers	(i) At-fault crash in previous year (ii) not-at-fault crash	For diabetes vs (i) at-fault crash involved controls: Adj OR: 0.7 (0.4-1.3) For diabetes vs (ii) not at-fault crash involved controls: Adj OR: 1.1 (0.7-1.9) For diabetes vs prior crash involved Adj OR for diab 2.5* (0.9-7.2) Treatment modalities and at-fault crashes: For OHA Adj OR: 1.3 (0.7-2.6) NS For Insulin Adj OR: 1.3 (0.6-2.9) NS Complicating Conditions Diab Retinopathy OR: 1.3 (0.3-5.2)NS Diab Neuropathy OR: 2.2 (0.4-11.2)NS
Sagberg (2006)	Case-Control n=4448 crash-involved drivers; 24 with diabetes , not on med. n=16 at-fault; n=8 not at fault	Self-reported medical condition amongst at fault (cases) and not at fault (controls)	Adjusted (age and driving annual driving distance) OR = 3.084 (p=0.05)
Salzberg et al., (1998)	Case-control; Cases n=27 with diabetes; passed Washington state special exam in 1994 Controls n= 449 drivers not in special exam program in 1994; age, gender, city of residence matched	(i) Crashes per 100 drivers per year (ii) Violations per 100 drivers per year	Pre-exam crash rate: Case:Control 6.4:3.8 Post exam crash rate: Case:Control 1.1:1.2 Pre-exam violations: Case:Control 8.5:7.5 Post-exam violations: Case:Control 2.3:2.3

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/Main Findings
Skurtveit et al. (2009)	Population-based prospective study; 3.1 million Noregian drivers 18-69 years Studied over Apr 2004-Sept 2006	Injury crashes and prescription medications was obtained from population-based registries in Norway. Exposure period = time from first prescription diabetic medication. Standardised incidence ratios (SIR): incidence of crashes in exposed vs. unexposed person time periods	SIR: 1.4, CI 1.2-1.6) drivers on insulin SIR: 1.2, 95%CI 1.0-1.3) drivers on oral glucose lowering agents
Staplin et al., (1999)	Cases n=363 with diabetes aged 68-89 years. Controls		OR 1.34 Females OR: 2.13
Songer et al (1988)	Cases n=158 IDDM Controls n=158 non-diabetic siblings	Crashes per 100 drivers/1,000,000 miles driven	Adj OR for diab: 0.99 (0.28-3.50) Adj OR for female cases:controls: 5.73 (1.04-31.6)* (p < .05)
Songer, (2002)	Cases n=428 IDDM Controls N/A	Crashes in previous year	Severe hypogl: Unadj OR 2.34*(1.13-4.83)* (p=.05) Hypogl w/o warning: Unadj OR 3.62 (1.64-7.98)* (p < .05)
Stevens et al. (1989)	Cases n=596 insulin-treated diabetics Controls n= 476 non-diabetics	(i) Rates of crashes; and (ii) Driving convictions in past 5 years	Crash rates for cases and controls: (23.2% vs 24.8%), $\chi^2=0.25$, p=0.62.
Vernon et al., (2002)	Pop/case-control; Cases (with diab, thyr, parathy; pituit; other metabolic conditions) n=10,105 (Restricted and unrestricted licence holders) Control (without medical conditions) n=20,210	(i) All Crash (ii) At-fault crash (iii) Citations Rates per 10,000 lic days	For low impairment cases (unrestricted): RR: 1.30 (1.23-1.38) * (p < .05), all crashes RR: 1.46 (1.36-1.58)* (p < .05, at-fault crash RR: 1.02 (0.98-1.07) NS, citations Higher impairment cases (restrictions): RR: 1.38 (0.75-2.54) all crash RR: 1.77 (0.87-3.61) at-fault crash RR: 1.39 (0.92-2.09) citations

Approaches to management

Assessing fitness to drive

Private licensing regulations relating to diabetes in a number of jurisdictions are summarised in Table 17. Overall, the regulations are reasonably in line with the evidence relating to crash risk for this driver group, as reviewed above. One general observation of the various guidelines, is that separate sets of guidelines are proposed based on diabetes type (diet controlled, NIDDM and IDDM). In the case of diabetes controlled by diet alone, there are generally no licence restrictions unless there is evidence of insufficient control (Canada; Sweden) or instability (Utah). However, guidelines generally lack definitions of what is “insufficient control”. Several guidelines also indicate the need for periodic review (Australia; Canada; Sweden). Particular mention is made of diabetic complications of vision (UK, Sweden). Recent revisions to the Canadian guidelines also specify a requirement for a good understanding of the condition. This is particularly interesting in view of studies by Cox and colleagues demonstrating poor rate of self-treatment practices in drivers with hypoglycaemia, although this condition is more problematic in drivers on insulin treatment (Clarke et al. 1999; Cox et al., 1993; 2000; 2001).

Similar guidelines to those for drivers with diet-controlled diabetes are proposed for drivers with non-insulin treated diabetes. Two notable exceptions are seen for Australia and the UK; namely, that full licence is retained until the age of 70 years in the UK provided there are no complications (UK) and a requirement for a 5-yearly review and conditional licence provisions if there are complications (Australia). In addition, Australian guidelines indicate that after the occurrence of a hypoglycaemic event, the driver must refrain for driving for a period of 6 weeks.

In the case of drivers with insulin-treated diabetes, there is quite a wide variation in guidelines across the various jurisdictions. For example, at the more liberal end of the spectrum, New Zealand guidelines state that individuals are generally considered fit to drive but recommends regular monitoring. Other jurisdictions offer unrestricted licences in cases where the condition is stable (Sweden), if no episodes of ketosis or altered consciousness for 6 months (Canada; Utah), and if the person can recognise onset of hypoglycaemic symptoms (UK). Regular medical supervision/assessment is required at varying intervals from 1 to 3 years. The most stringent requirements appear to be posed by Australia where only conditional (not unrestricted) licences may be issued to drivers with insulin-treated diabetes and only in cases where the person has the ability to detect hypoglycaemia in order to stop driving. As noted above, there is some scientific evidence for higher risk amongst drivers with Type 1 diabetes who have hypoglycaemia and unawareness of hypoglycaemia. Moreover, it is also important to consider the weight of evidence from studies by Cox and colleagues (Clarke et al. 1999; Cox et al., 1993; 2000; 2001) for low rates of self-regulation and self-treatment (glucose drink and stopping driving) amongst drivers with insulin-treated diabetes.

Commercial licensing regulations relating to diabetes in a number of jurisdictions are also summarised in Appendix D. Consistent with private licensing guidelines, separate sets of guidelines are proposed based on diabetes type (diet controlled, NIDDM and IDDM), with the most stringent guidelines recommended for insulin-treated diabetes. In the case of diabetes controlled by diet alone, there are generally no licence restrictions unless there is evidence of insufficient control (Canada; Sweden) or instability (Utah). Several guidelines also indicate the need for periodic review (Australia; Canada;

Sweden). Particular mention is made of diabetic complications of vision (Australia, USA, Sweden). Recent revisions to the Canadian guidelines also specify a requirement that drivers should stop driving and eat or drink something if blood glucose levels are below 6 mmol/L (108 mg/dL). Similar guidelines to those for commercial drivers with diet-controlled diabetes are proposed for drivers with non-insulin treated diabetes. However, commercial drivers in Australia and New Zealand cannot hold an unconditional licence, but they can hold a conditional licence if their diabetes is controlled, if there is no history of hypoglycaemia and if they comply with treatment. In addition, commercial drivers in Australia and New Zealand are required to undergo an annual review, and in New Zealand they are also required to undergo a two-yearly specialist review.

The UK licensing jurisdiction has the most stringent guidelines, in that drivers with insulin-treated diabetes are not allowed to drive commercial vehicles, however drivers may be allowed to drive CI vehicles (vehicles between 3500 and 7500kg), conditional on yearly medical assessments. However, as noted by Macleod (1999) “a blanket-restriction to all drivers with insulin treated diabetes is not supported by the available scientific evidence” (p289).

In the five remaining licensing jurisdictions, drivers with insulin-treated diabetes may hold a commercial licence if the condition is controlled and the driver complies with treatment, if the driver has not experienced hypoglycaemia episodes and the driver has hypoglycaemic awareness, if there are no significant diabetic complications (e.g., visual impairment or progressive retinopathy, peripheral neuropathy with functional loss, cardiovascular disease, ketosis or altered states of consciousness), and if they undergo periodic review. These guidelines appear to be consistent with the scientific evidence that suggests that the greatest risk is associated with drivers with “problematic hypoglycaemia” (Amiel, 1999, p 271).

In addition, Swedish drivers with an existing commercial licence who subsequently develop diabetes requiring insulin treatment may retain their licence if the condition is under control and if the driver requires the licence for their livelihood.

Conditional and restricted licences

Notwithstanding the relative uniformity of guidelines regarding diabetes and fitness to drive, Flanagan and colleagues have raised the question of whether clinicians’ advice to drivers is in line with regulations. Their study investigated clinicians’ responses (n = 73) to ‘real-life’ scenarios. Findings showed that while there was general agreement about hypoglycaemic unawareness, there was a lack of consensus in relation to patients with unstable control (Flanagan, Watson, Everett, Cavan, & Kerr, 2000). These issues become particularly important when clinicians are faced with making judgements about conditional or restricted licences.

One approach to dealing with driving risk amongst drivers with medical conditions, including those with diabetes is to impose conditions or restrictions on licence privileges and or require special assessments of fitness to drive. Despite the relatively widespread practice of such restrictions and assessment requirements, there has been little attempt to evaluate the effectiveness of such approaches. Vernon and colleagues reported that crash rates of drivers with diabetes who had licence restrictions according to speed, area and/or time of day (highest level of impairment) imposed by the licensing program for the state of Utah did not differ from drivers without diabetes, while those

without licence restrictions (lowest level of impairment) had significantly elevated crash rates and at-fault crashes (Vernon et al., 2002). These findings have been used to support the effectiveness of the licensing restriction approaches to medical review in Utah. However, as noted in more detail in the review above, there was no attempt to control for the effects of differences in driving exposure in either study and other possible explanations, including self-regulatory driving practices, may have contributed to the lower crash risks.

Training and rehabilitation

Various authors have highlighted the need for health clinicians to discuss fitness to drive with their patients who have diabetes. The Blood Glucose Awareness training programme (BGAT) was developed to with the aim of preventing driving mishaps in drivers with Type 1 diabetes (see Cox, Clarke, Gonder-Frederick & Kovatchev, 2001). The programme involved 8 sessions in which drivers receive training in recognition and interpretation of symptoms of hyper- and hypoglycaemia. Strategies for treatment and prevention of extreme hyper- and hypoglycaemia were also covered. After a 4.9 year follow-up period, 15% of drivers in the BGAT programme were involved in a crash compared with approximately 45% of those in a control group (participating in an unrelated stress-management programme), $p = 0.01$.

In a later version of the course (BGAT-2), drivers were asked to record incidences of severe hypoglycaemia, awareness of hypoglycaemia, judgements about whether to drive and driving violations for a period of 6 months (and repeated for a 12-month period). Cox et al. (2001) reported that in the latter period of study, significant improvements were observed in detection of hypoglycaemia and judgements about fitness to drive. Significant reduction (66%) in driving violations was also observed, over and above pre-programme levels, $p < 0.001$.

In a third type of intervention called Hypoglycaemia Anticipation Awareness treatment training (HAATT), Cox and colleagues (2001) specifically targeted drivers with recurrent, severe hypoglycaemia, using behavioural techniques. Significant reductions were found in driving violations in those who underwent HAAT (86%) compared to baseline levels while non-significant reductions were found for a second group who underwent an alternative programme involving empowerment training.

The findings of all three training studies reported by Cox et al. (2001) are particularly promising for reducing crash risk in drivers who are pre-disposed to hypoglycaemia. Although long-term maintenance of the training benefits post-training was not discussed in any of these studies, the longer-term safety benefits, particularly in terms of crash rates should be monitored in any future research on this topic.

Self-regulation

Hypoglycaemia is probably the most important problem for people with insulin-dependent diabetes (Essex, 1994) and for those who drive, accurate judgements about blood glucose levels are critical to decisions about driving. Self-regulation is an important issue for drivers with diabetes and particularly for those with Type 1 diabetes who have hypoglycaemia and hypoglycaemic unawareness. Monitoring of blood glucose levels before driving and during long journeys and having a supply of glucose in the vehicle at all times are common sense approaches to lessening crash risk. In addition to cautionary measures that drivers with diabetes may need to take to lessen the

potential for a hypoglycaemic event, it is expected that some drivers will regulate their amount of driving and other patterns of driving in a way that they believe is appropriate, taking into account reduction in functional ability associated with their condition.

A number of authors have noted that diabetes exerts a 'prophylactic effect' on driving habits. For example Eadington and Frier (1989) suggest that some diabetic drivers cease driving in response to declining health and driving skills. Further, they suggest that this may offset the potential increase in crash risk that might accompany hypoglycaemia. As discussed above, Eadington and Frier reported that in their 8-year follow-up study of 166 individuals with Type 1 diabetes, approximately 14% had voluntarily given up driving. What is not clear, however, is whether those who ceased driving had higher levels of impairment or were actually at a high risk of a crash. In contrast, 34% of drivers in this study still held an unrestricted licence suggesting that they had not reported their condition to the licensing authority.

Stevens and colleagues (1989) (see details of this study above) also reported that 50 of their 596 participants with diabetes (8.4%) were former drivers. Three ceased driving for medical reasons un-related to diabetes while 15 had ceased driving for reasons directly associated with their general condition of diabetes, or due to specific medical complications of their condition such as retinopathy and poor visual acuity and hypoglycaemia. Interestingly, the rate of driving cessation for medical reasons amongst controls was 10.3%. Approximately 66% of drivers with diabetes had declared their condition to the licensing authority, a legal requirement in the UK, and interestingly a slightly higher proportion (70%) had declared their status to their insurance company, despite the fact that this might impact negatively on their third party insurance status. Seventeen percent of drivers with diabetes admitted to having hypoglycaemic symptoms while driving in the previous year and nearly half of them said that they had experienced more than one episode. Eighty-one percent of drivers with diabetes said that they would stop immediately and would take glucose if they experienced hypoglycaemic symptoms and around 22% also said they would not continue to drive at that time. About 10% said they would take glucose but would continue to drive and about 7% said they would continue to drive home carefully or drive to a café or shop. The majority (around 83%) said they carried a supply of glucose in their car. These findings suggest a relatively good level of self-regulation amongst drivers with diabetes as well as a good level of preparedness in the event of hypoglycaemia while driving.

In contrast to these positive indications of self-regulatory behaviour, other studies suggest that there may be differences in what drivers *say* they would do and their *actual* decisions and behaviours in relation to self-treatment while driving. In one study demonstrating this point, Clarke, Cox, Gonder-Frederick and Kovatchev (1999) investigated drivers decisions about driving based on both perceived and actual BGL. Two groups (Group 1: n = 65 and Group 2: n = 93, replication group) of drivers with Type 1 diabetes (known levels of insulin treatment) were studied. Average age for the two groups was approximately 39 and 36 years and mean duration since diagnosis was 20.5 years (SD 10.6) and 17 (10.6) years respectively. Participants with psychiatric illness, substance abuse, or severe complications of diabetes were excluded from the study. Participants used a hand held computer to record their own symptoms and other information including estimated and actual BG recordings and whether he/she would drive. Data were collected over a 3-4 week period. The authors hypothesised that drivers would decide not to drive if they estimated their BG level to be low (<3.9 mmol/L [70mg/dL]) and that most would decide not to drive if their actual BG reading

was low. This level was based on previous findings that BG in this range that were associated with deterioration in driving performance. In addition, it was proposed that drivers would base their decisions on symptoms. Results showed that around 45% of the time when BG levels were estimated to be low (at levels associated with deterioration in driving), participants made a decision to drive. In addition, drivers indicated that they would drive more than 40% of the time when their actual BG levels were low (less than 2.2 mmol/l). These findings are consistent with findings of experiments by the same group in which drivers made decisions about driving during simulated driving (Cox, et al., 1993; Cox et al., 2000; Cox et al., 2001). These studies consistently showed that although drivers were aware of deterioration in their driving performance, they were not likely to treat their low BG while driving.

More recently, in a survey based study Watson, Currie, Lemon and Gold (2007) examined whether patients and their health practitioners were aware of and followed guidance provided by the UK Driving and Vehicle Licensing Agency (DVLA). A total of 117 drivers with insulin-treated diabetes and 106 health care practitioners (doctors, dietitians, nurses) completed an anonymous survey. Ninety five percent of patients indicated that they were aware of the need to inform the DVLA of their insulin treatment and 92% had done so. Ninety four percent indicated that they recognise symptoms of hypoglycaemia most of the time. However, few (17%) reported that they wait the recommended 45 minutes post hypoglycaemic episode before driving. Interestingly, only 15% indicated that they always check blood glucose levels prior to driving and 24% said they never do this. Analysis of the survey responses from the health practitioners showed a 100% awareness of the DVLA reporting guidelines and 62% were aware of the guideline to test before driving. Eight percent did not know that impaired hypoglycaemic awareness may be a contraindication for driving. The authors emphasised the need for regular reinforcement of DVLA driving recommendations as part of routine health care.

In a similar survey of current drivers with insulin-treated diabetes, Graveling et al. examined the extent of familiarity with and adherence to recommended safe practices in relation to hypoglycaemia and driving (Graveling, Warren & Frier, 2004). Participants were recruited from outpatients clinics at the Department of Diabetes at the Royal Infirmary of Edinburgh. Two hundred and five drivers were approached and 202 provided survey responses (112 had type 1 and 87 had insulin-treated type 2 diabetes). The majority of drivers (96.5%) indicated that they were aware of the need to notify the DVLA of their insulin-treated diabetes but 12 of these drivers had not done so. Additionally, 8 of the drivers with type 2 diabetes indicated they were aware of the rules for reporting and said they had reported, however their licences showed that they were not restricted, suggesting that they had not in fact reported their insulin status. Seven participants said they were unsure or thought there was no requirement but 6 of these participants had in fact reported their condition to the authorities, irrespective of their belief. A total of 21 participants (10.4%) had not reported their condition (insulin treatment) to the DVLA. Only about one third (32.7%) of drivers said that they always have a glucose meter with them while driving and 38.1% said they never carry one while driving. Only 3% reported always testing blood glucose before driving and around 11% said they test around half the time before driving. The majority had a good knowledge of blood glucose levels for safe driving. However, almost 60% reported that they either never test or only if they experience symptoms of hypoglycaemia. Just under one-third of all drivers said they had experienced hypoglycaemia while driving and 7 participants reported a crash which they attributed to a hypoglycaemic episode. The

authors highlighted the high compliance with DVLA statutory reporting requirements but given low reporting level of blood glucose monitoring before driving, they emphasised the need for patient education in this area.

Stork, van Haefen and Veneman (2007) investigated drivers' decisions about not to drive during hypoglycaemia. Participants were drivers aged 16-65 years with type 1 diabetes with normal awareness of hypoglycaemia (n = 24), drivers with type 1 diabetes with impaired awareness (n = 21) and drivers with type 2 diabetes with normal awareness of hypoglycaemia (n = 20). Participants were part of a larger study examining driving performance on a driving simulator in which they completed two sessions of three drives, each lasting 8 minutes: the first driven at constant plasma glucose concentration of 5.0 mmol/l; the second session was at 2.7 mmol/l and after at least 60 minutes. The results showed that many drivers with type 1 diabetes with impaired hypoglycaemic awareness (43%) failed to decide not to drive during the induced hypoglycaemic episodes. The authors noted that their decisions may lead to dangerous situations while driving. Amongst the drivers with type 2 diabetes, despite normal awareness of hypoglycaemia, one-quarter made decisions to drive while positive or in doubt that they were hypoglycaemic. The authors speculated that this result might be due to less frequent experience with hypoglycaemia (compared with type 1 diabetics) or they may have received less information about hypoglycaemia and driving from their clinicians.

These findings highlight the need for health care professionals to discuss safe driving practices and appropriate self-regulatory strategies amongst drivers with Type 1 diabetes. In particular, Clarke et al. counsel drivers with Type 1 diabetes "to be aware of the danger of relying on perceived driving skill, previous driving experience and low BG level to remain safe behind the wheel" (1999, p. 753). The findings also highlight the fact that low blood sugar may be better tolerated by some people than by others. This may also explain why drivers are prepared to self-restrict their driving at certain blood glucose levels while others do not. Someone whose blood glucose level is always relatively high may be less tolerant of a lower level than the person who is always relatively low. Hence, while it may be reasonable to provide specific glucose levels as a guideline for drivers to know when to avoid driving, these should not be interpreted as absolute limits that are applicable to all drivers.

Table 17 Private licensing guidelines for drivers with diabetes mellitus

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Diabetes	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Diabetes controlled by diet alone	May drive if patient has no other disqualifying complication.	No licence restriction. Periodic review by GP recommended.	Not required to notify DVLA unless complications develop eg visual acuity & visual field problems or if insulin treatment becomes necessary.	<i>Condition is Mild & Stable:</i> No licence restrictions. Yearly review required.	Generally considered fit to drive	Licence denial for diabetes that is not sufficiently controlled. Applications considered in light of road safety risk from diabetic complications eg vision & CVA conditions. Reappraisals carried out on a case-by-case basis or discontinued if unnecessary.
Non-insulin treated diabetes	May drive if patient is not subject to hypoglycaemia and has none of the following complications: 1. eye disease 2. renal disease 3. neuropathy or 4. cardiovascular disease	No licence restriction if there are no complications. 5-yearly review required. Conditional licence may be issued if end organ complications & hypoglycaemic episodes are adequately treated. After the occurrence of a hypoglycaemic episode, person must refrain from driving for 6 weeks. If accident involvement occurs, DLA must be notified.	Licence retained until 70 years of age provided complications do not develop eg hypoglycaemic episodes, circulatory problems with feet and legs, visual acuity, visual field problems, patient does not begin insulin treatment or worsening of any other co-existing conditions.	<i>Condition is Mild & Stable:</i> No licence restrictions. Yearly review required.	Generally considered fit to drive.	Licence denial for diabetes that is not sufficiently controlled. Applications considered in light of road safety risk from diabetic complications eg vision & CVA conditions. Reappraisals carried out on a case-by-case basis or discontinued if unnecessary.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Diabetes	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
		Periodic review required.				
Insulin-treated diabetes	May drive if patient is not subject to hypoglycaemia and has no other disqualifying complications.	<p>Person may not hold an unconditional licence.</p> <p>Conditional licence may be issued if diabetes is stable & no defined hypoglycaemic episodes & person has ability to detect hypoglycaemia in order to stop driving vehicle & no end organ effects.</p> <p>Medical exam required every 2 years.</p>	<p>Licence issued if person can recognise onset of hypoglycaemic symptoms & meets visual test requirements.</p> <p>Licences may be granted for periods of 1, 2, or 3 years.</p>	<p>Unrestricted licence issued if condition is stable & no episodes of ketosis or altered states of consciousness for 6 months. Medical supervision & annual review required.</p> <p>A restricted licence is issued if episodes of ketosis or altered states of consciousness have occurred in the last 3 months. Speed & area restrictions apply.</p> <p>Medical supervision & 3- 6 monthly review required.</p>	<p>Generally considered fit to drive.</p> <p>Regular monitoring required.</p>	<p>Licence denial for diabetes that is not sufficiently controlled.</p> <p>Applications considered in light of road safety risk from diabetic complications eg vision & CVA conditions.</p> <p>Reappraisals carried out after 1 year & if the disease is well-controlled subsequent appraisals done at 3-yearly intervals.</p>

References

- American Diabetes Association (2003). Facts about Type 1 diabetes. <http://www.diabetes.org/>
- American Diabetes Association (2009). Diabetes Statistics. <http://www.diabetes.org/diabetes-basics/diabetes-statistics/>
- Amiel, S.A. (1999). Diabetes and driving – an insular approach? (Editorial). *Diabetic Medicine*, 16, 271-272.
- Australian Bureau of Statistics 2006a, National Health Survey: Summary of Results, Australia, 2004-05, cat. no.4364.0, ABS, Canberra.
- Austrroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. 3rd Ed. Sydney, Australia.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Cheyne, E., Sherwin, R., Lunt, M., Cavan, D., Thomas, P., & Kerr, D. (2004). Influence of alcohol on cognitive performance during mild hypoglycaemia; implications for Type 1 diabetes. *Diabetic Medicine*, 21(3), 230-237.
- Chlup, R., Neoral, L., Chlup, R., & Neoral, L. (2004). Methods for retrospective assessment of an unusual behavior of a person with diabetes: ebrietas alcoholica and/or hypoglycaemia? [Case Reports]. *Biomedical Papers of the Medical Faculty of Palacky University in Olomouc, Czech Republic*, 148(1), 39-43.
- Clarke, W.L., Cox, D.J., Gonder-Frederick, L.A., & Kovatchev, B.P. (1999). hypoglycaemia and the decision to drive a motor vehicle by persons with diabetes. *Journal of the American Medical Association*. 282(8): 750-754.
- Cox, D.J., Clarke, W., Gonder-Frederick, L.A., & Kovatchev, B.P. (2001). Driving mishaps and hypoglycaemia: risk and prevention. *International Journal of Clinical Practice, Supp 123*, 38-42.
- Cox, D.J., Gonder-Frederick, L.A., Kovatchev, B.P., Julian, D.M., & Clarke, W.L. (2000). Progressive hypoglycaemia's impact on driving simulation performance: Occurrence, awareness, and correction. *Diabetes Care*, 23(2), 163-170.
- Cox, D.J., Gonder-Frederick, L.A., Kovatchev, B.P., Julian, D.M., & Clarke, W.L. (2001). Self-treatment of hypoglycaemia while driving. *Diabetes Research and Clinical Practice*, 54, 17-26.
- Cox, D.J., Gonder-Frederick, L.A. & Clarke, W.L. (1993). Driving decrements in type I diabetes during moderate hypoglycaemia. *Diabetes*, 42(2), 239.
- Cox, D. J., Kovatchev, B., Vandecar, K., Gonder-Frederick, L., Ritterband, L., & Clarke, W. (2006). hypoglycaemia preceding fatal car collisions. *Diabetes Care*, 29(2), 467-468.

- Cox, D. J., Penberthy, J. K., Zrebiec, J., Weinger, K., Aikens, J. E., Frier, B., et al. (2003). Diabetes and driving mishaps: Frequency and correlations from a multinational survey. *Diabetes Care*, 26(8), 2329-2334.
- Deary, I.J. (1999). Symptoms of hypoglycaemia and effects on mental performance and emotions. In B.M. Frier and B.M. Fisher (Eds.) *hypoglycaemia and Clinical Diabetes*. Wiley: Chichester, UK, 29-54.
- Diabetes Australia (2002). Diabetes Facts. www.diabetesaustralia.com.au.
- Diamond, T. H., Collins, J., & Rohl, P. (2005). Motor vehicle accidents during episodes of hypoglycaemia: case reports and lessons to be learnt. *Australian Family Physician*, 34(3), 151-154.
- Distiller, L. & Kramer, B.D. (1996). Driving and diabetics on insulin therapy. *South African Medical Journal*, 86(8), 1018-1020.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Dunstan, D., Zimmet, P., Welborn, T., de Courten, M., Cameron, A., Sicree, R. et al. (2002). The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study. *Diabetes Care*, 25(5), 829-834.
- Eadington, D.W. & Frier, B.M. (1989). Type 1 diabetes and driving experience: an eight-year cohort study. *Diabetic Medicine*, 6, 137-141.
- Essex, N. (1994). When can a diabetic patient drive? *The Practitioner*, 238, 667-674.
- Flanagan, D.E., Watson, J., Everett, D., Cavan, D. & Kerr, D. (2000). Driving and insulin – consensus, conflict or confusion? *Diabetic Medicine*, 17, 316-320.
- Flanagan, D.E.H., Watson, J., Everett, J., Cavan, D. & Kerr, D. (2000). Driving and insulin, consensus, conflict and confusion? *Diabetic Medicine*, 17, 316-320.
- Frier, B.M. (2000). hypoglycaemia and driving performance. *Diabetes Care*, 23(2), 148-150
- Frier, B. M. (2007). How hypoglycaemia can affect the life of a person with diabetes. *Diabetes/Metabolism Research and Reviews*, 24(2), 87-92.
- Frier, B.M. (1992). Driving and diabetes: by not notifying the licensing authority many diabetics are breaking the law (Editorial). *British Medical Journal*, 305(6864), 1238-1239.
- Graveling, A., Warren, R., & Frier, B. (2004). Hypoglycaemia and driving in people with insulin treated diabetes: adherence to recommendations for avoidance. *Diabetic Medicine*, 21, 1014-1019.
- Hansotia & Broste (1991) The effect of epilepsy or diabetes mellitus on the risk of automobile accidents. *The New England Journal of Medicine*, 324(1), 22-26.

- Heller, S. (2006). *Stratifying hypoglycaemic event risk in insulin-treated diabetes*. Rep. No. 61. Department of Transport, London.
- Hemmelgarn, B., Levesque, L.E., Suissa, S. (2006). Anti-diabetic drug use and the risk of motor vehicle crash in the elderly. *Canadian Journal of Clinical Pharmacology*, 13(1), e112-e120.
- Hulbert M.F.G. & Vernon, S.A. (1992) Passing the DVLC field regulations following bilateral pan-retinal photocoagulation in diabetics. *Eye*, 6, 456-460.
- Jude, E.B., Ryan, B., O'Leary, B.M., Gibson, J.M. & Dodson, P.M. (1998). Pupillary dilatation and driving in diabetic patients. *Diabetic Medicine*, 15, 143-147.
- Koepsell, T., Wolf, M. & McCloskey, L. (1994). Medical conditions and motor vehicle collision injuries in older adults. *Journal of the American Geriatrics Society*, 42, 695-700.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Leproust, S., Lagarde, E., Suissa, S. & Salmi, L.R. (2007). Association between road vehicle collisions and recent medical contact in older drivers : a case-cross-over study. *Injury Prevention*, 13, 382-387.
- Lindgren, M., Eckert, B., Sterberg, G. & Agardh, C.D. (1996). Restitution of neurophysiological functions, performance, and subjective symptoms after moderate insulin-induced hypoglycaemia in non-diabetic men. *Diabetic Medicine*, 13, 218-225.
- Lonnen, K.F., Powell, R.J., Taylor, D., Shore, A.C. & MacLeod, K.M. (2008). Road traffic accidents and diabetes: insulin use does not determine risk. *Diabetic Medicine*, 25, 578-584.
- McGwin Jr, G., Pulley, L., Sims, R.V. & Roseman, J.M. (1999). Diabetes and automobile crashes in the elderly. A population-based case-control study. *Diabetes Care*, 22(2), 220-227.
- McGwin Jr, G., Sims, R.V., Pulley, L. & Roseman, J.M. (2000). Relations among Chronic medical conditions, medications and automobile crashes in the elderly: A population-based case-control study. *American Journal of Epidemiology*, 152 (5), 424-431.
- MacLeod, K.M. (1999). Diabetes and driving: towards equitable, evidence-based decision-making. *Diabetic Medicine*, 16, 282-290.
- Mathers, C.D., Stein, C., Ma Fat, D., Rao, C., Inoue, M., Tomijima, N, Bernard, C., Lopez, A.D. & Murray, C.J.L. (2002) Global burden of disease 2000: Version 2. Methods and results. Global Programme on Evidence for Health Policy Discussion Paper No. 50. World Health Organisation. Available at http://www3.who.ch/whosis/discussion_papers/pdf/paper50.pdf
- Rehnova, V, Weinberger, J. & Kotal, R. (2005). Effects of diabetes mellitus on driving performance and relation to fatigue and alcohol effect. Report D-R1.6

IMMORTAL project for the European Commission under the Transport RTD programme, 5th Framework Programme.

- Pearson, A.R., Tanner, V., Keightley, S.J. & Caswell, A.G. (1998). What effect does laser photocoagulation have on driving visual fields in diabetics? *Eye*, 12, 64-68.
- Piotrowski, C. (1997). hypoglycaemia as a mitigating factor in vehicular accidents. *Perceptual and Motor Skills*, 84, 1241-1242.
- Ratner, R.E. & Whitehouse, F.W. (1989). Motor vehicles, hypoglycaemia, and diabetic drivers. *Diabetes Care*, 12(3), 217-222.
- Salzberg, P. & Moffat, J. (1998). The Washington State Department of Licensing Special Exam Program: An Evaluation. Washington Traffic Safety Commission. Olympia, WA.
- Sagberg, F. (2006). Driver health and crash involvement: A case-control study. *Accident Analysis & Prevention*, 38, 28-34.
- Skurtveit, S., Strom, H., Skriverhaug, T., Morlund, J., Bramness, J.G., & Engeland, A. (2009). Road traffic accident risk in patients with diabetes mellitus receiving blood glucose-lowering drugs. Prospective follow-up study.
- Songer, T. (2002). Low blood sugar and motor vehicle crashes in persons with Type 1 diabetes. 46th Annual Proceedings of the Association for the Advancement of Automotive Medicine. Tempe, Arizona, Sept 30-Oct 2.
- Songer, T.J., LaPorte, R.E., Dorman, J.S., Orchard, T.J., Cruickshanks, K.J., Becker, D.J. & Drash, A.L. (1988). Motor vehicle accidents and IDDM. *Diabetes Care*, 11(9), 701-707.
- Soule, J. B., & Egede, L. E. (2007). Challenges of managing diabetes in commercial truck drivers. *Clin Diabetes*, 25(3), 110-111.
- Staplin, L., Loccoco, K.H., Stewart, J., & Decina, L.E. (1999). Safe Mobility for Older People Notebook. National Highway Traffic Safety Administration Model Driver Screening and Evaluation program. DOT HS 808 853.
- Stevens, A.B., Roberts, M., McKane, R, Atkinson, A.B., Bell, P.M. & Hayes, J.R. (1989). Motor vehicle driving among diabetics taking insulin and non-diabetics. *British Medical Journal*, 299, 591-595.
- Stork, A. D. M., van Haefen, T. W., & Veneman, T. F. (2007). The decision not to drive during hypoglycaemia in patients with Type 1 and Type 2 diabetes according to hypoglycaemia awareness. *Diabetes Care*, 30(11), 2822-2826.
- Swedish National Road Administration. (1998). Swedish National Road Administration Statute Book. Sweden.
- Taylor, J.F. (1985). Diabetes and driving ordinary motor vehicles. *Practical Diabetes International*, 2, 35-37.

- The Diabetes Control and Complications Trial Research Group. (1993). The effect of intensive insulin therapy on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New England Journal of Medicine*, 329, 997-986.
- Tregear, S., Rizzo M., et al. (2007) Diabetes and motor vehicle crashes: A systematic evidence-based review and meta-analysis. Proceedings of the Fourth International Driving Symposium on Human Factors in Driver Assessment Training and Vehicle Design., Stevenson, Washington, 9-12 July, 343 - 350
- Veneman, Th. F. (1996). Diabetes mellitus and traffic incidents. *The Netherlands Journal of Medicine*, 48, 24-28.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C. & Deane, J. M. (2001). Further analysis of drivers licensed with medical conditions in Utah. National Highway Transport Safety Authority (NHTSA) Technical Report No. DOT HS 809 211. (www.nhtsa.dot.gov/people/injury/olddrive/utah/Utah_index.htm.)
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J. M. (2002). Evaluating the crash and citation rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, 34, 237-246.
- Watson, W., Currie, T., Lemon, J., & Gold, A. (2007). Driving and insulin-treated diabetes: who knows the rules and recommendations? *Practical Diabetes International*, 24(4), 201-206.
- World Health Organisation [WHO]. (2002). Diabetes mellitus. Fact Sheet No 138. www.who.int/inf-fs/en/fact138.html
- World Health Organisation [WHO]. 2008. *The global burden of disease: 2004 update*. Geneva: Switzerland. World Health Organisation Press.

3.6 EPILEPSY AND SEIZURE DISORDERS

Definition of epilepsy and seizure disorders

Epilepsy is a chronic neurological condition, characterised by recurring seizures, which may result in unusual sensations, emotions and behaviour, muscle spasms, loss of consciousness and convulsions (Adams & Victor, 1989; Dobbs, 2001). Epilepsy (also referred to as a seizure disorder) is defined by two or more unprovoked seizures (World Health Organisation [WHO], 2001).

The term epilepsy encompasses a group of syndromes that vary in pathology and seizure type (Nair, 2003). Epileptic syndromes identified by the National Institute of Neurological Disorders and Stroke (NINDS, 2001) include absence epilepsy, psychomotor epilepsy, temporal lobe epilepsy, frontal lobe epilepsy, occipital lobe epilepsy and Parietal lobe epilepsy. These are described in more detail below.

Although the cause of epilepsy is unknown in approximately 75% of cases, risk factors include: vascular disease; stroke; head trauma; syncope; congenital or perinatal factors; central-nervous-system infections; and neoplasms (The National Centre for Disease Control [CDC], 2002). Provocative factors, however are recognised in some participants. For example, certain flashing lights (television, discos, video games etc), over breathing, over-hydration, loss of sleep, emotional and/or physical stress may stimulate seizures. Although these factors do not cause epilepsy, they may influence timing and frequency of seizures. Research has shown that the cause of epilepsy in older individuals is more likely to be caused by an underlying brain disease, such as a brain tumour or cerebrovascular disease, or as the result of a head injury (WHO, 2001).

Prevalence of epilepsy

The WHO estimates that the prevalence of epilepsy is approximately 37 million worldwide (Mathers et al., 2002). In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at 1.7 million or around 1% of the total population. Similarly, the prevalence of epilepsy in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 2.1 million or around 1% of the total population. In 2004 the estimated prevalence in Australia was 0.7% (ABS, 2006). While epilepsy can present itself at any age, the prevalence and incidence is highest in infancy or late adolescence, and the likelihood of developing epilepsy rises again after the age of 65 (NSE, 2003).

Functional impairments associated with epilepsy relevant to driving

The most significant functional impairment associated with epilepsy, results from the consequences of seizures (Andermann, Rémillard, Zifkin, Troffier & Drouin, 1988; Hansotia, 1993).

Seizures

Seizures result from excessive electrical neuronal discharges in the brain that cause a variety of clinical manifestations that may vary from the briefest lapse of attention or muscle jerks to severe and prolonged convulsions (WHO, 2001). Researchers have

identified more than 30 different types of seizures, which vary in frequency, from less than once a year to several times per day. Seizures are generally divided into two main categories – partial or focal seizures and generalised seizures, however there are many different types of seizures in each of these categories (NINDS, 2001).

Partial or focal seizures: Partial or focal seizures arise from an electrical discharge or one or more localised areas of the brain regardless of whether the seizure is secondarily generalised (WHO, 2001). Depending on their type, they may or may not impair consciousness. There are two types of partial seizures: simple or complex.

During simple partial seizures, the individual will generally remain conscious but may experience unusual feelings or sensations that can take many forms. For example, the individual may experience sudden and unexplainable feelings of joy, anger, sadness or nausea (NINDS, 2001). However during complex partial seizures, the individual generally has a change or loss of consciousness often producing a dreamlike experience. People having a complex partial seizure may display strange, repetitious behaviours such as blinks, twitches, mouth movements, or even walking in a circle. These repetitious movements are called automatisms. These seizures usually last for a few seconds (NINDS, 2001).

Individuals with partial seizures, especially complex partial seizures, may experience auras – unusual sensations that warn of an impending seizure. These auras are actually simple partial seizures in which the individual maintains consciousness (NINDS, 2001).

Generalised seizures: Generalised seizures are a result of abnormal neuronal activity in many parts of the brain. These seizures may cause loss of consciousness, falls, or massive muscle spasms (WHO, 2001). There are many kinds of generalised seizures:

- Absence seizures in which the individual may appear to be staring into space and/or have jerking or twitching muscles. These seizures are sometimes referred to as petit mal seizures;
- Tonic seizures cause stiffening of muscles of the body, generally those in the back, legs, and arms;
- Clonic seizures cause repeated jerking movements of muscles on both sides of the body;
- Myoclonic seizures cause jerks or twitches of the upper body, arms or legs;
- Atonic seizures cause a loss of normal muscle tone. The affected person will fall down or may nod his or her head involuntarily;
- Tonic-clonic seizures cause a mixture of symptoms which include stiffening of the body and repeated jerks of the arms and/or legs as well as a loss of consciousness. Tonic-clonic seizures are sometimes referred to by an older term: grand mal seizures (NINDS, 2001).

It should also be noted that not all seizures are easily defined as either partial or generalised. Some individuals have seizures that begin as partial seizures but then spread to the entire brain. Others may have both types of seizures but with no clear pattern (NINDS, 2001).

Seizures which cause loss of consciousness have obvious and critical implications for driving ability, and should therefore be of principal concern when determining fitness to drive (Krumholz, Fisher, Lesser, & Hauser, 1991). In approximately 80% of those diagnosed with epilepsy, seizures can be successfully controlled with anti-epileptic drugs (AEDs) and/or surgical techniques. However, about 20% of individuals diagnosed with epilepsy continue to experience seizures – even with the best available treatment. This situation has been described as intractable epilepsy (NINDS, 2001).

Surgery

When seizures cannot be adequately controlled by AEDs, physicians may recommend that the participant be evaluated for surgery. Epilepsy surgery can render about two thirds of participants seizure free (Dam, 1996). The most common form of surgery for epilepsy is removal of a seizure focus, or small area of the brain where seizures originate. This type of surgery, often referred to as *lobectomy*, is only appropriate for partial seizures. However, if an individual has been diagnosed with generalised seizures, surgeons may perform a procedure called *multiple subpial transection*, where a series of cuts are designed to prevent seizures from spreading into other parts of the brain while leaving the person's normal abilities intact. About 70% of participants who undergo multiple subpial transection have satisfactory improvement in seizure control.

Visual field defects are a recognised complication of epilepsy surgery, particularly in its most common form: temporal lobe surgery for hippocampal sclerosis (see Manji & Plant, 2000). "In particular, homonymous upper quadrant deficits may be caused by damage to Meyer's loop of the optic radiations as it sweeps around the temporal horn of the lateral ventricle." (Lawden, 2000, p 6). This complication has important implications for an individual's ability to drive (Lawden, 2000) (see also section 3.13).

Pre-May 2003: Relationship between epilepsy and road safety outcomes

Due to the potential for rapid incapacitation of the driver, and of the unpredictability of the epilepsy illness, several studies have investigated the possible link between epilepsy and crash risk (Dobbs, 2001). A number of authors have reviewed early studies on epilepsy and crash risk dating back from 1960 to the early 1980s (see Fisher, Parsonage, Beaussart, Bladin, Masland, Sonnen, & Rémillard, 1994; Dobbs, 2001). However, the findings of these early studies may not be relevant to current risk estimates because medical practices have changed continuously since then through improved technology, new diagnostic techniques and treatment methods, and better general management (Hansotia, 1994). Consequently, the review provided in this section will focus on studies that were conducted post 1980. Table shows a summary of the findings of studies that have investigated the relationship between epilepsy, AEDs and road safety outcomes.

Crashes

In 2001, Lings (2001) conducted a 10-year historical cohort study to determine the driving crash frequency in a cohort of drivers with epilepsy. Specifically, Lings compared the crash rates per 1,000 person years for 159 drivers diagnosed with epilepsy (ICD-8) with 559 controls individually matched for age, gender, place of residence, and exposure period. Participants were excluded from the study if they had no driving licence, or if they had been admitted to a hospital with one of the following diagnoses: cerebrovascular disease, diabetes mellitus, dementia, psychoses, or alcoholism. In this

study, exposure period was defined as the period of time, after the date of diagnosis, in which the individual held a driving licence. The outcome measure used in this study was treatment at the emergency department after a motor vehicle crash as a car driver. Lings reported that over the period of 1980 and 1989, 10 participants and five controls had been treated. For the epilepsy group there were: four between-vehicle crashes, four crashes with fixed objects and two crashes without a counterpart (one overturning and one driving into an excavation). In the control condition there were three between-vehicle crashes and two fixed-object crashes.

Lings (2001) reported that the crude crash rate in the epilepsy group was 0.063 (10/159) and was 0.0089 (5/559) in the control group, resulting in a crude rate ratio of 7.07. The relevant exposure in the epilepsy group was 1,063.72 years and in the control group 3727.44 years. Therefore the crash rate per 1,000 person-years in the epilepsy group was 9.4 (i.e., $[10/1063.72] \times 1000$) and 1.34 for the control group (i.e., $[5/3727.44] \times 1000$). Lings reported that the crash rate per 1,000-years of exposure was 7.01 times higher in drivers with epilepsy compared to the control cohort (i.e., $9.4/1.34$, CI 2.18-26.13, $p < 0.001$). Examination of the records from the neurology department revealed that all drivers with epilepsy who had sustained injuries were experiencing grand mal attacks which are characterised by stiffening of the body and repeated jerks of the arms and/or legs as well as a loss of consciousness. The time interval between the last recorded seizure and the crash ranged from 6 months (for one participant who had been forbidden to drive) to 12 years. Lings concluded that drivers with epilepsy were significantly more likely to be treated at the emergency department after having a motor vehicle crash.

Lings (2001) noted that crash frequency was calculated on the basis of years of holding a driving licence after diagnosis and not in relation to actual driving distance (mileage). Lings argued that this method was selected because the question of mileage is complex. For example, drivers with epilepsy may drive less than healthy drivers because of self-regulation or as a consequence of decreased employment activity, thereby producing fewer crashes than others even if their mileage crash risk were great. However, it is possible that individuals with epilepsy drive more than others, for instance to seek treatment. This would increase the difference between groups. Lings notes that the outcome measure, driver's treatment at the emergency department after a crash, must be considered insensitive because such events are rare, and the small numbers is a patent weakness. Furthermore, this method does not take into account minor crashes or injuries leading to hospitalisation by other road users or passengers, nor does it take into account crashes that only involve material damage. Lings concluded that the seven-fold magnitude of increased risk was surprising, however suggests that previous studies had not adequately excluded participants with other neurologic diseases or addiction, but due to the small sample size, drastic consequences regarding driving regulations should be avoided until these results have been substantiated by further investigations.

Vernon, Diller, Cook, Reading, Suruda and Deane (2002) conducted a retrospective case control study and analysed crash rates (all crashes and at-fault crashes) and citation rates (see next section for information regarding citations) for 3,395 drivers with epilepsy or related episodic conditions (including syncope, cataplexy, narcolepsy, hypoglycaemia, and episodic vertigo). The authors argued that epilepsy includes any recurrent loss of consciousness or conscious control arising from intermittent changes in brain function and because of the similarity of consequences, other disorders affecting consciousness or control such as syncope have been included in this section.

Participants were also classified according to their licence status (restricted/no restrictions), with the majority of participants having no restrictions (n = 2620). However, Vernon et al. (2002) noted that drivers with unrestricted licences or restricted licences are not mutually exclusive, with approximately 27.5% (n = 745) of the drivers in this category fluctuating between restricted and non-restricted licensing privileges during the study period. In addition, drivers with epilepsy and other episodic conditions with no licence restriction (i.e., the lowest level of impairment) had significantly higher crash rates and at-fault crashes (RR: 1.81 and 2.11 respectively) than the general population drivers. Similarly, drivers with epilepsy and other episodic conditions with restricted licences (i.e., the highest level of impairment) had significantly higher crash rates and at-fault crashes (RR: 1.55 and 2.47 respectively) than the general population drivers. Vernon et al. (2002) concluded that drivers with epilepsy (both those with restrictions and those without restrictions) have a higher risk of crashing than the general population of drivers. One of the main limitations of this study was that the authors did not control for driver exposure, which assumes that drivers in the epilepsy group and matched controls drive similar distances. However, as noted by Lings (2001), it is reasonable to assume that medical conditions may influence driving distances. It should also be noted that the epilepsy group comprised drivers with other conditions such as syncope, cataplexy, narcolepsy, hypoglycaemia and therefore the elevated crash rates associated with this group may reflect the crash risks of other conditions.

Krauss Krumholz, Carter, Li and Kaplan. (1999) conducted a retrospective case-control study to determine possible risk factors for motor vehicle crashes due to seizures. Specifically, the authors compared 50 drivers with epilepsy who had a motor vehicle crash which could be attributed to a seizure (cases) with 50 drivers with epilepsy who have not had a motor vehicle crash which could be attributed to a seizure (controls). Case and control participants were recruited from the same epilepsy clinic and were matched on: having epilepsy (i.e., two or more seizures), gender and age. Participants were excluded if their epilepsy was in remission due to AED treatment during the study year or if they had had epilepsy surgery during the study year. Participants were also excluded if they crashed during their first seizure because the authors would be unable to collect clinical information regarding AED compliance, seizure-free intervals and number of seizure related crashes. The following clinical characteristics and driving histories of case and control participants were collected using a self-report questionnaire: demographic data; seizure information; treatment factors; driving history; crash variables; and regulatory factors.

The authors reported that the following factors were most strongly associated with *reduced* odds for crashing. Firstly, long seizure intervals (12 months or longer and 6 months or longer) were associated with reduced risk for seizure related crashes (OR: 0.075; CI 0.012 – 0.47; OR: 0.147, CI 0.031-0.691, respectively). Secondly, having reliable auras (i.e., where drivers reported always having auras at the start of seizures) also reduced the odds of having a seizure related crash (OR: 0.077). The authors noted that some drivers crashed despite auras, either because they continued to drive after the aura or because they were unable to stop driving before the seizure progressed because their auras were too brief. The authors were surprised to find that switching or reducing drivers' AED significantly reduced, rather than increased, the odds of crashing (OR: 0.111). The authors suggest that this finding could be due to drivers' having fewer seizures when their AEDs are consolidated (reduced from several to one) or switched. Finally, drivers who have had few prior crashes not related to seizures had significantly reduced odds of having a crash (OR: 0.465). Other findings noted by the authors were

that 25% of drivers had more than one seizure-related crash, 20% had just missed an AED dose prior to their crash, 4.6 times as many men with seizure related crashes compared to women, and that 54% of drivers who crashed were driving illegally with seizure free intervals shorter than legally permitted. The authors concluded that seizure free intervals, the presence of reliable auras, AED therapy modifications, and a history of non-seizure induced crashes should be considered when advising drivers with epilepsy about driving.

Taylor, Chadwick and Johnson (1996) attempted to estimate the risks of motor vehicle crashes over a three-year period in drivers with a history of single seizures or epilepsy and compare them with the risks in a cohort of drivers from the general population. Participants included 16,958 drivers with a history of single seizures or epilepsy and 8,888 non-epileptic drivers who all responded to a questionnaire. Drivers were asked to complete questions regarding demographics details, information about their driving history and if they had been involved in a crash as a driver over the previous three years. Drivers with epilepsy were also asked to complete questions regarding the history of their seizures, information about their prescribed medications and whether their seizures had ever resulted in a crash. Taylor et al. (1996) reported that after adjustments were made for age, sex, driving experience, and mileage between the two populations, there was no evidence of an overall increase in risk for drivers with epilepsy (OR: 0.95, CI 0.88-1.02). However, the authors noted that there was an increased risk of more severe crashes for drivers with epilepsy (OR: 1.37, CI 1.01-1.76, $\chi^2 = 4.3$, $p < 0.05$); furthermore there was evidence of a two-fold risk of increased driver fatalities.

Another interesting finding noted by Taylor et al. (1996) was that taking AEDs does not increase the risks of any form of crash in a population of drivers with a history of epilepsy (OR: 0.97, CI 0.87-1.07). Taylor et al. (1996) also reported that the absence of seizures over a three year period seems to halve the risk of serious injury or fatal crashes (OR: 0.56, CI 0.32-0.96). Taylor et al. concluded that the crash rates for individuals with epilepsy are no greater than the general population after adjusting for age, gender, driving experience and mileage. One limitation of this study is that the authors combined participants who had only had single seizures with those who had a history or diagnosis of epilepsy. As noted previously, epilepsy is only diagnosed after two or more seizures, therefore the non-significant findings may be due to the fact that participants in the epilepsy group did not actually have a diagnosis of epilepsy. In addition, although the authors made adjustments for important factors such as age, gender and driver exposure, they did not specify whether participants in either group were screened for other comorbid medical conditions.

Hansotia and Broste (1991) conducted a retrospective cohort study to assess the effect of epilepsy and diabetes mellitus on motor vehicle crashes (see section 3.5 for the results regarding diabetes mellitus). Specifically the authors studied the crash and citation rates (mishap ratios [MR] per 1000 person-years of licensed driving standardised for age) over a 4-year period (1985-1988) among 30,420 drivers (see next section for further information on citations). Participants were drivers aged 16-90 who had been recruited from the city and surrounding areas of Marshfield, Wisconsin. A total of 434 drivers with epilepsy were identified through the use of computerised ICD-9-CM diagnostic codes for epilepsy (345 to 345.9). Controls were active drivers who had no diagnostic code of epilepsy. The authors noted that participants with epilepsy had numerous other medical conditions including strokes, dementia, clinical depression and other psychiatric disorders (however the prevalence of these comorbid conditions

was not reported). Overall the study found significantly higher MRs for drivers with epilepsy for crashes (MR = 1.33, $p < 0.05$).

These findings should be interpreted cautiously because mishap ratios were not adjusted for exposure, nor were they adjusted for other important factors such as comorbid conditions, years since disease onset, disease severity, or disease treatment type. Furthermore, there are several potential sampling biases in this study. Firstly, the sample was recruited from a limited geographical area in Wisconsin and the authors made no attempt to determine whether the sample was an adequate representation of the population of all drivers in the US and/or other countries. Secondly, as pointed out by Earnest (1991), participants with epilepsy in this group were a highly selected group, in that 55% of drivers had not had a seizure during the study period. Thirdly, the authors did not specify the medical status of the control group, other than being identified as not having epilepsy. Consequently, participants in the control group may have other medical conditions which could be affecting their driving ability. Hansotia and Broste (1991) concluded that there was a slightly higher risk for crashes for drivers with epilepsy, however given the relatively small size of the population at risk, there was insufficient evidence to warrant further restrictions to driving privileges.

In 1988, Popkin and Waller (1988) examined the driving records of 112 drivers using six North Carolina Division of Health Services' clinics for the treatment of epilepsy during 1981–1982. Of those undergoing treatment in the clinic, 29 (26 %) were known to the DMV to have epilepsy. The group of epileptic drivers known to the DMV had a crash rate 1.4 times higher than the general population, where the crash rate for drivers with epilepsy who were not known to the DMV was 1.1 times the general population rate. While the group of epileptic drivers known to the DMV had a slightly higher crash rate than the group of epileptic drivers not known to DMV, differences within this small sample were not statistically significant. The authors also noted that because the participants were selected by virtue of being treated through local health departments, the results may not be representative of the entire population of drivers with epilepsy. Other methodological limitations of this study are that there is no information regarding driving exposure, medication use and stabilisation of condition, length of time since onset.

In 1987, Gastaut and Zifkin (1987) attempted to determine the risk of motor vehicle crashes posed by various seizure types when they occur during driving. 400 drivers with epilepsy were approached to participate in the study. Drivers were included in the study if they had a well-classified diagnosis of epilepsy and if they or one of their passengers could provide a good description of seizures that had occurred at the wheel. Of 400 drivers with epilepsy, 133 admitted having had one or more seizures at the wheel (33%). However, of the 133 drivers with seizures at the wheel, only 97 were able to describe or have a witness describe one or more of these attacks, and only 82 participants could be clearly classified. Of the 82 drivers, 64 had had one such seizure at the wheel (78%), 13 drivers had had two such seizures at the wheel (16%) and five drivers had had three to five seizures (6%). Thus the authors were able to identify 109 seizures at the wheel. Of the 109 seizures identified, 60 (55%) led to a crash. Of these 60 seizures, 4 (7%) were due to primary generalised epilepsy, 3 (5%) were due to generalised tonic clonic convulsions and 1 (2%) was due to prolonged absence. Three (5%) were simple partial seizures with no change in consciousness but with loss of motor control, one progressing to a generalised convulsion. 53 seizures (88%) were complex partial seizures, where 42 (72%) of these began with an initial alteration of

consciousness, and 11 with an aura. These 60 seizures leading to crashes were responsible for injury in 13 (22%) cases including 2 fatalities. In 20 cases there was a collision with another vehicle and in 30 cases there was a collision with another obstacle. The authors noted that although other seizures were not associated with crashes, this may be attributed to chance location and timing because if any of these had occurred on a busy street etc, then crashes would have almost surely have resulted. Gestaut and Zifkin (1987) concluded that seizures occurring while driving are very likely to lead to crashes unless the circumstances are fortunate and that complex partial seizures without aura, secondarily generalised seizures and generalised tonic clonic seizures are the types most implicated in crashes, whereas simple partial seizures, complex partial seizures with aura and absence seizures are less frequent, and myoclonic are rarely implicated. One of the main methodological limitations of this study is that it relies on self-report, and therefore the estimations of the crash rates may be underestimations given the fear of having the licence revoked in this population (Andermann, et al., 1988; Dobbs, 2001).

Citations

As outlined above, Vernon et al. (2002) conducted a retrospective case control study and analysed the citation rates for 3395 drivers with epilepsy or related episodic conditions (including syncope, cataplexy, narcolepsy, hypoglycaemia, and episodic vertigo). Unlike crash rates, the rate of violations for drivers with epilepsy was not significantly different than the general population comparison group.

In contrast, Hansotia and Broste (1991) found that while there was no evidence of higher overall citation rates (MR = 1.13, CI = 0.90-1.41, $p = 0.26$), drivers with epilepsy were more likely than drivers from the control group to commit careless driving citations (MR = 1.57, CI 1.05-2.25, $p < 0.05$) or to have alcohol or drugs citations (MR = 2.75, CI = 1.50-4.62, $p < 0.001$). However, these findings should be interpreted cautiously because these mishap ratios have not been adjusted for exposure, nor have they been adjusted for other important factors such as co-morbid conditions, years since disease onset, disease severity, or disease treatment type.

Driving Performance

No studies investigating epilepsy and driving simulator or real-world driving performance were found.

Treatment for epilepsy and road safety outcomes

The first-line treatment of epilepsy is administration of an antiepileptic drug (AED) (NINDS, 2001; Nair, 2003). While drug therapy has made remarkable progress in the treatment of epilepsy, no single drug is able to control all types of seizures, and many drugs carry undesirable side effects including: ataxia, blurred vision, confusion, day blindness, diplopia, dizziness, and drowsiness, and until tolerance develops, any of these side effects could impair driving skills (Novak et al., 1991; Popkin & Waller, 1989). It should be noted that some of the side effects affecting the central nervous system, such as drowsiness and dizziness, may be more apparent in the early days of taking the medication while the body is adjusting to the medication, and then these should lessen or disappear completely (Epilepsy Action, 2003). It should also be noted that, as with any medication, there is the potential for severe and life-threatening side

effects such as death due to aplastic anaemia, Stevens-Johnson syndrome or hepatotoxicity (Haslam & Koren, 1989; NINDS, 2001).

For most individuals with epilepsy, seizures are generally well controlled with just one AED at the optimum dosage (monotherapy). However, combinations of drugs are sometimes prescribed if monotherapy fails to effectively control a participant's seizures or if a physician is attempting to effect a "switch" in AED treatments (NINDS, 2001; Reubens, 2002). In participants with epilepsy, the issue of polypharmacy is particularly pertinent regarding the effects on cognitive ability. For example, while the cognitive effects of individual drugs have been evaluated, the effects of multiple-prescriptions of AED are in most cases unknown, and therefore physicians are limited in the information they can provide. Therefore physicians usually prescribe monotherapy whenever possible (Novak et al., 1991).

Regular monitoring of blood levels of AED, preferentially prescribing a nonsedating AED, and treating with a single AED whenever possible is highly recommended (Novak et al., 1991). These recommendations are especially important for older individuals who tend to become more sensitive to medications as they age (NINDS, 2001).

Recent studies in both developed and developing countries have shown that up to 70% of newly diagnosed children and adults with epilepsy can be successfully treated with AEDs. Furthermore, after 2-5 years of successful treatment, AED can be withdrawn in approximately 70% of children and 60% of adults without relapses.

However, while recent pharmacological advances have resulted in improved medications for controlling seizures, approximately 20% of participants with primary generalised epilepsy and 35% of participants with focal epilepsy have medically intractable seizures (Dobbs, 2001).

Crashes

The potential for anti-epileptic drugs (AEDs) to impair driving ability has not received much attention in the medical literature (Novak, Krumholz, Fisher, Lesser & Hauser, 1991). Taylor et al. (1996) reported that taking AEDs did not appear to increase the risks of any form of crash in a population of drivers with single seizures or a history of epilepsy (OR: 0.97, CI 0.87-1.07). In contrast, Krauss et al. (1999) (reviewed above) reported that switching or reducing drivers' AED significantly *reduced*, rather than increased, the odds of drivers with epilepsy crashing (OR: 0.111). The authors suggested that this finding could be due to drivers having fewer seizures when their AEDs are consolidated (reduced from several to one) or switched.

While there is a general consensus that AED at therapeutic doses would be unlikely to pose serious hazards for driving (Novak et al., 1991), no studies have specifically addressed the *overall* risk of a crash in drivers on AEDs. Future research in this area needs to take into account the issue of non-compliance, missed doses, gastric upsets etc which will affect the efficacy of a given therapy.

Post-May 2004: Relationship between epilepsy and road safety outcomes

The review provided in this section includes studies investigating the relationship between road safety outcomes and epilepsy and epilepsy medications since 2003. Table

18 summarises the findings of studies in the pre-May 2003 and post-May 2003 review periods.

Crashes

The impact of mandatory reporting on drivers with epilepsy was investigated by McLachlan, Starreveld and Lee, (2007) using a retrospective case-control study design. Drivers were recruited from Ontario, Canada - a jurisdiction with mandatory reporting - and Alberta, Canada which does not have mandatory reporting. Surveys were distributed to 500 adults with known epilepsy and 500 controls without epilepsy. A response rate of 80% was reported. The epilepsy population (n=425) was recruited from files of epilepsy clinics. The controls (n=375) were age matched (within five years) friends or relatives who lived in the same area. All of the participants were over the age of 16 which is the legal driving age. Patients with a physical impairment, psychiatric illness or mental impairment were excluded from the study. Seventy-three percent of participants with epilepsy had been, or were licensed drivers, compared to 94% of the controls. The following information was collected via a survey; demographic, driving history, life time crash history, accident history within the last year, employment status, and seizure activity. The authors found no difference in the number of crashes for the previous year between cases and controls irrespective of the different jurisdiction laws. A crash rate of 9% was reported for both groups in the previous year (OR 1.00, 95%CI, 0.95 - 1.06). The authors compared these rates with a general population crash rate of 6-8%. Overall the number of crashes experienced in a lifetime was higher for drivers with epilepsy aged over 45 years (62%), compared with patients aged less than 30 years (48%). Males without epilepsy had a higher lifetime accident risk (65%) compared to females with epilepsy (36%) (RR: 1.82, 95%CI 1.48 - 2.24). A limitation of the study is that data were obtained using self-report which means that the number of true crashes reported may have been lower than the actual number. Secondly, the number of years spent driving, and the average driving distances were not taken in to account in relation to accidents which reduces the credibility of the study.

Sheth, Krauss, Krumholz and Li (2004) investigated the fatal crash risk associated with seizures in drivers with epilepsy compared to other medical conditions and non-medical crashes using a cross sectional study design. The authors obtained 44 027 death certificates of people who died in the US from a MVC between 1995 and 1997. Participants either suffered from epilepsy and had a seizure related or non seizure related crash, or were diagnosed with another medical condition. The ICD-9 classification codes were used to classify the cause of death. Disease specific crash rates were calculated from the number of fatalities associated with each disorder compared to the annual prevalence rates. Only participants aged 18 years and above were included in the general prevalence rates. The relative risk of fatalities was determined according to each of four categories: seizure activity within 3 months, 6 months or 12 months. A proportionate mortality ratio (PMR) was determined for patients with epilepsy or convulsions. This calculates the proportion of people who died in a seizure related crash compared to the expected ratio from the general population. The authors reported that from the years 1995 to 1997, an average of 44 027 people died annually in fatal crashes, and only 86 (0.2%) were related to seizures. Of the 86 people involved in seizure related crashes, the majority were aged between 35-44 years. Rates for fatal seizure-related crashes in people with epilepsy were lower (8.6 per 1000 000) than rates for fatal crashes in the general population (22.4 per 100 000). The relative risk of fatal crashes for patients with seizures was 2.3 higher than for people with cardiovascular disease,

and 4.6 higher than people with diabetes. The authors also compared whether or not the number of seizure-related fatalities differed according to driving laws. No differences were found in the number of fatalities between states with laws about returning to driving after seizure activity in the last 3, 6 or 12 months. Two serious methodological limitations of this study is that there was no information regarding driving exposure and only fatal crashes were considered.

Citations

There were no studies that reported on the relationship between drivers with epilepsy and citations.

Driving Performance

No studies were found that assessed the driving performance of patients with epilepsy using an on road test vehicle or a driving simulator.

Treatment for epilepsy and road safety outcomes

Crashes

Faught, Duh, Weiner, Guérin and Cunnington, (2008) conducted a retrospective open cohort study in order to determine whether non-adherence to AED's was related to motor vehicle crashes in drivers with epilepsy. Medical claims data recorded between June 2006 and January 1997 were obtained for Florida, Iowa, and New Jersey. Participants were aged 18 years and above, had a diagnosis of epilepsy from a neurologist (ICD-9) or non-febrile convulsions, were prescribed AED's at least once, and recorded a period of six months or more of AED taking. The final sample consisted of 33 658 patients. The medication periods were obtained from medical and dispensing claims from Medicare/Medicaid. Adherence behaviour of AED taking was assessed using a medication possession ratio (MPR) which was calculated according to the number of days the drug was prescribed/number of days in a quarter. Therefore, each person had multiple MPR's over a typical five year period. The incidence of emergency department visits, hospital admissions, MVC injuries and fractures was investigated in relation to adherent and non adherent behaviour.

Sixteen percent of the total sample died during the data collection period, and the majority of people who died did so during a non-adherence quarter. Non adherent behaviour was associated with a five fold increase of mortality compared to adherence behaviour (adjusted multivariate hazard ratio was 4.96, (CI = 4.66-5.27)). Furthermore, the researchers found that the non-adherence quarters had a higher incidence rate of MVC injuries (incidence rate = 0.011) compared to adherence quarters (incidence rate = 0.005). Note the non-adherent/adherent IRR was 2.08 and the difference remained significant once age, medical co-morbidities and gender were taken in to account. A limitation of this study is that the researchers did not know the cause of death, only the outcome. Therefore a serious limitation of the study is that a number of deaths experienced by patients with epilepsy may not necessarily have been related to the disease. The authors noted that measurement of adherence status is not without its limitations. For example the researchers could not be certain that the patients took the drugs for the full 30 day period once they have been dispensed, therefore adherence may have been overestimated in the current study. A further limitation of the study is

that people who are covered by Medicaid are often of a lower socio economic status, which would lower the external validity of the study.

In the same year, Hovinga and colleagues (2008) investigated the factors associated with non-adherence to AED's. The cross sectional survey study included 408 epilepsy patients and 175 physicians. Patients were eligible to participate if they were US residents or citizens, aged between 18-64 years, had a self-reported diagnosis of epilepsy or seizure disorder, and were currently taking an AED. The physicians were eligible if they were a neurologist or epileptologist, practised for at least two years after residency, treated at least one patient with epilepsy or seizure disorder, and spend 50% of their time in direct patient care. The surveys were completed online and the patient survey included questions on demographics, seizure type, seizure history, AED medication, AED adherence (in the last week, month, and 3 months), productivity and work functioning, quality of life and cognitive functioning. In contrast, the physician survey obtained information regarding physician views on the physician-patient relationship, reasons for non-adherence and current prescribing behaviours. Of the 408 epilepsy patients, 29% were classified as non-adherent. Non-adherent patients were more likely to miss or stop going to work due to seizure related loss of driving ability (OR: 1.76, $p = .02$, 95%CI 1.10 - 2.82), and to experience an MVC due to a seizure (OR: 2.18, $p = 0.006$, 95%CI 1.25 - 3.81) compared to adherent patients. After adjusting for confounding variables (depression, employment status, seizure frequency, and number of seizure drugs) the non-adherent patients were still more likely to experience an MVC due to a seizure (OR: 1.92, $p = 0.03$, 95%CI 1.07 - 3.43). Patients and physicians identified forgetfulness and not having the medication on hand as the main reasons for non adherence. One major limitation of the study is that it relies on self-report, and therefore there was no confirmation of the epilepsy diagnoses using medical records. In addition, AED adherence rates may be overestimated. The authors noted that because the surveys were administered online the sample may have been bias towards people who use the internet, who may have different attitudes than the general population and tend to be of a higher education level and younger generation (Lusk et al. 2007). Overall, the findings from the study suggest that drivers with AED non-adherent behaviour are at a greater risk for seizure related motor vehicle crashes.

Citations

No studies investigating the relationship between treatment of epilepsy and citations were identified in the post-May 2003 review period.

Driving Performance

Mills and colleagues (2008) investigated the ability of patients with epilepsy to perform a cognitive task that has been correlated with driving performance and crash risk. Anti-epileptic drugs are often associated with unpleasant side effects including cognitive impairment. There is evidence to suggest that while some drugs (such as topiramate) can have an adverse effect on cognitive functioning, the side effects of other drugs (such as lamotrigine) are negligible. The aim of the randomised double blind study conducted by Mills et al. (2008) was to compare the effects of the anti epileptic drug lamotrigine (LTG) with topiramate (TPM) in a group of adults with partial seizure epilepsy. The study consisted of sixty-seven patients with seizure activity within the last 3 months, all of whom had a diagnosis of partial epilepsy within the last 6 months. Participants were eligible if they were receiving carbamazepine or phenytoin as a monotherapy combined with 1 additional AED. The LTG group consisted of 35 participants (17-88 years),

while the TPM group consisted of 32 participants (19-68 years). The mean duration of exposure to the TPG was 104.5 days, compared with 86.2 days for the LTG group. The study began with a two week screening phase, followed by an 8 week dose escalation phase followed by an 8 week double blind phase during which doses of medication were maintained. Cognitive tests were conducted three times; once in the screening phase, once at the end of the dose escalation phase and once at the end of the double blind phase. The battery of tests included; the Controlled Oral Association Word task, the Stoop task, Digit Cancellation, Lafayette Grooved Pegboard, Rey Auditory Verbal Learning Test, the Symbol Digit Modalities Test and the Performance On-Line test (POL). The POL is a version of the UFOV that has been shown to correlate well with driving performance (Mills et al. 1999). It is a computer task that assessed scanning, divided attention, and effective field of view. The field of view can become restricted by pharmacological agents (Mills et al. 1996). This restriction results in a ‘tunnel effect’ which places the driver at an increase risk for driving errors.

After eight weeks the LTG group had a significantly higher score on the POL compared to the TPM group ($p = 0.021$), indicating that the TMG group performance was significantly compromised. Mills and colleagues (2008) suggested that the decline in POL performance of the TMG group could be attributed to a restricted field of view, however, this finding cannot be translated directly to impaired driving performance per se. A major limitation of the study concerns the lack of a placebo group. Furthermore, patients were on adjunctive treatment, and therefore the solitary effects of TPM and LTG could not be determined. Although the researchers controlled for seizure frequency, seizure onset and age, they did not assess visual ability or record the level of education.

Summary

Wide differences in methodologies make it difficult to compare findings across studies on this topic. Several studies reported an elevated risk of crashing among drivers with epilepsy, however it should be noted that the size of the risk varied considerably across the studies. For example, the majority of studies reported that individuals with epilepsy are twice as likely to be involved in a motor vehicle crash compared to the general driving population (e.g. Vernon et al, 2002); one study reported that drivers with epilepsy were seven times more likely (Lings et al, 2001). One study reported higher risks associated with serious injury crashes and fatal crashes (Taylor et al., 1996). In contrast the two studies reviewed post-May 2003 provided no evidence for elevated risk associated with epilepsy. However, Sheth et al. (2004) analysed seizure related *fatal* crashes only and reported very low rates of crash involvement: only 0.2% of a sample of 44 027 drivers died from a seizure-related crash. McLachlan et al. (2007) found no difference between drivers with epilepsy and non-impaired drivers in self-reported crash rates.

Other studies published since 2003 focused on the relationship between AED adherence of drivers with epilepsy and the associated crash risk. The findings suggest that drivers who engage in non-adherent AED taking behaviour are at an increased risk for experiencing a motor vehicle crash. For example, Faught et al. (2008) reported that the IRR of experiencing an MVC is 2.08 for non-adherent drivers compared to adherent drivers. The data for this study was obtained from medical dispensing records in the US. In addition, the study by Hovinga et al. (2000) reported that the risk of epilepsy related crashes increases during non-adherent drug taking periods compared to adherent periods

(OR:1.76). This study relied on self-report data of crashes and adherence which most likely led to an overestimate of adherence, and an underestimate of MVC's.

There is a considerable lack of disagreement about the effectiveness of mandatory reporting laws pertaining to drivers with epilepsy. The study by McLachlan and colleagues (2007) investigated whether the crash risk differed according to the jurisdiction laws in Alberta versus Ontario. No differences in crash risk were found between the states which differ in terms of requirements for mandatory medical reporting. Similarly, Drazowski and colleagues (2003) found the number of seizure related crashes did not significantly increase once the law in Arizona was changed from seizure-free driving period of 12 months to three months. However, there is agreement that individuals with frequent seizures (short intervals) should not drive, and individuals with long intervals between their seizures can be considered capable of driving safely (Fisher et al., 1994).

Table 18 Summary of studies of risk associated with epilepsy

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Faught et al. (2008)	Retrospective open cohort study Cases = 33 658 people, ≥18 yrs old, ≥ 1 AED, epilepsy diagnosis ICD-9 AED adherence status was calculated separately for each quarter of a patient's observation period	1) AED adherence vs non adherence 2) Incidence of emergency department visits, hospital admission, MVA injuries, fractures and mortality	Non-adherence quarters had a higher incidence rate of MVA injuries (incidence rate = 0.011) compared to adherence quarters (incidence rate = 0.005)
Gestaut & Zifin (1987)	Cases = 82 drivers, ep clearly classified, 1 or more seizures at the wheel	Self report i) seizures while driving 3) crashes as a result of seizures	- Acc occurred w seizures in 55% -complex partial seizures occurred in seizures responsible for 88% of acc
Hansotia & Broste (1991)	Pop retrospective cohort study Cases = 241 with ICD-9 diag of ep Cases = 30,420 licence holders during a 4 year period.	Outcome measures: i) Self-reported crash rates ii) Self-reported citation rates - Mishap Ratios (MR)	MR Acc Inv: 1.33* MR Citation: 1.13
Hovinga et al. (2008)	Cross-Sectional (patients/physicians): Patients = 408, self-rep. diag. of epilepsy, currently taking an AED Physicians = 175, practiced for > 2 yrs, >50% time direct patient care	Survey Patients: demographics, seizure type, seizure history, AED medication, AED adherence, productivity and work functioning, quality of life and cognitive functioning Physicians: physician views on the physician-patient relationship, reasons for non adherence and current prescribing behaviours	Non adherent patients were still more likely to experience an MVA due to a seizure compared to adherent patients (OR: 1.92, $p = 0.03$, CI: 1.07-3.43)

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Krauss et al. (1999)	Retrospective case-control Cases = 50 ep p with seizure related crash Control = 50 ep p without seizure related crash	Self-report: i) seizure related crashes ii) seizure information iii) driving history regulatory factors	Factors reducing odds for crashing: ≥ 12 mon sz free ≥ 6 mon sz free ≥ 3 mon sz free reliable auras AEDs changed Few prior crashes
Lings (2001)	10 year historical cohort register study Cases = 159 Controls = 559 Cases = person with no history of other neur diseases, diab, psych, abuse or poisoning	Acc rate per 1000 person years	Acc/1000 person yrs: Ep > C **
Mills et al. (2008)	Randomised double blind study Patients = 67, > 18 yrs old	Medication - Lamotrigine (LTG) - Topirimate (TPM) Battery of cognitive tests including the Performance On Line test	<i>M</i> score for POL performance after 8 weeks was significantly better for the LTG group compared to the TPM group (<i>p</i> = 0.021)
McLachlan et al. (2007)	Retrospective survey case/control study Cases = 202 Ontario, 223 Alberta Controls = 200 Ontario, 175 Alberta	Jurisdiction: Ontario = mandatory reporting Albert = no mandatory reporting Survey - acc past year, acc in lifetime - seizure information	Crash rates: Cases=controls (9%) (OR 1.00, 95%CI, 0.95 - 1.06). Lifetime crash rates were not significantly different between states (OR 0.99, 95%CI, 0.67-1.47), nor were one year crash rates (rr = 1.38, 95%CI, 0.59-3.27).

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Popkin & Waller (1988)	Cases = 112 ep drivers - 29 known to DMV - 83 unknown to DMV	Driving records	Crash rate: Ep > gen pop* Crash rates: Ep known to DMV = Ep not known to DMV
Sheth et al. (2004)	Cross-Sectional 44 027 mortality data files from 1995-1997 were obtained ICD-9 epilepsy vs other medical conditions	Fatal crash rate for each medical condition Seizure related fatal crashes Relative risks based on population crash rates	0.2% of fatal crashes were related to seizures RR of a fatal crash for epilepsy patients was 2.3 higher than for people with cardiovascular disease, and 4.6 higher than people with diabetes.
Taylor et al. (1996)	Cases n = 16958 Control n = 8888 Cases = single seizures or epilepsy	Survey: - acc in past 3 yrs - acc with injury in past 3 yrs I. - acc with serious injury in past 3 yrs	OR Acc Inv: 0.95 OR Acc with injury: 1.08 OR Acc serious injury: 1.33*
Vernon et al. (2002)	Pop/case-control; Cases =3395 Control =20,210 'Cases' = epilepsy, syncope, cataplexy, narcolepsy, hypoglycaemia, episodic vertigo;	(i) Crash -all (ii) At-fault crash (iii) Citation Rates per 10,000 lic days	Not Restricted RR all crashes: 1.81* RR at-fault crashes: 2.11* RR citations: 1.03 Restricted RR all crashes 1.55* RR at-fault: 2.47* RR citations: 1.05

* signif diff from control, p < .05

Approaches to management

Assessing fitness to drive

The risk of losing consciousness while driving and the need to drive in today's society are opposing forces at play in determining the fitness and ability of individuals with epilepsy to drive (Andermann, et al., 1988). Seizures are the most common cause of loss of driving privileges for medical reasons (McLachlan & Jones, 1997), however a sample of recent medical and legal commentaries on this topic suggest that there is considerable disagreement as to the effect of epilepsy and seizure disorders on the ability to drive a motor vehicle (Black, 2001; Devereux, 2002; Lee, Wolfe & Shreeve, 2002).

As summarised in Table 19, all reviewed licensing jurisdictions for private licences specified that a diagnosis of epilepsy should be taken into account when determining a driver's fitness to drive. Specifically all jurisdictions emphasised the importance of seizure-free intervals when determining fitness to drive (Canada = 1 year; Australia = 3-6 months; UK = 1 year; USA = 6-12 months; NZ = 1 year, and Sweden = 1 year). This is consistent with the reviewed literature that suggests that one of the most useful and practical predictors of safe driving are the interval of time since the previous seizure (see Krauss et al., 1999; Taylor et al., 1996). In general, individuals with frequent seizures (less than 3 months) should not drive, and individuals with long intervals (6-12 months) between their seizures can be considered capable of driving safely. In addition, most jurisdictions recommend the provision of restricted or conditional licences for drivers who experiences seizures that offer no real danger with regard to driving ability given appropriate medical management (Austroads, 2006). For example, some individuals with epilepsy may have seizures that occur only during sleep and some seizures are consistently preceded by a prolonged warning or premonition (provided that full control is retained during the period of premonitory symptoms). There are also other examples where seizures only occur at a particular time of day, especially in the first hour after awakening. A restricted licence may be acceptable in such instances (Austroads, 2001). Finally, most jurisdictions also emphasise the importance of the individuals' medication compliance. For example, the driver should be considered conscientious and reliable and that they will continue to take the prescribed medication as directed.

Since 2003, few changes have been made to the guidelines, placing fewer restrictions on drivers with epilepsy. For example, the guidelines in Canada have changed from a ten year seizure free interval from the initial diagnosis to a five year seizure free interval. In addition, since 2006 Canadians who have had surgery treatment can resume driving five years after the surgery if they are seizure free. Whether the person is receiving medication treatment is no longer a restriction. In 2008 the UK inserted driving guidelines regarding medication withdrawal when previously there were no law regarding medication withdrawal.

The licensing jurisdictions for commercial licences are much more stringent: Most jurisdictions do not issue a commercial licence unless the driver has been seizure free for at least five years, have not taken AEDs for 3 years and have no evidence of epileptiform activity on EEG. However, progress has been made since 2003 in regards to licensing guidelines that take in to account individual circumstances. This is evident by the insertion of "may differ between patients" in the Canadian guidelines, and "exceptions can be made by the neurologist" in the UK guidelines.

Self-regulation

Bautista et al. (2006) investigated the prevalence of epilepsy and the characteristics of epilepsy drivers in the USA. An extensive survey was distributed to participants who were recruited from the University of Florida Health Sciences Centre/Jacksonville Comprehensive Epilepsy Program. The survey contained information about disease duration, seizure occurrence, employment, education level, marital status, receiving disability pensions, and medications. Sixty-five percent of surveys were completed by patients with epilepsy, while 34.7% of surveys were completed by carers on behalf of the patients. The mean age of participants was 43 years, and 42% were male. A total of 88 out of 319 people were current drivers. In comparison to the non-drivers, drivers were typically employed, earned a higher salary, and did not receive disability benefits. It was reported that 18% of people who experienced at least one seizure a year continued to drive, while an alarming rate of 24% of people with daily seizures continued to drive. The researchers found the following factors were independently related to driving; taking fewer AED's ($p = 0.02$), having less frequent seizures ($p = 0.01$), being employed ($p = 0.01$) and not receiving disability benefits ($p = 0.01$). The researchers claim that their study is the first to identify employment as a predictive factor of driving amongst people with epilepsy. It is acknowledged that the majority of patients in the study were likely to be at the severe stages of disease severity, however this is difficult to determine as there was no measure of disease severity. In addition, the participants did not represent a true sample of the population as the participants were typically of low socioeconomic status.

In another recent survey, Elliott and Long (2008) investigated factors that contributed to continuing to drive amongst people with epilepsy. A questionnaire was administered to 213 participants (Mean age = 39 years) who were recruited by mail from the Epilepsy Foundation of Central Ohio, in person from an epilepsy clinic, as well as by the internet from members of the Epilepsy Foundation. Participants were asked about demographic factors, seizures and driving, crashes, and questions from the Driver Perceptions and Practices Questionnaire (DPPQ). The sample comprised 69% of drivers who were currently driving. It was reported that 26% of the sample had been involved in a crash as a result of a seizure. It is interesting to note that 19% stated that they were not always honest with their doctor about their seizure frequency. Those who were driving were more likely to be employed compared to those who weren't driving (OR = 4.6(1.3-16.2) $p = 0.017$), confirming Bautista et al.'s finding. The methodological limitation of the study is that it relied upon self report data, and the researchers stated that there are no reliability estimates for the survey which weakens the empirical strength of the study.

Table 19 Private licensing guidelines for drivers with epilepsy

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Auras & minor epilepsy (absences)	<p>May drive if:</p> <ul style="list-style-type: none"> • seizures pattern has been stable for at least 3 years following a satisfactory neurological assessment. 	Not addressed.	<p>If patient suffered an attack whilst awake – must desist from driving for minimum 1 year from date of attack before licence may be issued.</p> <p>3 year licence issue is dependent on patient’s being treatment compliant and if driving is unlikely to cause danger to the public.</p> <p>70 licence restored if seizure free for minimum 7 years (with medication if necessary)</p>	<p>An unrestricted licence may be issued if seizure or episode-free for 5 years, without medication. OR seizure-free for 3 to 12 months without medication or with medication but no side effects.</p> <p>Reviews are required six-monthly, yearly or two-yearly.</p> <p>Speed, area & time of day restriction apply, depending on the length of time without seizures.</p> <p>Six-monthly review required.</p>	Regarded as a partial epilepsy attack & treat as uncontrolled epilepsy. May resume driving after 1 year free of any epileptic seizures. Upon specialist advice this period may be reduced if further seizures are unlikely.	Not addressed.
First, isolated epileptic seizure (prior to epilepsy diagnosis)	The individual is not eligible for a licence until a complete neurological assessment including an EEG has been performed and are satisfactory.	Desist from driving for 6 months. This may be reduced on medical advice.	<p>May resume driving until the age of 70 after 1 year free of any epileptic seizures.</p> <p>Medical opinion required before driving again.</p> <p>Special consideration may be given if a non-</p>	Whilst under evaluation, a restricted licence may be issued subject to medical advice.	May resume driving after 1 year free of any epileptic seizures. Upon specialist advice this period may be reduced if further seizures are unlikely.	<p>Licence denied due to any of the following:</p> <ol style="list-style-type: none"> 1. Seizure in the last 2 years. 2. EEG test & medical history show high risk of loss of consciousness. 3. No evidence of epileptiform activity on

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
			recurring cause of the seizure is clearly identified,			EEG. Exceptions may be made if a favourable prognosis is made eg seizures are unlikely to reoccur.
Epilepsy Diagnosis	The individual with a past history of epilepsy should not hold a licence unless: 1. The physician believes the individual is truthful about the frequency of the seizures 2. The physician believes the patient will take their medication in the manner prescribed 3. The applicant is under regular medical supervision 4. The seizures have been prevented by medication. If the patient has been seizure free on	Conditional licence granted if seizure-free for 3-6 months. Annual review required.	If patient suffered an attack whilst awake – must desist from driving for minimum 1 year from date of attack before licence may be issued. 3-year licence will be issued until the age of 70 if the driver is seizure free for at least 7 years since the last attack with medication, if required. Exceptions can be made if the seizure occurs during an acute head injury or intracranial surgery.	An unrestricted licence may be issued if seizure or episode-free for 5 years, without medication. OR seizure-free for 3 to 12 months without medication or with medication but no side effects. Reviews are required six-monthly, yearly or two-yearly. Speed, area & time of day restriction apply, depending on the length of time without seizures. Six-monthly review required.	May resume driving after 1 year free of any epileptic seizures. Upon specialist advice this period may be reduced if further seizures are unlikely.	Licence denied due to any of the following: 1. Seizure in the last 2 years. 2. EEG test & medical history show high risk of loss of consciousness. 3. No evidence of epileptiform activity on EEG. Exceptions may be made if a favourable prognosis is made eg seizures are unlikely to reoccur.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	or off medication for five years and receives a favourable report from their physician					
Seizures while sleeping	Eligible for a licence if the individual records a satisfactory waking EEG and is subject to medical review.	Conditional licence may be issued after 1 year seizure-free period since last seizure whilst awake.	If attack occurred whilst asleep, must desist from driving for minimum 1 year. If attacks occur for 3 years whilst asleep, and no attacks when awake then patient may be licensed. If attack when awake occurs, then as above. 3 year licence issue is dependent on patient's being treatment compliant and if driving is unlikely to cause danger to the public.	If seizures have occurred only whilst asleep over a period of 3 years or more & confirmed by a medical report, the person may be issued with a licence after a "suitable interval".	May resume driving after 1 year if no seizures whilst awake and seizure pattern upon waking or during sleep remains unchanged.	Licence denied due to any of the following: 1. Seizure in the last 2 years. 2. EEG test & medical history show high risk of loss of consciousness. 3. No evidence of epileptiform activity on EEG.
Withdrawal of Medication	Desist from driving for 3 months after withdrawal or change of medication. <i>If seizures recur:</i> Can resume driving on resumption of previously effective medications. Resume	Desist from driving during withdrawal period & for 3 months after this. On medical advice & with low risk of seizure, may not need to curtail driving.	Desist from driving during withdrawal period & for 6 months after this. Exceptions can be made depending on the physician's advice.	Person may qualify for a licence, subject to medical report & after a corrective adjustment to medication has been made & a "suitable interval" has elapsed.	A reduction in the requirement for a person to be seizure-free for 1 year prior to resuming driving may be considered if the seizure occurred whilst medication was being withdrawn or modified under medical direction.	Exceptions to the requirement for a person to be seizure free in the previous 2 years may be made if the seizures resulted from attempted withdrawal of medication on medical advice.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>driving after 3 months if seizure free.</p> <p><i>Long-term withdrawal</i> Patients can drive any class of vehicle after being seizure free for 5 years and if no epileptiform activity is recorded during a waking and sleep EEG obtained in the 6 months prior to driving</p>					The length of any post-seizure observation period may be specified on a case-by-case basis.
Epilepsy treated by surgery	Not addressed.	Conditional licence may be issued after 1 year seizure-free period after surgery.	Not addressed.	Not addressed.	Not addressed.	Not specifically addressed.

References

- Adams, R.D. & Victor, M. (1989). *Principles of neurology. 4th ed.* New York: McGraw-Hill.
- Andermann, F., Rémillard, G.M., Zifkin, B.G., Troffier, A.G. & Drouin, P. (1988). Epilepsy and Driving. *Le Journal Canadien des Sciences Neurologiques*, 15(4), 371-377.
- Australian Bureau of Statistics (2006). *National Health Survey: Summary of Results, Australia, 2004-05*, cat. no.4364.0, ABS, Canberra.
- Austroroads. (2003). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia.*
- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia.* Sydney, Australia.
- Bautista, R. E. D., & Wludyka, P. (2006). Driving prevalence and factors associated with driving among patients with epilepsy. *Epilepsy & Behavior*, 9(4), 625-631.
- Black, A.B. (2001). Epilepsy and driving: the perspective of an Australian neurologist. *Medicine & Law*, 20(4), 553-68.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers.* 1-13.
- Dam, M. (1996). Epilepsy surgery. *Acta Neurologica Scandinavia*, 96, 81-87.
- Devereux, J.A. (2002). Epilepsy and driving licences. *Medicine and Law*, 21 (121), 1-9.
- Dobbs, B. M. (2001). *Medical conditions and driving: current knowledge.* Technical Report, National Highway Transportation Safety Administration and The Association for the Advancement of Automotive Medicine Project.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive.* Swansea, UK.
- Drivers Medical Group DVLA Swansea. (2008). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive.* Swansea, UK.
- Drazkowski, J. (2007). An overview of epilepsy and driving. *Epilepsia*, 48(s9), 10-12.
- Eby, D.W., Trombley, D.A., Molnar, L.J. & Shope, J.T. (1998). The Assessment of Older Drivers' Capabilities: A review of the Literature (UMTRI 98-24). Ann Arbor, Michigan. The University of Michigan Transportation Research Institute.
- Epilepsy Action (2003). *Epilepsy Information Medical Management.* Retrieved April 2, 2003 from www.epilepsy.org.uk/info/drugsfrm.html

- Earnest, M.P. (1991). Comment: Hansotia & Broste. *The New England Journal of Medicine*, 324(21), 1510.
- Elliott, J. O., & Long, L. (2008). Perceived risk, resources, and perceptions concerning driving and epilepsy: A patient perspective. *Epilepsy & Behavior*, 13(2), 381-386.
- Faught, E., Duh, M. S., Weiner, J. R., Guérin, A., & Cunnington, M. C. (2008). Nonadherence to antiepileptic drugs and increased mortality: findings from the RANSOM Study. *Neurology*, 71(20), 1572-1578.
- Fisher, R.S., Parsonage, M., Beaussart, M., Bladin, P., Masland, R., Sonnen, A.E.H. & Rémillard, G. (1994). Epilepsy and driving: An international perspective. *Epilepsia*, 35(3), 675-684.
- Gastaut, H. & Zifkin, B.G. (1987). The risk of automobile crashes with seizures occurring while driving: Relation to seizure type. *Neurology*, 37, 1613-1616.
- Hansotia, P. (1993). Seizure disorders, diabetes mellitus, and cerebrovascular disease: Considerations for older drivers. *Clinics in Geriatric Medicine*, 9(2), 323-339.
- Hansotia, P. (1994). Epilepsy and Driving Regulations in Wisconsin. *Epilepsia*, 35(3), 685-687.
- Hansotia, P. & Broste, S.K. (1991). The effect of epilepsy or diabetic mellitus on the risk of automobile crashes. *The New England Journal of Medicine*, 324(1), 22- 26.
- Haslam, R.H.A. & Koren, G. (1989). Screening of the epileptic participant – Is it worth the trouble? *Le Journal Canadien des Sciences Neurologiques*, 16(3), 363-364.
- Hovinga, C. A., Asato, M. R., Manjunath, R., Wheless, J. W., Phelps, S. J., Sheth, R. D., Pina-Garza, J. E., Zingaro, W. M., & Haskins, L. S. (2008). Association of non-adherence to antiepileptic drugs and seizures, quality of life, and productivity: Survey of patients with epilepsy and physicians. *Epilepsy & Behavior*, 13(2), 316-322.
- Krauss, G.L., Krumholz, A., Carter, R.C., Li, G., & Kaplan, P. (1999). Risk factors for seizure-related motor vehicle crashes in participants with epilepsy. *Neurology*, 52, 1324-1329.
- Krumholz, A., Fisher, R.S., Lesser, R.P. & Hauser, W.A. (1991). *The Journal of the American Medical Association*, 265(5), 622-627.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Lawden, M. (2000). Epilepsy surgery, visual fields and driving. *Journal of Neurology, Neurosurgery, & Psychiatry*, 68, 6-7.
- Lee, W., Wolfe, T. & Shreeve, S. (2002). Reporting Epileptic Drivers to Licensing Authorities is Unnecessary and Counterproductive. *Annals of Emergency Medicine*, 39, 656-659.
- Lings, S. (2001). Increased driving crash frequency in Danish participants with epilepsy. *Neurology*, 57(3), 435-439.

- Lusk, C., Delclos, G. L., Burau, K., Drawhorn, D. D., & Aday, L. A. (2007). Mail versus Internet surveys: Determinants of method of response preferences among health professionals. *Evaluation & the Health Professions, 30*(2), 186-201.
- Manji, H. & Plant, G.T. (2000). Epilepsy surgery, visual fields, and driving: a study of the visual field criteria for driving in participants after temporal lobe epilepsy surgery with a comparison of Goldmann and Esterman perimetry. *Journal of Neurology, Neurosurgery, & Psychiatry, 68*, 80-82.
- Martin, R., Vogtle, L., Gilliam, F., & Faught, E. (2005). What are the concerns of older adults living with epilepsy? *Epilepsy & Behavior, 7*(2), 297-300.
- Mathers, C.D., Stein, C., Ma Fat, D., Rao, C., Inoue, M., Tomijima, N, Bernard, C., Lopez, A.D. & Murray, C.J.L. (2002) *Global burden of disease 2000: Version 2. Methods and results*. Global Programme on Evidence for Health Policy Discussion Paper No. 50. World Health Organisation.
- Medscape, (2009). *Seizures and epilepsy, overview and classification*. Retrieved on 11th June 2009 from <http://emedicine.medscape.com/article/1184846-overview>.
- McLachlan, R.S. & Jones, M.W. (1997). Epilepsy and driving: A survey of Canadian neurologists. *The Canadian Journal of Neurological Sciences, 24*, 345-349.
- McLachlan, R., Starreveld, E., & Lee, M. (2007). Impact of Mandatory Physician Reporting on Accident Risk in Epilepsy. *Epilepsia, 48*(8), 1500-1505.
- Mills, K. C., Drazkowski, J. F., Hammer, A. E., Caldwell, P. T., Kustra, R. P., & Blum, D. E. (2008). Relative influences of adjunctive topiramate and adjunctive lamotrigine on scanning and the effective field of view. *Epilepsy Research, 78*(2-3), 140-146.
- Mills, K., Parkman, K., & Spruill, S. (1996). A PC-Based Software Test for Measuring Alcohol and Drug Effects in Human Subjects. *Alcoholism Clinical and Experimental Research, 20*(9), 1582-1591.
- Mills, K., Parkman, K., Smith, G., & Rosendahl, F. (1999). Prediction of driving performance through computerized testing: high-risk driver assessment and training. *Transportation Research Record, 1689*, 18-24.
- Novak, A.J., Krumholz, A., Fisher, R.S., Lesser, R.P. & Hauser, A. (1991). Driving and epilepsy: the effects of medications. *The Journal of the American Medical Association, 265* (22), 296-297.
- Popkin, C.L. & Waller, P.E. (1989). Epilepsy and driving in North Carolina: An exploratory study. *Crash Analysis and Prevention, 21*, 389 – 393.
- Nair, D.R. (2003). *Epilepsy*. Retrieved April 2, 2003 from <http://www.clevelandclinicmeded.com/diseasemanagement/neurology/epilepsy/epilepsy>

- National Institute of Neurological Disorders and Stroke (NINDS, 2001). *Seizures and Epilepsy: Hope Through Research*. Retrieved on April 2, 2003 from http://www.ninds.nih.gov/health_and_medical/disorders/epilepsy.htm
- National Centre for Chronic Disease Prevention and Health Promotion (CDC, 2002). *Seizures and Epilepsy*. Retrieved on April 4, 2003 from www.cdc.gov/nccdphp/epilepsy/seizures
- Reuben, W. (2002). Epilepsy & Law. *Medicine and Law*, 21 (639), 1–9.
- Sheth, S. G., Krauss, G., Krumholz, A., & Li, G. (2004). Mortality in epilepsy: driving fatalities vs other causes of death in patients with epilepsy. *Neurology*, 63(6), 1002-1007.
- Swedish National Road Administration. (1998). *Swedish National Road Administration Statute Book*. Sweden.
- Taylor, J., Chawick, D. & Johnson, T. (1996). Risk of crashes in drivers with epilepsy. *Journal of Neurology, Neurosurgery, and Psychiatry*, 60, 621-627.
- The National Society for Epilepsy (NSE, 2002). *Epilepsy information: What is epilepsy?* Retrieved on April 2, 2003 <http://www.epilepsynse.org.uk/pages/info/leaflets/index.cfm>
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Utah State Driver License Medical Advisory Board. (2006). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J.M. (2002). Evaluating the crash and citations rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, 34, 237-246.
- World Health Organisation (WHO, 2001a). Epilepsy: Epidemiology, Etiology and Prognosis. Fact Sheet Number 165. Retrieved on March 3, 2003 www.who.int/inf-fs/en/fact165.html
- World Health Organisation (WHO, 2001b). Mental health: new understanding – new hope. Retrieved on June 26, 2003 from http://www.euro.who.int/mediacentre/PressBackgrounders/2001/20011128_1

3.7 MUSCULOSKELETAL DISORDERS

This section examines the literature pertaining to the effect of a range of diseases and injuries of the musculoskeletal system on driving and road safety outcomes, with particular attention given to the following disorders: arthritis, limb amputations, spinal injuries. These conditions differ widely in aetiology and the nature and extent of physical impairments. However, all have some impact on physical abilities. These impairments may affect fine and gross motor skills and co-ordination, and this, in turn, can interfere with the ability to drive which can lead to further mobility limitations. Due to the age-related changes that occur in the muscles, tissues, internal organs and bones, older people are vulnerable to a variety of disorders and degenerative diseases that affect the musculoskeletal system. Bone density and strength declines with age, with women exhibiting more profound changes than men (Buckwalter, Heckman & Petrie, 2003). Muscle mass and strength also diminishes with age and, by the age of 80 years may have decreased by as much as 60%, compared to people less than thirty years of age (Buckwalter et al., 2003).

Definitions of musculoskeletal disorders

Osteoarthritis

Osteoarthritis is the most common form of arthritis and mostly affects people in middle and old age. It is a degenerative disease and results from the “wear and tear” of joints. The cartilage, which provides a cushion between the joint and the bone, breaks down so that the bones grate against each other resulting in pain and restricted mobility. Osteoarthritis afflicts the weight-bearing joints (back, knees, hips and feet) as well as the hands (Arthritis Foundation, 2009). Predisposing factors include genetics (especially arthritis in the hands), prior joint injuries or joint surgery that resulted in damage, a family history of osteoarthritis, obesity (arthritis in the knees) and age (Arthritis Foundation, 2009).

Rheumatoid Osteoarthritis

Rheumatoid arthritis is a chronic form of arthritis that may affect the entire body. The lining of the joints becomes inflamed and this results in painful, tender, stiff and swollen joints and restricts movement. The inflamed cells release enzymes and these can attack and damage the cartilage and joint causing them to lose their “shape and alignment”. The joints in the feet or the hands are usually affected first. Other afflicted joints include the wrists, elbows, shoulders, neck, knees, hips and ankles. Rheumatoid nodules or lumps under the skin also appear at pressure-bearing sites, such as the back of the elbows. This disease is characterised by periodic flare-ups and can also afflict the internal organs of the body. While it is not known what causes rheumatoid arthritis, it is classified as an autoimmune disease because the body’s immune system attacks healthy joint tissue resulting in the symptoms described above (Arthritis Foundation, 2009). There are many forms of inflammatory arthritis with broadly similar effects but rheumatoid is probably the most common. Predisposing factors include hereditary causes, the presence of the genetic marker HLA-DR4 and other genes. It is thought that “agent-like viruses” trigger the disease in people who are susceptible to it (Arthritis Foundation, 2009).

Spinal cord injuries

Traumatic injuries to the spinal cord result from a variety of accidents. Approximately 42% of these are traffic-related, 27% occur from falls or jumping, 15% due to violence, 8% result from participation in sports and other leisure activities and a further 8% of unknown etiology (Spinal Cord Injury Information Network [SCIN], 2009). The degree of loss of muscle function and sensation that may result from traumatic spinal cord injuries depends on the location and extent of damage to the spinal cord. In general, the higher up the spinal cord that the trauma occurs, the more severe the damage. Two types of spinal injury are paraplegia and quadriplegia. *Paraplegia* refers to injuries to the spinal cord that occur in the lumbar or thoracic areas of the spine that result in either partial or total paralysis to the legs and feet. The trunk may also be affected. *Tetraplegia* (or *quadriplegia*) occurs when the spinal cord is injured in the cervical region resulting in either partial or total paralysis of both the legs and arms. Spinal cord injuries may also be congenital (e.g., deformities) or disease-related (e.g., resulting from polio) (Peters, 1998a).

Limb Amputations

Lower limb amputations fall into one of the following categories: partial foot, transtibial (i.e. below the knee), or trans-femoral (i.e., above the knee) (Coletta, 2000). Seventy-five percent of amputations are the consequence of circulation problems (Coletta, 2000) mostly as a result of atherosclerosis and also from diabetes (Marks & Michael, 2001). Another 20% of lower limb amputations occur from injury, although these types are more commonly performed on younger people. Following amputations, some people are fitted with prostheses or artificial limbs.

Other musculoskeletal conditions

Whiplash-associated disorder is used to describe the clinical manifestations of whiplash injury which is an injury to the cervical spine associated with rapid jerking of the head backwards and forwards, classically caused when a vehicle is struck from behind. The first axis corresponds to severity of the condition which increases categorically and the second axis to the time to recovery. The present literature review covers all categories across the first axis, but is limited to a recovery time greater than 180 days, designated as 'chronic'.

Prevalence of musculoskeletal disorders

Prevalence data for musculoskeletal disorders are not easily obtainable (Dobbs, 2001). However, Buckwalter et al. (2003) report that hip fractures and osteoarthritis of the hip and knee currently account for fewer than 10% of all musculoskeletal diseases.

Osteoarthritis

- An estimated 20.7 million Americans, predominantly 45 years or older have osteoarthritis. There is a higher incidence amongst women (Arthritis Foundation, 2009).

The WHO estimates that the prevalence of osteoarthritis is approximately 136.7 million worldwide (Mathers et al., 2002). In 2000, the prevalence of this disease in Northern American

countries (AMROA group which includes US, Canada and Cuba) was estimated at 11.1 million or around 3.4% of the total population. Similarly, the prevalence of this disease in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 16.7 million or around 4.1% of the total population.

Rheumatoid arthritis

The WHO estimates that the prevalence of rheumatoid arthritis is approximately 21.7 million worldwide (Mathers et al., 2002). In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at 1.8 million or around 0.6% of the total population. Similarly, the prevalence of this disease in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 2.8 million or around 0.7% of the total population.

- Rheumatoid arthritis affects 1.3 million Americans or approximately 0.7 % of the population;
- 1.5 million women (0.05%) and 600,000 (0.02%) men in the USA have rheumatoid arthritis (Arthritis Foundation, 2003).

An Australian report estimates prevalence statistics of arthritis (both rheumatoid arthritis and osteoarthritis) is approximately 2 million people, or 10.4% of all Australians. The report also notes that arthritis is generally more common in females than males, and that the prevalence statistics increase with age (Australian Institute of Health and Welfare, 2006).

Buckwalter et al. (2003) suggest that the demand for musculoskeletal health care will increase over the next two decades as a result of four factors: ageing population, increasing disease levels, the expectations of patients, and advances in technology. They forecast that the prevalence of musculoskeletal disorders will “increase rapidly” as the population ages. In particular, by the year 2020, it is estimated that 60 million people (or 21%) in the USA will have arthritis, of whom 36 million will be women. It will restrict the daily life of almost 12 million people.

Spinal cord injuries

- Approximately 259,000 people in the US have spinal cord injuries (SCIN, 2009).
- An estimated 2,500 people (or 0.00088 %) in Sweden have spinal cord injuries, with 55% of these injuries occurring near the neck.
- An estimated 10,500 people in Sweden (or 0.1%) drive modified vehicles (Peters, 1998a).
- In 2006-2007, an estimated 9,000 people in Australia had spinal cord injuries (Cripps, 2008).

Limb Amputations

- In 2005, it was estimated that 1.6 million people were living with limb loss in the US (Ziegler-Graham, MacKenzie, Ephraim, Travison & Brookmeyer, 2008);
- 54% had an amputation secondary to dysvascular disease with a comorbid diagnosis of diabetes mellitus (Ziegler-Graham et al., 2008);
- Limb loss secondary to trauma accounts for an additional 45% of the prevalent cases and cancer for the remaining less than 2% (Ziegler-Graham et al., 2008);
- An estimated 300,000 people in the USA (or 0.1%) have major lower limb amputations (Pandian & Kowalske, 1999);
- 75% of people with lower limb amputations are males (Colleta, 2000);
- The average age of people who undergo lower limb amputations is 51 to 69 years of age (see Colleta, 2000);
- In the UK, an estimated 5000 major amputations are performed each year (Marks & Michael, 2001).

Other musculoskeletal conditions

The incidence and prevalence of whip-lash associated disorders is difficult to determine. However by examining the pattern of compensation claims for whiplash-associated injury an estimation can be made. Approximately 4000 claims for compensation for whiplash injury were lodged with the Motor Accident Commission of South Australia in 2001. This suggests that the incidence can be said to be greater than 300 per 100,000 population in South Australia; in New South Wales, the claim incidence rate is 100 per 100,000 population. (Anderson, Gibson, Cox, Ryan & Gun, 2006). It is uncertain how chronic manifestations of this condition are represented in these statistics.

Functional impairments associated with musculoskeletal disorders relevant to driving

Musculoskeletal changes may interfere with the ability to control the car and make the appropriate manoeuvres. Specific impairments associated with various musculoskeletal diseases are described below.

Arthritis

Murray-Leslie (1991) notes that loss of strength and changes of bone structure, particularly in the hands, may occur with severe arthritis. In addition, joint pain and stiffness are also experienced by people with arthritis. There appears to be a general consensus amongst a number of studies regarding the specific nature of the problems encountered by people with arthritis whilst driving (e.g. Cornwall, 1987; Jones, McCann & Lassere, 1991; Murray-Leslie, 1991). To avoid repetition, the specific driving-related impairments associated with rheumatoid arthritis and osteoarthritis as reported by Jones et al. (1991) only are presented in Table 20.

Table 20 Driving difficulties experienced by people with rheumatoid arthritis and osteoarthritis (taken from Jones et al., 1991)

<i>Driving Disability</i>	<i>Rheumatoid arthritis</i>	<i>Osteoarthritis</i>
	<i>n=37</i>	<i>n=23</i>
Hand/Upper limb		
<i>Seat belt manipulation</i>	14%	4%
<i>Key manipulation</i>	19%	4%
<i>Hand brake use</i>	51%	9%
<i>Doors –open & close</i>	14%	0
<i>Mirror adjustment</i>	8%	0
<i>Gear use</i>	22%	4%
Upper limb/upper spine		
<i>Reaching for seat belt</i>	32%	9%
<i>Steering/cornering</i>	51%	30%
<i>Reversing</i>	38%	65%
Lower limb/lower spine		
<i>Car entry/exit</i>	14%	17%
<i>Footpedal use</i>	11%	17%
<i>Seat comfort & position</i>	32%	30%

As shown in Table 20, drivers with rheumatoid arthritis find steering and cornering the most difficult driving manoeuvres whereas drivers with osteoarthritis tend to find reversing the most difficult.

Spinal Injuries

People with spinal cord injuries have restricted mobility, those people with quadriplegia being more impaired than those with paraplegia (Peters, 1998a). Due to paralysis of the legs, the arms must be used to carry out all of the driving tasks. According to Peters (1998a; 1998b), the difficulties associated with driving that are encountered by drivers with paraplegia and quadriplegia include:

- getting in and out of the car;
- transferring to and from the wheel chair (paraplegics);
- fastening the seat belt;
- operating primary car controls, such as the brakes, accelerator and steering wheel;
- operating secondary car controls, such as indicators, horn, headlights and windscreen wipers;
- remaining upright due to a lack of stability in the trunk;
- task overload for the upper limbs and the resulting fatigue;

- dealing with multiple competing tasks whilst driving (eg hand controlled steering and braking);
- strength and agility problems;
- placing the wheelchair in the car and removing it (paraplegics).

Treatment of musculoskeletal disorders

There is a wide range of therapies and adaptive technologies that can help to alleviate the symptoms of musculoskeletal disorders and may also facilitate driving ability. These include:

- drug therapy;
- surgery;
- exercise and physical therapy;
- combination approaches.

Drug therapy

The following classes of drugs are used to provide relief from pain and inflammation: analgesics (paracetamol, aspirin and codeine); opiates (codeine, tramadol, oxycodone); non-steroidal anti-inflammatory drugs (NSAIDs); local corticosteroid injections; tricyclic antidepressants to alleviate chronic pain as well as for their sedative effects; anticonvulsants for neuropathic pain; and muscle relaxants to treat severe muscle spasm (Geffen, 2003). Disease-modifying antirheumatic drugs (DMARDs) are used in treatment of rheumatoid arthritis to retain functional abilities (Sokka et al., 2000). Opioid treatment may be used for the long-term management of chronic pain (Goucke, 2003).

Disease modifying antirheumatic drugs (DMARDs) have been found to preserve the functional capacity of rheumatoid arthritis sufferers over relatively long periods of time (8.5 years and 13 years) when treatment was begun within two years of disease onset (Sokka et al., 2000). NSAIDs are most commonly prescribed for pain relief for osteoarthritis. They are also used by people with rheumatoid arthritis but to a lesser extent. NSAIDs have been found to be better at managing pain than analgesics such as paracetamol (Freemantle, 2000). The long-term effects of opioids have not been fully researched. However, the cognitive impairment that accompanies them may impair driving ability, particularly when they are first taken or when the dose level is increased (Goucke, 2003).

Surgery

This option may include hip and knee replacements, tendon and ligament reconstruction, and joint and spinal arthrodeses (Buckwalter et al., 2003).

After being fitted with a prosthesis, the person will require gait training to enable them to walk properly (Pandian & Kowalske, 1999). In terms of driving, vehicular modifications may be

necessary as well as instruction in how to operate the vehicle with the prosthesis in place (Chadwick & Wolfe, 1992 cited in Coletta, 2000).

Exercise and physical therapy

Regular exercise helps to increase strength and muscle mass in older people. Exercises that incorporate stretching, muscle strengthening and range-of-motion movements can decrease the risk of soft tissue injuries that could occur when undertaking an exercise regime (Buckwalter et al., 2003).

Ostrow, Shaffron and McPherson (1992) examined the effect of a fitness training program that emphasized range-of-motion exercises on the driving skills of people aged 60 to 85 years old. The experimental group (n = 16) participated in a total of nine different joint flexibility activities which targeted the chin, neck, shoulders and trunk over an eight-week period. These exercises were chosen because they matched the skills required for driving. Controls did not receive this intervention. Subjects also completed a driving test that assessed various driving skills. It was found that the exercise regime was effective in improving shoulder flexibility and trunk rotation. In addition, the experimental group improved their scores for driving-related observation skills such as checking mirrors and turning the head to left or right and looking over the shoulders when appropriate while driving.

Combination approaches

Treatments of musculoskeletal conditions may also include any combination of the foregoing treatments. Chronic pain associated with several musculoskeletal is said to be difficult to treat with a small percentage of patients not responding despite receiving “optimal care” (Geffen, 2003). However, strengthening exercises, the application of heat, physiotherapy, drugs, and psychological treatment have been found to provide some improvement (Geffen, 2003). Due to its complexity, chronic pain is best treated using a biopsychosocial approach, that is, one that firstly takes account of the organic cause of pain but also includes a consideration of other contributory factors such as family and other interpersonal relationships, finances, employment record and personality. Early, non-drug interventions include weight loss, exercise, reassurance, and lifestyle changes (Goucke, 2003). Jones et al. (1991) have also suggested the use of analgesics and non-steroidal anti-inflammatory drugs to combat pain during long driving trips.

Pre-May 2003: Relationship between musculoskeletal disorders and road safety outcomes

The 2004 report reviewed five studies which examined this relationship. In 2009, the latest literature search found only two additional papers on this issue. A summary of all studies reviewed is provided in Table 21.

Crashes

Koepsell et al. (1994) conducted a study aimed at identifying injury crash risk of older drivers with various medical conditions, including arthritis (see section 3.5 for more details of the study methods). The results showed that approximately 53.8% of the injury crash-involved cases and 52% of non crash-involved controls were affected by osteoarthritis. Injury crash risk was not significantly different for drivers with osteoarthritis compared with controls (OR: 1.1, 95% CI: 0.8-1.5).

The study also examined risk associated with rheumatoid arthritis. Approximately 2.1% of the cases and 1.3% of controls had rheumatoid arthritis. Injury crash risk was not significantly different for drivers with rheumatoid arthritis compared with controls (OR: 1.6, 95% CI: 0.5-5.3). The authors note that adjustment for race, marital status and exposure (miles driven in previous year) resulted in only slight changes in these ORs, although no data are provided. Notwithstanding the relatively small number of drivers in this study, these findings suggest that older drivers with arthritis do not have an elevated risk of injury crashes.

In 2002, Vernon et al. conducted a retrospective case-control study to compare the relative risk of drivers with medical conditions and those without, during a five-year study period from 1992-1996 (for more detail regarding the study design see section 3.1). Crash rates per 10,000 licence days (Utah DOT official records) for 225 drivers with functional motor impairments (i.e., history of impaired functional motor ability including difficulties with muscular strength, coordination, range and motion, spinal movement and stability, amputations or absence of body parts and/or abnormalities affecting motor control) were compared with a control group of drivers matched by age, sex and place of residence. Drivers with functional motor impairments were also classified according to licence status (restricted/unrestricted) with the majority of cases (n = 208) having no restrictions. The authors reported that there were no significant differences between unrestricted drivers with functional motor impairments and control participants for overall crashes (RR: 1.11, CI 0.70-1.74) or at-fault crashes (RR: 1.79, CI 1.00- 2.93). Due to the fact that there were no reported crashes in the restricted licence group, it was not possible to calculate the relative risk.

In the same study, Vernon et al. investigated the crash rates (all crashes and at-fault crashes) and citation rates (see next section for information regarding citations) for 386 drivers with musculoskeletal disorders which the authors defined as a history of a condition or disease that may affect driving (e.g., osteoporosis or active infectious disease, including HIV). The licence status of most drivers with musculoskeletal disorders was unrestricted (n = 353). Drivers with musculoskeletal disorders with an unrestricted licence (i.e., the lowest level of impairment) had significantly higher crash rates and at-fault crashes (RR: 1.59, 95%CI 1.10 - 2.29; RR: 1.84, 95%CI 1.14 - 2.98 respectively) than the general population drivers. Similarly, drivers with musculoskeletal disorders with restricted licences (i.e., the highest level of impairment) had significantly higher crash rates and at-fault crashes (RR: 4.51, 95%CI 1.01-20.12; RR: 11.29, 95%CI 2.39-53.25 respectively) than the general population drivers. Confidence intervals were extremely large, thus, the findings need to be interpreted with caution and replication is necessary.

Vernon et al. concluded that while drivers with musculoskeletal disorders (both those with restrictions and those without restrictions) have a higher risk of crashing than the general population of drivers, drivers with functional motor impairments do not. One of the main limitations of this study was that the authors did not control for driver exposure, which assumes that drivers in each of the medical condition groups and matched controls drove similar distances.

It is important to note that in the studies reviewed above, no indication of use of adaptive cars is reported. This is likely to have a significant influence on driving performance of some groups with musculoskeletal problems. The studies reviewed below address the issue of adaptive technologies and driving performance with people with physical disabilities.

Henriksson (2001a; 2001b) undertook an analysis of the crash involvement of drivers of adapted cars (AC) in Sweden. Seven hundred and ninety-three people registered on Sweden's National Vehicle Register as having a modified car completed a postal survey. Respondents provided information on their disabilities, the nature of the car modifications, their driving exposure and the car crashes that they had been involved in over the period 1996-1999. 70-90% of respondents reported leg and/or foot problems (i.e. impaired function, no function or no limb) and 30% had spinal cord injuries. A total of 75% used a wheelchair to get around but only 7% of these drove from their wheelchair. The specific car modifications that had been carried out on respondents' cars were:

- 90% had automatic transmission;
- 64% had servo-powered steering;
- 42% had servo-powered braking;
- 27% had a swing seat; and
- 26% had a wheel knob.

The respondents were experienced drivers – 75% had driven for more than five years, 50% drove daily or almost daily, and their average annual mileage was 13,500km. In addition, 95% of respondents indicated that their confidence levels were relatively high or very high when driving their adapted cars.

Information on motor vehicle crashes (MVCs) that had been reported to the police were elicited from the drivers of AC cars. Eleven percent of drivers reported that they had been involved in motor vehicle crashes between 1996 and 1999 (a period of 3.5 years). These MVCs were mostly of a minor nature with 87% of them resulting only in property damage. The crashes that did lead to injury were commonly rear-end crashes or occurred because the other driver failed to give way to the disabled driver. Of the crashes that did occur, 11% were due to technical difficulties associated with the car modifications. Mimicking the trend in drivers in the general population, younger drivers of adapted cars were more frequently involved in MVCs than either middle-aged or elderly drivers of adapted cars. Drivers with spinal cord injuries were also over-represented amongst the group of AC drivers with MVCs. Henriksson (2001a) also calculated the MVC rate for AC drivers and compared it to that found in the general driving population. It was found that AC drivers had 0.85 crashes per million kilometres and drivers in the general population experienced 0.98 crashes per million kilometres. The risk of MVCs was computed to be 0.21 crashes/million kilometres driven for AC drivers and 0.20 crashes/million kilometres driven for general population. There was no significant difference in MVC risk for the two groups of drivers. Henriksson (2001a) points out that not all drivers of adapted cars are required to register their vehicle with Sweden's National Vehicle Register, for example those who use wheel knobs, have an accelerator fitted for the left foot, or foot brakes that are not controlled by the right foot or right hand. Thus, an accurate figure of the number of drivers of AC in Sweden cannot be provided, and this may impact on the overall results.

There is a lack of literature that specifically investigates the relative risk of driving with spinal cord injuries. While Henriksson (2001a), above, found that drivers with spinal cord injuries were over-represented in the group of AC drivers involved in car crashes, Peters (1998)

concluded that the sparse data to date do not indicate that drivers with spinal cord injuries have a higher MVC risk than other drivers as a result of “differences in driving performance” (p24).

McGwin, Sims, Pulley & Rossman (2000) investigated the effect of medical conditions and medications on the risk of being involved in an automobile crash, taking driving exposure and demographic factors into account. 901 drivers who were 65 years or older were selected from the Alabama Department of Public Safety records. Data on medical conditions, estimated annual distance driven, and self-reported driving quality were obtained. Information on motor vehicle crashes (MVCs) were obtained from the official Alabama Department of Public Safety database. The experimental group was divided into those involved in at-fault crashes (n = 244) and those who were not at fault (n = 182). The controls (n = 475) had not been involved in any crashes. At-fault drivers were older than the not-at-fault drivers and they drove more than the not-at-fault drivers and the no-crash drivers. Drivers with arthritis reported a 20% higher at-fault crash rate than those without arthritis, although this increased crash risk was apparent for females with arthritis only (OR: 1.8, 95%CI 1.1 - 2.9). Drivers with arthritis who were using non-steroidal anti-inflammatory drugs (NSAIDs) had a 70% higher crash rate than drivers who did not use these drugs (95% CI: 1.0, 2.6).

Hu, Trumble, Foley, Eberhard and Wallace (1998) used a panel data analysis to identify the factors that contributed to older drivers' crash risk. Gender-specific factors were uncovered, shedding some light on the finding by McGwin et al (2000) reviewed above. For older women, an inability to extend the arms above the shoulders increased their risk of being involved in an automobile crash. In fact, these women faced a two-fold increase in crash risk compared to women who had no difficulty in lifting their arms above their shoulders. It was also found that the distance driven affected women's crash risk. For example, older women who drove 6,000 miles per annum were 1.23 times more likely to be involved in a car crash than women who drove 3,000 miles per year. Crash risk for older men was also influenced by the distance driven. The risk ratios (RR) for different annual mileages for both genders were computed and it was found that the risk ratio for 3,000 miles, 6,000 miles and 12,000 miles was 1.25. For annual distances of 9,000 miles and 18,000 miles the risk ratio was 1.54.

Citations

As outlined above, Vernon et al. (2002) conducted a retrospective case control study of crash and citation rates of drivers with medical conditions during 1992 – 1996. The rate of citations amongst unrestricted drivers with functional motor impairments was significantly higher than that of control participants (RR: 1.42, 95%CI 1.04 - 1.94). In contrast, rate of citations amongst unrestricted drivers with musculoskeletal disorders was not significantly different from control participants (RR: 1.22, 95%CI 0.90-1.65).

Driving performance

No studies were found that addressed the relationship between musculoskeletal disorders and driving performance.

Post-May 2003 Relationship between musculoskeletal disorders and road safety outcomes

In 2009, the latest literature search found only two additional papers on this issue. A summary of all studies reviewed is provided in Table 21.

Crashes

No studies were found that addressed the relationship between musculoskeletal disorders and crash involvement.

Citations

No studies were found that addressed the relationship between musculoskeletal disorders and citations.

Driving performance

In a Canadian study Cranney et al. (2005) examined driving problems experienced by patients with rheumatoid arthritis. Participants were drawn from the South Eastern Ontario Medical organisation Health database, were aged over 25 years and had a diagnosis of rheumatoid arthritis. A total of 520 participants who were current drivers completed surveys (response rate 74%). More than half of the study sample (58%) of drivers with rheumatoid arthritis reported difficulty with driving: approximately 50% reported a little difficulty, 7% reported quite a bit and 1.5% a great deal of difficulty. Driving difficulties included activities such as sitting for long periods (54%) making head checks (34%), gripping the steering wheel (28%).

Pereira, Jolly and Treleaven (2008) studied self-reported driving habits in subjects with persistent whiplash-associated disorder. Thirty patients and 30 asymptomatic controls completed a number of questionnaires outlining their general health and neck disability and their driving habits. Patients complained of chronic neck pain attributed to a motor vehicle collision at least 3 months post injury. It is unclear whether the control group was also crash involved, however researchers noted that control group were drivers with “no history of neck pain or trauma”. It is not noted whether patients and controls were matched on any other relevant characteristics, however notable differences between the two groups include age (mean years 33.8 to 25.6 respectively) and driving experience (mean years 15.7 to 7.5 respectively). Persistent whiplash-associated disordered patients reported greater difficulty when reversing and parallel parking than the controls, despite their greater driving experience. The use of driving aids was not addressed.

Summary

Overall, while several studies describe driving difficulties experienced by people with physical impairments affecting the musculoskeletal system, the evidence suggests that there is only a slightly increased risk of crash associated with these disorders. Meta-analysis of studies on this topic by Vaa (2003) revealed a relative risk of 1.17 (95%CI, 1.004–1.36) and Dobbs (2005) concluded that musculoskeletal disorders were not considered a red flag for determining fitness to drive. As noted by Anstey and colleagues, this may be attributed to drivers’ ability to compensate for physical impairments when driving (Anstey, Wood, Lord and Walker, 2005). Two studies suggests that there may be specific driving movements (head check, steering etc) that may pose greater difficulty for people with arthritis and whiplash-associated disorders. Medication use may also impact upon driving performance. Specifically, one study reported that drivers with arthritis using non-steroidal anti-inflammatory drugs (NSAIDs) experienced a 70% higher crash rate than those who did not use these drugs. Driving aids or vehicle adaptation may assist drivers with similar medical conditions in completing these movements. Drivers of adapted

cars have the same risk of MVCs as drivers in the general population, although people with spinal cord injuries are over-represented amongst this group in terms of car crashes. However, the evidence on adapted vehicle use reviewed to date is very limited and no strong conclusions can be drawn.

Table 21 Summary of studies of risk associated with musculoskeletal disorders

Study: Author/date	Methods	Outcome Measure of Risk	Results
Cranney et al. (2005)	Cross-sectional, Drivers with rheumatoid arthritis (n=520)	Self-reported driving behaviours	58% of respondents with rheumatoid arthritis reported driving difficulties: shoulder checks gripping steering wheel and turning corners
Henriksson (2001)	Population/Case Population =drivers in the general population Case=drivers of adapted cars in Sweden (n=793)	Self-reported MVCs from 1996-1999.	From 1996-1999: 11% of drivers of adapted cars had MVCs, mostly of a minor nature. AC drivers had 0.85 crashes per million kilometers compared to general population who had .98 crashes per million kilometers. MVC risk =0.21 crashes/million kilometers driven for AC drivers versus 0.20 crashes/million kilometers driven for general population (no significant difference). Young AC drivers had more crashes than middle-aged & elderly AC drivers. Drivers with spinal cord injuries over-represented in MVC occurrence.
Koepsell et al (1994)	Case-control; n=234 (65yrs+) injury crashes n=446 no injury crashes;	Police-reported injury crashes requiring medical care	Osteoarthritis OR: 1.1, CI 0.8-1.5 Rheumatoid arthritis OR: 1.6, CI 0.5-5.3
McGwin, Sims, Pulley & Rossman (2000)	Population/Case-control Controls n=454 Case n=447 Cases: (1) 244 drivers in at-fault crashes (2) 182 drivers in not-at-fault crashes Controls=no crash involvement	1. At-fault crashes. 2. Not-at-fault crashes. Data on crashes obtained from official database.	<u>General</u> At-fault drivers older than not-at-fault drivers. At-fault drivers have higher annual mileage than not-at-fault drivers & no crash drivers. <u>Arthritis Sufferers:</u> Arthritics had 20% higher at-fault crash rate than non-arthritics.

Study: Author/date	Methods	Outcome Measure of Risk	Results
	Population = Mobile County Alabama residents ≥ 65 years with driver's licence in 1996. Chronic medical conditions: Arthritis, Cardiovascular, Diabetes, Visual problems, Renal,- Cognitive, Cancer, Stroke		Increased crash risk apparent for arthritic females only (OR=1.8, 95% CI: 1.1, 2.9). NSAID (non-steroidal anti-inflammatory drug) users had 70% higher crash rate than drivers who did not use these drugs (95% CI: 1.0, 2.6).
Pereira, Jully and Treleaven (2008)	Case-control Controls n=30 Case n=30 Cases with chronic whiplash-associated disorders	Self reported driving habits (measured by the Driving Habits Questionnaire), citation rates and crash rates.	Cases reported greater driving difficulty than controls, especially when reversing and parallel parking.
Vernon et al. (2002)	Pop/case-control; Functional motor impairment Cases =225 Control =20,210 'Cases' = history of impaired functional motor ability including difficulties with muscular strength, coordination, range and motion, spinal movement and stability, amputations or absence of body parts and/or abnormalities affecting motor control Muscolkeletal disorders Cases =386 Control =20,210 'Cases' = a history of a condition or disease that may affect driving (e.g., osteoporosis or active infectious disease, including HIV)	(i) Crash -all (ii) At-fault crash (iii) Citation Rates per 10,000 lic days	Functional motor impairment Not Restricted RR all crashes: 1.11 RR at-fault crashes: 1.79 RR citations: 1.42* Muscolkeletal disorders Not Restricted RR all crashes: 1.59* RR at-fault crashes: 1.84* RR citations: 1.22 Restricted RR all crashes: 4.51* RR at-fault: 11.29*

Approaches to management

Assessing fitness to drive

The licensing guidelines for private vehicles for each of the six countries surveyed all stipulate that prosthesis-wearers and arthritis sufferers may continue to drive subject to any necessary car modifications to enable the driver to safely operate the vehicle. Commercial drivers are subject to similar, although more stringent, guidelines with Sweden also requiring that bus and taxi drivers have the ability to assist passengers alight from the vehicle and buckle their seat belts. Fitness to drive for those with spinal cord injuries is also determined according to the severity of the injury and the resulting functional impairment (see Table 22 for more detailed description of licensing requirements).

Training and rehabilitation

A number of vehicle modifications have been recommended as a means of enabling drivers with arthritis to continue to drive. Jones, McCann and Lassere (1991) state that “almost all arthritic individuals are able to continue driving with the help of simple modifications” (p361). Haslegrave (1991) notes that car adaptations need to be undertaken with a view to the range and direction of movement that the driver has in their limbs as well as the amount of force required to operate the controls. Car modifications can range from very simple devices such as adding a knob to the steering wheel to complex adaptations such as converting the steering of the vehicle to a foot controlled operation. Haslegrave (1991) also comments that the interaction between a person’s disabilities and the car adaptations need to be formally assessed by a road test to determine the ability to drive safely.

Murray-Leslie (1991) has recommended the following car adaptations for drivers with arthritis:

- cars with one wide passenger and one wide driver door only;
- front seats that slide backwards and forwards as well as swivelling outwards to provide easier access and egress from cars;
- installation of secure head rests/restraints in cars to reduce the incidence and severity of neck sprains in the event of a rear-end collision;
- the fitting of additional rear view mirrors;
- padding the steering wheel to increase its girth;
- power-steering;
- electronically adjusted seats;
- vacuum-assisted braking;
- installing larger door handles;
- built-up keys.

Cornwall (1987) conducted a study of the driving skills of 83 people (82% female) suffering from arthritis who were assessed at a UK Mobility Centre over a period of two hours by a driving instructor and therapist. These patients were a subset of a larger study group of 908 individuals with a variety of disabilities undergoing assessment. Physical measurements, such as height (both standing and sitting), fingertip reach, and the distance from above and below the knee, were recorded for assessing functionality from a driving viewpoint. Similarly, the range of movement in the joints and muscle strength were also examined from a driving perspective. Of particular importance were the pain and fatigue that patients experienced, as these two factors are salient in arthritis sufferers and indicate which limbs should be used to manipulate the car controls, for example, steering, braking and acceleration. The ability to enter and exit the car was also assessed.

In terms of anthropometric measurements, it was found that the arthritic group had the fourth lowest height measurements out of the total sample of 908. Sitting height, however, was only somewhat affected. This finding was thought to be a consequence of hip and knee flexion abnormalities. The arthritic group also contained the second highest proportion of people exhibiting functional reduction in fingertip reach - those with congenital limb abnormalities had the highest. Arthritics displayed below average strength when braking due to pain, fatigue and restricted movement rather than muscle weakness per se. Male arthritis patients displayed marked weakness when steering, although female sufferers did not.

From the foregoing results, the following car modifications were recommended for many of the drivers with arthritis:

- Seat adjustments – 30% required the seat to be raised and 25% needed it to be tilted forwards;
- Side supports – 13% needed these to reduce fatigue and to provide stability;
- Head restraints – required by 41% of the arthritis patients to provide additional protection to the spine during hard braking;
- Steering wheel adjustment – 29% needed the diameter to be reduced due to difficulties in moving the shoulders, while some required the steering wheel to be padded to assist with grip;
- Power steering – 48% of the group required varying types of power assisted steering, with 11% of these also requiring the steering column to be moved closer to the driver;
- Brake modifications - 37% needed modifications to assist with braking. Parking brakes that had a pushbutton device were also recommended;
- Foot control modification - to decrease fatigue, approximately 50% needed adjustments made to the brake and accelerator pedals (larger pads, raised pedals or pedals that “cradled” the foot);
- Gears – 99% of the group needed automatic cars;

- Secondary control devices – controls on the dashboard (eg ignition) often needed repositioning to ensure that they were within the reach envelope of the arthritis patients;
- Miscellaneous modifications –door and boot handles, windows that opened and closed electronically, and an electric winch for wheelchair-bound patients.

Table 22 Private licensing guidelines for drivers with musculoskeletal disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Limb Amputation	<p><i>Amputation or deformity</i> May continue to drive subject to satisfactory driving assessment</p> <p><i>With prosthesis</i> May continue to drive subject to satisfactory driving assessment.</p>	<p><i>Complete or partial limb amputation:</i> May not hold an unconditional licence.</p> <p>A conditional licence may be issued following a practical driving assessment, car modifications & prosthesis requirements.</p>	<p>May be licensed if medical confirmation obtained that driving ability (e.g. nerves and circulation) is unimpaired.</p> <p>Vehicle modifications may be required.</p>	<p>If person has no “driving limitations” & subject to further driving assessment with prosthesis &/or car modifications, an unrestricted licence will be issued.</p> <p>Annual review required.</p> <p>A restricted licence may be issued according to the conditions (eg reduced speed or in limited areas) under which the person can operate the vehicle.</p>	<p><i>One arm amputated:</i> May drive if suitable vehicle modifications are made.</p> <p><i>Leg(s) amputated below the knee:</i> May continue to drive if prosthesis is worn & back, hips & joints are strong & have full range of movement & car is modified.</p> <p><i>Leg(s) amputated above the knee:</i> May drive if car has hand controls & other suitable modifications Assessment & training required.</p>	<p>Licence denied if ability to drive safely is impaired.</p> <p>May continue to drive if prosthesis &/or vehicle modifications can compensate for disability.</p>
Arthritis Joint Problems	<p>May be restricted if pain or range of movement adversely affects ability to drive safely; may be subject to satisfactory driving assessment.</p>	<p>May not hold an unconditional licence if person is unable to operate the vehicle safely.</p> <p>A conditional licence may be issued subject to an assessment of driving ability & treatment & car modification requirements.</p>	<p>May be licensed if driving ability is unimpaired.</p> <p>Vehicle modifications may be required.</p>	<p><i>With mild or moderate “residual loss of function”:</i> Person may hold an unrestricted licence. With or without vehicle modifications.</p> <p>One or two-yearly review required.</p> <p>A restricted licence may be issued according to the</p>	<p>Driving assessment is required if locomotor functioning is impaired.</p> <p>If condition interferes with ability to drive safely, then driving restrictions may apply.</p> <p>Vehicle modifications such as automatic transmission, spinner knobs may be required.</p>	<p>Licence denied if ability to drive safely is impaired.</p> <p>May continue to drive if modifications to vehicle can compensate for disability.</p> <p>Periodic review on a case-by-case basis is required for all progressive disorders.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
		Periodic review required.		conditions (eg reduced speed or in limited areas) under which the person can operate the vehicle.	If pain impairs driving ability, it is recommended that person refrains from driving.	
Spinal Conditions	<p><i>Cervical</i> If affected by loss of movement of head and neck, driver restricted to vehicles equipped with panoramic mirrors. Patients with severe pain or very restricted range of movement should be advised not to drive until pain and restrictions of movement are minimal.</p> <p><i>Lumbar</i> Subject to satisfactory driving assessment.</p>	<p>May not hold an unconditional licence if cervical spine movement is severely restricted “to only a few degrees of movement” (p69).</p> <p>A conditional licence may be issued subject to an assessment of driving ability & treatment & car modification requirements.</p> <p>Some reduction in head & neck movement is permitted providing vehicle is equipped with “adequate outside mirrors” (p68).</p> <p>Persons with severe neck pain & very restricted movement (including that from neck braces & collars) are advised not to drive until treatment is finished. Periodic review .</p>	<p>May be licensed if driving ability is unimpaired.</p> <p>Vehicle modifications may be required.</p>	<p><i>With mild or moderate “residual loss of function”:</i> Person may hold an unrestricted licence. With or without vehicle modifications.</p> <p>One or two-yearly review required.</p> <p>A restricted licence may be issued according to the conditions (eg reduced speed or in limited areas) under which the person can operate the vehicle.</p>	<p>If condition interferes with ability to drive safely, then driving restrictions may apply.</p> <p>Desist from driving if severe back, neck, shoulder or pelvic pain.</p> <p>May resume driving subject to driving assessment & car modification requirements.</p>	<p>Licence denied if ability to drive safely is impaired.</p> <p>May continue to drive if modifications to vehicle can compensate for disability.</p>

References

- Anderson, R. W. G., Gibson, T. J., Cox, M., Ryan, G. A., & Gun, R. T. (2006). *Whiplash associated disorders: A comparative review* (CASR016). Adelaide, Australia: University of South Australia, Centre for Automotive Safety Research.
- Anstey KJ, Wood J, Lord S, Walker JG. (2005). Cognitive, sensory and physical factors enabling driving safety in older adults, *Clin. Psychol. Rev.*, 25, 45–65.
- Arthritis Foundation (2009). Available at <http://www.arthritis.org> (Accessed May, 2009).
- Austrroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Buckwalter, J.A., Heckman, J.D. & Petrie, D.P. (2003). Aging of the North American population: New challenges for orthopaedics, *The Journal of Bone and Joint Surgery*, 85-A(4), 748-758.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Coletta, E.M. (2000). Care of the elderly patient with lower extremity amputation. *The Journal of the American Board of Family Practice*, 13(1), 23-34.
- Cornwell, M. (1987). The assessment of people with arthritis who wish to drive a car. *International Disabilities Studies*, 9(4), 174-177.
- Cranney AB, Harrison A, Ruhland L, Vaidyanath C, Graham I, Man-Son-Hing M, Jaffey J, Towheed TE, Anastassiades TP, Dwosh II. (2005). Driving Problems in Patients with Rheumatoid Arthritis, *J. Rheumatol.*, 32, 2337–2342.
- Cripps RA (2008). *Spinal cord injury, Australia, 2006–07*. Injury Research and Statistics Series No 48. AIHW Cat No INJCAT 119, Adelaide.
- Dobbs, B. M. (2001). *Medical conditions and driving: current knowledge*. Technical Report, National Highway Transportation Safety Administration and The Association for the Advancement of Automotive Medicine Project.
- Dobbs BM. (2005). *Medical Conditions and Driving: A Review of the Scientific Literature (1960–2000)*. Technical Report for the National Highway and Traffic Safety Administration and the Association for the Advancement of Automotive Medicine Project, Washington, DC.
- Drivers Medical Group DVLA Swansea. (2008). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Freemantle, N. (2000). Cost-effectiveness of non-steroidal anti-inflammatory drugs (NSAIDs)-what makes a NSAID good value for money? *Rheumatology*, 39, 232-234.

- Geffen, S.J. (2003). Rehabilitation principles for treating chronic musculoskeletal injuries. *Medical Journal of Australia*, 178(5), 238-242.
- Goucke, C.R. (2003). The management of persistent pain. *Medical Journal Australia*, 178, 444-447.
- Gyi, D.E., & Porter, J.M. (1998). Musculoskeletal problems and driving in police officers. *Occupational Medicine*, 48(3), 153-160.
- Haslegrave, C.M. (1991). Driving for handicapped people. *International Disability Studies*, 13(4), 111-120.
- Henriksson, P. (2001a). *Drivers with Disabilities – a Survey of Adapted Cars, Driving Habits and Safety*. VTI rapport 466, Swedish National Road and Transport Research Institute.
- Henriksson, P. (2001b). *Drivers of Adapted Cars in Sweden*. Conference proceedings from the 9th International Conference on Mobility and Transport for Elderly and Disabled People, Warsaw Poland, 2-5 July 2001.
- Hu, P.S., Trumble, D.A., Foley, D.J., Eberhard, J.W., & Wallace, R.B. (1998). Crash risks of older drivers: a panel data analysis. *Accident Analysis and Prevention*, 30(5), 569-581.
- Jones, J.G., McCann, J., & Lassere, M.N. (1991). Driving and arthritis. *British Journal of Rheumatology*, 30, 361-364.
- Koepsell, T., Wolf, M. & McCloskey, L. (1994). Medical conditions and motor vehicle collision injuries in older adults. *Journal of the American Geriatrics Society*, 42, 695-700.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Mathers, C. D., Stein, C., Ma Fat, D., Rao, C., Inoue, M., Tomijima, N., Bernard, C., Lopez, A. D., & Murray, C. J. L. (2002). *Global Burden of Disease 2000: Version 2, Methods and Results. Global program on evidence for health policy discussion. Paper 50*. WHO, Geneva.
- Martinez, R. (1995). Older drivers and physicians. *JAMA*, 274(13), 1060.
- Marks, L.J., & Michael, J.W. (2001). Artificial limbs. *BMJ*, 323, 732-735
- McGwin, G., Sims, R.V., Pulley, L., & Rossman, J.M. (2000). Relations among chronic medical conditions, medications, and automobile crashes in the elderly: A population-based case-control study. *American Journal of Epidemiology*, 152(5), 424-431.
- Murray-Leslie, C. (1991). Driving for the person disabled by arthritis. *British Journal of Rheumatology*, 30, 54-55.

- Ostrow, A.C., Shaffron, P., & McPherson, K. (1992). The effects of a joint range-of-motion physical fitness training program on the automobile driving skills of older adults. *Journal of Safety Research*, 23, 207-217.
- Pandian, G., & Kowalske, K. (1999). Daily functioning of patients with an amputated lower extremity. *Clinical Orthopaedics & Related Research*, 361, 91-97. Abstract.
- Pereira, M. J., Jull, G. A., & Treleaven, J. M. (2008). Self-reported driving habits in subjects with persistent whiplash-associated disorder: Relationship to sensorimotor and psychologic features. *Archives of Physical Medical Rehabilitation*, 89, 1097-1102.
- Peters, B. (1998a). *Car Drivers with Traumatic Spinal Cord Injury*. VTI report 426A-1998. Swedish National Road and Transport Research Institute.
- Peters, B. (1998b). *Spinal Cord Injuries and Driving*. Proceedings of the 8th International Conference on Transport and Mobility for Elderly and Disable People, Perth, Western Australia, September 1998.
- Sokka, T., Mottonen, T., & Hannonen, P. (2000). Disease-modifying anti-rheumatic drug use according to the 'sawtooth' treatment strategy improves the functional outcome in the rheumatoid arthritis: results of a long-term follow-up study with review of the literature. *Rheumatology*, 39, 34-42.
- SpinalCord Injury Information Network [SCIN], (2009). Spinal Cord Injury Facts & Figures at a Glance 2009. Available at <http://www.spinalcord.uab.edu/show.asp?durki=19679> (Accessed May, 2009).
- Swedish National Road Administration. (1998). *Swedish National Road Administration Statute Book*. Sweden.
- Utah State Driver License Medical Advisory Board. (2006). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J.M. (2002). Evaluating the crash and citations rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, 34, 237-246.
- Ziegler-Graham, K., MacKenzie, E. J., Ephraim, P. L., Travison, T. G., & Brookmeyer, R. (2008). Estimating the prevalence of limb loss in the United States: 2005 – 2050. *Archives of Physical Medical Rehabilitation*, 89, 422-429.

3.8 NEUROLOGICAL CONDITIONS (EXCLUDING EPILEPSY)

Neurological conditions are characterised by diseases, injuries and disorders of the brain, nerves, and spinal cord. Chronic neurological conditions include stroke, epilepsy, brain and spinal cord injury, multiple sclerosis, and Parkinson's disease. These conditions differ widely in aetiology and prevalence. Similarly, the extent and nature of impairment differs across conditions. In addition, individuals living with chronic neurological conditions may experience different levels of severity of impairment that may significantly interfere with health-related quality of life and functional abilities.

This section outlines the crash risk for individuals diagnosed with Parkinson's disease, multiple sclerosis, cerebral palsy, and spina bifida. The crash risk associated with other neurological conditions such as stroke and traumatic brain injury respectively and the crash risk associated with epilepsy is outlined in section 3.6.

In addition, several studies have investigated the crash risk associated with neurological conditions in general. These findings are presented at the end of this section.

3.8.1 *PARKINSON'S DISEASE*

Definition of Parkinson's disease (PD)

Parkinson's disease (PD) is a chronic and progressive neuro-degenerative disorder, which is characterised by a decrease in spontaneous movements, gait difficulty, postural instability, rigidity and tremor (NINDS, 2001). PD results from the degeneration of nerve cells in the basal ganglia which produce the neurotransmitter dopamine. Reduced levels of dopamine cause the nerve cells to fire out of control, leaving individuals unable to direct or control their movements (European Parkinson's Disease Association, EPDA, 2002; WHO, 1998). Parkinson's *Syndrome* is a similar clinical entity which may be drug induced or result from brain injury from ischaemic events.

PD produces four major symptom complexes:

- tremor (shaking);
- bradykinesia (slowness of movement);
- postural instability or impaired balance and coordination;
- rigidity (stiffness).

Individuals with PD may also experience a number of secondary symptoms. These include: depression, sleep disturbances, dizziness, and dementia (EPDA, 2002).

The severity or stage of the disorder is commonly assessed using the following instruments:

Hoehn and Yahr (H & Y) Staging of Parkinson's Disease:

- Stage One: signs and symptoms on one side only; symptoms mild; symptoms inconvenient but not disabling; usually presents with tremor of one limb; and friends have noticed changes in posture, locomotion and facial expression;

- Stage Two: symptoms are bilateral and minimal disability; posture and gait affected;
- Stage Three: significant slowing of body movements; early impairment of equilibrium on walking or standing; and generalised dysfunction that is moderately severe;
- Stage Four: severe symptoms; can still walk to a limited extent; rigidity and bradykinesia; no longer able to live alone; and tremor may be less than earlier stages;
- Stage Five: individual cannot stand or walk; and requires constant nursing care.

This rating system has been largely superseded by the Unified Parkinson's Disease Rating Scale, a more complicated assessment scale.

Unified Parkinson Disease Rating Scale (UPDRS):

The UPDRS is a rating tool to follow the longitudinal course of PD. It is made up of the assessment of: 1) Mentation, Behaviour, and Mood, 2) Activities of Daily Living (ADL) and 3) Motor functioning. Some sections require multiple grades assigned to each extremity. A total of 199 points are possible: 199 represents the worst (total) impairment and 0 represents no impairment.

Prevalence of PD

Identifying accurate prevalence estimates of the number of people with PD is difficult, especially in the early stages of the disease because many individuals attribute early symptoms to the "ageing process". PD is currently ranked as the fourth most frequent disorder of the nervous system, after epilepsy, cerebrovascular disease and Alzheimer's disease (WHO, 1998). It is estimated that the worldwide prevalence of PD is 6.3 million people (EPDA, 2008). The WHO estimates that the prevalence of PD is approximately 5.1 million worldwide (Mathers et al., 2002). In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at almost 1 million or around 0.03% of the total population. Similarly, the prevalence of PD in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 1.3 million or around 0.03% of the total population. In 2001 the prevalence of PD in Australia was estimated to be 104 per 100 000 regardless of age (Mehta et al. 2007).

The prevalence of PD increases with age: PD affects approximately 1% of individuals over the age of 65 years of age and increases to 2% in the population aged 70 years and older (Parkinson Society Canada, 2002). Although the incidence of PD is higher in the elderly population, it should be noted that approximately 10% of individuals diagnosed with the disorder are under the age of age 50 (EPDA, 2002). Prevalence rates by age group for the UK are listed below in Table 23 (Schrag et al., 2000). In consideration of the increased life expectancy worldwide, an increasing number of people are expected to develop PD (WHO, 1998).

Table 23: Prevalence rates of Parkinsons' Disease by age group for the UK

Parkinson's Disease	Total no. of people per 100 000
0-29	0
30-39	2
40-49	4
50-59	17
60-69	53
70-79	91
80+	68
Total	235

Functional impairments associated with PD relevant to driving

As outlined previously, the clinical appearance of PD is marked by four key symptoms: tremor, bradykinesia, rigidity and postural instability. These symptoms are outlined in detail below.

Tremor

Tremor at rest is the most recognised symptom of PD and is present in approximately 75% of individuals diagnosed with the disorder (NINDS, 2001). While tremors are one of the most obvious motor symptoms of PD, they are typically considered to cause the least amount of functional impairment. The tremor evident in PD is distinctive: It is a slow and rhythmic movement that is apparent when the limb is at rest and diminishes with movement. Initially the tremor may appear unilaterally, but eventually as the disease progresses, it may spread to the opposite side of the body (Parkinson Society Canada, 2002).

Bradykinesia

Bradykinesia refers to the slowness and poverty of movement experienced by all individuals diagnosed with PD (Parkinson's Disease Foundation, 2002). It is the ultimate expression of the brain's slowness in transmitting the necessary instructions to the appropriate muscles within the body (Parkinson Society Canada, 2002). Gait is shuffling, facial expression and gestures are lacking, eye blink frequency is decreased and the arms do not swing with walking. With advanced bradykinesia, the gait is paralysed, speech becomes muted and mumbled and swallowing becomes difficult (Parkinson's Disease Foundation, 2002).

Rigidity

Rigidity refers to an increased tone or stiffness in the muscles. Rigidity adds to the problem of bradykinesia, resulting in movements that are stiff as well as slow. Fluidity of movement is lacking and is replaced by hesitancy and even "freezing." (Parkinson's Disease Foundation, 2002).

Loss of postural reflexes

The individual with PD may develop poor posture and balance that may cause falls, gait or balance problems. The body becomes bent at the neck, spine and hips, leading to a stooped posture. The gait is hesitant at the start, followed by short, rigid steps that begin slowly but soon quicken to a peculiar running pace. Stopping can be as difficult as starting (Parkinson Society Canada, 2002).

As the disease progresses, new problems develop as the brain is further depleted of essential neurochemicals. For example, the APA (1994) reports that 20–60% of individuals with PD exhibit a spectrum of cognitive abnormalities, ranging from impairments in specific cognitive domains, to severe dementia.

A number of cognitive impairments have also been implicated in PD (Daum, & Quinn, 1991; Dubinsky, Gray, Hustead, Busenbark, Vetere-Overfield, Wiltfong, Parrish, & Koller, 1991; Gronin-Golomb, Gorkin, Growdon, 1994; Madeley, Hulley, Wildgust & Minham, 1990; Zimmerman, Sprengelmeyer, & Fimm, 1992). These include:

- Executive dysfunction - Individuals with PD often demonstrate executive function deficits (e.g., inability to plan, organise, regulate goal-directed behaviour). Difficulties in generation, maintenance, shifting, and blending of sets characterise executive function disorders, which manifest as mental inflexibility.
- Visuospatial difficulties – On neuropsychological tests, individuals with PD often demonstrate a typical progression of deficits with resulting development of difficulty with line orientation, block design, and picture arrangement.
- Memory deficits – Many individuals with PD demonstrate deficits in declarative memory and abnormalities in procedural memory.

In addition to deficits in visuospatial abilities, memory deficits and executive functioning, it has been shown that individuals with PD have difficulty with internally guided cognition (Georgiou et al., 1993; Berger et al., 1999). An example of internally guided cognition is the generation of a motor plan and the execution of a motor response. In relation to driving one can internally generate an action based upon prior knowledge to adjust driving behaviour to respond to changes in the road environment. Evidence suggests that individuals with PD are more responsive to external cues such as road signs (Stolwyck et al., 2005).

In addition, to the cognitive and physical impairments outlined above, it has been estimated that between 30- 90% of individuals with PD have a comorbid diagnosis of clinical depression (National Institute of Mental Health, (NIMH, 2002)). Some individuals experience this depression intermittently, while others chronically struggle with the mood disorder. It is still unclear whether the depression seen is a secondary reaction to the illness or an endogenous component of the illness (for a detailed description of the crash risk associated with depression and other psychiatric disorders, see section 3.9).

Sleep disturbances are also common in PD (NINDS, 2002; Parkinson's Disease Foundation, 2002). The earliest abnormality is sleep fragmentation or difficulty staying asleep. Reasons for sleep interruptions include pain, urination, stiffness, and difficulty turning in bed. Other problems include vivid dreaming, nocturnal vocalisations and

excessive daytime sleepiness, altered sleep-wake cycle and sudden onset of sleep (for a detailed description of the crash risk associated with sleep disorders, see section 3.11).

There is a lack of agreement about the standard definition of sudden onset of sleep (SOS). It can either be referred to as a “*sleep attack*” or as “*sudden onset of sleep*”. Furthermore, it is debatable as to whether or not a sudden onset of sleep actually exists as individuals with PD are unaware of the sleep signs before the attack (Pacchetti et al. 2003).

In summary, PD is frequently associated with varying combinations and degrees of impaired motor, sensory, and central coordination functions, as well as a spectrum of cognitive deficits (Madeley, et al., 1990). There are many manifestations of PD that may affect driving, including:

- generalised slowness of movement (bradykinesia);
- stiffness of limbs (rigidity);
- gait or balance problems (postural dysfunction);
- cognitive impairment.

Pre-May 2003: Relationship between PD and road safety outcomes

While it is well documented that PD impairs psychomotor and cognitive functions considered necessary for the safe operation of a motor vehicle, only a few studies have examined the relationship between PD and the ability to drive. Table 24 shows a summary of findings of studies on risk and PD.

Crashes

In a survey study by Dubinsky et al. (1991), 150 participants with PD and 100 control participants without PD were interviewed and their driving records and driving habits were compared. Drivers were included if they had two of the four characteristics of PD (rigidity, tremor, bradykinesia, and postural instability), a history of progression, and a responsiveness to levodopa. Controls were excluded if they had evidence of degenerative neurologic disease or if they were under 45 years of age. In order to measure impairment, participants with PD completed the Northwestern University Disability Scale (NUDS) and the Schwab and England activities of daily living scale (where 100 % represents an individual who is completely independent whereas 10 % represents an individual who is totally dependant on others), while the Hoehn and Yahr scale was used to measure the stage of the disease. A 40-question survey of driving habits and the MMSE were administered to all participants. There was a significantly higher crash rate per million vehicle miles of travel for participants with more severe PD (Hoehn and Yahr stage III) than participants with less severe PD (Hoehn and Yahr stage I) and control drivers ($p < 0.001$, Mann Whitney U test). The authors also reported that participants who demonstrated cognitive impairment (i.e., a MMSE score of 23 or less) were significantly more likely to have a motor vehicle crash per million vehicle miles travelled ($M = 93.9$, $SD = 236$) compared to PD participants without cognitive impairment ($M = 28.1$, $SD = 106$, $p < 0.02$). The authors concluded that the presence of cognitive impairment and more severe PD symptoms was significantly associated with an increased crash rate. Dubinsky et al. note that there is obvious bias in the recruitment

of the PD group since only those attending a clinic or support group meeting were approached to participate. Consequently, individuals with very mild or severe PD would have been excluded. In addition, both controls and PD participants travelled to the site of recruitment, introducing a bias against those who do not drive.

Citations

No studies reporting rates of citations or violations amongst drivers with PD were found.

Driving Performance

Heikkila, Turkka, Korpelainen, Kullanranta and Summala (1998) evaluated the driving ability of 20 individuals diagnosed with idiopathic PD and 20 age and sex matched controls using clinical evaluations, cognitive and psychomotor laboratory tests and a standardised on-road driving test. The inclusion criteria were male sex, mild to moderate PD (H&Y stages 1-3), general good health, and regular car driving. Participants with other medical conditions known to affect driving ability were excluded. Heikkila et al. reported that apart from three traffic crashes that had occurred in the PD group during the past two years compared with none in the control group, there were no differences in the driving histories of the members of the two groups. To rule out any on-off effects of medication, assessments and on-road driving tests were performed when the drivers with PD considered that they were at their optimal level of performance.

Both participants with PD and controls underwent computer laboratory tests which included tests of: visual short-term memory, perceptual flexibility and decision making, vigilance-continuous vigilance, complex choice reaction time, information processing capacity and reactive stress tolerance test. The on-road test was performed both in urban and rural surroundings on a standard and relatively difficult route. Two levels of errors were classified on the basis of their severity: 1) risky faults which could lead to danger, and 2) serious infringements of traffic regulations.

On all laboratory tests, participants with PD performed significantly worse than control participants. The differences were most pronounced in the tests for visual memory and choice reaction time. In addition, drivers with PD demonstrated impaired information processing capacity in complex situations. Heikkila et al. concluded that cognitive and psychomotor impairments are even evident in the mild to moderate stages of PD.

In the on-road test, drivers with PD committed significantly more “risky” manoeuvres and serious infringements than controls. In terms of faults, driving in a traffic flow was a considerably more difficult task for the participants with PD ($M = 3.9$, $SD = 2.4$) than for controls ($M = 1.6$, $SD = 1.4$; $p < 0.05$), as well as turning across traffic (PD group: $M = 1.7$, $SD = 2.1$; controls: $M = 0.6$, $SD = 0.6$, $p < 0.05$). The PD group's problems in driving appeared mostly in urban conditions. Disease indices (such as duration of disease, the Hoehn and Yahr scale, and the MMSE score) and dose of medication was not significantly linked to performance on the driving test.

The authors concluded that the driving ability of participants with even mild to moderate PD was clearly impaired and that the highly complex task of evaluating the driving ability of PD participants requires both psychological and psychomotor tests, and/or an on-road driving test. Methodological limitations of this study include a small

sample size. In addition, participants were excluded if they had very severe PD (H & Y stage 4 and above) and therefore the reported crash risk may be an underestimation of the actual risk for those with severe PD.

In 2002, Zesiewicz, Cimino, Malek, Gardner, Leaverton, Dunne and Hauser compared the driving ability of 39 PD drivers with 25 control participants using a driving simulator. Participants completed a Mini-Mental State Examination (MMSE) and a self-report questionnaire regarding driving history, including number of miles driven per month. Participants with PD were also evaluated with the Unified Parkinson's Disease Rating Scale (UPDRS) and Hoehn and Yahr (H&Y) scale by a movement disorder specialist immediately prior to testing. Control participants were neurologically and cognitively "normal" by self-report. The authors reported that miles driven did not significantly differ between the two groups ($t = -1.4, p = 0.10$). However within the PD group, 7 reported having stopped driving, 10 reported a decrease in the amount of driving, and 22 reported no change in driving habits. PD drivers who stopped driving had significantly lower MMSE scores ($M = 23.6 \pm 4.9$) than PD drivers who reported no changes in amount of driving ($M = 28.6 \pm 3.2$), PD drivers who decreased their driving ($M = 28.1 \pm 1.8$), and control drivers ($M = 29.7 \pm 0.9$) ($F = 10.1, p < 0.001$).

The group of all PD drivers (including those who limited their driving and those who stopped driving) had more collisions on the driving simulator than control drivers ($t = -3.7, p < 0.01$). PD drivers who were still driving (including those who had no change in driving and those who had limited their driving) had more simulator collisions than control drivers ($t = -3.1, p < 0.01$). When considering only those PD drivers who reported no change in driving, a trend was observed for these drivers to have more collision compared to control drivers ($t = -1.9, p = 0.08$). The percentage of PD drivers involved in one or more simulator collisions was associated with Hoehn and Yahr stage ($\chi^2 = 12.4, p < 0.001$). Furthermore, simulator collisions were also correlated with UPDRS motor score ($r = 0.5, p < 0.01$). Finally there was a trend for a significant correlation between collisions and MMSE scores in PD drivers ($r = -0.3, p = 0.06$).

Zesiewicz et al. (2002) concluded that drivers with more advanced PD were more likely to have a collision in the simulator than PD drivers with less advanced PD. While the authors noted that control participants were screened for cognitive and neurological disorders, they did not report whether drivers with PD were screened for other comorbid neurological conditions which may also affect their driving.

In 1992, Lings and Dupont conducted a controlled laboratory investigation of driving ability in individuals with PD. Using a mock car, they compared the performance of 28 drivers who had been diagnosed with PD (median age = 65) who reported that they were on optimal medications regimens and who did not have other complicating disorders with 109 younger controls (median age = 49). The authors reported that participants in the PD group were more likely to fail to react to stimuli such as a red light, more likely to have a high frequency of erroneous reactions (particularly directional errors), reduced speed and strength of movement, and prolonged reaction times. The results did not change when participants without a driver licence were excluded. The authors also noted the following observations: 21 participants could not adhere to the testing schedule because after reacting to a signal they were not ready to continue for some time; for 7 participants it was necessary to urge them verbally, and 5 participants failed to react at all, on at least one occasion. One major methodological limitation of this study is that the authors used a considerably younger control group,

and therefore it is impossible to determine if the difficulties demonstrated by drivers with PD were in fact due to the disease or just the natural ageing process.

Madeley et al. (1990) used a driving simulator to examine the effect of PD on driving ability. Participants included ten drivers diagnosed with PD who were volunteers from another longitudinal study and 10 healthy controls who were matched on age and sex. A further four participants with PD who were no longer driving were also included. In order to rule out “on-off” effects of medications, PD drivers were tested when they felt they were at their optimal level. The following outcome measures were generated from the driving simulator: simple and driving reaction times (in seconds), accuracy of steering (where lower scores indicated better performance), and number of red lights missed. The PD drivers were also rated on the Webster’s rating scale for severity of motor impairment (which contains 10 items including ratings of rigidity, bradykinesia, posture and gait). Mann-Whitney tests revealed that although there was no significant difference between the two groups in terms of simple reaction time ($U = 34.5, p = 0.12$), PD drivers had significantly impaired steering accuracy ($U = 21.0, p < 0.05$), slower driving reaction time ($U = 17.0, p < 0.01$) and missed more red lights ($U = 24.0, p < 0.01$). The authors also reported a significant correlation between the severity of PD measured by Webster’s rating scale and simulated driving reaction time ($r = 0.53, p < 0.05$), steering accuracy ($r = 0.78, p < 0.01$) and simple reaction time ($r = 0.63, p < 0.01$). However, there was no significant correlation with the number of red lights missed ($r = 0.05, p = 0.44$). The authors concluded that even PD drivers with moderate impairment will require careful consideration regarding their safety to drive. Madeley et al. (1990) noted that the sample in this study could be affected by an element of selection bias, in that the PD drivers who volunteered to participate may have been confident about their driving ability and other people diagnosed with PD who were less confident may have been less likely to volunteer. As outlined in Chapter 2, caution should also be exercised in extrapolating these simulator results to real world driving situations.

Treatment of PD and road safety outcomes

There is currently no known cure for PD and therefore treatment is aimed at controlling the symptoms (NINDS, 2001). PD is a highly complex neurological disease with an even more complex set of medications. There are several different groups of medicines that can be used by themselves or in combination with other drugs (Hubble & Berchou, 2003).

As noted previously, most symptoms of PD are attributable to the lack of dopamine within the basal ganglia of the brain. Thus, the majority of anti-Parkinson drugs are aimed at temporarily replenishing or mimicking dopamine (Parkinson Society Canada, 2002).

Administration of the drug Levodopa has been the standard and most effective treatment for PD (EPDA, 2002). Once it reaches the brain, Levodopa is converted to dopamine and is stored in nerve cells to replace the depleted dopamine. The drug reduces the tremor and muscle rigidity and improves movement (Parkinson Society Canada, 2002).

It is important to note that Levodopa preparations are not without side effects particularly with prolonged use over many years. The most common include nausea, vomiting, low blood pressure, dyskinesias (abnormal, involuntary writhing movements),

restlessness and, rarely, confusion (Hubble & Berchou, 2003; Parkinson's Disease Foundation, 2002; Parkinson Society Canada, 2002). Adverse effects of treatment such as dyskinesias may occur at any time, but are more common when the medication reaches its peak effect, typically 60-90 minutes after a dose (EPDA, 2002). Daytime sleepiness also occurs in some people early in therapy however these side effects typically subside over time (Parkinson's Disease Foundation, 2002). In addition, cells which react to dopamine and related neurotransmitters are present not only in those parts of the brain effected by PD, but throughout much of the nervous system, and consequently, dopaminergic drugs can overstimulate other cell groups, causing adverse side effects such as hallucinations (Hubble & Berchou, 2003). The simultaneous administration with levodopa of substances inhibiting the conversion of levodopa to dopamine in the peripheral tissues (e.g., carbidopa) allows a higher concentration of levodopa to reach the brain and also considerably decreases the side effects.

In addition, individuals with PD may experience unpredictable fluctuations in their symptom control, shifting from full-symptom control ("on-time") to periods of reduced voluntary movement ("off-time") (Hubble & Berchou, 2003). The most common time for an individual to experience an "off" episode is when their medication is losing its effect prior to time for the next dose. Altering the dosage or frequency of Levodopa may reduce fluctuations in motor control.

Despite potential side effects and fluctuations in motor performance that occur over time, carbidopa/Levodopa remains the gold standard in the treatment in PD (Hubble & Berchou, 2003).

Crashes

Over the past few years, there has been some concern that dopamine drugs commonly used in the treatment of PD may cause "sleep attacks": sudden episodes of falling asleep without warning, without being drowsy, similar to those described in narcoleptics (for a more detailed description of the crash risk associated with sleep disorders, see section 3.11).

The view that drivers with PD are particularly liable to have unforwarned sleep attacks at the wheel was largely initiated by Frucht, Rogers, Greene, Gordon and Fahn (1999). Among PD drivers monitored at three movement disorders centres, Frucht et al. identified 8 male individuals with PD who experienced sudden "sleep attacks" while driving and who had subsequently sustained automobile crashes. All 8 were receiving pramipexole. Five of whom apparently had no forewarning. The authors reported that none of these sleep attacks resulted in any injury. The authors concluded that pramipexole and ropinirole were responsible for the sleep attacks because all attacks occurred after participants began taking pramipexole or ropinirole and stopped after the drugs were discontinued. It should be noted that the Frucht et al. made no comparisons with age related healthy controls, to determine the extent that falling asleep at the wheel could have been due to normal ageing. Also, they provided no information about other individuals with PD who drive, but had no such attacks. The authors attributed the presumed sleep attacks to dopamine agonists, and that withdrawal of these drugs alleviated such attacks. Of course, drivers who have had the misfortune to fall asleep at the wheel usually are more careful not to allow this to happen again. So it is possible that in these drivers the likelihood of a further "sleep attack" whilst driving would have diminished anyway, with or without this medication being continued. Finally, although Frucht et al. reported that none of these individuals with PD had any history of sleep

disturbance, none was actually examined for this, and the evidence is only based on the participants' own opinions. As outlined in previous sections, this form of obtaining data is most unreliable.

In 2002, Homann, Wenzel, Suppan, Ivanic, Kriechbaum, Crevenna and Ott, conducted a review of publications between July 1999 and May 2001 in which sleep attacks or narcoleptic-like attacks were discussed in individuals with PD. Overall, 6.6% of individuals taking dopamine agonists who attended movement disorder centres had sleep events. Men were over-represented. Sleep events occurred at both high and low doses of the drugs, with different durations of treatment (0-20 years), and with or without preceding signs of tiredness. The authors concluded that sleep attacks are a class effect (i.e., depend on the type of medication), having been found in individuals with PD taking the following dopamine agonists: levodopa (monotherapy in 8 participants), ergot-based dopamine agonists (apomorphine in 2 participants, bromocriptine in 13, cabergoline in 1, lisuride or pramipexole in 23, pergolide in 5,) and non-ergot agonists (pramipexole in 32, ropinirole in 38). Reports suggest two distinct types of events: those of sudden onset without warning and those of slow onset with drowsiness. However, the authors concluded that there was insufficient data available to provide effective guidelines for prevention and treatment of sleep events in drivers taking dopamine agonists for PD and that prospective population based studies are needed to provide this information.

Post-May 2003: Relationship between PD and road safety outcomes

Since 2003 there has been considerable growth in the number of studies that have assessed predictors of road safety outcomes for drivers with PD. The majority of studies have focused on the neuropsychology predictors of driving performance, and have included driving simulator and on-road environments to assess motor, cognitive and visual skills specific to drivers with PD. Two studies addressed the risk of crashes amongst people with PD, specifically relating to sleep disorder in this population.

Crashes

Recently, driving studies in Parkinson's disease have started to focus on the relationship between excessive daytime sleepiness and crash risk. For example, Ghorayeb et al. (2007) investigated the prevalence of excessive daytime sleepiness and the occurrence of sudden sleep onset while driving in a sample of 1625 individuals with PD. Approximately half of the participants were regular drivers, and were recruited via a random sample of 400 neurologists. The neurologists were asked to select the first ten patients with PD who were eligible to participate in the study. Exclusion criteria consisted of a depressive illness, a cognitive MMSE score below 24, a daily living score above 50%, or a Hoehn and Yahr score greater than 4. The researchers obtained demographic information regarding driving habits, traffic crashes and medication history. The Epworth Sleep Scale (ESS) and a questionnaire about dozing off while driving were also administered to the participants in order to assess sleepiness. An ESS score ≥ 10 (indicative of excessive daytime sleepiness) was found for 29% of the sample, however ESS scores did not differ between drivers and non drivers. Also, ESS scores did not significantly differ according to demographic characteristics, or the mean number of crashes between drivers ($M = 7.3$, $SD = 5.2$) and non drivers ($M = 6.9$, $SD = 4.5$). The authors reported that reduced daily activity living scores independently predicted excessive daytime sleepiness ($p = 0.02$, OR, 0.98 95% CI, 0.97-0.99), as did daily Levodopa dosage ($p = 0.04$, OR, 1 95% CI, 1-1.05) and male gender ($p < 0.001$,

OR: 1.7, 95%CI 1.29 - 2.25). It is of interest to note that H and Y scores of disease severity were not predictive of excessive daytime sleepiness.

The chance of dozing while driving (never, slight, moderate, or high) was rated as slight to high by 16% of the sample and high by 0.8%. Results from a linear regression revealed that male gender ($p < .008$, OR: 1.83, 95%CI 1.42 - 4.27), a low H and Y score ($p = .002$, OR: 0.61, 95%CI 0.4 - 0.93), and an ESS score ≥ 10 ($p < 0.001$, OR 5.7, 95%CI 3.79 - 9.02), were predictive of dozing while driving. There was no significant distinction between ESS scores for the small number of participants (3.1%) who were previously involved in a traffic crash ($M = 7.3$, $SD = 5.2$) compared to those without an crash history ($M = 6.9$, $SD = 4.5$). The authors concluded that being male, taking a high dose of Levodopa, and having a reduced daily living activity score was predictive of excessive daytime sleepiness. Furthermore, excessive daytime sleepiness, a low disease severity score and being male predicted sudden onset of sleep episodes while driving. One of the main methodological limitations of this study was the small number of crashes reported by the sample, and the fact that the crash history period was not defined in the article. Furthermore, the sample did not include participants in the severe stages of the disease who may have experienced a greater number of traffic crashes. While the study failed to show an association between daytime sleepiness amongst drivers with PD and crashes, it will be important to explore this in future studies with a larger sample across different stages of disease severity.

Meindorfner and colleagues (2005) were also interested in the prevalence of sudden onset of sleep episodes (SOS), associated motor vehicle crashes and driving habits of drivers with PD. The study comprised an impressive sample of 6,620 patients with Parkinson's disease who were members of the German Parkinson Association. Forty percent of the sample had ceased driving and the remaining 60% were active drivers. The mean age of the sample was 68.5 years, and included 40.1% females and 59.9% male. A questionnaire containing information relating to driving habits, driving experience, driving exposure, disease duration and severity (using the H and Y scale), current medication and demographic characteristics was administered to participants. In addition, participants were asked whether or not they were involved in or had caused a motor vehicle crash in the last 5 years. In order to assess sleepiness participants completed questions from the Epworth Sleepiness Scale. In particular, the authors were interested in those participants who either experienced a sleep attack while driving ($n = 58$), reported an SOS while driving ($n = 134$), or had a crash in the past five years but never had a SOS at the wheel while driving ($n = 147$). Survey results indicated that 10.8% of the sample had caused as least one crash, while 14.5% had been involved in one crash while driving. When this statistic was compared to the age and crash rates in the German population, younger people with PD were found to be more likely to be responsible for causing the crash. Older drivers with PD in the study mimicked the general population trend of PD crashes increasing with age. A total of 8% of participants with current driving licences had experienced SOS at the wheel, and 28% of these people were involved in a crash resulting in mild or severe injuries.

One of the strengths of the study was the addition of specific details about the nature of the reported crashes. When the researchers compared crashes related to SOS compared to those without, the SOS accidents typically involved single vehicles where the participant drove off the road. In contrast, non SOS related crashes involved multi-vehicles, which typically occurred at cross roads or in parking lots. It is of interest to note that people who reported a sleep attack while driving generally reported a change

in their medication before their attack. Logistic regression analyses showed significant predictors for crash involvement were SOS at the wheel (OR: 3.16, 95%CI 2.33 - 4.30), disease severity (OR: Mod vs. Minor: 1.42, 95%CI 1.12 - 1.81; and Adv vs. Minor: 1.51, 95%CI 1.05 - 2.18) and annual distance driven (OR: 1.49, %CI:1.18-1.88). The regression models for crash causation showed significant predictors were higher ESS scores (OR: 1.61, 95%CI 0.97 - 2.68) (in addition to SOS at the wheel, OR:3.54, 95%CI 2.46-5.08) and a reported moderate disease severity (OR:1.45, 95%CI, 1.09-1.92). Age, sex and time since disease onset were not predictors of crash causation or involvement. In conclusion, the authors noted that SOS is a contributing factor to driving accidents experienced by people with PD. Major limitations of the study are the lack of a control group and the reliance on self-report data.

Citations

No studies were found since 2003 that investigated the relationship between PD and driving citations.

Driving Performance

In 2007, Amick and colleagues investigated the relationship between on-road driving performance and performance on cognitive and vision tests. Specifically, the authors hypothesized that contrast sensitivity would be a strong predictor of on-road driving performance in a sample of 25 participants with PD. Participants (aged between 54-72 years) comprised 17 males and 8 females. The inclusion criteria consisted of; a diagnosis of PD by a movement disorder specialist, presence of two or three cardinal manifestations and response to dopaminergic medication. All participants were current drivers and did not suffer from any psychiatric, neurological or physical disorders that could affect their driving. In addition, participants were screened for depression and dementia. Driving exposure, level of education, disease duration and disease severity were accounted for. Participants completed an on-road driving assessment and were either classified as a safe (60% of sample) or marginal (40%) based upon completion of a road test which was scored by a driving instructor. In addition to the driving assessment participants completed a battery of visual and cognitive tests. The tests included; Rey-Osterreith Complex Figure Test (ROCF), Trail making tests A and B (TMT-A and B), Useful Field of View (UFOV), Backwards visual masking, Functional Acuity Contrast Test (FACT), and the Pelli-Robson test of contrast sensitivity.

The authors compared performance measures between marginal and safe drivers. Significant group differences were found for TMT-B and FACT performance as a consequence of poor contrast sensitivity amongst marginal drivers. On-road driving performance correlated with performance on the divided attention component (subtest three) of the UFOV ($r = 0.49, p = 0.01$), TMT-A and B ($r = 0.49, p = 0.01$) and the ROCF ($r = -0.47, p < .005$). These findings suggest that tests requiring visuospatial skills, executive function, and rapid responding were the most predictive of on-road driving performance in this study. The authors concluded that neuropsychological tests which assess visual perceptual skills are better predictors of driving behaviour in people with PD compared to tests of contrast sensitivity and visual attention. One limitation of the study is the lack of experimental control in an on-road environment which increases the contribution of external confounding variables.

In a study published in the same year Amick and colleagues (2007b) explored the association between excessive daytime sleepiness (EDS) and on-road driving in

Parkinson's disease. The sample consisted of 21 current drivers with PD who were recruited from a movement disorders clinic in Rhode Island. The exclusion criteria consisted of a movement disorder characterised by the presence of two out of three cardinal manifestations, as well as an appropriate response to dopaminergic medication. Participants were not eligible to participate if they suffered from a psychiatric illness, neurological disorder or ophthalmological disorder, or had substance abuse within the previous year. The authors collected demographic information about education level, age, disease duration, disease severity as determined by the UPDRS and H and Y scale, cognitive status measured by an MMSE score, medication dose and sleepiness (using the Epworth Sleepiness Scale (ESS)). The researchers classified the participants according to scores on the ESS, where a score greater than ten implied the presence of excessive day time sleepiness. All participants completed an on-road driving test which was conducted by a driving instructor during daylight hours. Driving test performance resulted in a global rating of safe, marginal or unsafe.

A total of five out of the twenty-one participants were found to have EDS. These five people with ESS did not significantly differ according to driving safety rating compared to those without ($p = .56$, Fisher's Exact Test). Furthermore, all participants who were identified as suffering from EDS were taking DA medication. However levels of DA medication did not affect global safety levels of driving performance ($p = .56$, FET). The authors stated that in their study excessive daytime sleepiness and dopamine medication did not impact upon driving performance in patients with PD. There are a few methodological limitations of the study which could be addressed. Firstly, as noted by the authors, the measure of daytime sleepiness using the ESS questionnaire is not ideal as it is a self-report measure. A more objective measure of daytime sleepiness would have been more reliable. In addition, the authors suggested that participants may be susceptible to the Hawthorne Effect during the experimental drive. For example, participants are likely to be more alert when driving with an instructor as a part of a study than in their usual driving environment. Caution should be taken when interpreting the results due to the small sample size as well as the small number of drivers who suffered from EDS.

Devos and colleagues (2007) conducted a prospective case-control study investigating the visual and cognitive predictors of fitness to drive. In this study 40 participants with PD and 40 controls matched for age and gender completed a driving history survey, a simulator drive and the Clinical Dementia Rating. PD participants also underwent an assessment of fitness to drive which was obtained from cognitive assessments and an on-road assessment. Participants with PD were recruited from a movement disorder clinic and the PD society. The inclusion criteria consisted of; a diagnosis of PD based on the UK Brain Bank Diagnostic Criteria, a Hoehn and Yahr severity score of 1-3, a score ≤ 1 on the Clinical Dementia Rating scale, 20/20 vision obtained using the Snellen visual acuity chart, and a current driver's licence. Participants were ineligible to participate if they had deep brain stimulation implants or had motor fluctuations that were unpredictable. The researchers' collected demographic information in regards to; number of accidents in the past 5 years, driving habits (including driving exposure and experience), penalties, and self rated fitness to drive abilities. Participants completed the ESS, and were asked about medication dosage and disease severity. The test battery consisted of; the Pelli-Robson test of contrast sensitivity, Complex Rey Figure drawing task, an assessment of daily living as measured by the Unified Parkinson's Disease Rating Scale II (UPDRS II), and an assessment of motor deficits derived from the

UPDRS III, the Humphrey Visual Field Analyzer, The Rey Figure, and The Useful Field of View.

The assessment of fitness to drive was conducted by a team of health professionals who utilised the cognitive test scores from patient records to derive an overall score of driving ability. The participants were classified as either a pass or fail. On the basis of these assessments the majority of PD participants were classified as fit to drive (72.5%). However it is important to note that 27.5% current drivers were assessed as unfit to drive. The number of accidents ($p = 0.28$) and traffic violations ($p = 0.58$) reported by the cases did not distinguish between those who passed and those who failed. A logistic regression model was performed and indicated that the Clinical Dementia Rating scale, contrast sensitivity, disease duration and motor deficits (UPDRS III) were the greatest predictors of fitness to drive ($R^2 = .52$).

In the second phase of the study cases and controls completed a simulator task in which traffic accidents and traffic violations were recorded. In addition, participants completed a divided attention task while driving in which they were required to respond to symbols that appeared on the simulator screen. The variables of interest included reaction time, omissions and errors. The authors found that controls performed significantly better on the driving simulator task compared to PD participants. Cases recorded a greater number of total accidents and traffic violations in the driving simulator (Wilcoxon rank sum test = 2.012, $p < 0.0001$), and reported a slower reaction time in the divided attention task (W, $p = 0.01$). Once the driving simulator score was added, the model predicted pass/fail of fitness to drive to a greater extent ($R^2 = .60$), and correctly identified 97.5% of drivers with PD. This study is unique because the global measure of driving performance was derived from the combination of on-road test performance, as well as cognitive and visual test performance. The authors conclude that contrast sensitivity, disease severity, clinical dementia rating and motor deficits are important predictors of fitness to drive in individuals with PD. However, the study is not without its limitations. Caution should be taken when generalising the results of the study as the sample consisted of cognitively intact individuals, who do not represent the true population. Also, the prospective study design means that clinical assessments conducted in the past were evaluated against on-road driving performance conducted at the time of the study. It is likely that individuals would perform differently on the clinical assessments at a later date.

Lee and colleagues (2007) conducted a driving simulator study to firstly examine the differences in driving performance between individuals with PD and healthy controls, and secondly to evaluate the validity of using a driving simulator to assess driving performance of patients with PD. The sample consisted of 50 people (78% male) with idiopathic PD who were recruited from specialist clinics and neurologists, and 150 age-matched controls (81% male). Participants (aged between 60 to 80 years) had not received more than five demerit points in the last two years and were driving more than four hours a week. Exclusion criteria included a Mini Mental State of Examination (MMSE) score less than 26, visual acuity worse than 20/20 vision, and any psychiatric or medical condition. The study consisted of three components; a battery of clinical tests, an on-road driving assessment and a driving simulator assessment. Participants with PD were assessed during their optimum response to medication period. The clinical measures that were administered to the cases were; the timed up and go test which is a measure of motor function, the Hoehn and Yahr test of disease severity, the Unified Parkinson's Disease Rating Scale (UPDRS), the MMSE and the IQ code for

dementia. Disease duration, driving exposure and driving experience were also taken in to account.

The on-road assessment was conducted by both a driving instructor and a driver trained occupational therapist who graded a series of driving tasks to produce an overall road assessment index. The simulator driving task consisted of a 20 minute drive in an interactive PC based STISIM driving simulator. The variables of interest included; speed, lane position, use of the indicator, spotting distance, rear mirror use, observing traffic rules and the ability to perform two tasks simultaneously. Similar to the on-road assessment a simulated driving index was generated. The control group were found to perform better in the on-road driving test compared to the PD patients ($t(180) = 84.2, p < 0.0001$) although the groups did not differ according to driving experience or driving exposure. In particular, PD patients were slower to respond to hazards, displayed greater variations in speed and inconsistent brake applications, and had a greater number of collisions at roundabouts. Furthermore, PD participants had difficulty responding to changing traffic lights. PD participants also performed significantly worse on the simulator driving test compared with controls ($t(180) = 104.6, p < 0.001$). A simulated driving performance index was created and combined with the road assessment index in a linear regression model to predict driving performance for each participant group. After age, gender, driving exposure and disease duration were accounted for the model was found to be significant ($p < 0.01$). The simulated driving index explained nearly 40% of the road assessment index for PD participants and 68% for the control participants. The authors concluded that PD patients were poorer drivers than healthy controls and stated that driving simulators may be valid tools for assessing driving difficulties in patients with PD. It is important to note that the majority of PD participants were rated with an H and Y test score of low disease severity which restricts the range of participants with PD symptoms. Furthermore, the authors note that driving simulator stress and unfamiliarity with technology could have contributed to the poorer driving outcome behaviours of people with PD. The sample was also limited by the small number of female participants.

Singh et al. (2007) examined whether cognitive and clinical tests could predict on-road driving performance in a sample of 154 participants with PD. The researchers obtained the medical records of patients with PD who were referred to the Scottish Driving Assessment Service from 1989 – 2004. The mean age of the sample was 67.6 years and comprised 13% females. A total of 17 (10.9%) participants were no longer driving however they still completed the on-road driving assessment. Approximately half the participants (46.1%) suffered from another medical condition that could have affected their driving such as stroke. The most common medication prescribed was Levodopa with a peripheral dopa decarboxylase inhibitor (70.8%). The medical files included demographic information on; disease onset and severity as determined by the Hoehn and Yahr scale, as well as driving and medication history. The cognitive assessments included; Trails A and B, MMSE, forward and reverse digit span, sign recognition, visuospatial construction and story recall. Braking response time was recorded using a driving rig which required the participant to brake with the right and then left foot.

Participants were classified as suitable or unsuitable to drive from the results of the clinical, cognitive and on road driving assessments. Approximately one third of participants were classified as unfit to drive. The researchers then compared the suitable and unsuitable driver groups against each of the clinical and driving measures. The two groups were found to differ significantly according to disease severity ($X^2(2, N = 154)$

= 80, $p < .001$), duration of illness (MW, $z = -4.3$, $p < .001$), the presence of other medical conditions ($\chi^2 (1, N = 154) = 5$, $p < .026$), as well as the on-road driving assessment score (MW, $z = -7.4$, $p < .001$). A stepwise discriminant analysis was conducted to ascertain the factors that predicted fitness to drive. These factors included an H and Y score of stage 3 ($p < .001$), the on road driving score ($p < .001$), and an H and Y score of 2 when associated with reaction time and another medical condition ($p = .008$). It was concluded that the clinical test of disease severity, the duration of the illness and the presence of co-morbid conditions that affect driving ability all contributed to fitness to drive in patients with PD in this study. Although the study offers support for the linear relationship between disease severity and fitness to drive, it is not without its limitations. The authors claim that the on-road assessor was not blinded to the results of the clinical tests conducted in a previous session therefore there will be some bias. In addition, the prospective clinical assessments were compared to an on-road assessment conducted at the time of the study. Therefore, clinical scores will not necessarily be the same at the time the on-road assessment was conducted.

Uc and colleagues (2006a) were also interested in the cognitive predictors of on-road driving performance in Parkinson's disease, however their studies also evaluated the impact of a distraction task on driving. Their studies consisted of cognitive assessments, a distraction task, and an on-road driving task. PD participants were recruited from a movement disorders clinic in Iowa and consisted of 57 men and 14 women. Disease severity consisted of Stage 1 and 2 was assessed by the Hoehn and Yahr scale, and MMSE scores ranged from 22 to 30. Participants were not excluded from participating if there were any signs of cognitive impairment, however all participants recorded an MMSE score > 26 . A healthy age matched control group consisted of 147 people free of any psychiatric or medical conditions with 20/20 vision. All participants in the study were active drivers. The PD participants completed the test battery during their "on" time, and completed the; UPDRS, ESS, Geriatric Depression Scale, and the Schwab-England Activities of Daily Living. The visual ability of PD and control participants was assessed by the administration of tasks requiring contrast sensitivity, visual acuity, visual perception and Useful Field of View (UFOV). In addition, visual cognition was evaluated via the Rey-Osterreith Complex Figure Test Copy version, and the WAIS-III Block Design subset, and the CFT RECALL version of the Benton Visual Retention Test (BVRT) which is a measure of visual working memory. Executive functions were assessed using the TMT-A and B and the Rey Auditory Verbal Learning Test (AVLT). The distraction task was the Paced Auditory Serial Addition Test (PASAT) and was implemented twice, once before and once during the drive. The PASAT lasted for approximately two minutes and required participants to add pairs of serial numbers. The on-road driving test was conducted in an instrumented vehicle which recorded speed, accelerator and brake pedal position, steering wheel position and lateral position. The drive was administered on a straight stretch of road free of any challenging driving manoeuvres.

PD patients had significantly poorer (Median = 0.065) visual acuity compared with controls (Median = 0), ($p < .001$), and also displayed poorer performance (Median = 822) than controls (Median = 608) on the UFOV. Furthermore, a greater difference in TMT-B and A scores were found for PD patients (Median = 58.1) compared to controls (Median = 37.4). The PD patients ($60.9 \pm 19.3\%$) were also found to perform significantly worse than controls ($68.0 \pm 20.7\%$) on the PASAT for both the off road assessment. ($p < .05$), as well as during the experimental driving session ($50.9 \pm 17.8\%$ vs 58.1 ± 19.6 , $p < .01$). However, both groups scores decreased when they completed

the PASAT in the experimental session compared to the off road session. An experimenter sitting in the front of the instrumented vehicle recorded the number of at-fault safety errors made by the participant. It was found that the number of safety errors committed by the PD group was higher than controls for both the baseline drive and the drive which included the PASAT. However the PASAT did not significantly distinguish between safety errors in either participant group. A logistic regression revealed that for the PD group one error versus no errors in the baseline drive increased the chances of committing an error during PASAT by 177% (OR: 2.77, 95%CI 1.25 - 27.78), $p = 0.025$). When two errors were committed it increased the odds by 1530% (OR: 16.3, 95%CI 3.93 to 67.5), $p < .0001$). The predictors of committing a safety error during PASAT were determined and included MMSE (OR: 1.40, 95% CI = 1.06 to 1.87), $p = 0.020$), the TMT (B-A) (OR: 1.27, 95%CI 1.03 - 1.52) per 30 second increase, and the BVRT (OR: 1.15, 95%CI 1.01 - 1.30) per one more recognition error, ($p = 0.035$). In terms of driving performance PD participants drove at a slower speed and displayed greater variability in speed than controls.

In summary, the secondary identification task impaired PD patient driving performance to a greater extent than it did controls. The authors suggested that impaired cognitive functions rather than motor dysfunction were responsible for poor driving performance in PD, particularly in the distraction tasks that required additional cognitive load. The lack of an association between safety errors and clinical tests could be due to the small number of safety errors committed by the controls ($M = 0.45$, $SD = 0.81$). It should be noted that the sample was restricted to participants with mild to moderate disease severity. Therefore, the sample is not a true representation of all drivers with PD and caution should be taken when generalising to a broader population of interest.

A similar approach was used in a study by Stolwyk and colleagues (2006a) who firstly investigated the differences in driving behaviour between PD participants and controls, and secondly evaluated how performance on a concurrent task influenced driving behaviour. The sample consisted of eighteen participants with PD and eighteen healthy age matched controls who were recruited from a movement disorders clinic. All participants were current drivers and the majority of drivers in both the control and case group reported driving 50-200 km per week. Participants were not included if they suffered from a psychiatric illness, had a cognitive impairment as detected by an MMSE score of 23 or less, had a visual or hearing condition, suffered from drug or alcohol abuse, had a neurological impairment. Five control participants reported being involved in a crash within the past five years, compared with 7 participants with PD. The control group was similar to the PD group in terms of age, years of driving experience and level of education. PD drivers were in the mild to moderate stages of PD as assessed by the Unified Parkinson's Disease Rating Scale (UPDRS).

Participants completed a clinical evaluation as well as a number of drives in a fixed base Systems Technology Incorporated (STI) model driving simulator using STISIM Drive software. A concurrent distraction task was administered both before and during the simulator drives. The concurrent distraction task was an auditory task which contained three target sounds. The three target sounds were dispersed within sixteen non-target sounds. Participants were required to turn on the indicator each time they heard two target sounds simultaneously. This occurred on three separate occasions. Participants completed 20 concurrent simulator sessions and 20 non concurrent simulator sessions. The dependent variables consisted of driving performance measures at traffic signals and road curves. In regards to driving performance outcomes PD participants displayed

greater lane position variability than controls ($p = .002$), stopped further away past the lights ($p < .0001$) and started to decelerate later ($p < .001$). The researchers found that on approach to a traffic signal PD participants tended to decelerate later than controls during the concurrent task session ($p = .008$). Although PD participants travelled further past the traffic lights before stopping compared to controls, this was not influenced by the concurrent task ($p = .947$). PD participants were also less accurate ($p = .025$) and slower ($p < .0001$) at responding to target sounds than controls. Traffic signal stopping, mean speed around curves, speed variability and mean lane position were all comparable between participant groups in the concurrent task simulator session. This indicates that both groups adopted a more conservative driving approach in order to respond to the concurrent task. The authors suggested that PD participants may have traded concurrent task performance for driving performance. It should be noted that while driving simulators are useful and safe tools for assessing driving ability, simulated driving does not exactly equate to on-road driving.

In another case-control study Stolwyk and colleagues (2006b) conducted an experiment once again using the STI model driving simulator in order to determine the association between neuropsychological test performance and driving in PD. This study comprised 18 individuals with PD and 18 healthy controls matched for age, education level and driving experience. This study is unique because group differences in driving behaviour were examined with respect to each type of driving behaviour within the driving task (i.e. traffic signal approach speed) as opposed to the number of errors or pass/fail criteria. The driving performance measures included; traffic signal approach, traffic signal deceleration, traffic signal stopping point, mean speed around curves, effect of curve direction on mean lane position and variability of lateral lane position. The neuropsychological test battery consisted of tests relevant to driving skills such as; TMT-A and B, MMSE, Symbol Digit Modalities Test – auditory version (SDMT), Mean Simple Reaction Time test, Mean Choice Reaction Time, Up and Go test, Brixton Test of set shifting ability, Judgement of Line Orientation Test (JLO), Wechsler Adult Intelligence Scale-III Picture Completion, Digit Span Total, and Block Design.

To investigate the relationship between each driving performance and each neuropsychological test performance. Pearson correlations were conducted within each participant group. For the PD group TMT-A performance correlated with delayed traffic signal stopping, ($r = .495, p = .05$), while SDMIT correlated with delayed stopping at traffic lights ($r = -.587, p = .05$), late deceleration ($r = -.443, p = .05$) and adjustment of lane position to curve direction ($r = -.710, p = .01$). JLO performance correlated with delayed stopping at traffic lights ($r = -.628, p = .01$). Poor performance on Picture Completion was associated with reduction in the ability to maintain lane position around a curve ($r = -.501, p = .05$) and delayed stopping at traffic lights ($r = -.502, p = .05$). TMT-B correlated with slow approach speed ($r = -.710, p = .01$), late deceleration ($r = -.440, p = .05$), reduced speed around curves ($r = -.496, p = .05$), and maintenance of lane position around a curve ($r = .613, p = .01$). Performance on the Brixton Test correlated with slow speed around curves ($r = -.661, p = .01$), reduced ability to maintain lane position around curves ($r = -.710, p = .01$), delayed deceleration ($r = -.640, p = .01$) and slow approach speed ($r = -.643, p = .01$). Few correlations were found to be significant for the control group. Specifically, TMT-A was associated with mean speed around curves ($r = -.500, p = .05$), while JLO was correlated with approach speed to traffic lights ($r = -.510, p = .05$; $r = -.640, p = .01$) and speed around curves ($r = .562, p = .05$). MMSE and Digit Span performance did not correlate with any driving measures.

While a number of cognitive tests correlated with driving measures for the PD group there was a lack of an association between tests of motor functions and driving performance. Cognitive tests were better predictors of driving performance, specifically tests of information processing, working memory, and set shifting (i.e. TMT-B, SDMT, and the Brixton Test). The authors suggested that there may have been a lack of power in the study due to the sample size to detect any significant relationships between motor functioning in drivers with PD and driving performance. Once again caution should be taken when interpreting the results as there is an assumption that driving performance skills utilised in simulator driving are equivalent to skills used in on-road driving, which is not entirely true.

In a third study Stolwyk and colleagues (2005) investigated the impact of internal versus external cues on driving simulator performance in a sample of people with Parkinson's disease. The sample consisted of the same 18 participants with PD and 18 healthy age matched controls who participated in the previous two studies. A driving simulator task consisted of four conditions comprising the presence or absence of external and internal cues. For the first condition participants memorized a map of the driving route to enable internal cueing. In the second condition internal cueing was not possible as participants completed an unfamiliar driving route and were not allowed to read a map prior to the drive. In addition to the internal cueing, the impact of external cues was assessed by the presence or absence of warning signs which alerted the participant about an upcoming hazard. The outcome variables were the possibility of internal cueing, and the availability of external cues for drivers with PD compared to controls. The researchers investigated the impact of each condition on the driving measures of speed, speed variability, lane position, as well as approach and deceleration speeds.

Stolwyk et al. (2005) found that when external cues were present both controls and cases did not alter their approach speed in response to internal cues ($F(1, 34) = 0.68, p = 0.414$). However, when only internal cues were available the controls decreased their approach speed in response to the cues ($F(1, 17) = 20.71, p < .001$) while PD participants speed remained unchanged ($F(1, 17) = 0.12, p = 0.735$). These findings suggest that controls were more responsive to internal cues than drivers with PD. In relation to overall differences in driving performance PD participants decelerated significantly later than controls for the approach to traffic signals ($F(1, 43) = 21.58, p < .0001$). PD participants also travelled further when stopping at traffic lights compared to controls ($F(1, 34) = 26.76, p < .0001$) and travelled at a slower speed around curves ($F(1, 34) = 7.13, p = 0.012$). No differences in mean lane position were found between the groups. For the PD participants only age contributed to lower traffic signal approach speed ($F(1, 16) = 31.50, p < .0001$), slower deceleration ($F(1, 16) = 5.19, p = 0.037$), and lower mean curve speed ($F(1, 16) = 22.03, p < .0001$). In contrast to previous research UPDRS scores, BAI scales, disease duration, and previous motor vehicle crashes were not significantly related to driving performance measures in this study. The authors concluded that PD participants were more reliant on external cues than controls, even when internal cues were available. This was evident for deceleration and approach speed adjustment. This was particularly obvious at traffic lights where PD participants started to decelerate in response to external cues, while controls started to decelerate on their own accord. Although this is one of the few studies that has addressed the impact of external and internal cues on driving performance in a PD patient group, it is important to note that simulated driving does not fully equate to real world driving in a cohort group involving patients with PD. Therefore, PD participants

may have had greater difficulty adjusting to the simulator environment compared with controls.

In a recent study, navigation ability was assessed using a case-control study design by Uc et al. (2007). Drivers with Parkinson's disease were assessed on the number of safety errors they committed during a route following navigation task. The exclusion criteria included the presence of another medical illness, drug or alcohol abuse, visual acuity less than 20/50 vision. The MMSE scores of the PD group ranged from 22-30. A control group consisting of 152 healthy older adults were recruited to participate in the study. The control group were matched to the PD group on driving exposure and driving experience. All participants were active drivers and completed a testing session including cognitive, vision and motor assessments as well as a driving session conducted in an instrumented vehicle. The testing of PD participants occurred during their "on" time when they were most receptive to their medication.

The battery of tests included; the UPDRS, ESS, Geriatric Depression Scale, Pelli-Robson chart of contrast sensitivity, Snellen chart of near visual acuity, ETDRS chart of far visual acuity, and the UFOV. In addition, visual cognition was evaluated via the Complex Figure Test of copy and recall, and the Benton Visual Retention Test (BVRT). Executive functions were assessed using the TMT-A and B and the Controlled Word Association (COWA) test. Participants also completed the Auditory Verbal Learning Test-recall, the Geriatric Depression Scale, and a Functional Reach Test. The instrumented vehicle experiment included a drive along rural two-lane highways, suburban streets, and a freeway. The drive lasted for approximately 45 minutes and consisted of route following task sections dispersed between baseline driving sections. In the route following sections four different verbal instructions specifying driving directions were provided to the participant by the experimenter at the beginning of the section. Outcome measures included; the number of incorrect turns, the number of times the participant became lost, and the number of at-fault safety errors both on the task and on the baseline sections. In addition, the authors were interested in the time taken to complete the drive and the number of times the instructions had to be recited before participants learned the route.

PD participants performed significantly worse on all motor, cognitive and visual tests compared to controls. PD participants also performed more poorly on the experimental driving task. A greater proportion of people in the PD group (53.9%) made more incorrect turns (21.1%) compared to controls (OR: 2.8, 95%CI 1.4 - 5.7, $p < 0.0001$), and committed more at-fault safety errors (84.2%) than controls (46.7%) (OR: 7.5, 95% 3.3 - 17, $p < .0001$). A greater number of PD participants got lost (15.8%) compared to (2%) controls (OR: 4.7 95%CI 1.1 - 20.0, $p < 0.037$). These findings were significant after adjusting for age, gender, education and familiarity with the driving area. Controls completed the drive in significantly less time ($M = 177$ seconds, $SD = 71$) than PD participants ($M = 238$ seconds, $SD = 111$, $p < .0001$) and took fewer citations of the instructions to learn the route ($M = 2.75$, $SD = 0.86$ vs PD group, $M = 3.92$, $SD = 1.76$, $p < .0001$). PD participants committed a greater number of at-fault errors in the route following section than the baseline section compared to controls, suggesting navigation difficulties. The authors conducted Spearman correlations which revealed that in the PD group at-fault safety errors were correlated with AVTL-RECALL, CFT-RECALL, TMT (B-A), UFOV and MMSE. In contrast, CFT-RECALL, BVRT, TMT (B-A), COWA, MMSE, BLOCKS, CFT-COPY, JLO, UFOV, familiarity with driving area and FVA were predictors of making at least one wrong turn. In addition, MMSE and CFT-

RECALL were predictors of getting lost. It is of interest to note that in this study medication dosage was not found to be related to incorrect turns, getting lost, or the number of at-fault errors. The authors point out that the lack of an association between motor symptoms and driving performance may be due to the non representative sample which included PD patients who were active drivers who do not typically suffer from severe motor symptoms. Furthermore, it is not clear whether the control group was matched to the cases by age which would increase the susceptibility of the study to age effects.

Uc and colleagues (2006a) employed a case-control study design in order to investigate the impact of a distraction task on driving performance in a sample of participants with Parkinson's disease. The sample comprised 71 participants with PD who were recruited from a movement disorders clinic in Iowa and consisted of 57 men and 14 women. Disease severity consisted of Stage 1 and 2 as assessed by the Hoehn and Yahr scale. The MMSE scores ranged from 22 to 30 and participants were not excluded from participating if there were any signs of cognitive impairment. A healthy age-matched control group consisted of 147 people who were free of any psychiatric or medical conditions with 20/20 vision, and all participants were active drivers. The PD participants completed the following tests during their "on" time; UPDRS, ESS, Geriatric Depression Scale, and the Schwab-England Activities of Daily Living. The visual ability of PD and control participants was assessed by the administration of tasks assessing contrast sensitivity, visual acuity, visual perception and Useful Field of View (UFOV). In addition, visual cognition was evaluated using the Rey-Osterreith Complex Figure Test Copy version, the WAIS-III Block Design subset, and the CFT RECALL version of the Benton Visual Retention Test (BVRT) which is a measure of visual working memory. Executive functions were assessed using the TMT-A and B, and the Rey Auditory Verbal Learning Test (AVLT).

Participants also completed an on-road driving test in an instrumented vehicle, which recorded speed, accelerator and brake pedal position, steering wheel position and lateral position. The drive was administered on a straight stretch of road free of any challenging driving manoeuvres. The distraction task was the Paced Auditory Serial Addition Test (PASAT) and was implemented once before the drive, and at different times interspersed throughout the drive. The PASAT lasted approximately two minutes and required participants to add pairs of serial numbers. An experimenter sitting in the seat next to the driver recorded the number of at-fault safety errors. The PD group had significantly poorer (Median = 0.065) visual acuity compared to controls (Median = 0), ($p < .001$), and had greater difficulty (Median = 822) than controls (Median = 608) on the UFOV. Furthermore, a greater difference in TMT-B and A scores were found to exist for PD patients (Median = 58.1) compared to controls (Median = 37.4). The PD patients ($60.9 \pm 19.3\%$) also performed significantly worse than controls ($68.0 \pm 20.7\%$) on the PASAT during the off road assessment ($p < .05$), as well as during the experimental driving session ($50.9 \pm 17.8\%$ vs 58.1 ± 19.6 , $p < .01$). In agreement to the findings of Uc et al. (2006), both groups scores decreased when they completed the PASAT in the experimental session compared to the off road session. This is most likely due to the added complexity of the driving task, which impaired PASAT performance in both groups.

As predicted by the authors the number of safety errors committed by the PD group was higher than controls for both the baseline drive and the drive which included the PASAT. It is interesting to note that the PASAT did not significantly distinguish

between safety errors in either participant group. Although, for the PD group a logistic regression revealed that one error versus no errors in the baseline drive increased the chances of committing an error during PASAT by 177% (OR (95% CI) = 2.77, (1.25-27.78), $p = 0.025$). When two errors were committed it increased the odds by 1530% (OR, (95% CI), = 16.3 (3.93 to 67.5), $p < .0001$). The predictors of committing a safety error during PASAT included MMSE (OR: 1.40, 95%CI 1.06 - 1.87, $p = 0.020$), TMT (B-A) (OR: 1.27, 95%CI 1.03 - 1.52) per 30 second increase, and BVRT (OR: 1.15, 95%CI 1.01 – 1.15) times per one more recognition error, $p = 0.035$. In terms of driving performance PD participants drove at a slower speed, but displayed greater variability in speed than the control group. The authors were interested to find that the PASAT did not distinguish between patient and control groups during the driving task. However, cognitive decline, excessive daytime sleepiness, and verbal memory ability, predicted poorer driving performance for PD patients during the distraction task. The authors acknowledge that the driving task may not have been challenging enough to elicit significant changes in driving behaviour during the PASAT for patients with PD compared to controls.

In the same year, Uc et al. (2006b) investigated the relationship between impaired visual search and cognitive impairments associated with PD, and the ability to identify landmarks and traffic signs while driving. A case-control study design was employed and participants were administered visual and cognitive assessments as well as an on-road driving task in an instrumented vehicle. The sample consisted of 79 participants with PD who ranged from mild to moderate disease severity as measured by the Hoehn and Yahr scale. Participants were recruited from a Movement Disorders Clinic in Iowa and had MMSE scores ranging from 22-30. The PD group included 64 men and 15 women who were current drivers who had been driving for more than ten years. Exclusion criteria included the presence of another medical illness, drug or alcohol abuse, visual acuity less than 20/50 vision. An age-matched healthy control group consisted of 151 people who lived independently and were current drivers. All participants completed a battery of tests which consisted of: the UPDRS, ESS, Geriatric Depression Scale, Pelli Robson chart of contrast sensitivity, Snellen chart of near visual acuity, ETDRS chart of far visual acuity, and the (UFOV). In addition, visual cognition was evaluated via the Complex Figure Test of copy and recall, and the Benton Visual Retention Test (BVRT). Executive functions were assessed using the TMT-A and B and the Controlled Word Association (COWA) test. Participants also completed the Auditory Verbal Learning Test-recall, the Geriatric Depression Scale, and a Functional Reach Test. The testing of PD participants occurred during their “on” time when they were most receptive to their medication.

The on-road driving test was conducted in an experimental vehicle. The order of the cognitive tests and driving test were randomised across participants. The driving test consisted of a series of drive segments both with and without landmark and sign identifications tasks and included challenging manoeuvres such as stopping at signs and completing turns. Drivers were asked to read aloud the signs which differed in levels of saliency. The outcome measures were the percentage of landmarks and signs identified and the number of at-fault safety errors which were recorded by an experimenter. Safety errors were derived from unsafe driving performance behaviours such as lane deviation and unsafe behaviour at intersections. The researchers found that the PD group performed significantly worse on all the visual and cognitive tests compared to the control group. PD drivers committed a significantly higher number of safety errors during the identification task ($M = 1.97$, $SD = 1.56$, $p < .001$) than controls ($M = 0.45$,

$SD = 0.81$). Similarly, PD participants committed more safety errors in the baseline segments ($M = 0.64$, $SD = 0.40$, $p < .0001$) than controls ($M = 0.15$, $SD = 0.18$).

Fewer participants in the PD group identified 60% or more of the targets compared to controls (17.7% PD versus 49.7% controls). Results from a multivariate regression analysis revealed that at-fault safety errors committed by the PD group were predicted by TMT (B-A), and identification of targets was predicted by UFOV and Complex Figure Test-Copy. In contrast, depressive symptoms as measured by the GDS predicted at-fault safety errors in the control group. The identification of targets in the control group were predicted by measures of visual acuity, visual attention, spatial perception, verbal memory, and visuo-constructional ability, executive function and level of depressive symptoms. Medication dose was not associated with driving performance of PD participants during the identification task. It was concluded that the secondary identification task impaired PD patient driving performance to a greater extent than it did controls. This finding suggests that cognitive functions rather than motor dysfunction are responsible for impaired driving performance in PD, particularly in tasks that require additional cognitive load. In this study, a relatively small number of safety errors were committed by the controls ($M = 0.45$, $SD = 0.81$) therefore correlations between safety errors and clinical tests should be taken with caution. A methodological limitation common to a number of studies reviewed earlier is the fact that the sample was not a true representative of the population as no drivers with extreme cases of PD were included.

In a unique study Wood et al. (2005) assessed the association between self-reported driving ability and driving errors in Parkinson's disease. A driving history survey was administered to 25 patients with PD and 12 age-matched controls. None of the participants had any cognitive impairments as assessed by a score > 24 on the MMSE and all participants were active drivers. The patient group consisted of 4 females and 21 males, and were at stage 1-3 evaluated by the Hoehn and Yahr scale. The control group consisted of 3 females and 18 males. Participants also completed an on-road drive which lasted for 19.4km and alternated between direct navigation instructions by the occupational therapist (70% of drive) and sections where the participant drove independently. Throughout the drive the OT and a driving instructor independently scored the participant on seven different driving aspects at 147 locations in order to derive an overall driving safety rating. The OT and driving instructor scores were comparable therefore an average of the two was used in the analysis. PD patients were rated less safe ($M = 4.80$, $SD = 1.91$) compared with controls ($M = 6.56$, $SD = 1.72$). Fourteen out of 25 PD drivers scored less than 5, compared with 5 of the 21 controls meaning these participants would have failed the driving test. Driver safety ratings for the PD group were found to correlated with duration of the illness ($r = -0.60$, $p = 0.001$), specifically, the longer the duration the worse the safety rating. However no significant associations between disease severity H and Y ($r = -0.06$, $p = 0.79$) or Levodopa dosage ($r = -0.36$, $p = 0.11$) and safety ratings was found.

Results from the self-report survey showed that the groups were comparable on driving frequency, level of confidence while driving, and self reported crashes within the past ten years (7 controls, and eight cases). However, PD patients were less confident about driving alone compared with controls (modal category "confident" compared with "very confident", $U = 167.5$; $p = 0.015$). The PD patients found the following driving behaviours significantly more difficult than controls; moving foot between pedals ($t_{44} = 2.73$, $p = 0.0009$), steering ($t_{44} = 2.80$, $p = 0.0008$) and reading road signs in daylight (t_{44}

= 2.41, $p = 0.02$). Self ratings of driving ability were not associated with driver safety ratings in either the control or PD groups. In terms of driving performance PD patients had significantly greater difficulty maintaining lane position ($M = 4.62$, $SD = 1$) than controls ($M = 10.20$, $SD = 1.62$, $p = 0.01$), and had greater difficulty checking the blind spot ($M = 7.52$, $SD = 0.74$ vs controls $M = 5.19$, $SD = 0.66$, $p = 0.03$). The authors concluded that drivers with PD tend to misjudge their own driving ability. However in this study it appears that controls were also poor judges of their driving ability. As expected PD drivers displayed a greater number of safety errors than controls. In particular, the PD participant group had difficulty maintaining lane position, made fewer glances to the blind spot and had difficulty negotiating intersections.

Grace and colleagues (2005) reported on a study that aimed to identify cognitive and motor predictors of driving performance in a sample of 21 healthy participants, 21 participants with PD and 21 participants with Alzheimer's disease (AD). Diagnoses of neurological conditions were confirmed by a neurologist and participants were excluded if they had a psychiatric illness, physical disorder, ophthalmologic disorder, or a secondary neurological disorder. Medication doses were required to be stable for a six week period prior to participation. The participant groups were matched according to age, education and intelligence level. The neuropsychological test battery included; Hopkins Verbal List Learning Test-Revised (HVLT-R), the Rey-Osterrieth Complex Figure (ROCF) and the Neuropsychological Assessment Battery (NAB) Driving Scenes Test, the TMT-A and B, a computerised maze task and a finger tapping test. Participants completed an on road driving test within two weeks of completing the neuropsychological tests. A driving instructor blind to the participant group assessed the participants using the Washington University Road Test, and each participant was assigned a global rating score of safe, marginal or unsafe. Driving history related to the past three years was also recorded.

There was no difference in miles driven per week or frequency of trips between the experimental groups. While every person in the control group was classified as a safe driver, 67% of the PD group were safe and 45% of the AD group were safe. The majority of errors in the driving task were made by the AD group ($M = 13.9$), compared with the PD group ($M = 7.6$) and this difference was significant ($p = .003$). Only unsafe PD drivers scored significantly worse than controls for tests of memory on the HVLT ($ps < .01$, $\eta^2s > .26$). Unsafe and safe AD drivers were both impaired on the TMT-B, relative to controls, while only the PD unsafe group were impaired compared to controls ($p = .001$, $\eta^2 = .41$). Therefore the authors concluded that tests most likely to predict driving status in PD were the TMT- B, the ROCF, the HVLT and disease severity. Once again it should be noted that the small sample size is a methodological limitation of the study, as is the restricted range of disease severity of participants with PD.

An earlier study conducted by Radford et al. (2005) was carried out to identify the cognitive predictors of driving ability in PD in order to develop a screening tool to assess fitness to drive. Fifty-one participants were recruited from a fitness to drive centre and a movement disorders clinic in Nottingham. The mean age was 64 years and the majority (80%) were male. To be eligible for the study participants were required to be active drivers. Demographic variables included; age, gender, driving experience and driving exposure. The clinical assessment included; Webster's rating scale of physical performance, Unified Parkinson's Disease Rating Scale (UPDRS), Stroke Drivers Screening Assessment (SDSA) which assesses attention and reasoning skills, Adult Memory and Information Processing Battery (AMIPB), Stroop colour word test, the

Paced auditory serial addition task (PASAT) and the tapping task. The on-road driving component of the study was administered by a qualified driving instructor, and was completed in the participants own car. Participants were firstly classified as unsafe, probably unsafe, probably safe or definitely safe, and secondly ranked according to their ability to complete 26 driving aspects. The rankings consisted of no fault, minor fault, and major fault. A total of 43 drivers were classified as safe while six were classified as unsafe. The Webster Rating Scale differentiated between safe (median = 11.5, SD = 11-14) and unsafe (median = 6, SD = 5-9) drivers ($p = 0.02$). The study did not identify any cognitive predictors of driving performance between safe and unsafe drivers. However individual test performance on the SDSA dot cancellation errors ($r^2 = 0.32, p = 0.03$), the AMIPB story recall delay ($r^2 = 0.29, p = 0.05$), and the AMIPB information processing A ($r^2 = 0.31, p = 0.03$) correlated with the number of faults. The researchers attribute the limited findings to the small number of drivers in the unsafe group ($n = 6$) compared to the safe group ($n = 43$). Furthermore, the lack of a healthy control group weakens the study.

A study by Worringham et al. (2006) also attempted to identify cognitive predictors of driving performance in PD. However, in addition to the cognitive predictors the researchers also investigated motor and visual measures. A sample of 25 patients with PD and 12 age-matched controls (3 females and 18 males) completed an on-road drive and a driving history survey. None of the participants had any cognitive impairment as assessed by a score > 24 on the MMSE and all participants were active drivers. The patient group consisted of 4 females and 21 males, and were at stage 1-3 evaluated by the Hoehn and Yahr scale. The functional tests of motor, visual and cognitive functions consisted of; visual static visual acuity, the UFOV, Pelli-Robson contrast sensitivity, Visual Fields, Central motion sensitivity, Symbol digits modality, Trails A and B, Stroop Test, Beck Depression Inventory, Aiming task, Motor timing task, and the Purdue Pegboard Test. PD participants were tested in their optimum response to medication period. Participants completed a 19.4 km drive around a driving track and were assessed by an occupational therapist and a driving instructor. Driving performance was classified as either a pass or fail.

Only one test from each cognitive domain was chosen for comparison with on road driving safety scores. These tests showed the highest significant correlation between people who failed and people who passed. These included; contrast sensitivity ($r = 0.40, p < 0.01$), symbol digit modalities ($r = 0.46, p < 0.0005$), and the Purdue Pegboard Test ($r = 0.54, p < 0.0005$). The time since diagnosis and age were also significantly correlated with driving safety score for PD participants only. A discriminant function analysis was conducted using the specified tests, and age and time since diagnosis to predict driving assessment performance (pass or fail) for both groups separately and combined. For both groups combined the sensitivity was 85.2%, and the specificity was 63.2%. Results suggested that approximately 90% of PD cases who were predicted to have failed actually failed when years since diagnosis were included, while 75% of controls who were predicted to fail actually failed. The authors identified three tests which predicted on road driving performance; The Purdue Pegboard Test of motor performance, the Pelli-Robson test of contrast sensitivity, and the verbal version of the Symbol Digit Modalities test. Once again this study is limited by the small sample size which becomes problematic when conducting predictive analyses.

Treatment for PD and road safety outcomes

PD medication

Although PD is a chronic progressive disease there is medication that can be taken to relieve the symptoms and improve quality of life. The most common treatment for PD typically consists of Levodopa combined with Carbidopa (NINDS, 2009). As the response to medication types and dosage differ according to individuals, it is imperative that patients liaise with their doctor in order to determine the best treatment plan. The potential for medication dose to impair driving performance has been investigated using on-road and simulator studies. Recently, a number of studies that have been outlined above have typically found no relationship between dopamine antagonist medication dose and driving performance (Amick, 2007; Devos 2007; Worringham, 2006; Cordell, 2008). However, in all these studies participants were in the mild to moderate stages of the disease therefore it is less likely that the medication dose would be high enough to impair driving performance.

Avanzi (2008) reported on a case study of two individuals with PD in order to illustrate the consequences of excessive medication dosages. Case 1 suffered from PD for nine years and was prescribed Levedopa medication. He was aged 65 years old. Four years after the onset of his disease he started to self-regulate the dose of his medication by increasing the dose to improve his motor functions. Consequently, manic symptoms including decreased sleep, coarse speech and withdrawal dysphoria developed. During this time he engaged in risk taking behaviour which included driving over the speed limit, and was involved in three motor vehicle accidents. Case 2 was a 70 year old man who had suffered from PD for twenty years. Similar to Case 1, he increased his dose of L-dopa after fifteen years of suffering from the illness. The side effects included binge eating, gambling, withdrawal dysphoria and sexual disinhibition. Impulsivity also increased and contributed to risk taking behaviour which affected his driving. He typically drove at high speeds and was involved in a high speed motor vehicle crash. This study is important as it considers driving safety risk resulting from individuals with PD who mismanage medication dosages. Further research in to this phenomenon is warranted.

It remains unclear as to whether sleepiness associated with PD is related to the anti-parkinsonian medication or whether it is a consequence of a disrupted sleep-wake cycle that occurs in individuals with PD. The main aim of the study conducted by Avorn et al. (2005) was to investigate the nature of sudden uncontrollable somnolence in Parkinson's disease. Specifically, the researchers were interested in the number of times in the last six months when participants had experienced sudden onset of sleep in a group conversation or while driving. Neurologists from selected movement disorder clinics invited patients to participate in the study if they had a diagnosis of idiopathic PD, and did not suffer from a psychiatric illness or dementia. A total of 929 patients completed a telephone interview and information regarding the last six months was obtained. The Hoehn and Yahr Scale was used to assess the severity of the disease, a modified version of the Schwab and England scale was employed to assess daily living skills, the Epworth Sleepiness Scale measured sleepiness, and the frequency and medication dose for each patient was determined. Sudden onset of sleep was evaluated by two questions which asked whether in the last six months sudden onset of sleep had occurred either in a group conversation or while driving. The date of the occurrence and the specific situation were also recorded. Seventy-three percent of participants had driven a car within the past six months, and the majority of the sample were male

(62.6%). In regards to medication, 93% of participants were prescribed Levodopa either alone or in a combined with another medication. This group was further broken down in to patients who were taking levodopa plus a dopamine antagonist (DA) plus another class of antiparkinsonian medication, or Levodopa plus a DA or Levodopa alone.

A greater number of sudden somnolence events were recorded by people prescribed Levodopa plus a DA (22%) compared to those taking levodopa alone (13.4%). Typically those taking levodopa alone were older, and had greater severity and duration of the disease. However, the levodopa-DA group reported a great frequency of somnolence events (28.2%) compared to levodopa alone patients (13.4%). Odds ratio of 2.75 (CI 1.79 - 4.24) resulted in a greater risk for the occurrence of sudden somnolence for people taking either DA's alone or DA's in a combination compared to Levodopa alone. The authors concluded that the risk of uncontrolled somnolence is high for PD patients on dopamine agonist medication, and it appears that the risk is linearly associated with the dosage. It is difficult to determine the crash risk associated with driving and somnolence from this study because driving episodes were combined with sleep episodes that occurred during a conversation. It is of interest to note that 43% of participants changed medication within the six month period and therefore this is another factor that could have been explored. Caution should be taken when interpreting these results as the information collected relied upon self-report data.

Summary

The review post-May 2003 revealed only two published studies that have investigated the association between driving with PD and crash risk. The main focus of these studies was the association between sleepiness in drivers with PD and crash risk. It appears that a small proportion of drivers with PD experience a sleep episode while driving. Gorayheb et al. (2007) found no relationship between sleepiness and crash risk, while Meindorfer et al. (2005) reported a slight increase in crash occurrence for drivers with PD who experienced sleep episodes compared with those who do not have sleep disorders.

The ability to predict fitness to drive amongst individuals with PD has received a considerable amount of attention in the last few years. Studies have typically investigated the relationship between driving performance (as measured by on-road assessments and driving simulator tasks) and clinical test outcomes. Overall, it appears that drivers with PD have poorer driving performance than healthy controls (e.g. Lee et al., 2007; Stolwyk et al., 2005, 2006a, 2006b; Uc et al., 2006b; Wood et al., 2005; Worringham et al., 2006). In addition, there is increasing evidence that driver safety is correlated with neuropsychological test performance. Specifically, correlations have been found for tests requiring visual skills such as visuospatial ability (Amick et al. 2007), contrast sensitivity (Devos et al. 2007, Worringham et al. 2006), and visual working memory (Uc et al. 2006). Cognitive tests including tests of information processing, working memory, and set shifting, are generally better predictors of driving performance in PD than are motor tests (Stolwyk, 2006b). What remains to be demonstrated is how well these impaired cognitive functions in PD predict crashes.

Evidence from studies investigating distraction and drivers with PD has found mixed results. According to Stolwyk et al. (2006b) drivers with PD adopt a more conservative driving approach than controls when faced with a concurrent driving task however Uc et al. (2006a) did not find any differences in driving performance during a distraction task.

Studies concerning the navigation ability have shown that drivers with PD are more likely to get lost (Uc et al. 2007) and have difficulty responding to prior information such as reading a map to alter their driving behaviour (Stolwyk et al. 2005) and engage in risk taking behaviour associated with impulsivity (Avanzi, 2008). Further research in this area is warranted to aid the driving process.

Finally, there is insufficient evidence to determine whether individuals with PD taking prescribed medication are at an increased risk for a crash. Recent studies have not found any evidence to suggest that dopamine antagonistic medication dose is related to driving performance (Amick, 2007; Devos 2007; Worringham, 2006). However, these findings should be taken with caution due to inconsistencies in sample size, disease severity, duration of the illness and types of medication.

In conclusion, it appears that cognitive and visual deficits associated with PD contribute to a decline in driving performance. There are inconsistent findings about which tests accurately predict poor driving performance due to the use of small sample sizes, lack of a control group or an adequately matched control group, and the absence of PD participants across the broad spectrum of disease severity.

Table 24 Summary of studies of risk associated with PD

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Amick et al. (2007a)	Cases = 25 with PD drivers (54-72yrs)	On-road driving test - safe versus marginal drivers Neuropsychology tests - Rey-osterreith complex figure test (ROCF), - Trail making tests A and B - Useful Field of View (UFOV), - Backwards visual masking, - Functional Acuity Contrast Test (FACT) - Pelli Robson Test of contrast sensitivity.	On-road driving perf assoc with (subtest three) of the UFOV ($r = 0.49, p = 0.01$), Trails A and B ($r = 0.49, p = 0.01$), and the ROCF ($r = -0.47, p < .005$).
Amick et al. (2007b)	Cases = 25 with PD	On road driving test - safe, marginal, unsafe Excessive daytime sleepiness - Epworth Sleepiness Scale (ESS)	Driving performance did not differ between people with EDS and those without ($p = 0.56$, Fisher's Exact Test). DA medication did not affect global levels of driving performance ($p = .56$, FET).
Avorn et al. (2005)	Cases = 929 with PD (M age = 66.7 years) 347 females, 582 males 73% current drivers	Sudden onset somnolence in last 6 months - self-report Excessive day time sleepiness - ESS - self report Dose and frequency of medications in the last 6 months	DA medication resulted in a increase risk of sudden somnolence OR = 2.75 (CI 1.79-4.24) compared to Levodopa alone. High ESS scores were associated with greater risk of a somnolence event (OR = 6.86, 95% CI, 3.98-11.82).

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Devos et al. (2007)	Cases = 40 with PD Controls = 40 healthy age and sex matched participants	<ul style="list-style-type: none"> - On road driving test (pass/fail outcome) - Clinical evaluations Included visual and cog. tests: Clinical Dementia Rating (CDR), Pelli-Robson contrast sensitivity, Snellen Visual Acuity, UFOV, Humphrey Visual Field Analyzer, UPDRS, daily living skills. - Driving sim. test Included a divided attention task 	<p>No sig diff. in self reported acc ($p = 0.28$) and or violations ($p = 0.58$) for drivers who passed compared to those who failed</p> <p>CDR, contrast sensitivity, disease duration and motor deficits (UPDRS III) were the greatest predictors of fitness to drive ($R^2 = .52$)</p> <p>Accidents and violations in sim: PD > C*</p> <p>Divided attention task: PD > RT than C ($p = .0001$)</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Ghorayeb et al. (2007)	Cases = 1625 with PD 53.2% regular drivers	Survey - Excessive daytime sleepiness - Medication dose - activity of daily living - Disease severity (H and Y) - Accidents - chance of dozing while driving	No. of crashes between drivers ($M 7.3 \pm 5.2$) and non drivers ($M 6.9 \pm 4.5$) were not related to ESS. Reduced daily activity living scores predicted EDS ($P = 0.02$, OR, 0.98 95% CI, 0.97-0.99), as did daily levodopa dosage ($P = 0.04$, OR, 1 95% CI, 1-1.05) and male gender ($P < 0.001$, OR 1.7, 95% CI, 1.29-2.25) Male gender ($P < .008$, OR 1.83, 95% CI 1.42-4.27), and a Hoehn and Yahr score ($P = .002$, OR 0.61, 95% CI 0.4-0.93), and an ESS score ≥ 10 ($P < 0.001$, OR 5.7, 95% CI 3.79-9.02), were predictive of dozing while driving. No sig distinction between ESS scores for the small number of participants (3.1%) who were previously involved in a traffic accident ($M = 7.3$, $SD = 5.2$) compared to those without an accident history ($M = 6.9$, $SD = 4.5$).

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Grace et al. (2005)	<p>Cases = 21 with PD, 21 with AD</p> <p>Controls = 21 healthy matched on age and education level</p>	<p>On road driving test</p> <p>Neuropsychological test battery:</p> <ul style="list-style-type: none"> - Hopkins Verbal List Learning Test-Revised (HVLTR), - Rey-Osterrieth Complex Figure (ROCF) - Neuropsychological Assessment Battery (NAB) Driving Scenes Test, - Trail Making Test A and B, - Computerised maze task - Finger tapping test. 	<p>Errors in driving test: PD > C ($p = .001$)</p> <p>Errors in driving test: AD > PD ($p = .003$)</p> <p>The NAB driving scenes test did not distinguish between safe or unsafe drivers.</p> <p>ROCF performance: unsafe AD and PD drivers < C.</p> <p>TMT-A not ass with patient group, did distinguish between safe and unsafe drivers ($F(1, 34) = 9.04, p = .005, \eta^2 = .21$).</p> <p>TMT-B: AD safe and unsafe < C</p> <p>TMT-B: PD unsafe < PD safe ($p = .001, \eta^2 = .41$).</p>
Lee et al. (2007)	<p>Cases = 52 PD patients</p> <p>Controls = 129 healthy adults, age matched</p>	<ul style="list-style-type: none"> - Clinical evaluations - On road driving test - Simulator driving task 	<p>On road performance: PD < C ($t(180) = 84.2, p < .000.1$)</p> <p>Driving sim. performance: PD < C ($t(180) = 104.6, P < .001$)</p> <p>Simulated driving index explained nearly 40% of the road assessment index for PD participants and 68% for the control participants.</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Meindorfner et al. (2005)	Cases = 6,620	Survey <ul style="list-style-type: none"> - Driving habits (driving experience and driving exposure, cessation, restrictions), - disease duration and severity, (Hoen and Yahr scale) - current medication - Epworth Sleepiness Scale - Acc. past five years - Sleep episodes while driving 	<p>A total of 8% of participants with current driving licences had experienced SOS at the wheel.</p> <p>Accidents related to SOS typically involved single vehicles where the participant drove off the road. While non SOS related crashes involved multi-vehicles, at cross roads or in parking lots.</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Radford et al. (2007)	Cases = 51 with PD	<p>On-road driving test</p> <p>Clinical evaluations:</p> <ul style="list-style-type: none"> - Webster's rating scale of physical performance - Unified Parkinson's Disease Rating Scale (UPDRS), - Stroke Drivers Screening Assessment (SDSA) - Adult Memory and Information Processing Battery (AMIPB), - Stroop colour word test, - Paced auditory serial addition task (PASAT) and - The tapping task. 	<p>Webster Rating Scale differentiated between safe (median = 11.5, SD = 11-14) and unsafe (median = 6, SD = 5-9) drivers ($p = 0.02$).</p> <p>A regression equation consisting of SDSA dot cancellation errors, the AMIPB story recall delay the AMIPB information processing A and foot tapping accounted for 44% of the variability in number of driving faults for PD cases.</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Singh et al. (2007)	Cases = 154 with PD	<p>On-road driving test (suitable/unsuitable to drive)</p> <p>Clinical and cognitive tests – prospective</p> <ul style="list-style-type: none"> - TMT-A and B - MMSE - Reaction Time - Other medical conditions - Disease duration and severity (H and Y scale) - Forward and reverse digit span - Visuo-spatial ability 	<p>Suitable vs Unsuitable: disease severity ($X^2 (2, N = 154) = 80, p < .001$), duration of illness (MW, $z = -4.3, p < .001$), the presence of other medical conditions ($X^2 (1, N = 154) = 5, p < .026$) and the on-road driving assessment score (MW, $z = -7.4, p < .001$).</p> <p>H and Y score of stage 3 ($p < .001$), the on-road driving score ($p < .001$), and H and Y score of 2 when associated with reaction time and another medical condition ($p = .008$) predicted fitness to drive in 92% of patients with PD.</p>
Stolwyk et al. (2005)	<p>Cases = 18 PD drivers</p> <p>Controls = 18 age matched healthy adults</p>	<p>Clinical evaluations</p> <p>Driving Simulator Task</p> <ul style="list-style-type: none"> (i) presence of external cues (ii) absence of external cues, (iii) presence of internal cues (iv) absence of internal cues 	<p>Approach speed: There was a trend for PD < C ($F (1,34) = 3.42, p = .073$).</p> <p>When external cues were present the control and case participants did not alter their approach speed in response to internal cues ($F (1, 34) = 0.68, p = 0.414$).</p> <p>PD participants decelerated significantly later than controls ($F (1, 43) = 21.58, p < .0001$). PD participants also travelled further when stopping at traffic lights compared to controls ($F (1, 34) = 26.76, p < .0001$), and travelled at a slower speed around curves ($F (1, 34) = 7.13, p = 0.012$).</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Stolwyk (2006a)	Cases = 18 PD drivers Controls = 18 age matched healthy adults	Driving Simulator Task - concurrent task while driving	Lane position variability: PD > C ($p = .002$), PD stopped further away past the lights ($p = < .0001$) and PD started to decelerate later ($p < .001$). - On approach to a traffic signal PD participants tended to decelerate later than C in the concurrent task ($p = .008$). - PD participants were less accurate ($p = .025$) and slower ($p = < .0001$) at responding to targets in the concurrent task than controls.

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Stolwyk (2006b)	Cases = 18 PD drivers Controls = 18 age matched healthy adults	Driving Simulator Task Neuropsychological Test Battery <ul style="list-style-type: none"> - TMT-A and B, - MMSE, - Symbol Digit Modalities Test – auditory version (SDMT), - Mean Simple Reaction Time, - Mean Choice Reaction Time, - Up and Go test, Brixton Test of set shifting ability, - Judgement of Line Orientation Test (JLO), - Wechsler Adult Intelligence Scale-III Picture Completion, Digit Span Total, and Block Design. 	<p><u>PD group:</u> TMT-A corr with delayed traffic signal stopping, ($r = .495, p = .05$), SDMIT corr with delayed stopping at traffic lights ($r = -.587, p = .05$), late deceleration ($r = -.443, p = .05$) and adjustment of lane position to curve direction ($r = -.710, p = .01$).</p> <p>JLO corr with delayed stopping at traffic lights ($r = -.628, p = .01$). Picture Completion corr with reduction in the ability to maintain lane position around a curve ($r = -.501, p = .05$) and delayed stopping at traffic lights ($r = -.502, p = .05$).</p> <p>TMT-B corr with slow approach speed ($r = -.710, p = .01$), late deceleration ($r = -.440, p = .05$), reduced speed around curves ($r = -.496, p = .05$), and maintenance of lane position around a curve ($r = .613, p = .01$).</p> <p>Brixton Test corr with slow speed around curves ($r = -.661, p = .01$), reduced ability to maintain lane position around curves ($r = -.710, p = .010$), delayed deceleration ($r = -.640, p = .01$) and slow approach speed ($r = -.643 = .01$).</p> <p><u>Control Group:</u> TMT-A corr with mean speed around curves ($r = -.500, p = .05$). JLO corr with approach speed to traffic lights ($r = -.510, p = .05; r = -.640, p = .01$) and speed around curves ($r = .562, p = .05$).</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Uc et al. (2006a)	<p>Cases = 71 with PD active drivers</p> <p>Controls = 147 healthy age matched active drivers</p>	<p>Clinical Tests:</p> <ul style="list-style-type: none"> - UFOV - Visual acuity, contrast sensitivity, visual perception, UFOV - TMT A and B - UPDRS, Hoehn and Yahr Score - Geriatric Depression Scale - MMSE, - ESS - Benton Visual Retention Test (BVRT) - WAIS III Block Design - Rey Auditory Verbal Learning Test (AVLT) - Complex Figure Test-Copy <p>Instrumented Driving Experiment</p> <p>Paced Auditory Serial Addition Test (PASAT)</p> <p>(included in clinical and experimental assessments)</p>	<p>PD patients were worse than controls at the PASAT for the off road assessment. ($60.9 \pm 19.3\%$ vs $68.0 \pm 20.7\%$) ($p < .05$), as well as during the experimental driving session ($50.9 \pm 17.8\%$ vs 58.1 ± 19.6, $p < .01$).</p> <p>The predictors of committing a safety error during PASAT included MMSE (OR (95% CI) = 1.40 (1.06 to 1.87), $p = 0.020$), TMT (B-A) (OR (95% CI) = 1.27 (1.03 to 1.52) per 30 second increase, and BVRT (OR (95% CI) = 1.15 (1.01 to 1.30) per one more recognition error, $p = 0.035$).</p> <p>PD participants drove at a slower speed, but displayed greater speed variability than controls.</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Uc et al. (2006b)	Cases = 79 with PD Controls = 151 current drivers	Instrumented Driving Task - Identification of landmarks and traffic signs - At fault safety errors Clinical Test Battery - Refer to Uc et al. (2006a)	PD < C all visual and cog. measures PD > safety errors during the identification task ($M = 1.97$, $SD = 1.56$, $p < .001$) than controls ($M = 0.45$, $SD = 0.81$). PD > safety errors in the baseline segments ($M = 0.64$, $SD = 0.40$, $p < .0001$) than controls ($M = 0.15$, $SD = 0.18$). TMT (B-A), UFOV and Complex Figure Test-Copy predicted at fault safety errors for PD group. Depressive symptoms predicted at-fault safety errors for Controls.
Uc et al. (2007)	Cases = 77 with PD current drivers Controls = 152 healthy current drivers	Clinical Test Battery – Refer to Uc et al. (2006a) Instrumented Driving Task & Route Following Task - At-fault safety errors - No. of times lost - No. of incorrect turns	More people in the PD group (53.9%) made more incorrect turns (21.1%) compared to C (OR 95% CI = 2.8 (1.4-5.7), $p < 0.0001$), and committed more at-fault safety errors (84.2%) than C (46.7%) (OR 95% CI = 7.5, (3.3, 17.0), $p < .0001$). A greater number of PD participants got lost (15.8%) compared to C (2%) (OR 95% CI = 4.7 (1.1, 20.0), $p < 0.037$).

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Wood et al. (2005)	<p>Cases = 25 with PD</p> <p>Controls = 21 age matched healthy controls</p>	<p>Self Rating Survey on Driving Behaviours</p> <p>On road driving test</p>	<p>PD patients less confident about driving alone compared with controls (modal category “confident” compared with “very confident”, $U = 167.5; p = 0.015$).</p> <p>PD patients found moving foot between pedals ($t_{44} = 2.73, p = 0.0009$), steering ($t_{44} = 2.80, p = 0.0008$) and reading road signs in daylight ($t_{44} = 2.41, p = 0.02$) more difficult than controls.</p> <p>Overall driving behaviour was less safe for PD ($M = 4.80, SD = 1.91$) than controls ($M = 6.56, SD = 1.72$).</p> <p>Driver safety ratings for PD corr with duration of the illness ($r = -0.60, p = 0.001$).</p> <p>Self ratings of driving ability were poorer for PD patients ($M = 2.98$) than controls ($M = 2.57, t_{44} = -2.7, p = 0.0009$).</p> <p>PD patients had significantly greater difficulty maintaining lane position ($M = 4.62, SD = 1$ vs controls $M = 10.20, SD = 1.62, p = 0.01$), and checking the blind spot ($M = 7.52, SD = 0.74$ versus controls $M = 5.19, SD = 0.66, p = 0.03$).</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Worringham et al. (2005)	Cases = 25 with PD Controls = 21 age-matched healthy controls	<p>On road driving test</p> <p>Clinical evaluation:</p> <p>Visual:</p> <ul style="list-style-type: none"> - static visual acuity - UFOV - Pelli Robson contrast sensitivity - Visual Fields - Central motion sensitivity <p>Cognitive:</p> <ul style="list-style-type: none"> - Symbol digits modality - Trails A and B - Stroop Test - Beck Depression Inventory <p>Motor:</p> <ul style="list-style-type: none"> - Aiming task - Motor timing task - Purdue Pegboard Test 	<p>PD patients found moving foot between pedals ($t_{44} = 2.73$, $p = 0.0009$), steering ($t_{44} = 2.80$, $p = 0.0008$) and reading road signs in daylight ($t_{44} = 2.41$, $p = 0.02$) more difficult than C.</p> <p>Overall driving behaviour was less safe for PD ($M = 4.80$, $SD = 1.91$) than C ($M = 6.56$, $SD = 1.72$).</p> <p>Driver safety ratings for PD group corr with duration of the illness ($r = -0.60$, $p = 0.001$).</p> <p>Self ratings of driving ability were poorer for PD patients ($M = 2.98$) compared with C ($M = 2.57$, $t_{44} = -2.7$, $p = 0.009$).</p> <p>PD patients had significantly greater difficulty maintaining lane position ($M = 4.62$, $SD = 1$ vs controls $M = 10.20$, $SD = 1.62$, $p = 0.01$), and checking the blind spot ($M = 7.52$, $SD = 0.74$ versus C ($M = 5.19$, $SD = 0.66$, $p = 0.03$).</p> <p>No significant associations between disease severity H and Y ($r = -0.06$, $p = 0.79$) or Levodopa dosage ($r = -0.36$, $p = 0.11$) and safety ratings was found.</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Heikkila et al. (1998)	Cases = 20 with PD drivers Controls = 20 controls without PD matched on age and gender	- clinical evaluations - cognitive and psychomotor lab tests - on-road driving test (rural and urban)	Errors in lab tests: PD > C* PD committed more “risky” and serious infringements* heavy traffic errors: PD > C* turning across traffic errors: PD > C* - severity of disease and dose of meds not assoc with on-road performance
Zesiewicz et al. (2002).	Cases = 39 PD drivers Controls = 25 control participants	Performance on: - MMSE - self-reported driving history - driving simulator P with PD also completed: - UPDRS - Hoehn and Yahr staging (H &Y)	Miles driven: PD = C MMSE: PD who stopped driving < PD with no changes, PD who decreased their driving, and control drivers *** Collisions: PD> C ** - PD involved ≥ 1 sim collisions was assoc w H & Y stage *** - Sim collisions assoc w UPDRS score **
Lings & Dupont (1992)	Cases = 28 PD participants (median age = 65) Controls = 109 younger controls (median age = 49).	Driving performance using a mock car	PD group more likely to fail to react to stimuli such as a red light, a high frequency of erroneous reactions (particularly directional errors), reduced speed and strength of movement, and prolonged reaction times.

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Dubinsky, Gray, Hustead, Busenbark, Vetre-Overfield, Wiltfong, Parrish, and Koller (1991),	survey study Cases = 150 p with PD Control = 100 p	<ul style="list-style-type: none"> - 40-question survey of driving records and habits - MMSE - Northwestern University Disability Scale (NUDS) - Schwab and England activities of daily living scale, - Hoehn and Yahr scale 	<p>crash rate per million vehicle miles: severe PD > mild PD and C**</p> <p>- cog impair (MMSE \leq 23) assoc with increased acc/per million vehicle miles travelled *</p>
Madeley, Hulley, Wildgust & Mindham (1990)	<p>Cases = 10 drivers with PD</p> <p>Controls = 10 healthy controls who were matched on age and sex</p> <p>- A further four participants with PD who were no longer driving were also included.</p>	<p>driving simulator:</p> <ul style="list-style-type: none"> - simple and driving reaction times - accuracy of steering - number of red lights missed. - PD drivers rated on Webster's rating scale for severity of motor impairment 	<p>Simple reaction time: PD = C</p> <p>Steering Acc Impairment: PD > C*</p> <p>Driving reaction time: PD > C**</p> <p>Red lights missed: PD > C**</p> <p>- correlation b/w PD severity and:</p> <p>sim driving reaction time*</p> <p>steering accuracy **</p> <p>simple reaction time **</p>

Approaches to management

Assessing fitness to drive

As summarised below in Table 25, most licensing jurisdictions outline that in the early stages of PD, no driving restrictions may be necessary. However, most licensing jurisdictions do recommend periodic licence reviews (in most cases annually). In addition, most licensing jurisdictions suggest that there is a possibility of using conditional/restricted licensing criteria for drivers with PD. In the later stages of the disease, most licensing jurisdictions recommend the revocation of the driving licence. Sweden appears to have the most explicit recommendations, in that they suggest a risk assessment which includes an appraisal of the stage of the disease and the effect of treatment. Given that only one study has investigated the relationship between PD and crash risk, it is not impossible to evaluate whether these licensing guidelines are consistent with the scientific evidence.

Self-regulation

Few studies have investigated the self-regulatory habits of drivers with PD. In the survey study conducted by Dubinsky et al. (1991) outlined in the previous section, 21% of participants with PD reported that they had stopped driving because of their disease, whereas only 2% of control participants reported that they had stopped driving ($p < 0.0001$). However the authors did not specify the reasons as to why drivers with PD stopped driving.

In addition, Zesiewicz et al. (2002) compared the self-reported driving habits and driving ability of 39 PD drivers with 25 control participants using a driving simulator. Participants also completed a MMSE. The authors reported that within the PD group, 7 reported having stopped driving, 10 reported a decrease in the amount of driving, and 22 reported no change in driving habits. PD drivers who stopped driving had significantly lower MMSE scores ($M = 23.6 \pm 4.9$) than PD drivers who reported no changes in amount of driving ($M = 28.6 \pm 3.2$), PD drivers who decreased their driving ($M = 28.1 \pm 1.8$), and control drivers ($M = 29.7 \pm 0.9$) ($F = 10.1, p < 0.001$).

Table 25 Private licensing guidelines for drivers with PD

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Parkinson's	The degree and severity of the disease determine an individual's capacity to drive. If mechanical appliances are installed in the vehicle the applicant must demonstrate competency in a driving test.	An unconditional licence may not be held if the disease impairs driving. A conditional licence may be issued subject to the results of a driving assessment & treatment response & with appropriate vehicle modifications. Subject to yearly reviews (minimum).	May be licensed if medical confirmation obtained that driving ability (e.g. nerves and circulation) is unimpaired. Vehicle modifications may be required. Licence may be restricted to short-period licences requiring renewal every 1, 2 or 3 years .	An unrestricted licence may be issued if the person is able to control equipment & has no or minimal neurological impairment. Annual review required for minimal impairment. If the person is able to control equipment despite slight neurological impairment, a road test must first be passed before licensing can occur. Annual review required.	Driving to cease if person is unable to react appropriately to emergency situations or where quick responses are required. If a person has trouble with walking, it is likely that they will be unfit to drive. <i>Early stages of illness:</i> Driving may continue provided this can be done effectively. Yearly review may be required. <i>Other stages of illness:</i> Licence revocation.	Licence denial or revocation if disease impairs driving ability & so renders the person a traffic safety risk. Risk assessment to include an appraisal of the stage of the disease & treatment response. Periodic review required on a case-by-case basis.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				<p>A restricted licence with speed &/or area restrictions, may be issued if the person has moderate dexterity impairment.</p> <p>Annual review required.</p> <p>Greater restrictions (speed/area/time of day/must be accompanied by licensed driver) are imposed if there is <i>temporary</i> significant neurological impairment.</p> <p>Six-monthly review required.</p>		

3.8.2 MULTIPLE SCLEROSIS

Definition of multiple sclerosis (MS)

Multiple sclerosis (MS) is an incurable, autoimmune, chronic and progressive demyelinating disease of the central nervous system (Lings, 2002; Schultheis, Garay & DeLuca, 2001). MS is the most frequent cause of neurologic impairment in early to middle adulthood (20 – 40 years, Lings, 2002). MS symptoms result when the immune system attacks the myelin sheath which is the protective coating surrounding all the nerve fibres in the brain, the eye and the spinal cord (Roskar & Sever, 2001). Myelin facilitates the smooth, high-speed transmission of electrochemical messages between the brain, the spinal cord, and the rest of the body. The demyelination of the myelin sheath impedes the transmission of signals from the brain and therefore messages are slower, distorted or do not get through at all. Damaged areas of myelin are known as plaques or lesions (Lings, 2002; Roskar & Sever, 2001).

Depending on where the demyelination occurs (i.e., which nerves are affected), the symptoms of MS can mimic almost any neurological disorder (Roskar & Sever, 2001). The most frequent manifestations of this disorder include various degrees of paresis and spasticity, muscle weakness in the extremities, visual blurring, sensory disturbances, fatigue, vertigo, paroxysmal attacks (which are short, frequent and stereotyped symptoms associated with MS which include painful tonic spasms, ataxia, and numbness) and cognitive dysfunction (Lings, 2002). Symptoms of MS may be mild or severe and of long or short duration and appear in various combinations.

According to the clinical pattern of relapses and residual functional impairment experienced, individuals are classified as having one of four types of MS, representing a continuum of disease (Roskar & Sever, 2001):

Benign MS

Individuals remain relatively unimpaired for many years after an initial attack. Approximately 20% of individuals diagnosed with MS have this form of disease.

Relapsing-remitting MS

Individuals experience a course of relapses (“attacks”) where there is an increased level of symptoms, followed by remission during which there are less or no evident symptoms. The period of the acute attack occurs when the myelin sheath is inflamed, squeezing the nerve fibres so that messages do not pass clearly from the brain to other parts of the body. Approximately 25-35% of those with MS have this pattern at any one time. More than 80% of individuals with MS progress from relapsing-remitting MS to secondary progressive form.

Secondary progressive MS

This form of disease is marked by fewer remissions occurring after attacks and accumulating impairment between relapses. Approximately 40% of individuals with MS are in this category.

Primary progressive MS

This type of MS is characterised by a gradual, insidious and progressive deterioration with impairment developing from the onset of disease without remissions. Approximately 10% of individuals with MS have this type of MS.

Prevalence of MS

The WHO estimates that the prevalence of MS is approximately 2.3 million worldwide (Mathers et al., 2002). In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at 171,000 or around 0.05% of the total population. Similarly, the prevalence of MS in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 253,000 or around 0.06% of the total population. In 2007, it was estimated that 16 081 people suffered from MS in Australia (MSIF, 2007).

MS typically affects women more than men (approximately 2:1) and age of disease onset is usually between 20 and 40 years.

Functional impairments associated with MS relevant to driving

As outlined previously, individuals diagnosed with MS demonstrate widespread, multi-faceted impairments in many domains of physical and cognitive function (Ling, 2002). These include:

- Motor abnormalities - muscle weakness that can involve one side of the body (hemiparesis), both legs (paraparesis), or all four extremities (quadriparesis). Muscles in the affected area may tighten on minimal stimulus (called spasticity) and contract spontaneously and rhythmically (called spasm or clonus).
- Sensory disturbances (such as blurred or double vision, red-green colour distortion, or even blindness in one eye) and transitory abnormal sensory feelings such as numbness, prickling, or "pins and needles" sensations.
- Balance and equilibrium abnormalities (e.g., dizziness, vertigo, uncoordinated movements, tremor).
- Fatigue - Many people with MS experience fatigue and need to rest and sleep during the day in order to continue their activities. The degree of fatigue may not be related to the severity of other symptoms. The symptoms may be worse in hot environments.
- Psychological changes - Depression is a common feature of MS. In addition, about 10% of individuals with MS experience more severe psychotic disorders such as manic-depression and paranoia. Five percent may experience episodes of inappropriate euphoria and despair—unrelated to the participant's actual emotional state—known as "laughing/weeping syndrome." This syndrome is thought to be due to demyelination in the brainstem, the area of the brain that controls facial expression and emotions, and is usually seen only in severe cases.
- Cognitive impairment occurs in about half of all individuals diagnosed with MS (NINDS, 2001). These impairments can occur early in the course of the disease and can progress over time. The most commonly reported cognitive impairments are:

- Slowed information processing abilities: reduced ability to focus, maintain, and shift attention as needed in response to incoming information, particularly rapidly presented information;
- Changes in learning and memory capabilities: reduced ability to learn new information and recall it after a delay;
- Deficits of visuospatial ability: reduced ability to recognise objects, determine where they are in relation to each other, and to move objects, including ourselves, around in space;
- Executive dysfunction: reduced ability to perform complex tasks, such as planning and carrying out a sequence of activities or problem-solving;

In summary, several physical and cognitive impairments associated with the MS disorder, such as visual blurring, vertigo, paroxysmal attacks, cognitive dysfunction and impairment of muscular power and co-ordination appear to be deleterious to the safe handling of a vehicle (Brassington & Marsh, 1998; Lings, 2002).

Treatment of MS

According to the Multiple Sclerosis Foundation (2002) and the National Multiple Sclerosis Society (2003), treatment of MS can be divided into three categories:

Acute

Medications used to treat acute exacerbations or relapses are usually the corticosteroids such as methylprednisolone. They reduce inflammation in nerve tissue and shorten the duration of flare-ups and shorten the time to recovery after a relapse. They do not affect the course of MS and in any case could not be taken long term because of their well known side effects such as osteoporosis and high blood pressure (hypertension).

Symptomatic

Medications used to control symptoms experienced by MS participants include:

- Muscle relaxants: Tizanidine (Zanaflex) and baclofen (Lioresal) are oral treatments for muscle spasticity. Lioresal often increases weakness in the legs. Zanaflex appears to control muscle spasms without leaving the legs feeling weak but can be associated with drowsiness or a dry mouth.
- Medications to reduce fatigue: These may include the antidepressant medication fluoxetine (Prozac), the antiviral drug amantadine (Symmetrel) or a medication for narcolepsy called modafinil (Provigil).

Disease-modifying

The only drugs demonstrated to alter the natural course of MS include interferon beta-1b (Betaferon, Betaseron), interferon beta-1a (Avonex, Rebif) and glatiramer acetate (Copaxone). Beta interferons are genetically engineered copies of proteins that occur naturally in the human body. They help fight viral infection and regulate the immune system. These medications reduce flares of MS. It's uncertain which of their many

actions lead to a reduction in disease activity and what their long-term benefits are. Some people develop antibodies to beta interferons, which may make them less effective. Other people are unable to tolerate the side effects, which may include symptoms similar to those of flu (influenza).

Pre-May 2003: Relationship between MS and road safety outcomes

Despite recent evidence indicating the presence of decreased attentional and visual perceptual skills, slowed information processing speed, and executive dysfunction in individuals with MS, few studies have examined driving skills and abilities in MS (Schultheis, Garay & DeLuca, 2001). Table 26 shows a summary of the findings of studies that have investigated road safety outcomes and MS.

Crashes

In 2002, Schultheis, Garay, Millis and DeLuca investigated the incidence of motor vehicle crashes and citations as documented by driving reports from the DMV among drivers with MS (see the next section for more information regarding citations). Specifically, the authors hypothesised that individuals with MS and cognitive impairment would show a higher incidence of motor vehicle crashes than individuals with MS who are not cognitively impaired and controls. Participants included 27 drivers with a confirmed diagnosis of MS, with minimal to no physical impairment, and 17 control drivers. Participants with a history of other neurological disorders, psychiatric illnesses or a history of substance abuse were excluded from the study. Participants with MS who reported an exacerbation of symptoms within one month before testing were also excluded. In order to determine possible cognitive impairment, participants completed six neuropsychological tests (Paced Auditory Serial Addition Test, Wechsler Adult Intelligence Scale-Revised [WAIS-R] digit symbol, WAIS-R block design, Stroop Colour-Word Test, Trail Making Test, and the Motor-Free Visual Perceptual Test-Revised). Those who scored below the fifth percentile of performance on two or more of the tests were categorised as being cognitively impaired. On the basis of their performance on these cognitive tests, the sample of participants with MS was divided into two groups: MS participants without cognitive impairment ($n = 14$) and MS participants with cognitive impairment ($n = 13$).

Individuals with MS and cognitive impairment had a significantly greater incidence of 1 or more crashes compared to both the MS individuals without cognitive impairment ($\chi^2 = 6.9, p < 0.05$) and control participants ($\chi^2 = 8.4, p < 0.05$). Comparison of the MS group without cognitive impairment and the control group revealed no statistically significant difference in the incidence of crashes ($\chi^2 = 2.7, p = 1.0$). The authors also noted that individuals with MS with cognitive impairment reported the lowest frequency of driving activity (defined as total number of days driving per week), indicating that despite driving less they still demonstrated a higher incidence of crashes. The authors note that some limitations of their study include a small sample size and the inclusion of only individuals with MS without physical impairments. The authors conclude that health care professionals need to be aware of the importance of incorporating cognitive evaluations in their assessment and determination of an individual with MS's fitness to drive.

Lings (2002) conducted a 10-year historical cohort register-study, with 197 participants with MS and 546 control participants individually matched on age, gender, place of residence and exposure period. Participants were excluded from the study if they had no

driving licence, or if they had been admitted to a hospital with one of the following diagnoses: cerebrovascular disease, epilepsy, diabetes mellitus, dementia, psychoses, or alcoholism. In this study, exposure period was defined as the period of time, after the date of diagnosis, in which the individual held a driving licence. The outcome measure was treatment at the emergency department after a motor vehicle crash as a car driver. Lings reported that over the period of 1980 and 1989, five individuals with MS and four controls had been treated. The crude crash rate in the MS group was 0.025 (5/197) and in the control group 0.007 (4/545), resulting in a crude rate ratio of 3.46. The relevant exposure in the MS group was 1500.44 years and in the control group 4084.30 years. Therefore the crash rate per 1000 person-years in the MS group was 3.3 (i.e., $[5/1500.44] \times 1000$) and 0.98 for the control group (i.e., $[4/4084.30] \times 1000$). Lings reported that the crash rate per 1000-years was 3.4 times higher in drivers with MS compared to the control cohort (i.e., $3.3/0.98$, CI 0.73-17.15, $p < 0.05$). Lings concluded that drivers with MS were significantly more likely to be treated at the emergency department after having a motor vehicle crash.

Lings (2002) argued that selection bias is unlikely in this study because all registered participants with MS were included, and information bias was avoided by the use of register data only. However, the author did note that a potential source of confounding lies in the possibility that individuals with MS might be more prone to seek treatment because they are familiar with the hospital. However on the other hand, fear of losing their licence is known to play a significant role in individuals with medical conditions that affect their driving (see also section 3.5) and this may deter individuals from seeking treatment, resulting in the opposite effect.

Lings (2002) also noted that in the present study, crash frequency was calculated on the basis of years a driving licence had been held and not in relation to actual driving distance (mileage). Lings argued that this method was selected because the question of mileage is complex. For example, drivers with MS may drive less than healthy drivers because of self-regulation or as a consequence of decreased occupational activity, thereby producing fewer crashes than others even if their mileage crash risk were great. On the other hand, mileage may be a confounder as it is possible that individuals with MS drive more than others, for instance to seek treatment. This would increase the difference between groups. Lings notes that the outcome measure used in the present study: driver's treatment at the emergency department after a crash, must be considered insensitive because such events are rare, and the small numbers is a patent weakness. Furthermore, this method does not take into account minor crashes or injuries leading to a visit by other road users or passengers, nor does it take into account crashes that only involve material damage.

Citations

As outlined above, Schultheis et al. (2002) investigated the incidence of motor vehicle crashes and citations as documented by driving reports from the DMV among drivers with MS. Specifically, the authors hypothesised that individuals with MS and cognitive impairment would show a higher incidence of citations than individuals with MS who are not cognitively impaired and controls. The authors reported that there was no statistically significant difference in the incidence of citations across the three groups.

Driving Performance

The impact of cognitive impairment on driving skills and abilities has been documented in various neurologic populations including dementia, traumatic brain injury and stroke (see sections 3.3 and 3.4). Schultheis, Garay and DeLuca (2001) studied the impact of cognitive impairment on driving skills by comparing the performance of individuals with MS who demonstrated cognitive impairment ($n = 14$) with individuals with MS without cognitive impairment ($n = 13$) with a healthy control group ($n = 27$, matched on age and driving experience) using two computerised measures of driving skills. Cognitive impairment was scored using the same method outlined previously for Schultheis et al. (2002). Two instruments were used to assess driving-related skills: the Useful Field of View (UFOV) and the Neurocognitive Driving Test (NDT). The UFOV quantifies the visual field area over which a driver can process rapidly presented visual information (see section 3.13). As outlined in section 3.13, recent research on UFOV indicates that it is consistently and significantly associated with crash risk even after adjusting for other factors (Myers et al., 2000; Owsley, Ball et al., 1998; Sims et al., 2000). The NDT is comprised of five sections: 1) Self-Evaluation Questions, 2) Pre-Driving Questions, 3) Simple and Choice Reaction Time, 4) Driving Scenarios, and 5) Visual Task, and generates two composite scores: total error score (NDT-ERR) and total latency time score (NDT-LAT).

Participants with MS and cognitive impairment performed slower on measures of timed responses throughout the NDT ($M = 4416$ msec, $SEM = 313$ msec) than both controls ($M = 2785$ msec, $SEM = 201$ msec) and MS participants without cognitive impairment ($M = 2695$ msec, $SEM = 155$ msec, $p < 0.001$). However no significant difference was observed between MS participants without cognitive impairment and controls. Furthermore, there was no significant difference in the average number of errors committed during the driving related tasks of the NDT across the three groups.

On the UFOV overall score, there was a significant difference observed across the three groups ($\chi^2 = 12.49$, $p < 0.01$). Specifically, a higher proportion of MS participants with cognitive impairment (29%) compared to MS participants without cognitive impairment (0%) and controls (0%) were categorised in the high-risk group for probability of driving difficulties based on the overall UFOV performance. Analysis of the three subsections of the UFOV revealed that MS participants with cognitive impairment performed significantly poorer on two of the sub-tests. On the central vision and processing speed subsection of the UFOV, MS participants with cognitive impairment performed significantly worse ($M = 2.9$, $SEM = 0.13$) than both the control group ($M = 2.6$, $SEM = 0.12$) and MS participants without cognitive impairment ($M = 2.7$, $SEM = 0.12$, $F(2,44) = 3.6$, $p < 0.05$). On the divided attention subsection, there was no significant difference in the performance across the three groups. On the selective attention subsection of the UFOV, MS participants with cognitive impairment ($M = 5.3$, $SEM = 0.16$) performed significantly worse than the control group ($M = 4.8$, $SEM = 0.14$) but were not significantly different to the MS participants without cognitive impairment ($M = 5.1$, $SEM = 0.15$).

It should be noted that this study did not include individuals with MS who had severe physical impairments, and is thus not applicable to the more physically impaired MS population. Additional studies are needed to clarify what specific cognitive factors influence driving performance, how physical impairments affect driving skills, and the potential benefits of cognitive rehabilitation on driving ability. The authors conclude that cognitive impairment can negatively affect driving-related skills in individuals with MS and should be considered in the determination of fitness to drive.

Treatment of MS and road safety outcomes

The literature review did not identify any studies that specifically investigated the relationship between medications prescribed for MS and road safety outcomes including motor vehicle crash involvement, citations and driving performance using simulator or real-world driving measures.

Post-May 2003: Relationship between epilepsy and road safety outcomes

The review conducted for the period post-May 2003 identified one study addressing risk of crashes and two studies relating to driving performance measures amongst people with MS. Table 26 provides a summary of the findings of these studies.

Crashes

Ryan et al. (2009) investigated the relationship between characteristics of individuals with MS and their driving status. Patients were recruited from a neurological clinic in Detroit and were not eligible to participate if they suffered from a CNS disorder, psychiatric illness, suffered from substance abuse or had significant visual problems. Participants included 78 patients who were diagnosed with mild to severe MS as measured by the Kurtzke Expanded Disability Scale (Kurtzke, 1983), and 78 significant others who provided ongoing support to the patient and knew about the patient's driving habits. The sample comprised 60 patients who were currently driving and 18 non drivers. While the drivers and non drivers were of similar age, education level, income and gender, the driving groups differed according to the level of disability associated with MS. Participants completed a Barriers to Driving Questionnaire-Social Influences scale (BDQ Social) and a battery of neuropsychology tests, while their significant others were asked to rate their driving safety. Neuropsychological tests included Oral Symbol Digit Modalities Test, Paced Auditory Serial Addition Test, Judgment of Line Orientation-Short Form, Modified Stroop Test, Controlled Oral Word Association Test, Hopkins Verbal Learning Test-Revised, Trail Making Parts A and B and the Benton Visual Naming Test. Test performance scores were combined to form a neuropsychology composite score. Participant driving history for the last five years was obtained from the Department of Motor Vehicles. For analysis purposes the researchers combined crashes and citations to give an overall score of driving safety. The researchers found that the driving ratings of significant others ($sr_i^2 = .14$), the BDQ Social ($sr_i^2 = .12$) and the Kurtzke disability rating ($sr_i^2 = .07$) contributed the most to the prediction of driving safety (crashes and citations). Duration of illness was not related to the number of traffic incidents and citations, nor was neuropsychological test performance. Overall, non drivers performed significantly worse than drivers on all tests of neuropsychological function. A significant limitation of this study with respect to advancing evidence on risk status of drivers with MS was the absence of an unimpaired control group and hence the lack of relative risk estimates.

Citations

As reviewed above, Ryan and colleagues (2009) used a combined measure of crashes and citations as a road safety outcome to assess risk amongst drivers with PD. No other studies were identified in the period post-May 2003 addressing the association between MS and citations as a measure of road safety risk.

Driving Performance

Since 2003 two studies have assessed driving performance of patients with MS. Lincoln and Radford (2008) investigated whether or not a battery of cognitive tasks could predict on road driving performance in a sample of 34 individuals with MS. The patients were recruited from the Derby Regional Mobility Centre in the UK between the years 1995 to 1998. The eligibility criteria included a diagnosis of MS (no criteria provided), lived within 50 miles of the centre, driving for the past two years and successfully passed the driving test before the onset of MS. An interview was conducted in order to obtain information relating to driving behaviour, and the condition of MS. In addition, participants completed a small battery of cognitive tasks and a driving assessment. Cognitive tasks included; The Stroke Drivers Screening Assessment (SDSA), which assesses verbal reasoning, attention, concentration and executive functioning; The Paced Auditory Serial Addition Test (PASAT) used to assess speed of processing and sustained attention; The Stroop Task which assesses reasoning, selective attention and higher level functioning; The Test of Motor Impersistence which requires the participant to sustain a series of motor acts for 20 seconds and assesses self control; and the Adult Memory and Information Processing Test (AMIPB) which consists of a battery of tests designed to assess memory and information processing. All participants also completed the Extended Activities of Daily Living (AEDL).

The driving assessment consisted of a measure of visual acuity, visual attention, a medical history and a drive on a test track. The drive was assessed by an independent assessor, who was blind to the cognitive condition of the driver and the Nottingham Neurological Driving Assessment was used. The final driving test result was a pass or fail. Thirteen participants were considered unsafe to drive and were compared to the remaining twenty-one participants. No significant differences in age, years spent driving, or time since onset of MS were found between the safe and unsafe drivers. However, there was a significant difference in gender. A greater proportion of females failed compared to males ($p = .03$). In relation to cognitive test performance a significant association between on road driving test performance was found for the following tests; SDSA subtests of road sign recognition and dot cancellation, and the AMIPB figure copy, design learning interference, and information processing sub-tests. On the basis of these findings Lincoln and Radford (2008) generated a discriminant equation to predict safe/unsafe driving. The cognitive predictors that contributed to the model included tests related to visuo-spatial ability, information processing, executive function, visual memory and concentration. The authors claimed that the equation had a predictive accuracy of 88% and suggested that the equation could be used to identify unsafe drivers with MS-related cognitive impairments. The research neglected to consider individuals with MS who experience more severe symptoms who are unlikely to present at the specialist driving centre. Therefore, the study may be susceptible to sample bias. Similarly, the single-centre recruitment and lack of a control group increases the possibility of cohort effects.

Marcotte and colleagues (2008) also investigated the contribution of cognition as well as spasticity levels on driving performance of individuals with MS. The study had two aims: firstly, to compare driving performance of 17 individuals with MS to 13 healthy controls using a driving simulator; and secondly, to investigate the relationship between cognitive task performance levels of MS associated spasticity and driving performance. The inclusion criteria included; participants who had driven more than 400km in the past year, received a diagnosis of MS from a neurologist, had spasticity which was at a moderate level, aged 18-35 years, fluent in English and were on a stable dose of medication for at least 3 months. Participants were excluded from the study if they had

a psychiatric illness, were on unstable medication, had substance abuse, were using high doses of medication, or required benzodiazepines to reduce spasticity. The control group comprised individuals recruited from the general community and was similar to the cases in terms of age, number of years driven, education and gender. In the first instance cases were administered a small battery of cognitive tests and a test of spasticity to determine the level of MS affected abilities. The cognitive assessments comprised: Grooved Pegboard Test, TMT-A, WAIS- digit symbol, Paced Auditory Serial Test, TMT-B, and the Hopkins Verbal Learning Test-Revised (HVLTR). Spasticity levels were determined using the Modified Ashworth Scale (MAS).

Following the cognitive tests participants completed two simulator drives. The first simulator task was designed to increase cognitive load and consisted of a lane tracking task where participants were instructed to hold a constant speed for approximately 7 minutes. Throughout the drive symbols appeared on the screen and participants were required to respond by pressing either the right or left indicator or the horn. The second drive was a car following task whereby participants were instructed to drive behind a car which varied its speed. This driving task required a greater number of pedal movements compared to the first. The authors proposed that higher levels of spasticity would be associated with poor performance on driving tasks that required a greater number of foot movements. Intuitively, poor cognitive task performance was expected to be associated with difficulties completing the cognitive load task. Compared to the controls, drivers with MS drove faster in the simulator and recorded a greater standard deviation in lateral position (SDLP). In the car following task MS participants were slower to respond to the lead cars changes in speed compared to controls. Poor cognitive functioning was associated with variations in lane position. Specifically, SDLP was correlated with TMT-B, digit symbol and HVLTR total words. Levels of spasticity were related to variables which were associated with foot movements such as changing and maintaining speed. A methodological limitation of the study is the small sample size and the fact that participants were recruited from a clinical trial. For these reasons it is important to exercise caution when generalising the results. In addition, the authors note that the short length of the simulator drives may limit the sensitivity.

Treatment of MS and road safety outcomes

To our knowledge there were no studies conducted since 2003 that assessed the relationship between medications, MS and driving performance.

Summary

Few studies have investigated crash risk associated with drivers with MS. In the review period since May 2003, only one published study was identified examining crash involvement of drivers with MS, however this study provided no evidence on relative risk compared with unimpaired drivers. Nevertheless, the study by Lings (2002) provides strong evidence for a more than three fold elevated risk. To date, the majority of the research has been experimental work examining driving performance in laboratory-based, simulator and on-road driving settings and with an emphasis on the role of cognitive impairment in driving performance. The common finding from these studies is that MS-related cognitive impairments (when present) are associated with poorer driving performance. More recent studies have differentiated between cognitive and physical symptoms of the disease. Evidence from these studies suggests that physical impairments can negatively impact upon driving performance in different ways. For example there is evidence to suggest that physical impairment can negatively

impact upon driving manoeuvres that are directly related to foot movements (Marcotte et al. 2008). Future studies could incorporate larger sample sizes and include participants with a greater level of disease expression are warranted in future research. No evidence of the impact of treatment for MS on driving performance was found.

Table 26 Summary of studies of risk associated with MS

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Lincoln & Radford (2008)	Cases = 34 p w MS (24-69yrs)	<p>Cognitive tests: The Stroke Drivers Screening Assessment (SDSA), Paced Auditory Serial Addition Test (PASAT), The Stroop Task, Test of Motor Impersistence, Adult Memory and Information Processing Battery (AMIPB), Extended Activities of Daily Living</p> <p>Vision tests: visual acuity, visual attention</p> <p>On road driving performance: (pass/fail)</p>	<p>Failure on the driving performance test was correlated with scores on;</p> <ul style="list-style-type: none"> • SDSA road side recognition and dot cancellation components, • AMIPB figure copy, design learning interference and info. processing adjusted score components ($p < .05$).
Marcotte et al. (2008)	<p>Case/control</p> <p>Cases = 17 people with MS</p> <p>Controls = 14 people</p>	<p>Cognitive tests: Grooved Pegboard Test, TMT-A, Wechsler Adult Intelligence Scale-digit symbol, PASAT, TMT-B, and the Hopkins Verbal Learning Test Revised (HVLTR).</p> <p>Spasticity test: Modified Ashworth Scale (MAS)</p> <p>Two driving simulator test drives: including a divided attention task, and lane following task.</p>	<p>Spasticity level – no sig diff to standard deviation lane position (SDLP).</p> <p>Speed: MS faster than controls</p> <p>SDLP sig. correlated with TMT-B ($r = -.49, p = .052$), digit symbol ($r = -.51, p = .037$) and HVLTR total words ($r = -.63, p = .006$).</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Ryan et al. (2009)	<p>Case/control</p> <p>Cases = 78 p with MS, 60 currently driving</p> <p>Controls = 78 significant others</p>	<p>Cognitive tests: Oral Symbol Digit Modalities Test, PASAT, Judgment of Line Orientation-Short Form, Modified Stroop Test, Controlled Oral Word Association Test, HVLT-R, TMT-A and B, and the Benton Visual Naming Test.</p> <p>The Awareness Questionnaire, Social Barriers subscale of the Barriers to Driving Questionnaire.</p> <p>Driving records past 5 years as documented by Department of Motor Vehicles. Citations and accidents combined.</p>	<p>The duration of the illness was not related to driving incidents, nor was neuropsychological performance.</p> <p>Driver ratings of significant others ($sr_i^2 = .14$) the BDQ Social ($sr_i^2 = .12$) as well as the disability rating score ($sr_i^2 = .07$) contributed the most to the prediction of driving safety (citations and accidents).</p>
Lings (2002)	<p>10-year historical cohort register-study</p> <p>Cases = 197 p with MS</p> <p>Controls = 546 p</p>	<p>Acc rate per 1000 person-years</p>	<p>Crash rate per 1000-years 3.4 times higher MS compared to C*</p>
Schultheis, Garay, Millis & DeLuca (2002)	<p>Case control</p> <p>Cases = 27 p w MS (14 w/o cog impair and 13 w cog impair)</p> <p>Controls = 17</p>	<p>Incidence of MVCs and motor vehicle citations as documented by DMV reports for past 5 years</p>	<p>Crash rate: MS + cog impair > MS -cog impair & C*</p> <p>Crash rate: MS -cog impair = C</p> <p>Citations: MS + cog impair = MS -cog impair = C</p>

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Schultheis, Garay & DeLuca (2001)	<p>Case control</p> <p>Cases = 28 p w MS (13 w/o cog impair and 15 w cog impair)</p> <p>Controls = 17</p>	<p>Cognitive tests:</p> <p>Block Design and Digit Symbol subtests (WAIS-R), Stroop Colour-Word Test, Trail Making Test, Motor-Free Visual Perceptual Test-Revised and the Paced Auditory Serial Addition Test</p> <p>2 computerised driving tests:</p> <p>Useful Field of View (UFOV)</p> <p>Neurocognitive Driving Test (NDT)</p>	<p>Time on NDT: MS + cog impair > MS - cog impair & C***</p> <p>Errors on NDT: no sig diff</p> <p>central vision and processing speed errors: MS + cog impair > MS - cog impair & C*</p> <p>Divided attention: No sig diff</p> <p>selective attention errors: MS + cog impair > C* but were not sign diff to the MS - cog impair</p>

Approaches to Management

Assessing fitness to drive

As summarised below (see Table 27), most licensing jurisdictions outline that in the early stages of MS, no driving restrictions may be necessary, however most jurisdictions recommend periodic licence reviews (in most cases annually). In addition, most licensing jurisdictions suggest that there is a possibility of using conditional/restricted licensing criteria for drivers with MS. Sweden appears to have the most explicit recommendations in that they suggest a risk assessment which includes an appraisal of the stage of the disease and the effect of treatment.

Self-regulation

There is little information pertaining to the self-regulatory practices of drivers with MS. The secondary aim of the study described above by Ryan et al. (2009) was to investigate the relationship between the individuals' awareness of their deficits and their driving behaviour. Participants completed the Awareness Questionnaire to assess their awareness of their deficit before and after the onset of their MS and to evaluate how participants felt about their own driving ability. The extent to which the patients driving behaviour was influenced by external social influences was assessed using the Social Barriers subscale of the Barriers to Driving Questionnaire. The authors hypothesised that individuals who lacked awareness would be less likely to engage in compensatory strategies and would be more prone to adverse traffic safety outcomes. This is indeed what was found. Out of the number of patients currently driving 27% rated themselves as more functionally able than their caregivers, indicating a lack of awareness. Driver awareness was positively correlated with engaging in compensatory behaviours. Furthermore, drivers who drove fewer kilometers per week engaged in more compensatory behaviours. Ryan et al. (2009) found that the perception of significant others was positively associated with the number of traffic incidents, as was self-reported driving exposure, and significant others rating of driving ability. However, the duration of the illness since becoming licensed was not related to driving incidents, nor was neuropsychological performance. In conclusion, drivers were found to have less social pressures than non drivers, and drivers who were unaware of their deficits were not engaging in any compensatory driving behaviours. The authors acknowledged the small sample size of the sample and note the potential sampling bias as participants were recruited from a single clinic which limits the ability to generalize the findings. These findings should be interpreted cautiously because driving exposure was obtained via self-report in terms of frequency of trips per week rather than kilometers driven which would have been a more reliable estimate.

Table 27 Private licensing guidelines for drivers with MS

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Multiple Sclerosis	<p>The degree and severity of the disease determine an individual's capacity to drive. If mechanical appliances are installed in the vehicle the applicant must demonstrate competency in a driving test.</p>	<p>An unconditional licence may not be held if the disease impairs driving.</p> <p>A conditional licence may be issued subject to the results of a driving assessment & treatment response & with appropriate vehicle modifications.</p> <p>Subject to yearly reviews (minimum).</p>	<p>May be licensed if medical confirmation obtained that driving ability (e.g. nerves and circulation) is unimpaired.</p> <p>Vehicle modifications may be required.</p> <p>Licence may be restricted to short-period licences requiring renewal every 1, 2 or 3 years</p>	<p>An unrestricted licence may be issued if the person is able to control equipment & has no or minimal neurological impairment.</p> <p>Annual review required for minimal impairment.</p> <p>If the person is able to control equipment despite slight neurological impairment, a road test must first be passed before licensing can occur.</p> <p>Annual review required.</p> <p>A restricted licence with speed &/or area restrictions, may be issued if the person has moderate dexterity impairment.</p>	<p>Driving to cease if person is unable to react appropriately to emergency situations or where quick responses are required. If a person has trouble with walking, it is likely that they will be unfit to drive.</p> <p><i>Early stages of illness:</i></p> <p>Driving may continue provided this can be done effectively.</p> <p>Due to the aetiology of this disease (varying progression rates & periods of significant remission) it may be possible to permit driving during periods of remission & restrict driving during other active phases.</p> <p>Yearly review may be</p>	<p>Licence denial or revocation if disease impairs driving ability & so renders the person a traffic safety risk.</p> <p>Risk assessment to include an appraisal of the stage of the disease & treatment response.</p> <p>Periodic review required on a case-by-case basis.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				<p>Annual review required.</p> <p>Greater restrictions (speed/area/time of day/must be accompanied by licensed driver) are imposed if there is <i>temporary</i> significant neurological impairment.</p> <p>Six-monthly review required.</p>	<p>required.</p> <p><i>Other stages of illness:</i></p> <p>Licence revocation.</p>	

3.8.3 CEREBRAL PALSY

Definition of Cerebral Palsy (CP)

Cerebral Palsy (CP) is an umbrella-like term used to describe a group of chronic disorders of movement and posture that are caused by impaired development of, or damage to, motor centres in the brain (NINDS, 2001; Batshaw, 1997).

Symptoms of PD may include:

- deficits in fine motor control (such as writing);
- balance problems;
- problems with walking;
- involuntary movements.

The symptoms and severity differ on an individual basis, resulting in functional impairments ranging from mild to profound. The events or conditions that result in CP may also produce several other associated disorders including seizures, visual and auditory impairments, learning difficulties, cognitive impairment and behavioural problems (Batshaw, 1997). Clinicians diagnose CP on the basis of motor skills and reflexes, medical history, and other specifically designed measures.

Risk factors for CP may be congenital or acquired after birth including: genetic abnormalities that may lead to impaired brain development in the early stages of embryonic development; intrauterine infections that may impair developing fetal nervous systems; pregnancy related abnormalities that may lead to preterm delivery and related complications; adverse conditions during labour and delivery which may lead to oxygen and/or blood deprivation necessary for vulnerable areas of an immature brain; traumatic head injury; and rubella/German measles (Batshaw, 1997).

In summary, the damage or dysfunction causing CP generally occurs during an early period of the brain's development and is not progressive. This distinguishes CP from other progressive disorders of movement and posture such as PD which was outlined at the start of the neurological conditions section.

Prevalence of CP

The overall prevalence of CP has remained relatively stable in recent decades and is estimated as occurring in 2.36 births per thousand (Sanner, 1996; cited in Falkmer & Gregersen, 2000). Early signs of CP are usually apparent by 3 years of age. Infants with CP are generally slowed in physical development.

Functional impairments associated with CP relevant to driving

Although cerebral palsy is generally characterised as a disorder of movement and posture, impairments associated with CP have also been reported in other areas that are important to driving (Jahnsen, Villien, Stanghelle & Holm, 2003). These include:

- impaired range of motion and weakness;
- exaggerated startle reflex to loud noise;

- excessive muscle tone;
- problems coordinating movements;
- visual impairments (acuity, slowed tracking);
- learning difficulties;
- impaired judgement in complex situations;
- slow processing and reaction time;
- quick to fatigue.

People with CP often rely on wheelchairs, and vehicle adaptations are required to allow them to access and operate motor vehicles independently. As with drivers with cognitive impairment (see section 3.4), full and thorough evaluation on a case-by-case basis is required to assess the capabilities and needs of individuals when it comes to licensing, training and vehicle adaptation. Thus maximising the independence of the individual and their own safety and that of other road users.

One particular problem experienced by those people diagnosed with CP, which is of particular relevance to driving, is the quality of their visual search abilities (Maltz & Shinar, 1999). Individuals with CP appear to have less flexible visual strategies available to them, leading to slower information processing. This has also been shown in healthy novice drivers, but they are able to adopt effective strategies far quicker than people with CP (Underwood, Chapman, Brocklehurst, Underwood & Crundall, 2003).

Relationship between CP and road safety outcomes

Table 28 shows a summary of the findings of studies that have investigated CP and road safety outcomes including crashes, citations and driving performance.

Crashes

No studies reporting crash rates amongst drivers with CP were found.

Citations

No studies reporting rates of citations or violations amongst drivers with CP were found.

Driving Performance

Falkmer and Gregersen (2000) carried out a study aimed at isolating visual processing strategies within drivers with CP with a view to developing a way to teach them as a part of driver education. The authors compared the visual scanning patterns of learner drivers with CP (n=15), healthy learner drivers (n=20) and experienced drivers (n=20), over a 30-minute in car drive using an eye-tracking device. They found that novice drivers tended to concentrate more on a smaller area, nearer to their vehicle, and the learners with CP did this even more. The learners with CP also had more in-car eye fixations than the other groups. Also the learner drivers with CP were shown to have greater difficulty driving in complex traffic situations, due to their reduced scanning ability. The authors conclude that this is in support of the idea of teaching CP learners appropriate scanning strategies early to increase their ability to use them through the learning to drive phase and beyond. Again the small sample size is problematic for

making generalisations. Also the participants with CP were near to completion of their driver education, and shortly after the study they all obtained their licences. It is possible then that this group had already begun to compensate in some ways for scanning deficits and are not representative of the population of drivers with CP. Nevertheless these findings do indicate a strategy for improving driver education for drivers with CP.

Falkmer, Henriksson, Gregersen and Bjurulf (2000) investigated the driver education process in Sweden, with a view to finding particular difficulties encountered in the system by drivers with CP. They studied logbooks of lessons obtained from driving instructors. The learner drivers with CP tended to experience particular difficulties in multi-tasking, and to have perceptual problems. The authors advocated the development of test batteries to assess dual task performance and elements of perceptual skills to allow lessons to be programmed to the special needs of each candidate.

Post May 2003: Relationship between CP and road safety outcomes

The review of literature published between May 2003 and mid-2009 revealed no new studies concerning crashes, citations or driving performance and CP.

Crashes

No research papers assessing crash risk and CP were identified in the review of literature published between May 2003 and mid-2009.

Citations

There were no studies in the review period that investigated the association between citations and CP.

Driving Performance

There were no studies in the review period that investigated the association between driving performance and CP.

Treatment of CP and road safety outcomes

No studies reporting the relationship between treatment of CP and risk were found.

Summary

In conclusion, there are very few studies that have investigated the relationship between CP and driving ability. Most studies have focussed on the needs of individuals with CP in terms of driver education. Driver education programs may not be appropriate or adequate for individuals with CP. They may need more lessons or more individually tailored lessons specific to their impairment and adaptations within their motor vehicles.

There are unique methodological difficulties with CP and similar congenital conditions where the emphasis is on fitness to *learn* to drive rather than driving itself. This means in effect that every person with one of these conditions is subjected to assessment prior to commencing driving when they have no advantage of experience. This is in contrast to most of the other conditions discussed in the review, which are *acquired* once the person has been driving for some time. The result is a more thorough filtering out of

those deemed initially to be unfit to drive, as judged by a medical assessment and an on-road test. A crash risk study of those who get through the licensing hurdles would be most interesting.

Table 28 Summary of studies of risk associated with CP

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Falkmer & Gregersen (2000)	<ul style="list-style-type: none"> - CP learners (n=15), - novice control learners (n=20) - experienced drivers (n=20). 	- visual scanning over a 30-minute car drive using an eye-tracking device.	<ul style="list-style-type: none"> - novice drivers tend to concentrate more on a smaller area, nearer to their vehicle, and the CP learners did this even more. - CP learners more eye fixations in-car than the other groups. - CP learners had greater diff driving in complex traffic situations, due to their reduced scanning ability.
Falkmer, Henriksson, Gregersen & Bjurulf (2000)	77 learner drivers with CP	- logbooks of lessons obtained from driving instructors.	- CP learners had particular difficulty in multi-tasking, and to have perceptual problems.

Approaches to management

Assessing fitness to drive

As summarised below in Table 29, most licensing jurisdictions outline that an unconditional/unrestricted licence may be issued to a driver with CP if there is no or minimal neurological impairment, and if the disorder does not impair driving. In Canada and NZ, drivers are only required to undergo one medical examination and one on-road test, whereas in Australia, USA and Sweden drivers with CP are required to undergo periodic licence reviews (in most cases annually). Finally, most licensing jurisdictions recommend the use of vehicle modifications where necessary. Due to the fact that there have been no studies which have explicitly investigated the crash risk of drivers with CP, it is difficult to determine if these guidelines are adequate. More research in this area is needed.

Table 29 Private licensing guidelines for drivers with CP

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Cerebral Palsy	<p>The degree and severity of the disease determine an individual's capacity to drive. If mechanical appliances are installed in the vehicle the applicant must demonstrate competency in a driving test. A medical report must be provided when the individual applies for a driving test.</p>	<p>An unconditional licence may not be held if the disease impairs driving.</p> <p>A conditional licence may be issued subject to the following criteria:</p> <ol style="list-style-type: none"> 1. Severity of disabilities. 2. Effect of multiple disabilities. 3. Treatment response and 4. Appropriate vehicle modifications. <p>A driving assessment may be of use.</p> <p>Subject to periodic review.</p>	<p>May be licensed if medical confirmation obtained that driving ability (e.g. nerves and circulation) is unimpaired.</p> <p>Vehicle modifications may be required.</p> <p>Licence may be restricted to short-period licences requiring renewal every 1, 2 or 3 years</p>	<p>An unrestricted licence may be issued if the person is able to control equipment & has no or minimal neurological impairment.</p> <p>Annual review required for minimal impairment.</p> <p>If the person is able to control equipment despite slight neurological impairment, a road test must first be passed before licensing can occur.</p> <p>Annual review required.</p> <p>A restricted licence with speed &/or area restrictions, may be issued if the person has moderate dexterity impairment.</p> <p>Annual review required.</p>	<p>No licence restrictions if the person passes the driving test.</p> <p>Car modifications may be required if there are problems with joint & limb flexibility, subject to assessment by an occupational therapist.</p>	<p>Licence denial or revocation if disease impairs driving ability & so renders the person a traffic safety risk.</p> <p>Periodic review required on a case-by-case basis.</p>

3.8.4 SPINA BIFIDA

Definition of Spina Bifida (SB)

Spina bifida (SB) is a neural tube defect resulting from incomplete development of the brain, or spinal cord, which occurs when the foetus's spinal cord does not completely close during pregnancy. Although this can be closed using surgery shortly after birth the neurological damage is permanent, leading to degrees of paralysis, generally in the lower limbs. There are three types of spina bifida which are outlined below.

- **Spina Bifida Occulta** - Spina bifida occulta literally means a hidden split in the spine. This is a very mild and common form and very rarely causes impairment. There is a slight deficiency in the formation of (usually) one of the vertebrae. It may have visible signs of a dimple or small tuft of hair growth on the lower back. However, many people are unaware that they have spina bifida occulta as they have no symptoms or signs.
- **Meningocele** - In this type of spina bifida, the meninges (covering of the spinal cord) protrude through the opening, causing a lump or sac on the back. The spinal cord is often undamaged. There are usually no long-term problems, although problems can arise. This is the least common form of spina bifida.
- **Myelomeningocele (or Meningomyelocele)** - This is the most common form of spina bifida and also the most severe. The sac that has protruded on the back contains cerebrospinal fluid, blood vessels and the damaged spinal cord and meninges. As a result, there is always some paralysis and loss of sensation below the damaged region. The amount of impairment depends very much on where the spina bifida is and the amount of nerve damage involved.

Spina bifida most often occurs in the lumbar region of the spinal cord, but may occur anywhere along the length of the cord. The level and severity of damage to the cord influences the severity and location of motor and sensory impairments.

Most individuals with SB also have hydrocephalus (from the Greek hydro = water, cephalie = brain) which is an accumulation of cerebrospinal fluid which arises from an imbalance in the production and drainage of that fluid. Hydrocephalus is a major cause of intellectual disability, but can be avoided or reduced by the insertion of a "shunt" to remove the fluid that accumulates (Jenkinson & Wilson, 1996).

Prevalence of SB

Spina bifida is the most frequent permanently impairing birth defect. It affects approximately one out of every 1,000 newborns in the US (Spina Bifida Association of America, 2003). Females are generally affected 3-7 times more frequently than males, and the incidence also increases with maternal age and lower-socio-economic status (Batshaw, 1997).

Functional impairments associated with SB relevant to driving

According to the Association for Driver Rehabilitation Specialists (2003), functional impairments associated with SB which could adversely affect driving include:

- limited range of motion and strength;
- difficulty with coordinated movements;
- visual impairments (poor acuity);
- trouble visually scanning or tracking quickly;
- learning difficulties;
- impaired judgment in complex situations;
- slow processing and reaction time.

Pre-May 2003: Relationship between SB and road safety outcomes

Few studies have attempted to investigate the relationship between SB and road safety outcomes. Table 30 summarises the few studies that have been conducted to date in this area.

Crashes

Simms (1991) reported a case-control study comparing the driving experiences of 36 participants with Spina Bifida with Hydrocephalus (SBH) and 36 healthy control drivers. Using a self-report questionnaire, Simms reported that in the first year after their licence assessment, participants with SBH were twice as likely to have been involved in one or more crashes than the control participants. Furthermore, Simms also reported that participants with SBH were driving far fewer miles than the controls. Simms concluded that the results of this study indicate a need for improved training strategies for drivers with SB to allow them to feel more capable and confident on the road and to fully develop driving skills. Methodological limitations of this study include the small sample size and the well documented limitations of obtaining information via self-report.

Citations

No studies reporting rates of citations or violations amongst drivers with SB were found.

Driving Performance

Although vehicle adaptations would allow many people with SB to be able to physically control a car, there are concerns surrounding the high incidence of associated cognitive impairments (visual perceptual skills and learning problems), as they may preclude safe driving and ability to learn to drive (Simms, 1987). Simms (1987) studied the cognitive abilities of 32 drivers with spina bifida (SB, n = 7) and spina bifida and hydrocephalus (SBH, n = 25) who attended a driving assessment. All participants were deemed to be physically capable of controlling a car. The cognitive tests included visual discrimination and scanning, visual memory, memory span, and reasoning. The cognitive performance of participants with SB was comparable to the non-impaired range, whereas the participants with SBH performed around a low average range. Two years following the original assessment, 15 participants had passed their driving test, 4

participants were still learning, and 9 participants had decided against driving (the full sample could not be contacted). Analysis comparing driver status and cognitive test battery indicated that the battery was a poor predictor of future driving status. Simms argued that this is encouraging as people with SB are able to find strategies to overcome their deficits in a driving situation.

Treatment of SB and road safety outcomes

No studies were found which examined the relationship between treatment of SB and driver risk.

Post May 2003: Relationship between SB and road safety outcomes

The review of literature published between May 2003 and mid-2009 revealed no new studies concerning crashes, citations or driving performance and SB.

Crashes

No research papers assessing crash risk and SB were identified in the review of literature published between May 2003 and mid-2009.

Citations

There were no studies in the review period that investigated the association between citations and SB.

Driving Performance

There were no studies in the review period that investigated the association between driving performance and SB.

Summary

In conclusion, there are very few studies in this area. While the two studies reported above provide some evidence for decrements in driving and elevated crash risk in SB, sample sizes were small and restricted sampling may have biased the findings. There were no new studies conducted since 2003 related to driving and SB. Hence, it would be premature to claim that a clear link has been established.

Table 30 Summary of studies of risk associated with SB

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Simms (1987)	32 SB adults SB only n=7: SB and hydrocephalus (SBH) n = 25	Cognitive abilities - visual discrimination and scanning, - visual memory, - memory span, and - reasoning.	SB group were comparable with the non-impaired range SBH group was lower than average range. - clinical assessment of cog functioning did not discriminate b/w drivers and non-drivers
Simms (1991)	case control study Cases = 36 SBH drivers Controls = 36 control participants	Questionnaire on: - driving tuition - car use - car adaptations - current driving patterns - route planning and using service stations - crash inv following licence test - miles travelled	Crashes in first year of driving : SBH (47%) > controls (22%).

Approaches to management

Assessing fitness to drive

As summarised below in Table 31, the USA, Canada and NZ licensing guidelines recommend that an unconditional/unrestricted licence may be issued to a driver with SB if there is no or minimal neurological impairment, and if the disorder does not impair driving. In Canada and NZ, drivers with SB are only required to undergo one medical examination and one on-road test, whereas in USA and Sweden drivers with SB are required to undergo periodic licence reviews (in most cases annually). Interestingly, the Australian guidelines do not list SB as a condition to take into account when determining fitness to drive. Given the nature of the condition, individual medical assessment with advice from an occupational therapist would almost always be required. Due to the fact that only one study explicitly investigated the crash risk of drivers with SB, it is difficult to determine if these guidelines are adequate. More research in this area is needed.

Table 31 Private licensing guidelines for drivers with SB

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Spina Bifida	The degree and severity of the disease determine an individual's capacity to drive. If mechanical appliances are installed in the vehicle the applicant must demonstrate competency in a driving test. A medical report must be provided when the individual applies for a driving test.	Not specifically addressed.	<p>May be licensed if medical confirmation obtained that driving ability (e.g. nerves and circulation) is unimpaired.</p> <p>Vehicle modifications may be required.</p> <p>Licence may be restricted to short-period licences requiring renewal every 1, 2 or 3 years</p>	<p>An unrestricted licence may be issued if person is able to control equipment & has no or minimal neurological impairment.</p> <p>Annual review required for minimal impairment.</p> <p>If the person is able to control equipment despite slight neurological impairment, a road test must first be passed before licensing can occur.</p> <p>Annual review required.</p> <p>A restricted licence with speed &/or area restrictions, may be issued if the person has moderate dexterity impairment.</p> <p>Annual review required.</p>	<p>No licence restrictions if the person passes the driving test.</p> <p>Car modifications may be required if there are problems with joint & limb flexibility, subject to assessment by an occupational therapist.</p>	<p>Licence denial or revocation if disease impairs driving ability & so renders the person a traffic safety risk.</p> <p>Periodic review required on a case-by-case basis.</p>

References

- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders (4th ed.)*. Washington, DC: Author.
- Amick, M. M., D'Abreu, A., Moro-de-Casillas, M. L., Chou, K. L., & Ott, B. R. (2007). Excessive daytime sleepiness and on-road driving performance in patients with Parkinson's disease. *Journal of the Neurological Sciences*, 252(1), 13-15.
- Amick, M. M., Grace, J., & Ott, B. R. (2007). Visual and cognitive predictors of driving safety in Parkinson's disease patients. *Arch Clin Neuropsychol*, 22(8), 957-967.
- Austrroads. (2003). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Austrroads. (2003). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Avanzi, M., Baratti, M., Cabrini, S., Uber, E., Brighetti, G., & Bonfa, F. (2008). The thrill of reckless driving in patients with Parkinson's disease: An additional behavioural phenomenon in dopamine dysregulation syndrome? *Parkinsonism & related disorders*, 14(3), 257-258.
- Avorn, J., Schneeweiss, S., Sudarsky, L. R., Benner, J., Kiyota, Y., Levin, R., & Glynn, R. J. (2005). Sudden Uncontrollable Somnolence and Medication Use in Parkinson's Disease. *Arch Neurol*, 62(8), 1242-1248.
- Berger, H. J. C., van Es, N. J. C., van Spaendonck, K. P. M., Teunisse, J.-P., Horstink, M. W. I. M., van, M. A., Hof, t., & Cools, A. R. (1999). Relationship between Memory Strategies and Motor Symptoms in Parkinson's Disease. *Journal of Clinical and Experimental Neuropsychology*, 21(5), 677 - 684.
- Boullion, L., Mazer, B., & Gelinas, I. (2006). Validity of the cognitive behaviour driver's inventory in predicting driving outcome. *American Journal of Occupational Therapy*, 60, 420-427.
- Batshaw, M.L. (1997). *Children with Disabilities (4th ed.)*. Philadelphia: MacLennan and Petty P/L.
- Brassington, J.C. & Marsh, N.V. (1998). Neuropsychology of multiple sclerosis. *Neuropsychological Review*, 8, 43-77.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Centre for Cerebral Palsy (2009). Retrieved 5th June 2009 from http://www.tccp.com.au/About_Cerebral_Palsy.
- Daum, I. & Quinn, N. (1991). Reaction times and visuospatial processing in Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology*, 136, 972 - 982.

- Devos, H., Vandenberghe, W., Nieuwboer, A., Tant, M., Baten, G., & De Weerd, W. (2007). Predictors of fitness to drive in people with Parkinson disease. *Neurology*, *69*(14), 1434-1441.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Dubinsky, R.M., Gray, C., Hustead, D., Busenbark, K., Vetere-Overfield, B., Wiltfong, D., Parrish, D. & Koller, W.C. (1991). Driving in Parkinson's disease. *Neurology*, *41*, 517-520.
- European Parkinson's Disease Association (EPDA, 2002). *Parkinson's Disease Fact Sheet*. Retrieved on June 10, 2003 from <http://www.epda.eu.com>
- European Parkinson's Disease Association (EPDA, 2008). *Parkinson's Disease Fact Sheet*. Retrieved on June 5, 2009 from http://www.epda.eu.com/patientGuide/LWP_1_01_whatIsPD.shtm
- Falkmer, T., & Gregersen, N.P. (2000). The prevalence of learner drivers with cerebral palsy who are in need of specialized driver education. *Journal of Traffic Medicine*, *28*, (3/4), 22-31.
- Frucht, S., Rogers, J.D., Greene, P.E., Gordon, M.F. & Fahn, S. (1999). Falling asleep at the wheel: motor vehicle mishaps in persons taking pramipexole and ropinirole. *Neurology*, *52*, 1908-1910.
- Georgiou, N., Iansek, R., Bradshaw, J.L., Phillips, J.G., Mattingley, J. B., Bradshaw, J.A. (1993). An evaluation of the role of internal cues in the pathogenesis of parkinsonian hypokinesia. *Brain*, *116*, 1575-1587.
- Ghorayeb, I., Loundou, A., Auquier, P., Dauvilliers, Y., Bioulac, B., & Tison, F. (2007). A nationwide survey of excessive daytime sleepiness in Parkinson's disease in France. *Movement Disorders*, *22*(11), 1567-1572.
- Goodkin, D.E. (1998). Commentary, *Lancet*, *352*, 1486-1487.
- Grace, J., Amick, M. M., D'Abreu, A., Festa, E., Heindel, W., & Ott, B. (2005). Neuropsychological deficits associated with driving performance in Parkinson's and Alzheimer's disease. *Journal of the International Neuropsychological Society*, *11*, 766-775.
- Gronin-Golomb, A., Gorkin, S., Growdon, J.H. (1994). Impaired problem solving in Parkinson's disease: impact of a set-shifting deficit. *Neuropsychologia*, *32*, 579-593.
- Heikkila, V.M., Turkka, J., Korpelainen, J., Kallanranta, T. Summala, H. (1998). Decreased driving ability in people with Parkinson's disease. *Journal of Neurology, Neurosurgery, & Psychiatry*, *64*(3), 325-330.
- Homann, C.N., Wenzel, K., Suppan, K., Ivanic, G., Kriechbaum, N., Crevenna, R. & Ott, E. (2002). Sleep attacks in participants taking dopamine agonists: review. *British Medical Journal*, *324*(22), 1483-1487.

- Hubble, J.P. & Berchou, R.C. (2003). Parkinson's Disease Medications. *The National Parkinson Foundation*. Retrieved on June 10, 2003 from <http://www.parkinson.org/medications.htm>
- Jahnsen R, Villien L, Stanghelle JK, Holm I. (2003). Fatigue in adults with cerebral palsy in Norway compared with the general population. *Developmental Medicine and Childhood Neurology*, 45(5), 296-303.
- Jenkinson, J.C. & Wilson, L.J. (1996). Causes and prevention of disability. In J. Annison, J. Jenkins, W. Sparrow and E. Bethune (Eds.). *Disability – A guide for Health Professionals*. Melbourne, Australia: Thomas Nelson Australia.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Lee et al. (2007). Validity of driving simulator in assessing drivers with Parkinson's disease, *Advances in Transportation Studies an International Journal*, Special Issue.
- Lincoln, N. B., & Radford, K. A. (2008). Cognitive abilities as predictors of safety to drive in people with multiple sclerosis. *Multiple Sclerosis*, 14(1), 123-128.
- Lings, S. (2002). Driving crash frequency increases in participants with multiple sclerosis. *Acta Neurologica Scandinavia*, 105, 169-173.
- Lings, S. & DuPont, E. (1992). Driving with Parkinson's disease. A controlled laboratory investigation. *Acta Neurologica Scandinavia*, 86(1), 33 – 39.
- Madeley, P., Hulley, J.L., Wildgust, H. & Mindham, R.H.S. (1990). Parkinson's disease and driving ability. *Journal of Neurology, Neurosurgery and Psychiatry*, 53, 580-582.
- Maltz M, & Shinar D. (1999). Eye movements of younger and older drivers. *Human Factors*, 41(1), 15-25.
- Marcotte, T. D., Rosenthal, T. J., Roberts, E., Lampinen, S., Scott, J. C., Allen, R. W., & Corey-Bloom, J. (2008). The contribution of cognition and spasticity to driving performance in Multiple Sclerosis. *Archives of Physical Medicine and Rehabilitation*, 89(9), 1753-1758.
- Mathers, C.D., Stein, C., Ma Fat, D., Rao, C., Inoue, M., Tomijima, N, Bernard, C., Lopez, A.D. & Murray, C.J.L. (2002) *Global burden of disease 2000: Version 2. Methods and results*. Global Programme on Evidence for Health Policy Discussion Paper No. 50. World Health Organization.
- Medscape (2007). *Spina Bifida*. Retrieved June 8, 2009 from <http://emedicine.medscape.com/article/1266529-overview>
- [Mehta P](#), [Kifley A](#), [Wang JJ](#), [Rohtchina E](#), [Mitchell P](#), & [Sue, CM](#). (2007) Population prevalence and incidence of Parkinsons' disease in an Australian community. *Internal Medicine Journal*, 37(12),812-4.

- Meindorfner, C., Körner, Y., Möller, J., Stiasny-Kolster, K., Oertel, W., & Krüger, H. (2005). Driving in Parkinson's disease: Mobility, accidents, and sudden onset of sleep at the wheel. *Movement Disorders, 20*(7), 832-842.
- Multiple Sclerosis Foundation (2002). *MS Info*. Retrieved June 11, 2003 from <http://www.msfacts.org/treatment.htm>
- Multiple Sclerosis International Federation (MSIF) (2007). Retrieved 5th June 2009 from <http://www.atlasofms.org/query.aspx?pq=yes&s=1&q=1&r=Global&year=2007>
- National Institute of Neurological Disorders and Stroke (NINDS, 2009). *Parkinson's disease: Hope Through Research*. Retrieved on June 2, 2009 from http://www.ninds.nih.gov/disorders/parkinsons_disease/parkinsons_disease.htm
- National Institute of Mental Health (NIMH, 2002). *Depression and Parkinson's Disease*. Retrieved on June 10, 2003 from <http://www.nimh.nih.gov/publicat/depparkinson.cfm>
- National Institute of Neurological Disorders and Stroke (NINDS, 2001a). *Parkinson's Disease Information Page*. Retrieved on June 10, 2003 on http://www.ninds.nih.gov/health_and_medical/disorders/parkinsons_disease.htm
- National Institute of Neurological Disorders and Stroke (NINDS, 2001b). *Multiple Sclerosis Information Page*. Retrieved on June 10, 2003 from http://www.ninds.nih.gov/health_and_medical/disorders/multiple_sclerosis.htm
- National Multiple Sclerosis Society (2003). *Living with MS*. Retrieved on June 10, 2003 from <http://www.nationalmssociety.org/Treatments.asp>
- Neurology channel (2003). *Multiple Sclerosis*. Retrieved from June 11, 2003 from <http://www.neurologychannel.com/multiplesclerosis/>
- Pacchetti, C., Martignoni, E., Terzaghi, M., Zangaglia, R., Mancini, F., Nappi, G., & Manni, R. (2003). Sleep attacks in Parkinson's disease: a clinical and polysomnographic study. *Neurological Sciences, 24*(3), 195-196.
- Parkinson's Disease Foundation (2002). *Parkinson's Disease: An Overview*. Retrieved from June 10, 2003 from <http://www.pdf.org/AboutPD/>
- Parkinson Society Canada (2002). *Parkinson's Disease*. Retrieved on May 29, 2003 from <http://www.parkinson.ca/pd/nd.html>
- Radford, K. A., Lincoln, N. B., & Lennox, G. (2004). The effects of cognitive abilities on driving in people with Parkinson's disease. *Disability & Rehabilitation, 26*(2), 65 - 70.
- Rokšar, S. & Sever, A. (2001). *Cognitive Impairment in Multiple Sclerosis*. Retrieved on June 10, 2003 from <http://www.ff.uni-lj.si/Psihologija/iLibrary/IS2001-CN/PDF/21-Roskar-Sever.pdf>
- Ryan, K. A., Rapport, L. J., Telmet Harper, K., Fuerst, D., Bieliauskas, L., Khan, O., & Lisak, R. (2009). Fitness to drive in multiple sclerosis: Awareness of deficit

- moderates risk. *Journal of Clinical and Experimental Neuropsychology*, 31(1), 126-139.
- Salzberg, P. & Moffat, J. (1998). *The Washington State Department of Licensing Special Exam Program: An Evaluation*. Washington Traffic Safety Commission. Olympia, WA.
- Schrag A., Ben-Shlomo, Y., & Quinn, N, P.(2000). Cross sectional prevalence survey of idiopathic Parkinson's disease and Parkinsonism in London. *BMJ*, 321(7252), 21-2.
- Scultheis, M.T., Garay, E., & DeLuca, J. (2001). The influence of cognitive impairment on driving performance in multiple sclerosis. *Neurology*, 56(8), 1089-1094.
- Scultheis, M.T., Garay, E., Millis, S.R. & DeLuca, J. (2002). Motor vehicle crashes and citations among drivers with multiple sclerosis. *Archives Phys Med Rehabilitation*, 83, 1175-1178.
- Singh, R., Pentland, B., Hunter, J., & Provan, F. (2007). Parkinson's disease and driving ability. *J Neurol Neurosurg Psychiatry*, 78(4), 363-366.
- Simms, B. (1997). A 3-year follow-up of the driving status of 32 young adults with spina bifida. *International Disability Studies*, 9(4), 177-9.
- Simms, B. (1991). The Car Use of Young Drivers with Spina Bifida and Hydrocephalus. *European Journal of Pediatric Surgery, Supplement 1*, 31-34.
- Stolwyk, R., Triggs, T., Charlton, J., Iansek, R., & Bradshaw, J. (2005). Impact of internal versus external cueing on driving performance in people with Parkinson's disease. *Movement Disorders*, 20(7), 846-857.
- Stolwyk, R., Triggs, T., Charlton, J., Moss, S., Iansek, R., & Bradshaw, J. L. (2006a). Effect of a concurrent task on driving performance in people with Parkinson's disease. *Movement Disorders*, 21(12), 2096-2100.
- Stolwyk, R. J., Charlton, J. L., Triggs, T. J., Iansek, R., & Bradshaw, J. L. (2006b). Neuropsychological function and driving ability in people with Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology*, 28(6), 898 - 913.
- Swedish National Road Administration. (1998). *Swedish National Road Administration Statute Book*. Sweden.
- Uc, E. Y., Rizzo, M., Anderson, S. W., Sparks, J. D., Rodnitzky, R. L., & Dawson, J. D. (2006a). Driving with distraction in Parkinson disease. *Neurology*, 67(10), 1774-1780.
- Uc, E., Rizzo, M., Anderson, S., Sparks, J., Rodnitzky, R., & Dawson, J. (2006b). Impaired visual search in drivers with Parkinson's disease. *Annals of Neurology*, 60(4), 407-413.

- Uc, E. Y., Rizzo, M., Anderson, S. W., Sparks, J. D., Rodnitzky, R. L., & Dawson, J. D. (2007). Impaired navigation in drivers with Parkinson's disease. *Brain*, *130*(9), 2433-2440.
- Underwood G, Chapman P, Brocklehurst N, Underwood J, Crundall D. (2003). Visual attention while driving: sequences of eye fixations made by experienced and novice drivers. *Ergonomics*, *46*(6), 629-46.
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J.M. (2002). Evaluating the crash and citations rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, *34*, 237-246.
- Wood, J. M., Worringham, C., Kerr, G., Mallon, K., & Silburn, P. (2005). Quantitative assessment of driving performance in Parkinson's disease. *J Neurol Neurosurg Psychiatry*, *76*(2), 176-180.
- World Health Organisation (WHO, 1998). *Parkinson's Disease – A Unique Survey Launched. Press Release WHO/71*. Retrieved on May 14, 2003 from www.who.int/inf-pr-1998/en/pr98-71.html
- Worringham, C., Wood, J., Kerr, G., & Silburn, P. (2006). Predictors of driving assessment outcome in Parkinson's disease. *Movement Disorders*, *21*(2), 230-233.
- Zesiewicz, T.A., Cimino, C.R., Malek, A.R., Gardner, N., Leaverton, P.L., Dunne, P.B. & Hauser, R.A. (2002). Driving safety in Parkinson's disease. *Neurology*, *59*(11), 1787-1788.
- Zimmerman, P., Sprengelmeyer, R. & Fimm B. (1992). Cognitive slowing in decision tasks in early and advanced Parkinson's disease. *Brain Cognition*, *18*, 60-69.

3.9 PSYCHIATRIC ILLNESSES

Psychiatric illnesses or disorders refer to the existence of clinically recognisable symptoms or behaviours which are characterised by abnormalities in cognition, emotion or mood and often associated with distress and interference with personal functions (World Health Organization, 2001a).

Definition of psychiatric illnesses

The term “psychiatric illness” encompasses numerous cognitive, emotional and behavioural disorders such as schizophrenia, depression, anxiety disorders, personality disorders, substance abuse disorders and dementia. The disorders differ widely in aetiology and symptoms and each condition is described separately below. For the purpose of the current review, substance abuse disorders and dementia are addressed elsewhere (see sections 3.1 and 3.4 respectively).

In the past decade, researchers have begun to recognise that individuals with specific disorders such as Attention-Deficit Hyperactivity Disorder (ADHD) which affect many areas of learning and social development in childhood may also be at a high risk for motor vehicle crashes. Therefore the relationship between ADHD and road safety outcomes will also be addressed at the end of this section.

Prevalence of psychiatric illnesses

Psychiatric illnesses are relatively common, with recent studies estimating that approximately 25% of the general population will develop at least one psychiatric illness at some stage in their lifetime (WHO, 2001a). Furthermore, the WHO recently ranked mental illness first in terms of causing disability in the United States, Canada, and Western Europe when compared with all other diseases such as cancer and heart disease. Approximately 20% of Australians experience some form of mental illness every year, and about 3% are seriously affected long term (Australian Bureau of Statistics, 1998; Australian Bureau of Statistics, 2008).

Prevalence figures for specific psychiatric illnesses are presented below.

General functional impairments associated with psychiatric illnesses relevant to driving

As outlined previously, driving is a complicated psychomotor performance which depends on fine coordination between the sensory and motor system, and is influenced by a number of factors such as arousal, perception, learning, memory, attention, concentration, emotion, reflex speed, time estimation, auditory and visual functions and decision making (Cremona, 1986). According to Metzner, Dentino, Godard, Hay, Hay and Linnoila (1993) specific areas of impairment that are associated with psychiatric illnesses that may affect driving include:

- impaired information-processing ability, which includes attention, concentration, and memory components;
- reduced sustained attention (i.e., vigilance);
- impaired visual-spatial functioning, including motor response latency;

- poor impulse control, including and increased degree of risk taking;
- poor judgment, including the ability to predict/anticipate;
- reduced problem solving or a reduced ability to respond to simultaneous stimuli in a changing environment (e.g., in potentially dangerous situations).

However, it should be noted that the assessment of drivers with psychiatric illnesses regarding fitness to drive is quite complex and presents a challenging problem for the examining physician. For example, a number of psychiatric illnesses may fluctuate in their degree of functional impairment and transience and therefore their precise effect on driving ability may be unclear (Niveau & Kelley-Puskas, 2001).

3.9.1 SCHIZOPHRENIA

Definition of schizophrenia

According to the DSM-IV (APA, 2000), schizophrenia is characterized by profound disruption in cognition and emotion, affecting the most fundamental human attributes: language, thought, perception, affect, and sense of self. The array of symptoms, while wide ranging, frequently includes psychotic manifestations, such as hearing internal voices or experiencing other sensations not connected to an obvious source (hallucinations) and assigning unusual significance or meaning to normal events or holding fixed false personal beliefs (delusions). No single symptom is definitive for diagnosis; rather, the diagnosis encompasses a pattern of signs and symptoms, in conjunction with impaired occupational or social functioning.

Schizophrenia is a chronic and debilitating illness which is characterised by abnormal perceptions (hallucinations), alterations in the way individuals experience the world (delusions), and profound distortions in thinking (APA, 1994; Silverstone, 1988).

The symptoms of schizophrenia are generally divided into three broad categories: positive, disorganised and negative symptoms (NAMI, 2003):

- Positive or “psychotic” symptoms tend to reflect overt thoughts or behaviours that should not normally be present such as hallucinations and/or delusions. For example, hallucinations are disturbances of perception where individuals hear or see things that are not there, or delusions where individuals have false, fixed beliefs such as the delusion that other people control their thoughts.
- Disorganised symptoms generally involve marked disturbances in logical thought processes such that they are loose, disorganised, illogical and/or bizarre. These disturbances in thought processes frequently produce observable patterns of behaviour that are also disorganised and bizarre.
- Negative symptoms tend to reflect the absence of thoughts and behaviours that would be otherwise expected. For example, individuals with schizophrenia are often limited in their ability to think abstractly (“concrete thinking”), have a general reduction in the ability to express emotion (“blunted affect”), are unable to experience pleasure or to react appropriately to pleasant situations (“anhedonia”) or an inability to initiate activities or to become motivated.

Many individuals with schizophrenia have recurring acute “psychotic” attacks (i.e., severe disturbances of thought content and process that comprise the positive and disorganised symptoms) throughout their life, which are typically separated by intervening periods in which individuals usually present demonstrate residual or negative symptoms. While the psychotic phase of this illness is often responsible for much of the acute distress associated with disorder, it is actually the negative symptoms of schizophrenia that appear to be responsible for most of the chronic and long-term impairments associated with the disorder (NAMI, 2003; NIMH, 1999; WHO, 2001a).

Medications and other treatments for schizophrenia, when used regularly and as prescribed, can help reduce and control some of the distressing psychotic symptoms of the illness. However, for many individuals, the illness can follow a chronic or recurrent course with residual symptoms and serious limitations in daily activities (WHO, 2001a).

Prevalence of schizophrenia

The WHO estimates that in 2004 there were approximately 26.3 million people suffering from Schizophrenia (WHO, 2004). One in a hundred Australians experience schizophrenia with the age at onset being commonly in the late teens or early thirties (SANE, 2009). Current estimates for Australia suggest there are approximately 2000 people diagnosed each year (Schizophrenia Research Institute, 2009). In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at 1.5 million or around 1% of the total population. Similarly, the prevalence of this disease in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 2 million or around 1% of the total population.

Although schizophrenia affects men and women with equal frequency, the disorder often appears earlier in men, usually in their late teens or early twenties, than in women, who are usually affected in their twenties to early thirties (NIMH, 1999). Women also tend to have a better course and treatment outcome (WHO, 2001a).

Functional impairments associated with schizophrenia relevant to driving

Research has shown that individuals with schizophrenia have widespread, multifaceted impairments in many domains of cognitive function (Velligan, Mahurin, Diamond, Hazelten, Kert, Miller, 1997). For example, individuals with schizophrenia typically demonstrate:

- a reduced ability to selectively attend to relevant information while ignoring unimportant information;
- a reduced ability to sustain concentration or attention;
- reduced cognitive and perceptual processing speeds, including reaction time;
- a reduced ability to perform in more complex conditions (in presence of distraction) than in simpler control conditions.

These functional impairments have obvious consequences for driving ability. However, it should be noted that the functional impairments associated with schizophrenia are particularly difficult to determine because the degree of impairment fluctuates between the acute and residual phase of the illness (Iancu, Spivak, Weiner & Weizman, 1996).

3.9.2 DEPRESSION

Definition of depression

According to the DSM-IV (APA, 2000), major depressive disorder is defined a depressed mood and/or loss of interest or pleasure in life activities for at least 2 weeks and at least five of the following symptoms that cause clinically significant impairment in social, work, or other important areas of functioning almost every day:

- Depressed mood most of the day;
- Diminished interest or pleasure in all or most activities;
- Significant unintentional weight loss or gain;
- Insomnia or sleeping too much;
- Agitation or psychomotor retardation noticed by others;
- Fatigue or loss of energy;
- Feelings of worthlessness or excessive guilt;
- Diminished ability to think or concentrate, or indecisiveness;
- Recurrent thoughts of death (APA, 2000, p. 356).

Unlike transient sadness or “the blues”, clinical depression causes significant distress and interferes with an individual’s ability to perform routine daily functions (Webb, Dietrich, Wood, Katon & Schwenk, 2000).

Major or unipolar depression is characterised by a severe, persistent depressed mood and loss of interest or pleasure in normal activities, accompanied by decreased energy, changes in sleep and appetite, and feelings of guilt or hopelessness. These symptoms must be present for at least two weeks, cause significant distress, and be severe enough to interfere with functioning. If the depression is very severe, it may be accompanied by psychotic symptoms or by suicidal thoughts or behaviours.

On the other hand, manic or bipolar depressive disorder is a mood disorder characterised by mood swings from mania (exaggerated feeling of well-being, stimulation, and grandiosity in which a person can lose touch with reality) to depression (overwhelming feelings of sadness, anxiety, and low self-worth, which can include suicidal thoughts and suicide attempts).

Prevalence of depression

According to the World Health Organisation, depression affects about 121 million people worldwide (WHO, 2010). Each year, in Australia, 160,000 young people (16-24

years) and one million adults, experience depression (Australian Bureau of Statistics, 2008).

In 2000, the prevalence of depressive disorders in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at 10.9 million or around 3% of the total population. Similarly, the prevalence of this disease in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 12 million or around 3% of the total population (Mathers et al., 2002)..

Due to rapid global transformation, poverty, and a generalised ageing of the world's population, the number of people with depression is set to rise significantly over the next two decades (Murray & Lopez, 1996; WHO, 2001a).

Certain subgroups have a higher incidence of depression – women are more often affected than men, as are the elderly compared with younger individuals. Among people with a general medical illness, especially illnesses that involve chronic pain, the prevalence of depression may be as high as 20-30% (WHO, 2001a).

The co-occurrence of depression and generalised anxiety disorder is the most common combination of psychiatric illnesses. In addition depression often co-exists with substance abuse disorders (APA, 1994).

Functional impairments associated with depression relevant to driving

As noted previously, clinical depression can be quite debilitating (Noyes, 1986; Silverstone, 1988; WHO, 2001a). Specifically, research has shown that individuals who have been diagnosed with depression demonstrate:

- disturbances in attention;
- impaired information processing and judgement;
- psychomotor retardation;
- diminished concentration and memory ability,
- decreased reaction time;
- sleep disturbances and fatigue;
- suicidal ideation.

All of these impairments may theoretically affect driving ability. According to Silverstone (1988), drivers with severe depression are therefore at-risk of being involved in a motor vehicle crash on two counts: their slowed responsiveness and poor concentration put them at risk from the vehicle-handling point of view, while their suicidal ideation may cause them to crash their car in an attempt to end their lives (for more information regarding motor vehicle crashes due to suicide see Routley, Staines, Brennan, Haworth & Ozanne-Smith, 2003).

3.9.3 ANXIETY DISORDERS

Definition of anxiety disorders

Anxiety disorders are characterised by symptoms of overwhelming anxiety, fear and avoidance behaviour. Unlike the relatively mild, brief anxiety caused by a stressful situation, such as a conference presentation, anxiety disorders are chronic, relentless and can grow progressively worse if not treated (NIMH, 2003).

Anxiety disorders include panic disorder, obsessive-compulsive disorder, post-traumatic stress disorder, generalized anxiety disorder, and phobias (social phobia, agoraphobia, and specific phobia). While each anxiety disorder has its own distinct features, they are all bound together by the common theme of excessive, irrational fear and dread (NIMH, 2003).

Prevalence of anxiety disorders

Anxiety disorders as a group are the most common or frequently occurring psychiatric illness (NIMH, 2003). Approximately 19.1 million American adults aged 18 to 54, or about 13.3% of people in this age group in a given year, have an anxiety disorder (Narrow, Rae & Regier, 1998). Approximately 10 % of Australians will be affected by anxiety disorders at some point in their life. (Andrews et al. 1999).

Functional impairments associated with anxiety disorders relevant to driving

Anxiety, which may be understood as the pathological counterpart of normal fear, is manifest by disturbances of mood, as well as of thinking, behaviour, and physiological activity. Eysenck (1997) has shown that due to features of anxiety such as heightened alertness for threat and the tendency to worry, individuals with anxiety disorders typically:

- have decreased working memory;
- are more easily distracted;
- have less attentional capacity available to them.

In addition, there is also the potential for some individuals with an anxiety disorder to experience a “panic attack” while driving which has obvious consequences for driving ability.

3.9.4 PERSONALITY DISORDERS

Definition of personality disorders

The DSM-IV defines a personality disorder as deeply ingrained and enduring patterns of pervasive and inflexible personality traits that deviate from cultural norms and that cause distress or functional impairment (APA, 1994).

Currently, there are 10 distinct personality disorders identified in the DSM-IV which are usually divided into three clusters:

- Cluster A personality disorders, which are mainly characterised by odd, eccentric behaviour, include paranoid personality disorders, schizoid personality disorders and schizotypal personality disorders;
- Cluster B personality disorders, which are mainly characterised by dramatic, explosive, emotional or erratic behaviour, include antisocial personality disorders, borderline personality disorders, histrionic personality disorders and narcissistic personality disorders;
- Cluster C personality disorders, which are mainly characterised by anxious, fearful, dependent and introverted behaviour, include avoidant personality disorders, dependent personality disorders, and obsessive-compulsive personality disorders.

Prevalence of personality disorders

The prevalence of personality disorders has been estimated from 1 to 10% of the general population, depending on the criterion being used. Some diagnoses are made more commonly in men (such as anti-social personality disorder), while others are more common in women (such as histrionic and borderline personality disorders) (Martin & Sugarman, 1997) The population prevalence of Borderline Personality Disorder in Australia is approximately 1% (Jackson & Burgess, 2002).

Functional impairments associated with personality disorders relevant to driving

Individuals with severe personality disorders are at high risk of alcohol or drug abuse, and violent or self-destructive behaviours. Furthermore, specific personality traits have been shown by several researchers to be associated with a propensity for (Cremona, 1986; Noyes, 1985; Petch, 1996; Tsuang et al., 1985):

- aggression;
- egocentricity;
- impulsiveness;
- resentment of authority;
- intolerance of frustration;
- irresponsibility.

These functional impairments have obvious consequences for driving ability.

3.9.5 *PYSCHIATRIC ILLNESSES - GENERAL*

Pre-May 2003: Relationship between psychiatric illnesses and road safety outcomes

Individuals with psychiatric illness have often been viewed as dangerous drivers, sometimes without serious epidemiological basis (Silverstone, 1988). For example, it seems reasonable to assume that psychotic drivers will be distracted by hallucinations

and delusions, particularly if they involve other drivers, that depressed drivers will have poor concentration and may not be concerned if they are involved in a crash, and that anxious drivers would be incapacitated by indecision (Cremona, 1986). Despite the prevalence of psychiatric illness in the general population, the relationship between history of psychiatric illness and motor vehicle crashes has received limited attention (Elwood, 1986).

Dobbs (2001) notes that the majority of the available research was conducted more than 30 years ago (for reviews see Tsuang et al., 1985; Noyes, 1985), and therefore findings from these early reviews may not be relevant to current risk estimates because the treatment and management of psychiatric illnesses has changed substantially in the past three decades, particularly through improved medication. Therefore, the literature presented in this review will only focus on studies conducted post 1980. Table 28 shows a summary of the findings of studies that have investigated psychiatric illness, psychotropic medication and risk as determined by crash involvement, citations and driving performance.

In relation to driving and crash risk, a number of studies have considered drivers with psychiatric illness as a homogenous group, while others have, more appropriately, studied the independent effects of these disorders on road safety outcomes. These studies are reviewed below.

Crashes

In 2002, Vernon et al. conducted a retrospective case control study of crash rates of drivers with medical conditions during 1992 – 1996. Crash rates per 10,000 licence days (Utah DOT official records) for 6808 drivers with psychiatric illnesses and other emotional conditions (history of psychiatric or emotional conditions, psychotic illness, suicidal tendencies, perception distortions, psychomotor retardation, schizophrenia, major depressive disorders, bipolar disorders and/or organic syndromes) were compared with a control group of drivers matched by age, sex and place of residence (see section 3.1 for a more detailed description of the study methodology). Drivers with psychiatric illnesses were also classified according to licence status (restricted/unrestricted) with the majority of cases (n = 6481) having no restrictions. Overall, the authors reported that both unrestricted and restricted drivers with a psychiatric illness had significantly higher rates of at-fault crashes (unrestricted: RR: 1.85, 95%CI 1.69 - 2.01; restricted: RR: 2.89, 95%CI 1.67 - 5.07) and all crashes (unrestricted: RR: 1.57, 95%CI 1.46 - 1.67; restricted: RR: 1.87, 95%CI 1.11 - 3.17). Vernon et al. concluded that restricted drivers with psychiatric illnesses or emotional conditions had a relative risk of having an at-fault crash almost two and a half times greater than controls. One of the main limitations of this study was that the authors did not provide information on the independent effects of the various psychiatric illnesses. Furthermore, the authors did not control for driver exposure, which assumes that drivers in the psychiatric illness group and matched controls drive similar distances.

In a study focussing specifically on schizophrenia, Edlund, Conrad and Morris (1989) compared the incidence of motor vehicle crashes for 70 out-patients with schizophrenia with 122 age-matched controls. The authors reported that all out-patients met the DSM-III-R criteria for schizophrenia of at least one year's duration. There was no significant difference between the two groups for self-reported motor vehicle crashes over the previous 12 months (10% for the psychiatric group and 9% for the control group, $p > 0.05$). However, when crash rates were adjusted for driver exposure, the authors

reported that there were considerable differences between the two groups. For example, only 68% of participants with schizophrenia reported that they drove at all, compared with 99% of controls ($p = 0.00001$). At each level of miles driven per year (i.e., 0-100, 100-5000, 5000-10000, and over 10,000 miles) the proportion of control drivers in the category was approximately 2.2-2.5 times that of participants with schizophrenia. Of those driving, 40% of participants with schizophrenia reported that they drove more than 100 miles per year, whereas 98% of controls reported that they drove more than 100 miles in the past year ($p = 0.00001$). The authors concluded that as the self-reported motor vehicle crash rates were equivalent for both groups, individuals with schizophrenia who drove have double the risk of motor vehicle crashes per distance driven compared to age-matched controls. As outlined in Chapter 2, one of the major methodological limitations using self-reported outcome measures is the potential for selection bias (see Chapter 2). In this study, 20 additional out-patients with schizophrenia were approached for inclusion in the study but refused to participate. Chart reviews conducted for 15 of the 20 out-patients refusing to participate revealed that 20% had been involved in a major motor vehicle crash in the last year. It should also be noted that the authors did not indicate whether the control participants had been screened for psychiatric or medical comorbidities.

Armstrong and Whitlock (1980) compared self-report crash rates of 100 participants with a psychiatric illness with 100 participants with a physical illness matched for age, sex and social background who had been admitted to a private hospital. Psychiatric diagnoses included schizophrenia ($n = 12$), manic depression ($n = 34$), neuroses ($n = 28$), personality disorder ($n = 8$), alcoholism ($n = 15$), and drug abuse ($n = 2$). Armstrong and Whitlock reported that during the six months before admission there were no significant differences between the two groups with respect to crash and traffic code infringements. However driving exposure for the psychiatrically ill drivers was substantially less than the physically ill group, suggesting that the risk of crashes in the psychiatric group is substantially higher than the physically ill participants when adjusted for driving exposure. The authors noted that participants with psychiatric illnesses were more likely to report driving problems since becoming ill (60%) compared to the participants with a physical illness (23%, $p < 0.001$). The authors concluded that no specific psychiatric diagnosis was associated with an increased risk of having a motor vehicle crash. Limitations of this study include the use of self-report crash data, small sample size per diagnostic group and that a description of illnesses in physically ill group was not provided. In addition, the authors note that confining the study to outpatients in private hospitals may have excluded those in lower socio-economic groups whose driving records could be very different from those who participated in the study.

Citations

Vernon et al. (2002) investigated citation rates of drivers with psychiatric conditions during 1992 – 1996 and found some evidence of a higher citation rate, but only amongst those with unrestricted drivers (lower level of impairment) with a psychiatric illness (RR: 1.23, 95%CI 1.17-1.30, $p < 0.05$). In contrast, citation rates of those with restricted licences (higher level of impairment) were no different from controls (RR: 0.84, 95%CI 0.53-1.33, $p > 0.05$).

In view of the limited amount of evidence available, it is difficult to make any definitive statement about psychiatric illness and its impact on citation rates. At best, the evidence

in this regard suggests a modestly elevated citation rate but only for those who have a low level of impairment. It is possible that those with higher levels of impairment self-regulate their driving in such a way as to reduce their exposure or drive slower or more cautiously. Neither of the two studies reported here investigated the link between crashes and citations, so what remains unclear is how the findings on citations might relate to crash risk.

Driving performance

No studies reporting the relationship between psychiatric illness (considered as a group) and driving performance were found.

Treatment of psychiatric illnesses and road safety outcomes

Prescribed psychotropic medications are often the first-line of treatment for most individuals who have been diagnosed with a psychiatric illness. However, some psychotropic medications have been shown to impair perception, vigilance and psychomotor skills (Cremona, 1986), and are therefore thought to have a potentially detrimental effect on driving (Elwood, 1998). A review of the studies examining the effects on specific categories of medications used for psychiatric illnesses on driving is provided below. A number of these studies have explored the effects of treatment on driving performance using driving simulators or driving-related psychomotor tasks. Few have examined treatment effects and crash risk directly and clearly more research on this topic is needed.

In 1980, Armstrong and Whitlock investigated the effects of prescription medications for psychiatric and physical illnesses on crash rates (for more details regarding the study design see the previous section). Not surprisingly, participants with a psychiatric illness were consuming greater quantities of psychotropic drugs than the group with physical illnesses. However, the authors reported that medication did not appear to influence the outcome in statistical terms: neither the physically ill or psychiatrically ill participants who reported crashes were taking more medications than participants who had not crashed.

Antipsychotics

Antipsychotic medications, also known as neuroleptics, are the mainstay of the pharmacological treatment of serious psychiatric illnesses such as schizophrenia (Judd, 1985). Antipsychotics have the capacity to diminish the positive symptoms of schizophrenia such as delusions, hallucinations and disorganised thinking, and may have some impact on the negative symptoms such as lack of motivation and blunted affect (NAMI, 2003).

Antipsychotic medications, like virtually all medications, have unwanted side effects along with their beneficial effects. During the early phases of drug treatment, individuals may be troubled by side effects such as drowsiness, restlessness, muscle spasms, tremor, dry mouth, or blurring of vision, however there is evidence that over time individuals will develop tolerance to the sedation, drowsiness and decreased alertness which may be evident in the early phase of treatment (Judd, 1985). In addition, most of these effects can be corrected by lowering the dosage or can be controlled by other medications.

However, it is the long-term side effects of antipsychotic medications that may pose considerably more serious problems. For example, Tardive dyskinesia is associated with prolonged use of antipsychotic medication, and is a complex syndrome of involuntary hyperkinetic movements, most frequently affecting the mouth, lips, tongue, jaw, and sometimes trunk or other parts of the body such as arms or legs.

Crashes

No studies reporting the relationship between antipsychotic medications for psychiatric illness and crashes were found.

Citations

No studies reporting the relationship between antipsychotic medications for psychiatric illness and driving citations or traffic violations were found.

Driving performance

Antipsychotic medications also have the potential to impair driving ability (Metzner et al., 1993). For example, motor dysfunction due to Parkinsonism, akathisia (motor restlessness), dystonia (sustained muscle contractions), and tardive dyskinesia (bizarre motor behaviours) can impair coordination and response time. Sedation, which is a common side effect of antipsychotics, can slow response times and reduce attentiveness. Reduction of visual accommodation and papillary reactivity, which are usually anticholinergic side effects, can negatively affect driving performance

Despite the widespread use of antipsychotic medications and the potential for side effects such as sedation and impaired psychomotor performance, there is little evidence in the literature to suggest that they are significantly implicated in motor vehicle crashes (Judd, 1985).

In a review of the literature prior to 1980, Judd reports that when participants without schizophrenia were administered an acute dose of antipsychotic medication, they demonstrated increased sedation, impaired performance on visual motor coordination tasks and specific attentional behaviours. Judd concluded that the acute administration of antipsychotic medication deleteriously effects driving behaviour in control participants. Judd also noted that antipsychotics are rarely used on an acute basis and tolerance to sedation and decreased alertness generally develops over long-term treatment. In contrast, Judd reports that there is a general agreement that individuals with schizophrenia who require maintenance on antipsychotic drugs manifest *improved* psychomotor performance while on these medications, and therefore it is possible that antipsychotic medication may have a beneficial effect on driving in individuals with schizophrenia. Judd suggests that future studies should investigate the effect of long-term maintenance of antipsychotic drugs on driving performance of individuals with schizophrenia.

Antidepressants

Anti-depressants are the cornerstone of treatment for major depression (Dobbs, 2001). Besides the beneficial effects of anti-depressants, these drugs can also produce side effects such as sedation, lethargy, impaired psychomotor function and sleep

disturbances (Ramaekers, 2003). Therefore in situations requiring individuals to engage in potentially dangerous activities, i.e., operating a vehicle, these side effects could increase the risk of injury or death through performance related crashes (Ramaekers, 2003).

Crashes

In 1992, Ray, Fought and Decker conducted a study to determine whether commonly used psychoactive drugs (antidepressants and benzodiazepines) increase the risk of involvement in motor vehicle crashes for drivers over the age of 65 years (see the next section for the results regarding the effect of benzodiazepines). Specifically, the authors conducted a retrospective cohort study, obtaining data from computerised files from the Tennessee Medicaid program, drivers licence files, and police reports of injurious crashes. Cohort members were Medicaid enrollees, aged between 65-84 years, who had a valid driver's licence. There were 16,262 individuals in the study cohort study, which had 38,701 person-years of follow-up. These participants were involved in 495 injurious crashes; a rate of 12.8 per 1,000 person-years which the authors report is slightly higher than the rate for all drivers of comparable age in Tennessee (10.6 per 1,000 person-years). Current users of tricyclic antidepressants were associated with an increased relative risk of injurious crash involvement (RR: 2.2, 95%CI 1.3 - 3.5). Concurrent use of two different tricyclic antidepressants was also associated with a significant increase in risk of involvement in an injurious crash. For tricyclic antidepressants, the risk increased from 2.2 for current use of a single drug to 9.8 (95%CI 2.4 - 39.5) for use of more than one ($p < 0.05$). The risk of crash involvement did not vary significantly with duration of tricyclic antidepressant use. Finally, the authors noted that the relative risk increased with dose and was substantial for high doses: for doses greater than 125mg of amitripyline the relative risk was 5.5 (95%CI 2.6 – 11.6). Information regarding drug use in this study was ascertained from computerised records of prescriptions filled at the pharmacy. While this method of obtaining data avoids the potential for participants involved in a crash to underreport their medication use, it does not take into account non-compliance or use of drugs from other sources. Other potential confounding factors in this study that were not controlled for include health status, alcohol use, and driving exposure. Notwithstanding these limitations, others have reported a similar association where participants taking tricyclic antidepressants had a 2.3 increase in crash risk compared to matched controls (Leveille, Buchner, Koepsell, McCloskey, Wolf & Wagner, 1994).

Citations

No studies reporting the relationship between antidepressant medications for psychiatric illness and driving citations or traffic violations were found.

Driving Performance

Antidepressants are generally divided into older tricyclic antidepressants and newer selective serotonin reuptake inhibitors (SSRI, Dobbs, 2001). In 1998, O'Hanlon, Robbie, Vermeeren, van Leeuwen and Danjou compared the effects of venlafaxine (Effexor, a SSRI) to that of mianserin, a cyclic antidepressant, on driving, psychomotor and vigilance performance. Results from 37 healthy volunteers revealed that venlafaxine had no significant effect on psychomotor performance. On the other hand, mianserin profoundly affected both psychomotor and driving performance. Vigilance was significantly affected by both antidepressants.

Similar results have been reported by van Laar, van Willgenburg and Volkerts (1995). Simulated driving and psychomotor performance of 24 healthy participants was examined following the administration of nefazodone (SSRI) and imipramine (cyclic antidepressant). Using a double blind, cross-over, placebo controlled methodology, impairments were noted on the lateral position control following single doses of imipramine compared with no impairments following single doses of nefazodone. The authors also noted that minor impairments in psychomotor performance were evident with imipramine compared with no impairment with nefazodone.

In conclusion, significant impairments in psychomotor and driving performance have been noted with cyclic anti-depressants. On the other hand, fewer impairments are evident with the newer SSRIs.

Anti-anxiety

Benzodiazepines are the most commonly used medication for the treatment of anxiety and insomnia and one of the most frequently used classes of medication taken by elderly individuals (Ray, Purushottam, & Shorr, 1993).

Benzodiazepines⁴ can be divided into those with a short half-life (e.g., lorazepam/Ativan, alprazolam/Xanax, triazolam/Halcion⁵, oxazepam/Serepax, temazepam/Normison) and those with a long half-life (e.g., clonazepam/Klonopin, mainly used in Australia for the treatment of certain types of Epilepsy, clordiazepoxide/Librium, diazepam/Valium, nitrazepam/Mogadon, halazepam/Paxipam², prazepam/Centrax², clorazepate/Tranxene², flurazepam/Dalmane²). In general, the duration of action for those with a short half-life is 2 to 8 hours and 8 to 24 hours for those with a long half-life (Dobbs, 2001). Side effects that may adversely affect driving include sedation, drowsiness, prolonged psychomotor reaction times, incoordination, memory loss, vertigo, dizziness, and double vision (see Ray et al., 1993 for a complete review).

Benzodiazepines are very frequently abused and there are many published studies regarding the effects of high doses of benzodiazepines on driving. This section is not concerned with benzodiazepine abuse but with the effects of these drugs on driving when they are taken as prescribed for proper therapeutic purposes.

Crashes

In 2000, McGwin, Sims, Pulley and Roseman conducted a population-based control study, examining chronic medical conditions and motor vehicle crashes among older drivers. Specifically, the authors were interested in estimating the association between medical conditions and at-fault involvement in crashes among older drivers after adjusting for demographic factors and driving exposure. A total of 447 drivers aged 65 years and older were selected from Alabama Department of Public Safety driving records who had at least one automobile crash in 1996. Police records corresponding to the crashes incurred by the participants were judged according to criteria to determine

⁴ Some benzodiazepines are marketed under several trade names. The names quoted are the most commonly recognised for each drug

⁵ Not available in Australia

whether the driver was at least partially at-fault in the crash. Of the participating cases, 249 were found to be at least partially at-fault. Control participants comprised 454 drivers also selected from Alabama Department of Public Safety driving records who were not involved in crashes. Information on demographic factors, chronic medical conditions, medications, driving habits, visual function, and cognitive status was collected and participants from both groups did not differ in age or gender. Analyses were adjusted for mileage and previous crash involvement (for the results regarding heart disease, stroke, diabetes and arthritis, see sections 3.2, 3.3, 3.5, and 3.7 respectively). The authors reported that benzodiazepine use was associated with an increased risk for at-fault crash involvement (OR: 5.2, 95%CI 0.9 – 30.0). Unfortunately, the authors did not have any information regarding the specific types of benzodiazepines, and therefore could not compare the differences between short and long half-life benzodiazepines. Methodological limitations of this study include the well documented problems associated with data obtained via self-report.

Barbone, McMahon, Davey, Morris, Reid, McDevitt and MacDonald (1998) undertook a case-crossover study using a record-linkage database in an attempt to investigate the association between road traffic accidents and the use of benzodiazepines. 19,386 drivers aged over 18 who were involved in a road traffic accident during the study period between August 1, 1992 and June 30, 1995 were linked to their pharmaceutical prescriptions via a unique patient identifier (their community health number). Data on driver's age, sex, date of accident, day of week, hour of day, lighting conditions, severity of injuries, number of vehicles involved, driver at fault, positive or negative alcohol breath test, date of prescription, drug code, drug dose, dosing instructions and number of tablets dispensed was collated. For each case, logistic regression models were studied to estimate the odds of having an accident whilst unexposed to the study drugs against the odds of crashing while exposed. Odds ratios were calculated as a measure of association between drug use and a road traffic accident. Of all the drivers, 1,731 were users of the following study drugs: 793 were users of tricyclic antidepressants; 334 users of selective serotonin-reuptake inhibitors (SSRIs); 916 users of benzodiazepines; and 138 users of other psychoactive drugs. The sample size of users of 'other psychoactive drugs' was too small for any meaningful analyses. The odds ratios for each of the other drug types are: 1.62 for benzodiazepine use; 0.93 for tricyclic antidepressants and 0.85 for SSRIs. Although other drug classes were examined, only results for benzodiazepine exposure are reported in the paper. The authors found that use was highest among drivers younger than 30, and that risk decreased with increasing age; risk was not higher for people aged 65 and over (test for trend $p = 0.01$). The odds ratio for benzodiazepine users decreased as the number of vehicles involved in the crash increased, although this was non-significant. Use was also associated with risk in accidents where the drivers was judged to be at fault ($n = 162$; OR: 1.88, 95%CI 1.36 - 2.60). A strong association was also found for drivers who returned a positive breath test and used benzodiazepines ($n = 7$; OR: 8.15, 95%CI 2.06 - 32.34). Benzodiazepines were also classified as hypnotics or anxiolytics. Users of hypnotic benzodiazepines were found not to be at increased risk, arguably because their 95% were prescribed as a single nightly dose. Anxiolytic benzodiazepine users were found to be at a significantly increased risk of crash (OR: 2.18; 95%CI 1.52 - 3.13). This study is only appropriate for transient exposure data, and is limited to residents of city of Tayside, UK. Regardless, this study provides strong evidence that use of anxiolytic benzodiazepines represents an increase in crash risk for individuals.

Hemmelgarn, Suissa, Huang, Boivin and Pivan (1997) compared the injurious rate of 5,579 older drivers (aged between 67 and 84) using benzodiazepines to a group of 13,256 controls during a period of 1990 to 1993. Exclusion criteria included residence in a long-term care facility, hospitalisation in the past 60 days, or hospitalisation for greater than 30 days in the past year. Data on benzodiazepine use were taken from a provincial prescription drug database. Benzodiazepines were classified as having a long elimination half-life (i.e., greater than 24 hours clonazepam, diazepam, clorazepate, chlordiazepoxide, flurazepam, nitrazepam) or a shorter elimination half-life (less than 24 hours, alprazolam, bromazepam, lorazepam, oxazepam, temazepam, triazolam). Duration of exposure was classified as 1-7 days, 8 to 30 days, 31-60 days or 61-365 days. The data were adjusted for age, sex, residence, chronic disease score (derived from drug use), benzodiazepine dose, exposure to other benzodiazepine or central nervous system drug use, and previous motor vehicle crash involvement. The authors reported that the prevalence of older drivers who had motor vehicle crashes and who were taking long-half life benzodiazepines was 6.9% and 5.2% for control participants, the prevalence of shorter half-life benzodiazepines was 14.4% and 14.7% respectively. The use of long half-life drugs was associated with an increased risk for motor vehicle crashes (adjusted RR: 1.28, 95%CI 1.12 - 1.45), however the use of shorter half-life drugs was not (RR: 0.96, 95%CI 0.88 - 1.05). For those individuals taking benzodiazepines with a longer half-life, the risk was highest in the first week (RR: 1.45, 95%CI 1.04 - 2.03) and remained higher than controls for continued use over a period of 61-365 days (RR: 1.26, 95%CI 1.09 - 1.45). The authors concluded that participants taking longer acting benzodiazepines are at a higher risk of crashing whereas there was no evidence of increased crash risk for those on the short acting benzodiazepines. It should be noted that observational data such as this is susceptible to selection bias.

In contrast to the results of Hemmelgarn et al. (1997) and Barbone et al. (1998), Leveille et al. (1994) failed to find a relationship between benzodiazepines use and motor vehicle crashes resulting in injuries. In this investigation, injurious crash rates of 234 elderly drivers were compared to those of 447 controls matched for sex, age, and country of residence. Difference in the results between the two studies may, in part, be due to the fact that the most widely used benzodiazepine used in the Leveille study was triazolam, a short acting benzodiazepine.

Based on prescription and driving records of 16,262 seniors (i.e. aged 65 years and over), Ray et al. (1992) conducted a retrospective cohort study and reported that current users of benzodiazepines had injurious crash rates 1.5 times higher compared to individuals with no psychoactive drug use (RR: 1.5, 95%CI 1.2 - 1.9). Concurrent use of two different benzodiazepines was also associated with a pronounced increase in risk of involvement in an injurious crash, with the relative risk increased from 1.5 for current use of a single benzodiazepine to 4.8 (95%CI 1.6 - 14.5) for use of more than one ($p = 0.05$). The authors reported that the risk of crash involvement did not vary significantly with duration of benzodiazepine. In addition, Ray et al. reported that there was a dose-dependant relationship: crash rates of benzodiazepine users at the lowest therapeutic level were approximately equal to that of controls. In contrast, drivers with benzodiazepine levels at the highest therapeutic dose (more than 20mg of diazepam per day) had crash rates 2.4 times higher than controls (95%CI 1.3 - 4.4). As outlined in the previous section, limitations of this study are that the authors did not control for health status, alcohol use, medication non-compliance, use of medication from sources other than the pharmacy, or driving exposure.

Citations

No studies reporting the relationship between anti-anxiety medications for psychiatric illness and driving citations or traffic violations were found.

Driving performance

Törnros, Vikander, Ahlner and Jönsson (2001) conducted a study to determine if benzodiazepine users exhibit impaired performance in simulated car driving and in laboratory tests. The authors also studied the effects of a small dose of alcohol on performance. Participants included 20 outpatients who had used prescribed benzodiazepines for treatment of anxiety or insomnia for years and 20 control participants who were individually aged and sex matched. Participants were excluded if they had a history of drug or abuse dependence or if they drove less than 1000 km annually. Driving performance was examined using a driving simulator, where the outcome measures studied were brake reaction time, lateral position variation, and speed variation. The two groups were also compared on three laboratory tests: simple reaction time, choice reaction time, and short-term memory. The authors reported that there was no overall difference between the two comparison groups for brake reaction time ($F(1, 18) = 2.37, p > 0.05$) or lateral position variation ($F(1, 18) > 1, p > 0.05$). On the other hand, the speed variation for participants using benzodiazepines was greater than among control participants ($F(1, 18) = 17.02, p > 0.001$). For example, the speed variation in the first session was 4.6 km/h for the benzodiazepine users and 3.4 km/h for control participants. Participants using benzodiazepines also demonstrated impaired performance on the simple reaction time and short-term memory tests (simple reaction time: $F(1, 18) = 5.07, p < 0.05$) and short-term memory: ($F(1, 18) = 6.29, p < 0.05$). However there was no significant difference in performance between the groups for the choice reaction time task ($F(1, 18) = 2.89, p > 0.05$). The authors concluded that the results of study do not suggest that individuals using prescribed benzodiazepines would constitute an increased risk for motor vehicle crashes. One limitation of this study is that it is not possible to determine if the differences are actually caused by the benzodiazepine use or by the underlying illness. In addition, the generalisability of these findings to “real world driving” and crash risk is not clear. The only conclusion that can be made from this study is that the differences were not due to age or sex because they were the only two factors controlled for.

O’Hanlon, Vemeeren, Uiterwijk, van Vegal and Swijgman (1995) examined the effects of benzodiazepines (diazepam and lorazepam), benzodiazepine-like anxiolytics (alpidem and suriclone), and a 5-HT agonist (ondansetron) on a standardised road tracking test. Participants were healthy young controls (22 to 43 years) and anxiety patients (24 – 64 years). In a double-blind, placebo-controlled design, participants were tested on the road tracking test 2 to 3 times after taking one of the drugs for 8 to 15 days. There were no significant differences in driving performance between the two groups in the baseline, placebo and ondansetron conditions. However, significant impairments in driving performance were noted in the benzodiazepine and benzodiazepine-like drug conditions.

In conclusion, benzodiazepines have been shown to impair vision, attention, information processing, memory, motor coordination, and combined skilled tasks (Ray et al., 1993). Most case-control studies suggest that benzodiazepine use in general is associated with increased crash risk. In addition, it appears as though longer acting benzodiazepines are of particular concern and that the risk appears highest in the first

four weeks of therapy, after which tolerance generally develops to the sedation and dysfunctional effects on coordination (Silverstone, 1988). This drug class may also be especially hazardous for elderly drivers (Ray et al., 1993).

Post-May 2003: Relationship between psychiatric illnesses and road safety outcomes

In the review period from May 2003 to mid-2009, three studies were identified which addressed road safety outcome measures associated with psychiatric disorders: one study addressed crash risk and two studies used driving performance-based outcome measures. Nine studies were identified addressing psychiatric medications or treatments, including one relating to crashes and eight studies addressing other driving performance measures.

Crashes

Dumais, Lesage, Boyer, Lalovic, Chawky et al. (2004) conducted a case-control study investigating psychiatric risk factors associated with MVCs. In this study, cases (n = 61) were young male drivers (aged 18-36) who died in a road crash who were identified through the Montreal Central Morgue. Cases were excluded if the MVC was classified as a suicide. Controls (n = 61) were living male participants matched for age, sex, employment and marital status with cases. The authors determined psychiatric diagnoses through the psychological autopsy method (Kelly & Mann, 1996). The authors noted that cases were more likely to have cluster B personality disorders (borderline and [or] antisocial) (OR: 3.54, 95%CI 1.38 - 16.01) and substance use disorders in the past 6 months (OR: 4.33, 95%CI 1.42 - 9.25) compared to controls. In addition, the authors observed an age effect, where differences in cluster B personality disorders and substance use disorders in the last 6 months were only significantly more prevalent in cases aged 26 years or over, compared with controls of the same age. Drivers under age 25 years appeared to be comparable with control subjects on all measures of psychopathology. Finally, the authors noted that the interaction between cluster B personality disorders and age over 26 years was the only significant predictor of car fatalities (adjusted OR: 16.25, 95%CI 1.67 - 158.10). The authors concluded that borderline and antisocial personality disorders in which impulsive-aggressive behaviours play a central role and substance use disorders appear to be risk factors for young male deaths in MVCs. Interestingly, this effect seems to be specific to MVC case subjects aged 26 years or over. The main limitations of this study are the reliance on proxy-based interviews for cases and absence of data on crash fault. The study addresses psychiatric illness as a risk factor for *fatal* crashes and therefore does not contribute specifically to the broader question of whether people with a psychiatric illness are at increased risk of crashes.

Citations

No studies reporting the relationship between psychiatric illness (considered as a group) and crashes were found.

Driving performance

Bulmash, Moller, Kayumov, Shen, Wang and Shapiro (2006) compared the driving performance of 18 outpatients who met the DSM-IV criteria for Major Depressive disorder (MDD) (Cases) with 29 controls on four 30-minute simulated driving trials. All

participants were aged between 18-65 years, had a valid driver's licence and had at least five years of driving experience. Participants were excluded if they had previously suffered serious head injuries, had a neurological or medical condition, or presented with a psychotic or substance use disorder. Controls were assessed for the presence of MDD based on DSM-IV. Both cases and controls were required to be free of antidepressant medication. The authors reported cases exhibited slower steering reaction times across trials ($p < 0.05$) and experienced significantly more crashes across trials ($p < 0.05$) when compared to controls. However, no significant differences were found for road position, speed or speed deviation. The authors concluded that individuals with untreated MDD demonstrate impaired simulator driving performance. The authors noted that the driving course consisted of a rural highway drive with few passing vehicles, changes in speed limits or pedestrians and may not have been as challenging as an urban drive. Also, it should be noted that due to the relatively small sample size and the use of a simulator to measure driving performance, it is not clear how these findings might generalise to actual driving risk for drivers with MDD.

Brunnauer, Laux, Geiger, Soyka and Moller (2006) considered the driving performance of 100 patients aged between 20 and 78 (mean age 46.8 ± 13.6) who met the ICD 10 and DSM-IV criteria for Major Depressive disorder (MDD) using a naturalistic and non-randomised study design. Inclusion criteria for the study were (1) antidepressant monotherapy, (2) steady-state pharmacological conditions (all patients were considered for discharge within at least 3 days) and (3) possession of a valid driver's licence. Patients were excluded if they had a history of neurologic illness, substance abuse or mental retardation. The experimental procedure, conducted at 9:00AM, consisted of computerised psychomotor tests of visual perception, selective attention, vigilance, reactivity and stress tolerance. Patients were considered to fail, in accordance with German guidelines for road and traffic safety, if the patient fell short of the threshold of one standard deviation below the mean of normative data derived from a representative sample of car drivers. 76% of the sample did not pass the threshold criterion according to German guidelines. Using less conservative criteria (allowing patients to fail in up to 40% of test parameters), 60% of patients were found to be mildly-moderately impaired with regard to fitness for driving and the psychomotor performance of 16% of cases was found to be severely impaired (unfit to drive). The authors concluded that the results suggest that most depressive patients considered ready for discharge to out-patient treatment did not reach the level of psychomotor performance of healthy controls on tasks related to driving ability. It should be noted that as a non-randomised study design, causal relationships cannot be claimed. Further to this, selection bias of participants cannot be excluded. However, the authors only tested patients who were sufficiently well enough to participate in a 150 minute experimental procedure, suggesting that the results may reflect an overestimation of MDD patient fitness to drive.

Treatment of psychiatric illnesses and road safety outcomes

Antipsychotics

Crashes

No studies reporting the relationship between antipsychotic medications for psychiatric illness and crashes were found.

Citations

No studies reporting the relationship between antipsychotic medications for psychiatric illness and driving citations or traffic violations were found.

Driving performance

Recent studies have begun to investigate possible differences between conventional and atypical (more modern) neuroleptics with respect to driving-relevant cognitive aspects. Kagerer, Winter, Moller and Soyka (2003) investigated the effects of atypical neuroleptics in comparison with a conventional dopamine antagonist (haloperidol) on several dimensions of psychomotor performance (visual perception, attention, reaction time, and sensorimotor performance) considered to be of relevance in evaluating fitness to drive. All participants ($n = 49$) were recruited from a Psychiatric Hospital in Munich, Germany. Cases ($n = 29$) were defined as participants who were being treated with atypical neuroleptics (5 received clozapine, 4 received amisulpride, 7 received riperidone, 8 received quetiapine, 4 received olanzapine and 1 received ziprasidone, all in different dosages). Although a monotherapy was favoured, 7 received an additional medication when clinically indicated. Controls ($n = 20$) were defined as participants who were being treated with a conventional dopamine antagonist (haloperidol) (dosage of 4–30 mg/day). All participants met the ICD-10 and DSM-IV criteria for schizophrenia or a schizoaffective disorder. All participants were clinically stabilized, had a steady state of neuroleptic medication and were ready for discharge. There was no significant difference in age, sex, education and Brief Psychiatric Rating Scale (BPRS) scores between the haloperidol-treated group and the group treated with atypical neuroleptics. Participants were excluded if they had a disabling physical disorder, organic brain disorder, acute substance abuse, or any serious concurrent medical condition. All participants had either a valid driver's licence or intended to obtain one within the next few months.

The authors reported that participants with schizophrenia currently being treated with haloperidol performed significantly worse on several dimensions of psychomotor performance (visual perception, reaction time, and sensorimotor performance), considered to be of relevance in evaluating fitness to drive, compared to participants with schizophrenia currently being treated with atypical neuroleptics. Relevant psychomotor skills for driving fitness were assessed by the act-and-react test system (ART 90), a standardized and computerized test unit developed by the Austrian road safety board (Grabe et al. 1998). The reliability and validity of this test battery have been confirmed in large samples of both community and clinical participants. The authors note that the tests have been found to predict driving performance under different traffic situations (Risser et al., 1993; Bukasa et al., 1990 and Bukasa et al., 2003). On a Peripheral Vision Test with Tracking Task (PVT), a task assessing peripheral visual perception, divided attention, sensorimotor performance and reaction time, participants treated with atypical neuroleptics demonstrated significantly better performance on the tracking task ($M = 4.1$, $SD = 2.0$) compared to participants who were being treated with haloperidol ($M = 4.9$, $SD = 1.8$) ($p < 0.05$), however there was no significant difference between the reaction times for this test ($p < 0.5$). On the Tachistoscope Test (TT15), a task assessing the ability to quickly extract relevant information from typical traffic situations presented for 0.75 s, participants being treated with haloperidol demonstrated significantly shorter reaction time ($M = 3.4$, $SD = 0.8$) participants being treated with atypical neuroleptics ($M = 4.4$, $SD = 1.6$) ($p < 0.01$), however no differences were found in the number of correct response items. On the attention test (Q1), a task assessing attention under a monotonous condition, there were

no significant differences across the groups. On the Reactive Stress Tolerance Test (RST3), a test measuring stress resistance and the capacity to integrate information, participants being treated with atypical neuroleptics demonstrated better performance on all dimensions. For example, in the 'highest stress level', compared with participants treated with haloperidol, participants treated with atypical neuroleptics provided significantly more correct responses (Atypical: $M = 134.2$, $SD = 37.6$; Haloperidol: $M = 106.8$, $SD = 26.2$; $p < 0.05$) and more correct responses in time allowed (Atypical: $M = 70.7$, $SD = 50.3$; Haloperidol: $M = 39.0$, $SD = 43.6$; $p < 0.05$), a tendency of a less percentage of delayed responses (Atypical: $M = 53.1$, $SD = 26.4$; Haloperidol: $M = 69.2$, $SD = 24.7$; $p = 0.058$) and less omissions (Atypical: $M = 39.8$, $SD = 32.7$; Haloperidol: $M = 60.7$, $SD = 26.7$; $p = 0.061$). Several limitations of this study are noted. First, the sample size is very small ($n = 49$). Second, participants were recruited from one psychiatric hospital and it is not clear whether the sample is adequately representative of the population of all drivers. Third, the authors noted that participants had either a valid driver's licence or intended to obtain one within the next few months, however the authors do not control for driver exposure, which assumes that drivers from each of the groups drive similar distances. Finally, the generalisability of these findings to real world driving and crash risk is not clear.

Brunnauer, Laux, Geiger and Moller (2004) also investigated possible differences between conventional and atypical neuroleptics with respect to driving-relevant cognitive aspects in individuals with schizophrenia. Consecutively admitted individuals with schizophrenia ($n = 120$) were tested before discharge to outpatient treatment. All participants met the ICD-10 criteria for schizophrenia. Nineteen participants were being treated with typical neuroleptics, 53 participants were being treated with atypical neuroleptics, 24 received clozapine, 24 received flupenthixol, all in different dosages. All participants were (1) neuroleptic monotherapy, (2) steady state pharmacological conditions and (3) in possession of a driver's licence. Participants with a history of neurologic illness, substance abuse or mental retardation were excluded.

Data were collected with the computerized Act & React Test system and were analyzed according to medication, severity of illness, and age. Only 32.5% of participants passed the tests without major impairments. Patients treated with atypical neuroleptics or clozapine demonstrated better test performance on skills related to driving ability when compared with patients on typical neuroleptics. Differences were most pronounced in measures of divided attention (PVT), stress tolerance (RST3), and attention (Q1). Data also suggest that treatment with clozapine had an overall positive impact on measures of reactivity and stress tolerance. These results show that even under steady state pharmacological conditions, psychomotor functions of most participants with schizophrenia are impaired. The authors concluded that individuals being treated with typical neuroleptics performed worse than those treated with atypicals with respect to choice reaction, orientation, attention and tracking. Several limitations of this study are noted. Participants were recruited from one psychiatric hospital and it is not clear whether the sample is adequately representative of the population of all drivers. Second, the authors noted that participants had either a valid driver's licence, however the authors do not control for driver exposure, which assumes that drivers from each of the groups drive similar distances. Finally, the generalisability of these findings to real world driving and crash risk is not clear.

Most recently, Soyka, Winter, Kagerer, Brunnauer, Laux & Möller (2005) extended the findings of Kagerer, Winter, Moller and Soyka (2003) and investigated the effects of

risperidone (an atypical neuroleptic) and haloperidol (a conventional dopamine antagonist) on several dimensions of psychomotor performance (visual perception, attention, reaction time, and sensorimotor performance) considered to be of relevance in evaluating fitness to drive with a group of healthy controls. Cases ($n = 40$) were defined as participants who met the ICD-10 and DSM-IV criteria for schizophrenia or a schizoaffective disorder. Cases were recruited from a Psychiatric Hospital in Munich, Germany. Twenty cases were being treated with risperidone, average dosage of 4.6 mg/day (4–8 mg). The other 20 cases were being treated with haloperidol, with a dosage of 10, 4 mg/day (5–30 mg). Controls ($n = 19$) were defined as ‘healthy’ controls. There was no significant difference in age, sex, education and Brief Psychiatric Rating Scale (BPRS) scores between the haloperidol-treated group and the group treated with atypical neuroleptics. Patients were excluded if they had a disabling physical disorder, organic brain disorder, acute substance abuse, or any serious concurrent medical condition. There was no evidence of extrapyramidal motor symptoms in any of the study patients. All participants had either a valid driver’s licence or intended to obtain one within the next few months.

Overall, the findings demonstrate that (1) participants with schizophrenia showed significantly worse results in most psychometric tests compared to healthy controls, (2) participants with schizophrenia currently being treated with haloperidol showed considerable psychomotor and cognitive impairment compared to participants who were being treated with risperidone. On a Peripheral Vision Test with Tracking Task (PVT), significant differences across groups were found with regards to both reaction time and the tracking performance. Participants with schizophrenia being treated with the risperidone demonstrated significantly faster reaction times ($M = 1.28$, $SD = 0.53$) than the participants with schizophrenia being treated with haloperidol ($M = 1.94$, $SD = 1.18$, $p < 0.05$). In addition, participants with schizophrenia being treated with haloperidol demonstrated significantly longer reaction times than the controls ($M = 1.06$, $SD = 0.27$, $p < 0.0001$). On the tracking task, no significant differences were observed between the haloperidol and the risperidone treated groups, however they were both observed to have significantly reduced performance compared to the controls (Risperidone: $M = 4.50$, $SD = 1.93$; Haloperidol: $M = 4.92$, $SD = 1.78$; controls: $M = 3.06$, $SD = 0.65$; Risperidone vs. controls, $p < 0.01$; Haloperidol vs. control, $p < 0.001$). On the Tachistoscope Test (TT15), there was no significant difference observed between the haloperidol ($M = 30.00$, $SD = 4.27$) and risperidone ($M = 31.80$, $SD = 3.21$) treated participants with schizophrenia, while both groups showed significantly less correct answers than controls (34.65, $SD = 2.46$, Risperidone vs. controls: $p < 0.001$, Haloperidol vs. controls: $p < 0.01$, respectively). On the attention test (Q1), there was a trend towards more completed trials by participants treated with Risperidone ($M = 419.55$, $SD = 90.84$) compared participants treated with haloperidol ($M = 367.55$, $SD = 80.15$, $p = 0.052$), however, both group demonstrated significantly lower performances than the controls ($M = 513.25$, $SD = 64.65$, Risperidone vs. controls: $p < 0.01$, Haloperidol vs. controls: $p < 0.001$, respectively). On the Reactive Stress Tolerance Test (RST3), participants treated with Risperidone performed significantly better on all dimensions compared to the participants treated with haloperidol ([More correct responses: Risperidone $M = 137.56$, $SD = 26.21$; Haloperidol: $M = 109.23$, $SD = 28.15$; $p < 0.01$] [More correct responses in time allowed: Risperidone: $M = 69.78$, $SD = 45.88$; Haloperidol: $M = 39.38$, $SD = 40.78$; $p < 0.05$] (Less omissions: Risperidone: $M = 35.28$, $SD = 25.03$; Haloperidol: $M = 60.24$, $SD = 25.13$; $p < 0.01$). However, the authors also noted that cases treated with Risperidone performed significantly worse than the controls ([More correct responses: Risperidone $M = 137.56$, $SD = 26.21$; Controls $M = 170.95$, $SD = 7.56$, $p < 0.001$] [More correct responses in time allowed:

Risperidone: $M = 69.78$, $SD = 45.88$; Controls: $M = 128.53$, $SD = 35.22$, $p < 0.001$] [Less omissions: Risperidone: $M = 35.28$, $SD = 25.03$; Controls: $M = 7.16$, $SD = 6.84$, $p < 0.001$]. There are also several potential sampling biases in this study. First, the sample size is very small. Second, cases were recruited from one psychiatric hospital and it is not clear whether the sample is adequately representative of the population of all drivers. Third, there is no information provided about how the control participants were recruited into the study. Also the authors did not indicate whether the control participants had been screened for psychiatric or medical comorbidities. Fourth, the authors noted that cases had either a valid driver's licence or intended to obtain one within the next few months, whereas controls were recruited if they held a valid driver's licence. However the authors did not control for driver exposure, which assumes that drivers from each of the groups drive similar distances. Finally, while the authors indicate that the tests are considered to be of relevance in evaluating fitness to drive, the generalisability of these findings to real world driving and crash risk is not clear.

Anti-depressants

Crashes

No studies reporting the relationship between antidepressant medications for psychiatric illness and crashes were found.

Citations

No studies reporting the relationship between antidepressant medications for psychiatric illness and driving citations or traffic violations were found.

Driving Performance

Wingen, Ramaekers and Schmitt (2006) investigated the driving and cognitive performance of individuals diagnosed with depression who were receiving long-term anti-depressant medication compared to healthy controls. Cases were 24 individuals with a primary diagnosis of unipolar disorder with an active depressive episode according to the DSM-IV criteria, with scores on the Hamilton Depression Rating Scale above 17. Cases were undertaking antidepressant treatment with a selective serotonin reuptake inhibitor (SSRI) venlafaxine for 6-52 weeks. Controls were 24 age, gender and years of driving experience matched healthy volunteers, free from psychiatric illness at present or in the past and had no first-degree relative with a history of psychiatric illness and were not taking any medication. Cases were recruited through regional psychiatric centres and by advertisement in the local newspaper. Controls were recruited by advertisement in the local newspaper. All participants were free from neurological, cardiovascular, respiratory, metabolic, hepatic or renal disorders or a history of these disorders, did not use illicit drugs and were devoid of any motor or sensory deficits that could be reasonably expected to affect test performance. All participants had a valid driving licence of three years and driving experience of at least 5,000 km/year during each of the preceding years. The authors noted poorer driving performance on the on-road driving test by cases, as evidenced by significantly higher standard deviation of lateral position or 'weaving motion' compared to controls ($p < 0.01$). The 'time to speed adaptation' in the car following test was also significantly impaired in cases compared to controls ($p < 0.05$). However, other outcome measures such as brake reaction time, speed and headway did not differ significantly across

groups. In terms of cognitive measures, the average critical flicker fusion threshold frequency was reduced in cases compared to controls ($p < 0.01$), however there were no other significant differences across the two groups. The results of this study suggest statistically significant impairment of driving performance in depressed participants receiving long-term antidepressant treatment as compared to healthy controls. It should be noted that due to the relatively small sample size and the use of a simulator to measure driving performance, it is not clear how these findings might generalise to actual driving risk for drivers diagnosed with depression receiving long-term antidepressant medication.

Brunnauer, Laux, Geiger, Soyka and Moller (2006), outlined earlier, also considered the effects of antidepressant monotherapy on psychomotor functions related to car driving skills. One hundred patients aged between 20 and 78 (mean age 46.8 ± 13.6) who met the ICD 10 and DSM-IV criteria for Major Depressive disorder (MDD) using a naturalistic and nonrandomised study design. Inclusion criteria for the study were (1) antidepressant monotherapy, (2) steady-state pharmacological conditions (all patients were considered for discharge within at least 3 days) and (3) possession of a valid driver's licence. Antidepressant use across the patients was divided across tricyclic antidepressants (TCA; $n = 40$), selective serotonin reuptake inhibitors (SSRI; $n = 25$), a noradrenergic and specific serotonergic antidepressant called mirtazapine ($n = 20$) and a serotonin-norepinephrine reuptake inhibitor called venlafaxine ($n = 15$). The experimental procedure, conducted at 9:00am, consisted of computerised psychomotor tests of visual perception, selective attention, vigilance, reactivity and stress tolerance. Statistically significant differences were found between patients treated with TCAs and SSRIs versus mirtazapine, indicating a better test performance for patients prescribed mirtazapine. Patients using TCAs also significantly differed from those using SSRIs ($F = 6.53$, $df = 1,64$, $p < .05$) and mirtazapine ($F = 12.76$, $df = 1,59$, $p < .001$) indicating impaired performance for TCAs. Significant differences were not found between patients treated with TCAs or venlafaxine. Overall, the results suggest that patients treated with SSRIs or mirtazapine may be advantaged over those treated with TCAs or venlafaxine. However, it should be noted that as a non-randomised study design, causal relationships cannot be claimed. Further to this, selection bias of participants cannot be excluded: only patients who were sufficiently well enough to participate in a 150 minute experimental procedure were recruited, suggesting that the results may reflect an overestimation of MDD patient fitness to drive. Given that patients were recruited from a population derived from clinical psychiatric practice which overcomes many of the generalisability issues associated with clinical trials, these results are worth taking note.

Iwamoto, Kawamura, Takahashi, Uchiyama, Ebe, Yoshida et al. (2008) and Iwamoto, Takahashi, Nakamura, Kawamura, Ishihara, Uchiyama et al. (2008) looked at the effects of antidepressants and individual pharmacokinetic differences on driving performance. In these studies Iwamoto et al. recruited 17 healthy male volunteers (age range 30 – 42, mean 35.8 years) who had held a driver's licence for at least 10 years, drove a car daily, were drug-free, and had no physical or psychiatric disorders. They were also prohibited from alcohol or caffeinated beverages for 12 h before testing, on test days participants prohibited from ingesting caffeine, chewing gum, supplement drinks to avoid a stimulating effect on their performance. Driving performance was measured on a simulator and involved a road tracking test (RTT), a car following test (CFT), and a harsh braking test (HBT). The study was a randomised, double-blind, placebo-controlled, 3-way crossover design. In Iwamoto, Takahashi et al. subjects received doses of 10mg paroxetine (a selective serotonin reuptake inhibitors [SSRI]), 25mg amitriptyline (a tricyclic antidepressant [TCA]) and matched placebo in 3

different treatment sessions, held one week apart (washout period). Medications and placebo were presented identically. Participants were given training in the driving simulator until they'd reached the plateau level (to minimize learning effects). Participants' driving test took 15 minutes, conducted at baseline, 1 hour after dosing, and 4 hours post dosing. Friedman's χ^2 r-test showed statistically significant effects of treatment on the differences between baseline and 4-h post-dosing on the RTT ($\chi^2 = 12.0$, $df = 2$, $p = 0.0025$) and CFT ($\chi^2 = 8.82$, $df = 2$, $p = 0.0121$). Post hoc testing demonstrated that RTT was significantly greater under the amitriptyline condition than the two other conditions ($p < 0.05$ versus placebo, $p < 0.01$ versus. paroxetine), and CFT was significant greater under the amitriptyline condition than under the paroxetine condition ($p < 0.01$). In Iwamoto, Kawamura et al. the 17 subjects received doses of 25mg amitriptyline (a TCA). Baseline assessment was followed by dosage of the antidepressant. Blood samples were collected 4 hours after administration (when maximum plasma concentration occurs). Subjects were re-tested. Resultant blood samples were centrifuged at 1700g for 10 mins and frozen at -30°C . Plasma concentration determined on high-performance liquid chromatography, 5-point calibration curves set up for range 2 – 200 ng/mL. A linear response function was obtained and limit of quantification was 2 ng/mL. Interday coefficient of variation for 4 days for plasma amitriptyline at 20ng/mL was 11.2%. Intraday coefficients of variation were 1.1-1.2% ($n=2$). Driving performance was measured as in the study described earlier. Significant correlation was observed between plasma amitriptyline concentration and percentage change in RTT (baseline deviation from the centre of the road was $38.9 \pm 10.8\text{cm}$, at 4 hours post-dosage it increased to $51.3 \pm 12.7\text{cm}$). This increase in deviation may be compared to lateral swerving in the real world, which might lead to road traffic accidents, suggesting that amitriptyline may have a detrimental effect on concentration and road tracking. However, there are a number of issues associated with these two studies: it is unclear how or where participants were recruited, only male participants were recruited because of the changes in hormone levels occurring during the menstrual cycle which may affect cognition in healthy women and only used single doses of each antidepressant. Despite these limitations, these studies had a number of strengths; the study design controlled well for between and within subject differences and tested participants at low concentrations of the antidepressant, equivalent to the starting dose prescribed by medical professionals.

Anti-anxiety

Crashes

Herbert, Delaney, Hemmelgarn, Levesque and Suissa (2007) attempted to reconcile the differences between two experimental designs employed in studies by Hemmelgarn et al. (1997) and Barbone et al. (1998). The investigators re-analysed data from the Hemmelgarn et al. study using both a case-control and a case-crossover design. The case-control design used 6.2% of the sample as unmatched controls. Index dates were selected at random and cases were considered to be exposed to benzodiazepines if the duration of their last prescription covered the index date. Odds ratios (OR) were estimated by adjusting for the number of previous crashes, age, sex, chronic disease score and other central nervous system acting agents. For the case-crossover design, control periods were created using the exposure of each case during 18 weeks prior to the week of the crash. Results suggest that use of benzodiazepines with a long half life (clonazepam, diazepam, clorazepate, chlordiazepoxide, flurazepam and nitrazepam) can

be found to be associated with an increased risk of crash in elderly drivers when a case control design is used (OR: 1.45, 95%CI 1.12 - 1.88). When a case-crossover design is used, an increased risk of crash is associated with the use of long half-life benzodiazepines when infrequently used (1-4 prescriptions filled in the year prior to index date) was found (OR: 1.53, 95%CI 1.08 - 2.16). The authors concluded that a case-crossover design with its ability to remove time-invariant between-subjects differences may be more appropriate for investigation of the impact of benzodiazepines upon driving due to their time dependent effects and cautioned users of long-acting benzodiazepines who continued to drive. The study's large sample size (5,579 participants) and consistent findings of increased crash risk with the use of long half-life benzodiazepines regardless of study design is worthy of note.

Citations

No studies reporting the relationship between anti-anxiety medications for psychiatric illness and driving citations or traffic violations were found.

Driving performance

In 2007, Boucart, Waucquier, Michael and Libersa investigated the effect of a benzodiazepine (diazepam) on selection attention and dual task performance. Thirty-six healthy volunteers (all drivers, French speakers, without a history of chronic illness, alcoholism or drug abuse and with a tobacco consumption of no more than 10 cigarettes per day) were recruited. Participants' urine samples were tested to ensure that they were not chronic users of benzodiazepines, had not taken concomitant medication for at least 21 days prior and also abstained from caffeine and alcohol 24 hours prior to the study. Participants were randomly assigned into one of three groups of 12: a placebo group (mean age of 24.5 years, 8 women and 4 men); a diazepam experimental group who received a dosage of 0.1 mg/kg (mean age of 25.2 years, 5 women and 7 men); a second diazepam experimental group who received a dosage of 0.3 mg/kg group (mean age 26.2 years, 6 women and 6 men). The diazepam dosages were selected so that the peak plasma concentration of the benzodiazepine would correspond to the average plasma concentration under the usual given doses in practice. The dosages were prepared by an unblinded nurse with 100mL of water, and were distributed to participants by a blinded nurse to preserve the double-blind procedure. The placebo administered was an extract of bitter orange peel and syrup. A Rapid Serial Visual Presentation (RSVP) paradigm was used with photographs depicting roads and 10 well known French city names of five letters each. All participants were given a practice run and started with the dual task to minimise learning effects. In the dual task condition participants were asked to identify the city name (the target) and to detect the presence of a vehicle (probe); in the single task control condition, participants were asked to detect the presence of a vehicle and ignore the city name. To ensure optimal blood concentration of drug, pharmacokinetics were performed more than 10 days before the experiment. These tests involved administering 0.1 or 0.3 mg/kg diazepam to each participant (on different days). A blinded nurse collected blood samples to measure blood concentration of diazepam before intake and at 20, 30, 40, 50, 60, 75, 90, 120, 180, and 240 min after intake. Concentrations were analysed and a T max (time of optimal concentration) were provided for each participant). During the experiment, participants were administered diazepam 10 minutes before their T max; a random T max was generated for those administered the placebo. No significant differences were found between the placebo condition and 0.1 mg/kg diazepam condition with regard to target identification. A

significantly higher difference (compared to placebo) was found for the 0.3 mg/kg diazepam condition ($F(1, 22) = 25.6, p < 0.001$). The accuracy of probe detection was higher for placebo treated participants than for both diazepam exposure groups ($F(2, 33) = 23.6, p < .001$). A significant main effect of task condition was found ($p < .001$). Task condition also interacted significantly with group, ($F(2, 33) = 10.8, p < .001$). A larger impairment in probe detection was found in the benzodiazepine group than the placebo group in the dual task condition; performance was not significantly affected by diazepam in the single task condition. The magnitude of the attentional blink effect was larger for 0.1 mg/kg diazepam than for placebo, ($F(1, 22) = 6.4, p < .019$); 0.3 mg/kg diazepam versus placebo, ($F(1, 22) = 59, p < .001$); and diazepam 0.1 mg/kg versus 0.3 mg/kg, ($F(1,22) = 14.4, p < .001$). No gender effects were found. The authors concluded that diazepam degraded dual task performance, by half when exposure was 0.1 mg/kg and by a quarter when exposure was 0.3 mg/kg compared to the placebo. No limitations were discussed within the paper however whether the effects of the drug mediate the negative effects of chronic illness or compound their effects is not discussed.

Dubois, Bedard and Weaver (2008) examined the impact of benzodiazepine exposure on driver error through a case-control design with drivers aged 20 and over. The authors used measurements of toxicity through standardised testing across all benzodiazepine half-life types. Fatality Analysis Report System (FARS), a database of fatal crash information from the United States was used to extract information on drivers from 1975 to the present. The study used the following database information: age, sex, drug test results (blood or urine), blood alcohol concentration results, type of vehicle driven and information on drivers' past driving record. 72,026 driver fatalities with a BAC of zero were analysed using logistic regression models. Of the sample, 2,200 (3%) tested positive for benzodiazepines. Benzodiazepine exposure was further broken down according to half-life duration: < 6 hours (short half-life; $n = 161$); greater than 6 but less than or equal to 24 hours (intermediate half-life; $n = 369$); and > 24 hours (long; $n = 1020$). Six hundred and fifty cases were excluded as they tested positive for unclassified benzodiazepines and/or positive for multiple half-lives. When adjusted for age, sex, other medications and driving records, odds ratios were: short half-life = 1.00 (95% CI = 0.79 – 1.49); intermediate half-life = 1.54 (95% CI 1.21 – 1.96); and long half-life = 1.44 (95% CI 1.25 – 1.66). When analysed by age and benzodiazepine exposure, it was found that a 25 year old driving using a long half-life benzodiazepine had an odds ratio of 1.68 (95% CI 1.34 – 2.21) compared to an odds ratio of 1.13 for a driver aged 75 (95% CI 0.84 – 1.53). The authors concluded that the odds of an unsafe driver action (recorded by police as actions which contributed to the crash, no record if driver not at fault) was found to increase from 33% to 68% for a driver exposed to benzodiazepines. The impact of exposure decreased with age, but remained statistically significant for intermediate and long half-life benzodiazepines through middle age. Drivers exposed to short half-life benzodiazepines did not demonstrate increased odds of an unsafe driver action. There are a number of limitations associated with this study: a much smaller group of drivers aged 65 and over were used (suggestion that the group may have lacked statistical power), ages of drivers under 20 (arguably frequent users of benzodiazepines) were not considered and the effect of concentrations of benzodiazepines were not considered (potentially underestimating the risk).

Summary

Despite the prevalence of psychiatric illness in the general population, the relationship between history of psychiatric illness and motor vehicle crashes has received limited

attention (Elwood, 1986). In terms of crash risk, several studies have shown that drivers with psychiatric illness have an increased crash risk, as well as an increased at-fault crash risk, compared to drivers without psychiatric illness (Armstrong & Whitlock, 1980; Vernon et al., 2002). In addition, several studies have shown that schizophrenia and personality disorders may be associated with an increased crash risk (Edlund, Conrad & Morris, 1989; Dumais, Lesage, Boyer, Lalovic, Chawky, Ménard-Buteau, Kim & Turecki, 2004).

In terms of citations, the evidence in this regard suggests a modestly elevated citation rate but only for those who have a low level of impairment (Vernon et al., 2002). It is possible that those with higher levels of impairment self-regulate their driving in such a way as to reduce their exposure or drive slower or more cautiously. Given the limited amount of evidence available, it is difficult to make any definitive statement about psychiatric illness and its impact on citation rates.

In terms of driving performance, recent evidence suggests that drivers with an untreated Major Depressive Disorder perform significantly worse than healthy controls on simulated driving trials (Bulmash, Moller, Kayumov, Shen, Wang & Shapiro, 2006).

As noted by Dobbs (2001) and others, most of the available literature investigating the relationship between psychiatric illness and driver risk is limited by the following methodological weaknesses:

- The use of self-report data or data obtained from medical records, the crash victim and their families and/or police records is likely to result in an underestimation of crashes (McDonald & Davey, 1996).
- Sample sizes per diagnostic category are often too small.
- Estimating prevalence of psychiatric disorders through the use of non-standardised interviews and reliance on obtaining psychiatric information from medical records will result in underestimation of the true rates of psychiatric disorders, as only those that have been formally diagnosed and entered on the available records will be recorded (Kolman, 1983).
- A number of psychiatric illnesses may fluctuate in their degree of impairment and transience, and unless the duration and severity of illness is specified, the precise effect on driving ability may be unclear. In addition, the use of medical histories will also leave unanswered the question of whether or not the disorder was in remission at the time of the crash (Kolman, 1983).
- The use of different diagnostic criteria or categories across studies makes direct comparisons difficult. Use of standardised criteria (e.g., DSM-IV) would help to alleviate this limitation.
- Most studies failed to specify the type of prescription medication and medication compliance. Dobbs (2001) suggests that future studies should include data on medication use and use statistical controls for drug use.
- Finally, many studies failed to consider driving exposure. It is not unreasonable to expect that individuals with psychiatric illnesses drive substantially less than

age- and sex-matched controls in the general population. Thus, the available estimates of crash risk are likely to be underestimations.

The limited available evidence suggests that crash rates may be higher among drivers with a psychiatric illness, however much more research on specific types of psychiatric illness involved and their specific relations to crashes is needed. Furthermore, most drugs used in psychiatric therapy have some effect on driving ability, particularly when prescribed in high doses.

For example, antipsychotic medications, also known as neuroleptics, are the mainstay of the pharmacological treatment of serious psychiatric illnesses such as schizophrenia (Judd, 1985). Recent studies have begun to investigate possible differences between conventional and atypical neuroleptics on several dimensions of psychomotor performance (visual perception, attention, reaction time, and sensorimotor performance) considered to be of relevance in evaluating fitness to drive. Overall, the findings of these studies have demonstrated that (1) participants with schizophrenia have significantly worse results in most psychometric tests compared to healthy controls (Soyka et al., 2005), and (2) participants with schizophrenia currently being treated by conventional neuroleptics showed considerable psychomotor and cognitive impairment compared to participants who were being treated with atypical neuroleptics (Brunnauer et al., 2004; Kagerer et al., 2003; Soyka et al., 2005).

Anti-depressants are the cornerstone of treatment for major depression (Dobbs, 2001). Besides the beneficial effects of anti-depressants, these drugs can also produce side effects such as sedation, lethargy, impaired psychomotor function and sleep disturbances (Ramaekers, 2003). Research suggests that cyclic antidepressants are associated with an increased risk of injurious crash involvement (Ray et al., 1992). In addition, recent research suggests significant impairment of driving performance in depressed participants receiving long-term antidepressant treatment (SSRI) as compared to healthy controls (Wingen et al., 2006). Antidepressants are generally divided into older tricyclic antidepressants and newer selective serotonin reuptake inhibitors (SSRI, Dobbs, 2001). Current research suggests that older tricyclic antidepressants are more likely to affect psychomotor and driving performance compared with SSRIs (O'Hanlon et al., 1998; Laar, et al., 1995).

Benzodiazepines are the most commonly used medication for the treatment of anxiety and insomnia and one of the most frequently used classes of medication taken by elderly individuals (Ray, Purushottam, & Shorr, 1993). Benzodiazepines have been shown to impair vision, attention, information processing, memory, motor coordination, and combined skilled tasks (Boucart, Waucquier, Michael & Libersa, 2007; Ray et al., 1993). Most case-control studies suggest that benzodiazepine use in general is associated with increased crash risk (Ray et al., 1992; Barbone et al., 1998), at-fault crash risk (Barbone et al., 1998; McGwin et al., 2000) and impaired driving ability (O'Hanlon et al., 1995). In addition, it appears as though longer acting benzodiazepines are of particular concern (Barbone et al., 1998; Dubois, Bedard & Weaver, 2008; Hemmelgarn et al., 1997) and that the risk appears highest in the first four weeks of therapy, after which tolerance generally develops to the sedation and dysfunctional effects on coordination ((Hemmelgarn et al., 1997; Silverstone, 1988). This drug class may also be especially hazardous for elderly drivers (Ray et al., 1993, Herbert et al., 2007).

However, it should be noted that simply finding an association between the use of prescribed psychotropics and an increased risk of road crashes is insufficient evidence to suggest that the medication played a part in the crash process, there are many other factors such as personality factors, driver fatigue, age, individual drug tolerance, concurrent alcohol use, driving experience and number of hours spent driving (The University of Western Australia, 1995). Furthermore, the difficulty here lies in the fact that psychiatric illnesses themselves impair driving, and therefore it is extremely difficult to assess whether the effects of the medication or the effects of the illness are responsible for the crash (Cremona, 1986). It may well be that individuals are safer drivers with psychotropic medications than without them (Cremona, 1986).

Despite methodological strengths and weaknesses of numerous studies, the findings suggest that medication treatments for psychiatric illnesses certainly have the potential for causing motor vehicle crashes (Silverstone, 1988).

Table 32 Summary of studies of risk associated with psychiatric illness and treatments for psychiatric illness

Study: Author/date	Methods	Outcome Measure of Risk	Results
Armstrong & Whitlock (1980)	Cases = 100 psych ill: Controls = 100 physically ill	Self-report interviews: - 6 months pre-admin - 2-3 years pre-admin - Yrs driving experience	6 month: Psyc = Phys 2-3 yrs: Psyc = Phys Yrs driving: Psyc < Phys *
Barbone et al. (1998)	19,386 individuals registered with a Tayside, UK Gap between January 1992-1995 and involved in an MVC. Cases = individuals aged 18 + who had a MVC linked by Community Health Numbers to prescription data.	Case-crossover design - the odds of having a crash while exposed to one of the study drugs were compared with the odds of having a crash while unexposed. Odds ratio calculated as a measure of association between drug use and crash.	916 individuals prescribed a benzodiazepine (OR = 1.62, confidence intervals not given). <ul style="list-style-type: none"> • use highest among drivers younger than 30, decreased with increasing age, and not raised in people aged 65+ (trend p=.01) • OR for users of benzodiazepines decreased as number of vehicles involved increased (ns) • associated with significant risk in accidents where driver at fault • risk of road accident associated with benzodiazepine use was significant in drivers who had a negative breath test for excess alcohol but association was much stronger for those who had a positive breath test (p=.02) (exposure same as nonexposed drivers)
Boucart, Waucquier, Michael & Libersa (2007)	36 healthy individuals divided into 3 groups: placebo, 0.1 mg/kg diazepam and 0.3 mg/kg diazepam	Performance on dual tasks and selective attention	Dual task performance degraded by 2x for 0.1 mg/kg diazepam and 4x for 0.3 mg/kg diazepam c.f. to placebo.
Brunnauer, Laux, Geiger, Souka & Moller (2006)	100 depressive inpatients (ICD 10 & DSM IV criteria for major depressive disorder) Mean age 46.8y (range 20-78).	Performance on psychomotor tests of visual perception, RT, selective attention, vigilance, stress tolerance.	SD between patients treated with TCAs versus mirtazapine (p<.05, z=-2.49), mirtazapine versus SSRIS (p<.01, z=-2.04).

Study: Author/date	Methods	Outcome Measure of Risk	Results
			SD between TCAs and SSRIs (F=6.53, df = 1,64, p<.05) and mirtazapine (F=12.76, df = 1,59, p<.001) indicating impaired performance for TCAs.
Dumais et al. (2004)	<p>Case – Control</p> <p>Cases = 61 male drivers (18-36 yrs) who died in a road crash.</p> <p>Controls = 61 living males matched for age, sex, employment & marital status</p>		<p>Cases were more likely to have:</p> <p>Cluster B personality disorders (borderline and [or] antisocial) (OR 3.54; 95%CI, 1.38 to 16.01)</p> <p>Substance use disorders (OR 4.33; 95%CI, 1.42 to 9.25)</p> <p>Interaction between cluster B personality disorders and age over 26 years was the only significant predictor of car fatalities (adjusted OR 16.25; 95%CI, 1.67 to 158.10).</p>
Dubois, Bedard & Weaver (2008)	72,026 killed drivers in USA with BAC = 0, 2,200 (3%) tested positive for benzodiazepines. Grouped according to half-life: short (n=161), intermediate (n=369) and long (n=1020).	<p>Outcome – Unsafe driver actions (UDA)</p> <p>Recorded by police as actions that contributed to the crash, if no UDA</p>	<p>When adjusted for age, sex, other medications and driving record, odds ratios of any potentially unsafe driver action occurring were:</p> <ul style="list-style-type: none"> • short half life = 1.00 (95% CE = 0.72, 1.39); • intermediate half life = 1.54 (95% CE = 1.21, 1.96); • and long half life = 1.44 (95% CE = 1.25, 1.66)
Edlund et al. (1989)	<p>Cases = 70 outpatient schiz</p> <p>Control = 122 age matched</p>	<p>Self-report questionnaires</p> <p>Crude incidence of crashes over past 12 months</p>	<p>Self-report crash rate: schz = C</p> <p>Distance driven: schz < C*</p> <p>Acc ratio/ mile driven: Schz > C*</p>

Study: Author/date	Methods	Outcome Measure of Risk	Results
Hemmelgarn, Suissa, Huang, Boivin & Pivan (1997)	Cases = 5,579 older drivers using BZ (aged b/w 67 and 84) Controls = 13,256 controls during a period of 1990 to 1993.	Data on BZ use was taken from a prescription drug database.	Long half-life BZ RR acc (adjusted) : 1.28* RR in first week: 1.45* Short half-life BZ RR acc: 0.96 Small sample size P recruited from one psychiatric hospital – is sample representative of all drivers? Did not control for driver exposure. Generalisability of findings to “real world driving” and crash risk is not clear.
Herbert, Delaney, Hemmelgarn, Levesque & Suissa (2007)	5,579 injurious motor vehicle collisions between 1 June 1990 and 31 May 1993, aged 67 to 84. Control population 6.2% of sample. Analysed data using two study designs	Database of collisions linked to prescription database maintained in province of Quebec analysed via case-crossover and case-control experimental designs.	Regardless of design, use of long half-life benzodiazepines found to be associated with increased risk of motor vehicle collision in elderly drivers (OR = 1.45, 95% CI 1.12 – 1.88).
Iwamoto, Kawamura et al. (2008)	17 male volunteers (30-42 years, mean 35.8), who had held a driver's licence for at least 10 years, drove a car daily, drug-free, no physical or psychiatric disorders. Double-blind design. Subjects received doses of 25mg amitriptyline (a TCA). Baseline assessment, followed by dose.	Driving performance on a simulator	Significant correlation was observed between plasma amitriptyline concentration and percent change in vehicle lateral position maintenance (Baseline was 38.9 +- 10.8, at 4 hours increased to 51.3 +- 12.7 cm).

Study: Author/date	Methods	Outcome Measure of Risk	Results
	Blood samples collected 4 hours after administration (when maximum plasma concentration occurs). Plasma concentration and calibration curves set up for range 2 – 200 ng/mL. Linear response function was obtained.		
Iwamoto, Takahashi et al. (2008)	<p>17 male volunteers (30-42 years, mean 35.8), who had held a driver's licence for at least 10 years, drove a car daily, drug-free, no physical or psychiatric disorders.</p> <p>Subjects received doses of 10mg paroxetine (an SSRI), 25mg amitriptyline (a TCA) and matched placebo in 3 different treatment sessions, held one week apart (washout period).</p>	<p>Randomised, double-blind, placebo-controlled, 3-way crossover design.</p> <p>Driving performance on a simulator</p>	4 hours after taking a single 25 mg dose of amitriptyline, there was significant impairment of lateral position maintenance and braking on a car following test.
Bulmash et al. (2006)	<p>Case Control</p> <p>Cases = 18 outpatients met DSM-IV criteria for Major Depressive disorder (MDD).</p> <p>Controls = 29 individuals from the community who met screening criteria for inclusion and did not meet criteria for a current episode of MDD.</p> <p>All participants were aged between 18-65, had a valid driver's licence and at least five years of driving experience.</p>	Driving performance of individuals on four 30-min simulated driving trials	<p>Steering reaction time: Cases > Controls*</p> <p>Number of Crashes: Cases > Controls*</p> <p>Road position: Cases = Controls</p> <p>Speed: Cases = Controls</p> <p>Speed deviation: Cases = Controls</p>

Study: Author/date	Methods	Outcome Measure of Risk	Results
Brunnauer et al. (2004)	Ps with schizophrenia (n = 120) 19 Ps = typical neuroleptics, 53 Ps = atypical neuroleptics, 24 Ps = with clozapine, 24 Ps = with flupenthixol	Performance on the Act-and-react test system (ART 90)	Overall performance: Atypical and Clozapine > Typical and Flupenthixol PVT: Typical < Clozapine *** Typical < Atypical*** Typical < Flupenthixol** TT15 – Number of correct numbers: Typical < Atypical*** Q1: Typical < Atypical* Typical < Clozapine * RST3 – Omissions: Clozapine < Flupenthixol**
Kagerer et al. Soyka (2003)	Cases = 29 inpatients with schizophrenia or a schizoaffective disorder treated with atypical neuroleptics Controls = 20 inpatients with schizophrenia or a schizoaffective disorder treated with conventional dopamine antagonist	Performance on the Act-and-react test system (ART 90)	PVT: Cases < Controls* Cases = Controls TT15 – Mean reaction time: Controls < Cases ** TT15 – Number of correct responses: Cases = Controls Q1: Cases = Controls RST3 – Number of correct responses: Cases > Controls * RST3 – Number of correct responses in allowed time: Cases > Controls *
McGwin, Sims, Pulley & Roseman (2000).	- pop-based control study - Cases = 447 drivers 65 yrs and older inv in crash - Control = 454 drivers not inv in crash	- Police records judged if case was at least partially at-fault	OR At-fault crash inv: 5.2*
O'Hanlon, Robbie, Vermeeren,	Cases = 37 healthy volunteers	Driving and psychomotor	SSRI no sign effect on psychomotor.

Study: Author/date	Methods	Outcome Measure of Risk	Results
van Leeuwen & Danjou (1998)	given venlafaxine (SSRI) and Mianserin (TCA)	performance	TCA sig effect on both psychomotor and driving performance. Vigilance sign affected by both
Soyka et al. (2005)	<p>Cases – atypical = 20 inpatients with schizophrenia or a schizoaffective disorder treated with atypical neuroleptics</p> <p>Cases – typical = 20 inpatients with schizophrenia or a schizoaffective disorder treated with typical neuroleptics</p> <p>Controls = 19 ‘healthy’ individuals</p>	Performance on the Act-and-react test system (ART 90)	<p>Overall performance: Cases < Controls*</p> <p>Cases – Typ < Cases – Atyp*</p> <p>PVT – reaction time: Cases – Typical > Cases – Atypical * Cases – Atypical > Controls***</p> <p>PVT – tracking performance: Cases – Typical = Cases – Atypical Cases < Controls **</p> <p>TT15 – Number of correct numbers: Cases – Typical = Cases – Atypical Cases < Controls **</p> <p>Q1: Cases < Controls**</p> <p>RST3 – Number of correct responses: Cases – Typical < Cases – Atypical** Cases – Atypical < Controls***</p> <p>RST3 – Number of correct responses in time allowed: Cases – Typical < Cases – Atypical* Cases – Atypical < Controls**</p> <p>RST3 – Number of omissions: Cases – Typical > Cases – Atypical** Cases – Atypical > Controls**</p>
Törnros, Vikander, Ahlner & Jönsson (2001)	<p>Cases = 20 outpatients taking BZ</p> <p>Controls = 20 p who were individually aged and sex matched</p>	<p>driving simulator:</p> <ul style="list-style-type: none"> - brake reaction time, - lateral position variation, - speed variation. <p>lab tests:</p>	<p>brake reaction time: BZ = C</p> <p>lateral position variation: BZ = C</p> <p>speed variation: BZ > C **</p> <p>simple reaction time: BZ < C*</p> <p>short-term memory tests: BZ < C*</p>

Study: Author/date	Methods	Outcome Measure of Risk	Results
		<ul style="list-style-type: none"> - simple reaction time, - choice reaction time, and - short-term memory. 	choice reaction time: BZ = C
Ray, Fought & Decker (1992)	retrospective cohort study n = 16,262 drivers Cases = 65-84 yr-olds taking AD or BZ Controls = 65-84 yr- no drug use	<ul style="list-style-type: none"> - drivers licence files, - police reports of injurious crashes - drug use 	AD RR inj crash : 2.2* RR taking > 1 AD: 9.0* RR taking highest dose: 5.5* BZ RR of inj crash inv: 1.5* RR taking > 1 BZ: 4.8 RR taking highest dose: 2.4*
van Laar, van Willgenburg and Volkerts (1995)	double blind, cross-over, placebo controlled methodology - Cases = 24 healthy participants given nefazodone (SSRI) and imipramine (cyclic antidepressant).	Driving and psychomotor performance	Driving: TCA: lateral position impair* SSRI: no impair Psychomotor: TCA: minor impair* SSRI: no impair
Vernon et al. (2002)	Psychiatric/emotional disturbances Unrestricted = 6481 Restricted = 45	Questionnaire data on medical conditions	Not restricted RR all crashes: 1.67* RR at-fault crashes: 2.1 * RR citations: 1.30* Restricted RR all crashes: 1.87* RR at-fault crashes: 2.89* RR citations: 0.84
Wingen et al. (2006)	Cases = 24 individuals with unipolar disorder with an active depressive episode taking SSRI Controls = 24 age, gender and years of driving experience matched healthy volunteers.	Performance on on-road test Cognitive measures	SD of lat pos: Cases > Controls** Time to speed adapt: Cases > Controls* Brake reaction time: Case = Controls Speed : Case = Control Headway: Case = Controls Crit flic thres freq: Cases < Controls** Other cog measures: Case = Controls

Antipsychotics (AP), Antidepressants (AD) and Benzodiazepines (BZ)

Approaches to management

Assessing fitness to drive

As summarised in Table 33, drivers with psychiatric illnesses are fit to drive if their condition is stable (i.e., not in the acute phase), the risk of functional impairments due to symptoms is assessed as minimal, they are deemed compliant and medication side-effects are minimal. Most jurisdictions also recommend periodic reviews (6-12 months). The American guidelines for fitness to drive state that a restricted licence may be issued if the prescribed medication minimally impairs psychomotor functioning. For example, speed restrictions may apply.

Self-regulation

Currently, there is no available information regarding the extent to which people with psychiatric illnesses adopt self-regulatory practices. The issue of self-regulation for individuals with a psychiatric illness may be quite complicated, due to the fact that some individuals diagnosed with psychiatric illnesses such as schizophrenia, psychosis and depression are generally unaware of having an illness (Amador, Flaum, Andreasen, Strauss, Yale, Clark, & Gorman, 1994). This is likely to result in limited insight as to how their illness may affect their driving ability.

Table 33 Private licensing guidelines for drivers with psychiatric illnesses

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)**</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Anxiety or Depression	If a physician believes that a patient's judgement or psychomotor activity has been severely affected by their emotional state the patient should be advised not to drive until sufficiently recovered. The possible side effects of drugs should be considered when making this decision.	<p><i>Severe Depression or Anxiety</i> May not hold an unconditional licence if the condition is severe, or taking medication that impairs driving in the long-term.</p> <p>A conditional licence may be issued if the condition is under control & the side effects of medication minimally interfere with driving.</p> <p>Subject to periodic review.</p>	<p><i>Without Significant Symptoms:</i> May continue to drive.</p> <p>If medication is taken which adversely affects driving ability (particularly the older tri-cyclic antidepressants), driving is to cease.</p> <p>No need to notify DVLA.</p> <p><i>Severe anxiety or depression (including significant memory or concentration problems, agitation or behavioural disturbances):</i> Driving to cease until medical evaluation is undertaken.</p> <p>Driving may resume after a period of stability.</p> <p>Of special concern are</p>	<p>Unrestricted licence may be issued if the condition is stable without medication, or with medication that does not impair alertness or psychomotor functioning. A restricted licence may be issued if the medication minimally impairs psychomotor functioning.</p> <p>Yearly or six-monthly review required.</p> <p>When determining fitness to drive, should consider prior accident and violation records which are a more valid predictor of crash risk than psychiatric diagnosis.</p> <p>Impairments that may increase crash risk include:</p> <ol style="list-style-type: none"> 1. Impulsiveness, 	<p><i>Mental Disorder that May Impair Driving:</i> Assessment is to be based on the impact that the disorder has on behaviour, mood & psychomotor functioning. Other factors to consider are the insight the person has into the illness & medication (side effects & effectiveness).</p> <p>It is recommended that the person refrains from driving during periods of suicide ideation.</p> <p><i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive.</p> <p>Driving may resume if:</p> <ol style="list-style-type: none"> 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. 3. Person has undergone an observation period of 6 months. 4. Psychiatric assessment 	<p><i>Condition stable & minimal risk of symptom manifestation:</i> Licence may be retained.</p> <p><i>Serious disorder:</i> Licence denial if the disorder results in serious disturbances of behaviour, judgement or adaptability.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)**</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
			those people who might try to commit suicide whilst driving.	explosive anger and impaired social judgement 2. Inattentiveness Suicidality, perceptual distortion, or irrationality	is required prior to resumption of driving.	
Manic-Depression (Bi-polar Disorder)	If a physician believes that a patient's judgement or psychomotor activity has been severely affected by their emotional state the patient should be advised not to drive until sufficiently recovered. The possible side effects of drugs should be considered when making this decision.	<i>Acute phase of illness:</i> Desist from driving. May not hold an unconditional licence if the condition is severe, or taking medication that impairs driving in the long-term. A conditional licence may be issued if the condition is under control & the side effects of medication minimally interfere with driving. Subject to periodic review.	<i>Acute phase of illness:</i> Desist from driving. Re-licensing may occur after an isolated episode if person is: 1. Well & has been stable for a minimum of 3 months. 2. Has insight into their illness. 3. Compliant with treatment. 4. Has no side-effects from medication. 5. Receives a favourable psychiatric report. <i>Repeated Mood Swings:</i> (Defined as more than 4 swings in the previous year). Re-licensing may occur if person is:	<i>Acute phase of illness:</i> No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.	<i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive. Driving may resume if: 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. 3. Person has undergone an observation period of 6 months. 4. Psychiatric assessment is required prior to resumption of driving.	Licence denial or revocation in cases of serious disturbance. May continue to drive if the condition is stable & the risk of symptoms assessed as minimal. Desist from driving for 1 year following a relapse of the illness. This period may be reduced if the relapse was into a depressive phase.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)**</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
			<p>1. Well & has been stable for a minimum of 6 months. AND complies with conditions 2, 3, 4 & 5 as listed above.</p> <p>If the condition is associated with substance misuse, continuing misuse is not acceptable for licensing.</p>			
Chronic Schizophrenia	Not specifically addressed.	<p><i>Acute phase of illness:</i> Desist from driving.</p> <p>May not hold an unconditional licence if the condition is severe, or taking medication that impairs driving in the long-term.</p> <p>A conditional licence may be issued if the condition is under control & the side effects of medication minimally interfere with driving.</p>	May drive if behaviour is stable for 3 months, driver complies with treatment, no adverse effects from medication & subject to specialist advice.	<p><i>Acute phase of illness:</i> No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.</p>	<p><i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive.</p> <p>Driving may resume if:</p> <ol style="list-style-type: none"> 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. 3. Person has undergone an observation period of 6 months. 4. Psychiatric assessment is required prior to resumption of driving. 	<p>Licence denial or revocation in cases of serious disturbance.</p> <p>May continue to drive if the condition is stable & the risk of symptoms assessed as minimal.</p> <p>Particular attention is to be given to the existence of delusions, hallucinations, disorganised behaviour, anger & rage outbursts, alcohol/substance abuse & any residual problems after an active phase of the illness.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)**</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
		Subject to periodic review.				Desist from driving for 1 year following an active phase of the illness.
Psychotic Disorders	<p>Fit to drive if: Condition is stable for a six month period subject to a physician report</p> <p>The patient compliance with medication is monitored</p> <p><i>Acute phase of illness:</i> Desist from driving. May return to driving once acute episode has settled and is demonstrated by a psychiatrists report.</p>	<p><i>Acute phase of illness:</i> Desist from driving.</p> <p>May not hold an unconditional licence if the condition is severe, or taking medication that impairs driving in the long-term.</p> <p>A conditional licence may be issued if the condition is under control & the side effects of medication minimally interfere with driving.</p> <p>Subject to periodic review.</p>	<p><i>Acute phase of illness:</i> No driving.</p> <p>Re-licensed if condition is stable for 3 months & complies with treatment & no adverse effects from medication & subject to specialist advice.</p> <p>Even when insight is limited, licensing is not necessarily precluded. However, drivers whose psychotic symptoms relate to other road users may be particularly dangerous.</p>	<p><i>Acute phase of illness:</i> No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.</p>	<p><i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive.</p> <p>Driving may resume if:</p> <ol style="list-style-type: none"> 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. 3. Person has undergone an observation period of 6 months. 4. Psychiatric assessment is required prior to resumption of driving. 	<p>Licence denial or revocation in cases of serious disturbance.</p> <p>May continue to drive if the condition is stable & the risk of symptoms assessed as minimal.</p> <p>Particular attention is to be given to the existence of delusions, hallucinations, disorganised behaviour, anger & rage outbursts, alcohol/substance abuse & any residual problems after an active phase of the illness.</p> <p>Desist from driving for 1 year following an active phase of the illness.</p>
Personality Disorders	Should not be allowed to drive without careful consideration and psychiatric assessment if the person presents	People with personality disorders frequently exhibit a disregard for social values & the law & may have a history of aggressive & erratic	If the driver is likely to be a danger behind the wheel, the licence would be revoked or not granted. If medical advice confirms that the behavioural disturbance	Unrestricted licence may be issued if the condition is stable without medication, or with medication that does not impair alertness or	<i>Mental Disorder that May Impair Driving:</i> Assessment is to be based on the impact that the disorder has on behaviour, mood & psychomotor functioning. Other factors	<p>Licence denial or revocation in cases of serious disturbance.</p> <p>May continue to drive if the condition is stable & the risk of symptoms</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)**</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
	with any of the following: disregard for social values, history of erratic, aggressive or irresponsible behavior which may include repeated violations.	behaviour. Psychiatric, legal & administrative assistance may be required with driver licensing. A conditional licence may be issued if: 1. The illness is controlled. 2. Medication side-effects are minimal. Subject to periodic review.	is not likely to impact upon driving or road safety, then licensing may be permitted.,	psychomotor functioning. A restricted licence may be issued if the medication minimally impairs psychomotor functioning. Yearly or six-monthly review required. When determining fitness to drive, should consider prior accident and violation records which are a more valid predictor of crash risk than psychiatric diagnosis. Impairments that may increase crash risk include: 1. Impulsiveness, explosive anger and impaired social judgement 2. Inattentiveness 3. Suicidality, perceptual distortion, or irrationality <i>Acute phase of illness:</i>	to consider are the insight the person has into the illness & medication (side effects & effectiveness). It is recommended that the person refrains from driving during periods of suicide ideation. <i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive. Driving may resume if: 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. 3. Person has undergone an observation period of 6 months. 4. Psychiatric assessment is required prior to resumption of driving.	assessed as minimal. Particular attention is to be given to anti-social & borderline personality disorders.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)**</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
				No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.		

** No distinction is made in this manual between types of psychiatric disorders. Distinction is made in terms of functional ability.

3.9.6 ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

In the last decade, there has been an emerging interest in the broad learning and behavioural ramifications of ADHD and specifically in the road safety implications of the disorder. Researchers have recently begun to recognise that individuals with specific childhood disorders such as Attention-Deficit Hyperactivity Disorder (ADHD) may actually be at a high risk for motor vehicle crashes due to symptoms and functional impairment which continue into adolescence and young adulthood (Barkley, Murphy & Kwasnik, 1996). This mirrors the impact on adult function (de Graaf et al. 2008, Davidson et al. 2008, Rostain, 2008).

Guidelines for management of ADHD in Australia have been drawn up nationally in 1997 and 2009, under the auspices of the National Health and Medical Research Council, which is yet to make full approval. These are based on existing international evidence and consensus, with wide professional and public consultation in Australia. These documents provide comprehensive and rigorous modern sources of extensive references regarding ADHD, of interest to readers of this chapter. They specifically address ADHD and driving (NHMRC, 2009, pages 180-182).

Definition of ADHD

ADHD is a disruptive childhood behaviour disorder, which is characterised by developmentally inappropriate degrees of inattention and/or hyperactivity-impulsivity (American Psychiatric Association, (APA 2000), Pliska, 2007).

According to the DSM-IV⁶, there are three types of ADHD according to which symptoms are strongest in the individual. These types are described below:

Predominantly Inattentive Type

It is hard for the individual to organise or finish a task, to pay attention to details, or to follow instructions or conversations. The person is easily distracted or forgets details of daily routines.

Predominantly Hyperactive-Impulsive Type

The individual fidgets and talks a lot. It is hard to sit still for long (e.g., for a meal or while doing homework). Smaller children may run, jump or climb constantly. The individual feels restless and has trouble with impulsivity. Someone who is impulsive may interrupt others a lot, grab things from people, or speak at inappropriate times. It is hard for the person to wait their turn or listen to directions. A person with impulsiveness may have more crashes and injuries than others.

Combined Type

This type involves symptoms of the above two types which are equally predominant in the person.

⁶ DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition), published in 1994 preceded the DSM-III R published in 1987.

In each case, the symptoms must be present for at least six months to a degree that is maladaptive and inconsistent with developmental level. In addition, some symptoms must be present prior to age seven, and in two or more settings (e.g., at school, work and home). There must be clear evidence of clinically significant impairment in social, academic or occupational functioning, and the impairment cannot be caused by other disorders such as anxiety, psychosis or pervasive developmental disorder (APA, 2000).

Once believed to be only a childhood syndrome, ADHD is now generally regarded as a life span disorder with a high risk for continued symptoms into adolescence and young adulthood (Barkley, et al., 1996). The severity can diminish with age, and previously it was believed that most adults no longer cross the threshold of the disorder. However, this is now shown to be incorrect and is dependent upon whether the individual or others report the severity of the impairment.

Prevalence of ADHD

ADHD prevalence estimates are rare in the published literature, especially in relation to DSM-IV (APA, 2000) and ICD-10 criteria (WHO, 1992). The reported prevalence of ADHD in school age children varies from 3-7% depending on the criteria used, with males being over represented, on average, 3:1 (NHMRC, 2009, Pliska, 2007). Currently, approximately 1.6 to 2 million people are estimated to have this disorder (APA, 2000). The incidence is thought to diminish with age however, and the prevalence by the early 20s have been quoted in some studies as less than 5% of that in the previous decade and modern cross-sectional studies suggest that ADHD occurs in 4% of adults (Fayard et al. 2007).

Functional impairments associated with ADHD relevant to driving

In the past decade, researchers have begun to recognise that ADHD is not simply a problem with paying attention, but rather is a developmental impairment of a complex range of executive functions (EFs) (Barkley et al., 1996). The term “executive function” is relatively recent in origin, and is generally regarded as encompassing skills necessary for goal-directed behaviour (Shallice, 1982; Stuss & Benson, 1986). The outcome studies of cohorts from 20 years ago, mainly followed individuals with hyperactivity. Studies did not necessarily separate those with inattentive ADHD or those at high risk for conduct disorder. The results can not be easily generalised to all individuals recently diagnosed with ADHD. This is discussed below.

Individuals diagnosed with ADHD often display the following impairments (Barkley et al., 1996):

- difficulty in planning, organising and prioritising tasks;
- difficulty estimating time;
- difficulty focussing, sustaining focus, shifting focus from one task to another, or filtering out distractions;
- an inability to persist on a task in the face of temptation, frustration, or interruption;
- difficulty managing frustration and modulating emotions

- impaired processing speed;
- difficulty utilising working memory and accessing recall;
- difficulty monitoring and regulating self-action or impulsivity.

The impairments associated with ADHD are considered to be very important for the tactical operations of a motor vehicle in traffic, and therefore could contribute to an increased crash risk (Barkley et al., 1996; Parker West, Stradling & Manstead, 1995; Pless, Taylor & Arseneault, 1995).

Pre-May 2003: Relationship between ADHD and road safety outcomes

A growing number of studies are beginning to examine the longer-term outcomes of children with attentional difficulties such as ADHD (for review see Barkley, 1998). One long-term outcome that has received increasing research attention concerns the driving behaviour of adolescents and young adults with earlier attentional difficulties. Table 30 shows a summary of the findings of studies that have investigated the relationship between ADHD and rates of crashes, citations and driving performance.

Crashes

Barkley, Murphy, DuPaul and Bush (2002) compared the driving ability of 105 adults with ADHD with 64 participants without ADHD. Participants were aged between 17 and 28 years and were screened for comorbid physical and psychiatric illnesses through a clinical diagnostic interview (SCID). The study compared driving citations and driving performance of participants (see below for details) as well as crash records of the two groups. Based on official driving records of crash events, participants with ADHD were involved in more vehicular crashes as the driver ($p = 0.06$), being more at fault ($p = 0.08$), and having more severe crashes as reflected in the cost of damage ($p = 0.05$). One obvious limitation of this study is that the authors did not control for driver exposure, and make an assumption that the two groups drive similar distances which may not be the case.

Woodward, Fergusson and Horwood (2000) conducted a study to investigate the relationship between attentional difficulties at age 13 and a range of adverse driving outcomes at age 21 years. Data were gathered over a 21-year longitudinal study of an unselected birth cohort of 941 New Zealand children. Data collection included the following: parent and teacher measures of attentional difficulties at age 13 years; number of motor vehicle crash involvement (both injurious and non-injurious) from age 18-21 years; history of driving and driving from age 18 to 21 years (examples of this included drunk/over the legal limit, seriously intoxicated, arrested for DUI); and the number of traffic violations from age 18-21. The authors also investigated the extent to which the relationship between attentional difficulties at age 13 and later adverse outcomes could be explained by the effects of confounding factors such as gender, conduct problems, IQ, socio-familial background, number of months participants had held their licence, and the total distance driven by the participant (in kilometres). Participants were classified into five groups according to the extent of parent and teacher reported difficulties at age 13. The authors reported that after controlling for key confounding factors (gender, distance driven, length of time since licence obtained, and co-morbid conduct disorders), increasing levels of attentional problems were associated with increases in participants' subsequent risks of involvement in a motor vehicle crash

causing injury ($p < 0.001$). This relationship held even after making appropriate adjustments for multiple statistical comparisons. The profile of those at greatest risk of later driving problems identified in this study was that of a young male, with a conduct disorder and significant attentional problems who, despite limited driving experience, spends a lot of time on the road. The authors argue that the use of a large, general population sample avoids many of the problems associated with the use of small and unrepresentative sample of young adults with ADHD. However, one limitation of this study was that the authors did not report whether participants were taking any psychotropic medication (such as Ritalin). Another serious limitation is that cases were not actually diagnosed with ADHD using a standardised measure such as the DSM-IV. Therefore, it is difficult to generalise the findings of this study to other studies in this area.

In an earlier study, Barkley, Murphy and Kwasnik (1996) investigated the motor vehicle driving competencies and risks in adolescents and young adults with ADHD. Participants comprised 25 young adults aged 17 to 30 years old who met the DSM-IV criteria for a diagnosis of ADHD. The control group comprised 23 young adults without ADHD. The two groups were equated for age, gender, and educational level, and both groups were screened for other psychiatric illnesses, epilepsy, serious sensory or motor impairments. Five out of 25 participants were taking psychotropic medication (4 = stimulants, 1 = antidepressant). The participants taking stimulants were requested to refrain from taking their medication at least 24 hours before testing because stimulant medication has been shown to improve sustained attention, inhibition, motor speed and co-ordination in individuals with ADHD (see next section) Each participant was interviewed about their driving history, which included questions regarding how long they have had their licence, average amount of driving per week, number and type of traffic violations (see below), number of crashes while driving (both at-fault and not), and whether crashes were associated with bodily injuries or not. Official DMV records were also obtained for number and type of violations and number of crashes. Driving performance measures were also recorded and are reported below. The two groups did not significantly differ in the length of time they had been driving or the average distance they estimated they drove a typical week. Participants with ADHD were found to be more likely to be involved in crashes ($p = 0.08$), and their crashes were more likely to cause bodily harm than participants without ADHD. Inspection of the official driving records corroborated these self-reported outcomes.

Citations

In the study described above by Barkley and colleagues (2002), citation rates of drivers with ADHD were compared with those without ADHD. The results showed that in addition to an elevated crash risk, individuals with ADHD reported significantly more traffic citations than the control group ($p < 0.05$), with most of these corroborated in the official DMV records. Specifically, participants with ADHD had more than twice the number of driving citations, particularly for speeding ($n = 88$) than controls ($n = 44$, $p = 0.06$), more licence suspensions/revocations ($n = 105$) compared to controls ($n = 64$, $p < 0.01$). These findings confirmed earlier results reported by the same authors (Barkley et al., 1996; see above) showing that participants with ADHD were nearly twice as likely to be cited for speeding ($p < 0.07$) and more than twice as likely to have had their licence suspended ($p < 0.05$). Inspection of the official driving records corroborated these self-reported outcomes.

In their longitudinal study of young drivers Woodward et al. (2000) (see above for details) also examined the relationship between attentional problems and traffic citations (for example driving without a driver's licence, driving without vehicle warrant of fitness or registration, speeding, overtaking illegally, running red lights, reckless driving). After controlling for key confounding factors (gender, distance driven, length of time since licence obtained, and co-morbid conduct disorders), increasing levels of attentional problems were associated with increases in participants' subsequent risks driving without a licence ($p < 0.05$) and general traffic violations ($p < 0.05$). However, once adjustments were made for the large number of statistical comparisons, these relationships were found to be not significant.

Nada-Raja, Langley, McGee, Williams, Begg, and Reeder (1997) investigated the relationship between the symptoms of ADHD, conduct disorder, anxiety and depression at the age of 15 years on the rates of driving offences and involvement in motor vehicle crashes between the ages of 15 and 18 years. The sample comprised 916 participants from a New Zealand birth cohort. Specifically, at age 15, participants' mental health was assessed using a modified version of the Diagnostic Interview Schedule for Children (DISC-C). Parent reports and other questionnaires on family background, were used to confirm adolescent report of disorder. The sample was divided into four groups. The first group comprised participants who met the DSM-III criteria for ADHD ($n = 101$), the second group comprised participants who met the DSM-III criteria for conduct or oppositional disorder ($n = 46$), the third group comprised those who met DSM-III criteria for anxious or depressive disorders ($n = 85$) and the fourth group comprised participants who did not meet the DSM-III criteria for any of the DSM disorders assessed in this study ($n = 684$). Official motor vehicle driving offences for each participant were obtained from the Land Transport Safety Authority (LTSA). Finally, participants provided information on their own driving behaviour and offences for the 12-month period preceding assessment. The authors reported that a significantly greater proportion of young women with high levels of ADHD symptoms were involved in one or more driving offences (11%) than participants with conduct disorder group (7%) and no disorder group (2%, $p < 0.05$). In contrast, a greater proportion of males in the conduct disorder group reported that they had committed one or more driving offences at age 18 than the rest of the sample ($p < 0.05$). The authors concluded that adolescents with a history of ADHD or conduct disorder are significantly more likely to commit traffic offences.

Driving Performance

In addition to examining crash and citation rates, Barkley and colleagues (2002) (see above), also compared driving performance of adults with and without ADHD. Participants were administered a battery of executive function tasks and their driving performance was measured using the Elemental Driving Simulator (EDS, Gianutsos, 1994). The EDS is a computer software program employing a personal computer, monitor and a driving console, on which participants were scored on seven items: steering control; response time; field responding; adjusting to change; consistency; self-control; and self-appraisal. Finally, the participants' driving knowledge and rapid decision making abilities were measured using the Driver Performance Analysis System (DPAS; Weaver, 1990). Performance of the ADHD group was comparable to the control group on basic visual discrimination and reaction time tasks, which the authors concluded suggests no perceptual impairments that might affect driving. In contrast, participants with ADHD manifested some limitations in basic cognitive functions

related to driving, such as attention. They made more errors during the visual reaction task when the rules were reversed, implying difficulties in rule-governed behaviour ($p = 0.05$). The ADHD group also scored lower scores on a test of driving rules and decision-making but not on the driving simulation (EDS) task. Several executive functions, inattention, interference control and inhibition, were significantly yet modestly related to crash frequency and total traffic violations after controlling for severity of ADHD. Finally the authors reported that driving difficulties were not a function of co-morbid oppositional defiant disorder, depression, anxiety, or frequency of alcohol or illegal drug use. One of the limitations of this study was that the assessor was not blinded to the group membership of the participants. However the authors argue that since most of the tests were computer administered, or obtained from official records then this may not have significantly affected the results.

In their earlier study, Barkley et al. (1996; see above for details) also compared driving performance of adults with and without ADHD using the EDS computerised simulated driving test (Giannutsos, 1994). Participants also rated their own “real world” driving habits (e.g., braking properly at intersections, driving within the speed limit) using the Driving Performance Rating Scale, where higher scores reflected better driving behaviour. This rating was compared with ratings made by their parent or someone who knew them well. In addition, participants completed the DPAS test to assess driving knowledge regarding high risk driving situations. Results of the EDS driving performance task showed that participants with ADHD had significantly more scrapes and crashes than controls on least complex driving trial, but not on more complicated trials. There was no significant difference between the two groups on the DPAS test of driving knowledge and traffic procedures. Participants with ADHD were rated as using significantly poorer driving habits, by both their own reports and those of others, than were members of the control group. The authors suggested that driving difficulties in ADHD are more likely to be the result of driving performance, specifically motor control impairments, than driving knowledge.

Co-morbidity and Risk

One of the difficulties in diagnosing ADHD is that it is often accompanied by other disorders such as Conduct Disorder and Oppositional Defiant Disorders. These two disorders are the other disruptive behaviour disorders described in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV). It is clear that there is a large overlap between ADHD and these disruptive behaviour disorders. The symptoms may include refusing to comply with commands from adults such as parents, teachers, and coaches; doing the opposite of what is expected; disrupting the play of others; being verbally or physically aggressive; being destructive, such as breaking objects that do not belong to the child; lying; stealing; being truant; and committing other forms of delinquent behaviour as the child gets older. Oppositional-noncompliant behaviour occurs early in the course of ADHD if it is going to occur at all. It may be a forerunner of a later diagnosis of conduct disorder and antisocial personality disorder as the child matures into adolescence and adult life. The presence of aggression and conduct symptomatology and of oppositional- noncompliant behaviour is a predictor of negative outcome, primarily the development of antisocial spectrum disorders in later adult life among children with ADHD.

Only one study was found that addressed the question of comorbidity, ADHD and driver. The study was one of the earliest studies to examine driving difficulties in

adolescents and young adults with ADHD (Barkley, Guevremont, Anastopoulos, DuPaul & Shelton, 1993). Participants were 35 adolescents and young adults diagnosed with ADHD and 36 control participants without ADHD, aged between 16 and 24 years of age, all of whom were licensed drivers. Parents of the participants were mailed a survey, which asked them to rate their child's current symptoms of ADHD, oppositional defiant disorder and conduct disorder. In addition, parents were asked to rate their child's driving behaviour and to report any negative driving outcomes. The authors reported that significantly more participants with ADHD had driven a car illegally without having a licence (37%) than the control participants (11%, $p < 0.05$). More participants with ADHD had also had their licences revoked or suspended (23%) than the control participants (0%, $p = 0.051$). Significantly more participants with ADHD had experienced multiple crashes (2+) as the driver than control participants ($p < 0.05$). The groups did not differ in the number of injurious crashes they had been in as the driver, but there was a trend ($p < .061$) for the ADHD group to have had more such injuries in the crashes in which they were involved. Significantly more subjects with ADHD had been a driver in a crash in which they were at fault (49%) than control participants (11%, $p < 0.01$). Significantly more participants with ADHD had had a traffic citation (77%) than the control participants (47%, $p < 0.05$). Finally, participants with ADHD were more likely to be using less sound driving habits in their current driving performance (40%) compared to control participants (11%, $p < 0.001$).

Of particular interest in Barkley et al.'s study, was the finding that these negative outcomes were further increased by the degree of co-morbid oppositional and conduct problems demonstrated by the participants. For example, the combination of ODD and CD symptoms in the equation accounted for more than 37% of the variance in the driving skill ratings. The authors concluded that participants with ADHD, and especially when associated with ODD/CD symptoms is associated with substantially increased risks for driving teenagers and young adults. The generalisability of these findings is limited by several factors including a reliance on parental reports for driving-related outcomes, use of a predominantly male sample, no measure of exposure to driving, and a brief window of driving history.

Treatment of ADHD and road safety outcomes

Psychostimulant medications are often used to control the symptoms of ADHD. The most commonly prescribed medication used to treat ADHD is Methylphenidate (usually known as Ritalin). Preparations available in other countries are not available in Australia. For example, dexamphetamine as Adderall and pemoline (Cylert), are no longer used. In Australia, Methylphenidate only become as equally available as dexamphetamine on the Pharmaceutical Benefits Scheme in 2006, so for decades in Australia dexamphetamine was at least as equally prescribed (Salmalinen, 2002,2004).

In 2000, Cox, Merkel, Kovatchev and Seward conducted a double-blind (Ritalin vs. placebo) cross-over, counter-balanced design to determine the effect of stimulant medication on driving performance of young adults with ADHD. Specifically the authors compared the driving performance of seven young male adults with a diagnosis of ADHD (according the DSM-III criteria) with six young male adults without a diagnosis of ADHD. Participants were excluded if they had any other psychiatric illnesses as assessed by the Structured Clinical Interview for Diagnosis (SCID). In addition, participants with ADHD had to have previously taken Ritalin, but could not be taking any medication within the past six months. Participants with ADHD reported that they had more crashes ($n = 2.7$) in their driving careers compared to participants without

ADHD ($n = 0.8$, $p < 0.05$) and more citations (2.6 vs. 1.5, $p = 0.06$). However these findings were not compared to official driving records.

Driving performance was obtained over two drives using a high-fidelity driving simulator. Participants rated their driving performance after both drives. Participants with ADHD drove worse on the simulator under placebo condition compared to participants without ADHD ($t = 2.4$, $p < 0.05$) however demonstrated a significant improvement in their driving performance under the Ritalin condition ($t = 1.68$, $p = 0.05$). In addition, participants with ADHD rated their driving performance lower in the placebo condition ($M = 3.0$) than participants without ADHD ($M = 3.9$, $p = 0.05$). On the other hand, participants with ADHD rated their driving performance better in the Ritalin condition ($M = 3.5$, $p = 0.07$).

The authors concluded that individuals with ADHD should have the therapeutic benefit of a stimulant medication while operating a vehicle. Limitations of this study include the fact that this study was a short-term clinically controlled observation over a very short period of time with a single exposure to stimulants, with an extremely small sample, with only male participants and that they did not control for driving exposure.

Another issue is that drivers, especially commercial drivers, treated with stimulants for ADHD can be tempted to abuse their medication such as taking them in excessive doses or in conjunction with other illicit drugs and/or alcohol (M. Odell, personal communication, July 07, 2003).

Post-May 2003: Relationship between ADHD and road safety outcomes

During the review period from May 2003, only one study was identified which addressed the relationship between ADHD and crashes. In contrast, there has been a growing interest in research investigating driving performance measures and ADHD. A total of 8 papers on this topic were identified. Additionally, 5 studies were identified for this review period which addressed treatment of ADHD including one examining crashes.

Crashes

Thompson et al. (2007) studied driving outcomes in a group of 355 adolescents and young adults as part of the Pittsburgh ADHD Longitudinal Study (PALS) and 240 controls. Subjects had been part of the study since the ages of 5 to 17 and all met the diagnostic criteria of the DSM-III-R or IV. All subjects were interviewed according to a standard protocol using the Young Adult Driving Questionnaire (Donovan et al 1983). The study relied solely on self reporting which was acknowledged by the investigators as a limitation.

The findings showed significant differences between the study and control groups in having driven unlicensed (OR = 3.97), having ever received a ticket (1.46), having a licence suspension (1.65) and having been directed to go to traffic school (4.45). Smaller associations were found for the numbers of crashes. No differences were found for risky driving or alcohol impaired driving.

The authors concluded that the increased risks for citations and crashes associated with ADHD were small especially for the group with hyperactivity and impulsivity. A limitation of the study is that a proportion of participants had had some form of

specialist driving education (traffic school). No exposure measure was used in analysis of risk.

Citations

See above for study citing multiple road safety outcomes by Thompson (2007) and below for study by Fischer et al. (2007). No other papers were found dealing solely with citations.

Driving Performance

Reimer et al. (2005) used the Driving Behaviour Questionnaire (DBQ; Reason et al., 1990) to explore the relationship between age, gender and ADHD status on error, lapse and violation report scores. The subjects were 83 drivers between 16 and 55 years (average age 35 for ADHD, 30 for controls) who were participating in an unrelated driving simulation study. Forty-five of these satisfied the DSM-IV criteria for ADHD and had IQs over 80. Forty-five were males and 38, females. All subjects were interviewed by trained psychometricians and were administered a battery of psychological tests including the DBQ.

Drivers with ADHD generally showed significantly increased scores for most of the risky and aggressive driving traits in the questionnaire. Gender was a significant predictor of violations and the incidence of all three measures of violations, driving lapses and driving errors decreased with age.

This study showed that the effects of age and gender on crash risk that are well known for the driving population in general also apply to drivers with ADHD. The authors speculate that pharmacological treatments for the condition may not be as effective as subjects age since their driving performance and crash risk may be already falling to levels similar to that of the general driving population.

In a more recent study, the same research group (Reimer et al., 2007) investigated the difference between 25 adult drivers with ADHD and 23 controls in a simulator study designed to assess the effects of fatigue. Subjects had to have ADHD according to the DSM-IV criteria, an IQ over 80, have a valid driver licence and no psychiatric comorbidities. They were asked to abstain from stimulant medication for the experiment. Seven participants (5 ADHD, 2 controls) withdrew because of simulator sickness. The study was conducted in a driving simulator where drivers were required to avoid cyber animals on the road at two times after starting driving. The initial presentation was soon after familiarisation and the second after an interval of relatively boring driving. The trials were conducted at different times of the day to include circadian effects.

Prior to the simulation the ADHD drivers reported the expected increased rate of crashes compared to controls. In the first simulated drive, there were no collisions with cyber animals but the ADHD group had an increased rate of collision at the second presentation. As expected, both groups had an increased rate of collisions in the early morning and late evening compared to the early and later afternoon. The result suggested that drivers with untreated ADHD become fatigued more quickly than controls which could increase their risk if crashing in monotonous fatiguing driving situations..

An interesting study investigating the accuracy of self evaluation by ADHD drivers was reported by Knouse et al (2005). This study compared ADHD drivers using self evaluation, a simulator study and a Driving History Survey. The Driving History Survey had previously been validated against driving records from the DMV (Barkley & Murphy 2002). Forty-four drivers with ADHD who had been recruited for another study participated. They were selected using the DSM-IV criteria and the Adult ADHD Rating Scale and subjected to a series of psychological screening tests. There were 44 controls from the same general community. All participants undertook a brief (12 minute) drive in a simulator and completed several self reporting assessment instruments.

The results showed that adults with ADHD performed worse on the naturalistic measure of driving skill but tended to overestimate their driving abilities. This did not extend so strongly to the simulated driving task but this was a brief drive not necessarily able to provide a realistic evaluation of true driving ability. A weakness of this study however was the reliance on a previously validated instrument rather than true driving histories for the comparison. Despite this, the study showed that ADHD drivers, in common with the rest of the driving population, tend to overestimate their abilities on the road.

ADHD was one of the conditions investigated by the IMMORTAL project (Impaired Motorists, Methods of Roadside testing and Assessment for Licensing) coordinated by the University of Leeds. The ADHD study (Immortal, 2004) recruited 17 male subjects aged from 19 to 47 years and 28 healthy controls of similar ages. The study group met the DSM-IV criteria for ADHD and all had a positive response to stimulant therapy for at least three months. All participants held a valid driver licence for more than six months. All subjects were interviewed by an experienced clinical psychologist to confirm the diagnosis and exclude other comorbidities.

Participants were asked to not take their regular medication for 24 hours prior to testing in a driving simulator and on the days of the study were randomised into a double blind comparison between 20 mg of methylphenidate or a placebo. Testing was performed on a SINTEF driving simulator with a mixed rural and village driving scenario.

The results of the study indicated that in general the un-medicated ADHD study group drove as well as, or even slightly better than, the control group. The lack of difference was seen both in rural and village driving. The report did not report any detailed differences in the medicated group other than the observation that an improvement would not be expected since the un-medicated group already drove so well. The study design did not allow for any prediction or estimate of crash risk in the study group.

The lack of difference between the ADHD and control groups was attributed to methodological problems with the simulator situation. This has been a problem with other studies which have found minimal difference between the performances of ADHD study groups compared to controls.

Weafer et al. (2008) used the common yardstick of alcohol impairment as a measure of the difference between an ADHD group and controls in two experiments using a driving simulator. In the first experiment fifteen ADHD drivers and 23 age matched controls participated. Thirteen of the ADHD subjects had their diagnoses confirmed by a review of their medical records, the other two were subjected to a screening procedure based on the DSM-IV criteria and the Adult ADHD rating scale. The simulator used was a computer screen based device with a simple country driving task. ADHD subjects were

tested once, in the absence of medication. The control group were tested twice, once with a zero blood alcohol concentration (BAC) and once after being given a dose of alcohol sufficient to raise their BAC to about 80 mg/dl (0.08%).

As expected there were significant differences between the sober and intoxicated control groups in deviation of lane position, steering rate (jerkiness) and driving speed variation. The ADHD group showed no significant difference to the intoxicated control in all three measures and significant differences to the sober controls only in the first two.

The second experiment was conducted to compare alcohol induced impairments between ADHD and control groups. Eight ADHD participants from the first experiment and eight aged matched controls were tested. Subjects were tested at zero BAC and with two doses of alcohol sufficient to raise the BAC to 0.05% and 0.08%. Subjects were assessed as in the first experiment and also on self rated intoxicated and fitness to drive. As expected there was a progressive impairment of all five measures as the BAC increased and that, perhaps paradoxically, the impairment was worse for the ADHD group and more so only at the lower dose of alcohol.

The authors provided an extensive discussion and concluded that the effects of alcohol on ADHD drivers is greater possibly because of its detrimental effect on divided attention in subjects whose attentional abilities are already impaired by their condition.

Richards et al. (2006) investigated whether increased anger is responsible for the adverse driving outcomes associated with ADHD. Fifty-six adults with a DSM-IV diagnosis of ADHD, a valid driver licence and an IQ over 80 completed a series of self reporting questionnaires measuring driving anger, its expression, thoughts and associated risk taking and aggressive behaviours. Two control groups of 106 people from the same communities and 432 college students were used. ADHD drivers consistently reported increased levels of anger, aggression and risk taking on a number of measures compared to controls. The differences were least when the ADHD group were compared to the younger more angry aggressive and risk taking college student groups. The authors concluded that the result partially supported their hypothesis that ADHD drivers experience more anger, express it in aggressive and less adaptive ways and experience more crashes. These findings are to be expected in a study of a group of drivers with a condition which increases activity and impulsiveness however it is hard to distinguish the effects of "pure" ADHD from common comorbidities that may also contribute to anger and aggression.

Fischer et al. (2007) followed a cohort of 147 children with ADHD into young adulthood (mean age 21.1 years) and compared their official driving records, driving instructor reports on a road test and simulated driving performance with a control group of 71. The children were followed for more than 13 years. The ADHD subjects had been recruited as children and all met the DSM-III-R criteria and had an IQ over 80; 91% of them were males. Only 8% were taking medication and the results did not differ when they were excluded from a supplementary analysis.

The study found that self reporting of adverse traffic events was higher than DMV records for both groups, probably because of the exclusion of minor incidents from official records. Using DMV data, the ADHD group had a significantly higher incidence of citations and licence suspensions. The cost of their crashes was also higher. Road testing and simulator results showed increased impulsive errors with an increased

crash and scrape rate in simulated driving. These findings were consistent with previous longitudinal studies of ADHD children followed into adulthood.

Treatment of ADHD and road safety outcomes

The effect of stimulant medication on driving performance of ADHD patients continues to be a subject of intense research.

Crashes

Sobanski et al (2008) conducted a total of 27 drivers with ADHD and 27 age and education matched controls to investigate the risks and impact of methylphenidate (MPH) treatment on ADHD drivers. The test subjects were recruited from an ADHD clinic and all satisfied the DSM-IV criteria for the condition. All were screened for psychiatric comorbidities and drug use. Assessment was by neuropsychological testing and an interview. Nine of the subjects were treated with sustained release MPH and drug levels monitored in blood.

The subjects with ADHD had 2.5 times higher crash rate than controls prior to the study with significant differences in at-fault, major & minor damaging and parking incidents. There were also significant differences in the rate of traffic violation point loss and speeding offences. MPH treatment resulted in statistically significant improvements in neuropsychological indices to do with visual orientation, tracking complex traffic situations and reaction behaviour. The study did not allow for assessment of crash risk or incidence associated with treatment.

Citations

No studies were found dealing solely with citations.

Driving Performance

Barkley et al. (2005) reviewed the effect of stimulants on driving performance. Barkley et al. (2005) studied the effects of two doses of the stimulant methylphenidate (MPH) on 56 patients with ADHD using a virtual reality driving simulator. Patients were selected on the basis of a clinical diagnosis of ADHD, age between 18 and 65 years, IQ greater than 80, adequate visual function for driving, and an absence of significant comorbidities. All subjects were interviewed by an experienced clinical psychologist who confirmed the diagnosis according to the DSM-IV criteria and administered several psychological screening tests including the Adult ADHD Rating Scale (Barkley & Murphy 1998). Eighty-seven percent of the subjects were diagnosed with the combined inattentive/hyperactivity type, 11% were predominantly inattentive, none were predominantly hyperactive/impulsive and 2% were ADHD Not Otherwise Specified. These proportions would now be very different in a population of adolescent and adult drivers.

Subjects were randomly assigned to 2 doses of MPH or placebo on a randomised double blind crossover basis. Dosage was timed so that the peak levels of medication occurred during the simulator tests. All participants underwent baseline testing with no medication as well as with all three combinations.

Performance was assessed using the simulator scores both on standard and obstacle type courses. A computerised continuous performance test was also conducted (Conners 1995). Somewhat surprisingly the findings indicated that there were significant improvements over the baseline for all three medication regimes including the placebo. These differences also extended to a reduction in simulator sickness for all three medication groups compared to the baseline. The authors speculate that this may have been due to a learning effect. There was some evidence for increased effectiveness of the higher dose of MPH and this was presumably the basis for the conclusion that the study endorsed the use of stimulant medication to reduce driving risk. Previous studies have demonstrated a linear dose response up to 0.9 mg/kg individual doses but real world prescribing is much less than this. Current guidelines in NSW limit the dose to 2mg/kg per day which is usually given in three immediate release doses per day or extended release doses with possible 'top ups' of immediate release to suit lifestyle and demand.

The hypothesis that driving deficits in ADHD are related to inattention and that stimulants improve driving performance by acting on inattention was the stimulus for an interesting pilot study into another method of overcoming attention in ADHD drivers. Cox et al (2006) compared the performance of ten adolescent drivers with ADHD on simulated driving using either a manual or automatic transmission crossover scenario. The subjects were selected from a group screened for a previous study and all satisfied the criteria for a diagnosis of ADHD. Nine were of the inattentive subtype and one was of the mixed inattentive/hyperactive subtype. All subjects were asked to refrain from medication on the days of the study. Objective driving performance was rated by means of the Impaired Driving Score (IDS, Cox et al 1998) and the subjects also rated their attentiveness on a 1-4 scale.

The results showed a small increase in self rated attentiveness for the manual transmission group and significant increases in IDS. This suggests that the requirement for attention to the relatively complex procedure of gear changes reduces inattention to other aspects of the driving task. This was an essentially qualitative study after which the authors suggested that a possible synergistic effect with stimulant medication may be worthy of future research.

The effectiveness of two different types of long acting stimulant therapy were compared in a study by Cox et al. (2005) and funded by the manufacturer of one of the drugs. The subjects were 35 adolescent drivers (19 males, 16 females, mean age 17.8 years) who were tested in a driving simulator in a double blind placebo controlled crossover study after taking 72 mg of sustained release OROS methylphenidate (MPH) or 30 mg of amphetamine salts Extended Release. Subjects were enrolled on the basis of a previous diagnosis of ADHD, a structured interview, a history of response to stimulants and a current driver licence. Exclusion criteria were a history of adverse reactions to stimulants, a medical contra-indication or a history of substance abuse. There were 12 subjects with the Inattentive subtype, two with the hyperactive subtype and 21 with the combined form.

Information from the simulator data log was used to compile an Impaired Driving Score (see above). The results showed improved performance of the MPH group compared to placebo ($p < 0.001$) and amphetamine ($p = 0.03$) but no improvement for the amphetamine group over placebo. Subjects were aware of the improvement with MPH but not with amphetamines.

Summary

The effects of ADHD and its treatment on driving behaviour and crash risk continue to interest road safety researchers although there are a limited number of workers who are responsible for much of the research. Many of the studies continue to suffer from similar methodological deficiencies noted in the initial review including:

- Self reporting and uncertain veracity of the data. Only one study used DMV data to confirm the subjects' driving record. Another study appeared to be purely a comparison between a questionnaire validated previously with DMV data and a non validated one;
- uncertain validation of methods of assessment (simulations, computer based neuropsychological testing and questionnaires);
- small sample sizes with skewed gender ratios;
- unrepresentative controls.

Since the previous review period, selection and definition of ADHD subjects seems to have settled into a commonly used set of criteria including:

- DSM-IV diagnosis, usually confirmed by repeat assessment during the study;
- IQ over 80;
- Vision adequate for driving in the relevant jurisdiction;
- Possession of a valid driver licence;
- Absence of psychiatric comorbidities (usually as defined in DSM-IV) screened for during the study.

Noteworthy amongst the research reviewed in the post May 2003 period were studies investigating the effects of other sources of driving impairment on ADHD drivers. Fatigue and alcohol are two common contributors to crash causation and initial studies appear to show that both can have more profound effects on ADHD drivers than on the general community. Most studies confirmed previous findings that ADHD drivers have higher crash and traffic law infringement rates of various types and that they are more prone to angry aggressive and risky driving behaviour. Research into the therapeutic effects of stimulants continues with increasing evidence of a beneficial effect of methylphenidate treatment, at least in younger subjects. It is vital however that studies into therapeutic agents for all conditions include adequate disclosure where the studies have been supported by drug manufacturers. There was only one such disclosure in the studies reviewed here and that one did not specifically mention the role of the sponsor in marketing the drug under investigation.

A comprehensive meta-analysis of thirteen previous observational studies on ADHD and driving was performed by Jerome et al (2006). The studies dated from 1997 to 2006 and included many of those discussed in this document. There was also a review of seven experimental studies of various stimulant medications and one of a non-stimulant. The authors concluded that there was enough information now available to allow a

quantification of the relative driving risk for ADHD drivers of 1.54. This is more evident in citations and violations than crashes (the 'gold standard' used for RR estimates in the current study), although the latter are statistically more uncommon than the former. There is as yet not enough evidence to extend the risk calculation to the different subtypes of ADHD although observational studies give some insight into the mechanisms by which ADHD may contribute to various types of aberrant driving. Experimental studies of the effects of medications showed a clear effect of stimulants, with MPH appearing to be more effective than amphetamines.

Table 34 Summary of studies of risk associated with ADHD

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Barkley, Murphy, DuPaul & Bush (2002)	Cases = 105 Controls = 64- -P aged between 17 and 28 yrs -P screened for co-morbid psych and phys illness	- Computer simulated driving test (EDS): - performance on a battery of EF tasks - Video test of driving knowledge and rapid decision making abilities (DPAS) - Official motor vehicle records	Driving citations: ADHD > C** Licence suspensions: ADHD > C** Crashes as driver: ADHD > C At-fault crashes: ADHD > C Severity of crashes: ADHD > C* No sig diff in performance on EDS
Cox, Merkel, Kovatchev & Seward (2000)	Double-blind (Ritalin vs placebo) cross-over counter-balanced design Cases = 7 ADHD Controls = 6 non-ADHD	Sim "Driving impairment" score Self-reported driving history Self-rating of driving performance	Acc: ADHD > C* Cit: ADHD > C Impair on sim under placebo cond: ADHD > C* ADHD rated themselves as driving poorer in placebo cond* improve driv perfor under Ritalin cond* - ADHD rated themselves as driving better under Ritalin cond
Nada-Raja et al. (1997)	Cases = 916 p from birth cohort of NZ children ADHD = 101 Conduct = 46 Anx/dpress = 85 No disorder = 684	parent reports mental health assess at 15 yrs self report data on driving behavior over past 12 months -official driving records from LTSA between ages of 15 and 18	Males w ADHD and conduct disorder sig more driving offenses other groups* - Females w ADHD sig more driving offenses and traffic crashes *
Barkley, Murphy & Kwasnil (1996)	Cases = 25 with ADHD Controls = 23 w/o ADHD 17-30 years	- sim driving test (EDS): - perform on EF tasks - Self reported viol and acc - Behaviour ratings by self and others - Video test of driving knowledge and rapid decision making abilities (DPAS) - Official records	Cit for speeding: ADHD > C Licence susp: ADHD > C Acc Inv: ADHD > C Injurious Acc: ADHD > C - ADHD rated by selves and others as using poorer driving habits EDS: Only differed from C on steering control

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Barkley, Guevremont, Anastopoulos, DuPaul & Shelton (1993)	3- to 5-year follow-up survey Cases = 35 p with ADHD Controls = 36	- parent ratings of current ADHD symptoms, ODD and CD - Survey of negative driving outcomes - parent rating of driving skills	Driven illegally: ADHD > C * Lic susp: ADHD > C Repeated traff cit: ADHD > C * Multiple crashes as driver: ADHD > C* At-fault crash: ADHD > C* - ADHD less likely to be employing sound driving habits as reported by their parents***
Barkley, Murphy, O'Connell, Connor (2005)	56 patients with ADHD. Double blind crossover study comparing 10 and 20 mg doses of methylphenidate (MPH) with placebo and baseline	Virtual reality simulator and computerised continuous performance test	All 3 treatment groups (including placebo) improved compared to baseline*. Some increased improvement in the higher dose MPH group
Cox, Punja, Powers, Merkel, Burket, Moore, Thorndike Kovatchev (2006)	10 ADHD adolescents in crossover study on simulated driving using manual vs automatic transmission	Self rated attentiveness, Impaired Driving Score (IDS)	Drivers using manual transmission reported higher levels of attentiveness and generated lower IDS values on simulated driving.
Cox, Merkel, Moore, Thorndike, Muller, Kovatchev (2006)	35 ADHD, double blind crossover simulator study comparing long acting methylphenidate and amphetamines.	As above	MPH group drove better than placebo (p<0.001) and amphetamine group (p=0.03). Study funded by manufacturer of MPH.
Fischer, Barkley, Smallish, Fletcher (2007)	Longitudinal cohort study of 147 children with ADHD followed into young adulthood. 71 controls Comparison of DMV records, on road testing and simulated driving	Crashes, violations, citations and reports from instructors and observers	ADHD drivers had increased incidence of suspensions, driving unlicensed, hit & run crashes and traffic tickets compared to controls*. Increased impulsive errors on real and simulated driving* .
IMMORTAL (2004)	17 males with ADHD 28 controls. Driving simulator study with and without methylphenidate compared to placebo	Driving simulator score of driving errors, speed, headway and crashes.	No difference between subjects and controls. Thought to be due to deficiencies in simulator
Jerome, Segal, Habinski (2006)	Meta-analysis of 13 observational and 8 experimental studies	Various	Arrived at a relative risk factor of 1.54 for ADHD drivers. Risk more apparent for citations and violations than for crashes.
Knouse, Bagwell, Barkley, Murphy (2005)	44 ADHD 44 controls	Simulator driving ability self rated and rated by observer, Rates	ADHD drivers had higher rates of tickets, collisions and citations but tended to

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
	Self reported estimates of driving ability compared to validated questionnaire of driving history and brief simulator study	of tickets, collisions and citations	overestimate their driving ability based on naturalistic events reported on the validated questionnaire. *
Thompson, Molina, Pelham, Gnagy (2007)	355 adolescents and young adults with ADHD 240 controls	Self reported traffic crash and citation histories	Significant differences in: having driven unlicensed (Odds Ratio=3.97) having ever received a ticket (1.46) having a licence suspension (1.65) having been directed to go to traffic school (4.45). Smaller associations for the numbers of accidents. No differences for risky driving or alcohol impaired driving
Reimer, D'Ambrosio, Coughlin, Fried, Biederman, (2007)	20 ADHD not on medication 21 controls Fatigue inducing simulator task at different times of the day	Crash rate with simulated animals	ADHD drivers had higher crash rates after a period of fatigue induction. Conclusion that ADHD drivers become fatigued more rapidly than controls which may increase crash risk in monotonous driving situations
Reimer, D'Ambrosio, Gilbert, Coughlin, Biederman, Surman, Fried, Aleari (2005)	45 ADH, 38 controls 45 males, 38 females Self reported driving history using DBQ	Incidence of lapses, errors and violations	Confirmation that ADHD drivers have a higher incidence of crashes and risky/aggressive driving behaviours than controls. Incidence is related to age and gender in a similar way to the general driving population.
Richards, Deffenbacher, Rosen, Barkley, Rodricks (2006)	56 ADHD adults (41 male) 2 control groups of 106 and 432, not matched Self reporting of anger, aggression and risk parameters using a variety of tests	Measures of angry thoughts and actions, aggressive thoughts and actions and risky driving behaviours from questionnaires.	ADHD drivers more prone to angry and aggressive thoughts and actions compared to all controls. Differences least in younger college student controls.
Sobanski, Sabljic, Alm, Skopp, Kettler, Mattern, Strohbeck-Kuhner (2008)	27 ADHD 27 controls matched for age & education Neuropsych testing and crash history interview. Nine subjects	Scoring on neuropsych test battery, comparison of self reported traffic history	ADHD group had 2.5X crash rate of controls, more likely to be at fault and have major or minor damage, and have parking collisions. MPH improved performance on some neuropsych tests,

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Weafer, Camarillo, Fillmore, Milich, Marczinski (2008)	<p>on methylphenidate</p> <p>Experiment 1: 15 ADHD, 23 controls Simulator study comparing ADHD with sober & intoxicated controls (BAC = 0.08%)</p> <p>Experiment 2: 8 ADHD, 8 controls Simulator study comparing controls & ADHD at 0.05%, 0.08% BAC</p>	<p>Deviation of lat position, steering speed, speed variation</p> <p>As above plus self rating of intoxication & driving fitness</p>	<p>effect on crash risk not assessed.</p> <p>Sober ADHD as bad as intoxicated controls at 0.08%*</p> <p>ADHD worse than controls under all circumstances. More difference at 0.05% than 0.08%. *</p> <p>Increased alcohol effect in ADHD drivers</p>
Woodward, Fergusson & Horwood (2000)	21 year longitudinal study of a birth cohort of NZ children 941 young individuals with measure of att diff at 13yrs and driving outcomes at 21yrs	<ul style="list-style-type: none"> - parent & teacher measure of att diff (13yrs) - acc inv (18-21) - driving and driving (18-21 yrs) - traff viol (18-21 yrs) 	<ul style="list-style-type: none"> - Att diff at 13 yrs sig predictor of: - MVA causing injury***, - driving without a licence* - general traffic violations* - Once adjusted for large number of stat comp, only r'ship b/w att diff & inv in injury acc were sig.

* significant diff from control, $p < .05$

Approaches to management

Assessing fitness to drive

Despite the findings that ADHD is associated with an increased crash risk most jurisdictions do not specifically list ADHD as a medical condition to be taken into consideration when assessing fitness to drive. The two exceptions are Canada and Australia. In Canada, individuals with ADHD may be licensed subject to clinical assessment and if they are seen to be responding positively to treatment. This recommendation is consistent with the one study that suggested that psychostimulants may have a beneficial effect on driving for individuals with ADHD. Similarly, the Australian Austroads Guidelines (2006) recommend that specialist advice be sought when assessing ADHD drivers. More research is necessary in this area to determine if these guidelines accurately reflect the scientific evidence.

Self-regulation

As mentioned previously, there is currently no available information on the extent to which individuals with a psychiatric illness adopt self-regulatory practices. This is likely to result in a limited insight into how their illness may affect their driving ability.

Table 35 Private licensing guidelines for drivers with ADHD

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
ADHD	May be licensed subject to clinical assessment & positive treatment response. Must be able to comprehend & respond to traffic situations.	Subject to specialist advice.	Presence of ADHD, per se, is not a reason to bar licensing. Factors such as impulsivity and limited awareness of the impact of their behaviours need to be considered.	Not specifically listed.	Not specifically listed.	Not specifically listed.

References

- Amador, X.F., Flaum, M., Andreasen, N.C., Strauss, D.H., Yale, S.A., Clark, C.C., & Gorman, J.M. (1994). Awareness of Illness in Schizophrenia and Schizoaffective and Mood Disorders. *Archives of General Psychiatry*, *51*, 826-836.
- American Psychiatric Association (APA, 2000). *Diagnostic and statistical manual of mental disorders (DSM-IV-TR)*. Washington, DC: APA.
- Andrews, G., Hall, W., Teesson, M., & Henderson, S. (1999). *The Mental Health of Australians*. Canberra, ACT: Commonwealth of Australia.
- Armstrong, J. L., & Whitlock, F. A. (1980). Mental illness and road traffic accidents. *Australian and New Zealand Journal of Psychiatry*, *14*, 53-60.
- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Barbone, F., McMahon, A. D., Davey, P. G., Morris, A. D., Reid, I. C., McDevitt, D. G., et al. (1998). Association of road-traffic accidents with benzodiazepine use. *The Lancet*, *352*(9137), 1331-1336.
- Barkley, R.A. (1998). *Attention Deficit Hyperactivity Disorder: A handbook for diagnosis and treatment (2nd ed.)*. New York: Guilford.
- Barkley, R.A. (1997). Behavioural Inhibition, sustained Attention, and Executive Functions: Constructing a Unifying Theory of ADHD. *Psychological Bulletin*, *121*(1), 65-94.
- Barkley, R.A., Guevremont, D.C., Anastopoulos, A.D., Dupaul, G.J. & Shelton, T.L. (1993). Driving-related risks and outcomes of attention deficit hyperactivity disorder in adolescence and young adults: a 3-to 5-year follow-up survey. *Paediatrics*, *92*, 212-218.
- Barkley, R.A., Murphy, K.R., Dupaul, G.J. (1996). Psychological adjustment and adaptive impairments in young adults with ADHD. *Journal of Attention Disorders*, *1*, 41-54.
- Barkley, R.A., Murphy, K.R., Dupaul, G.J. & Bush, T. (2002). Driving in young adults with attention deficit hyperactivity disorder: Knowledge, performance, adverse outcomes, and the role of executive functioning. *Journal of the International Neuropsychological Society*, *8*, 655-672.
- Barkley, R.A., Murphy, K.R. & Kwasnik, D. (1996). Motor vehicle competencies and risks in teens and young adults with attention deficit hyperactivity disorder. *Paediatrics*, *98*, 1089-1095.
- Brunnauer, A., Laux, G., Geiger, E., Soyka, M., & Möller, H.-J. (2006). Antidepressants and driving ability: results from a clinical study. *The Journal of Clinical Psychiatry*, *67*(11), 1776-1781.

- Bukasa, B., Wenninger, U., & Brandstätter, C. (1990). *Validierung verkehrspsychologischer Testverfahren*. Wien, Literas: Kleine Fachbuchreihe des Kuratoriums für Verkehrssicherheit.
- Bukasa, B., Christ, R., Pocomny-Seliger, E., Smuc, M., & Wenninger, U. (2003). Validitätsüberprüfung verkehrspsychologischer Leistungstests für die Fahreignungsbegutachtung. *Zeitschrift für Verkehrssicherheit*, 49(4), 193.
- Bulmash, E., Moller, H., Kayumov, L., Shen, J., Wang, X., & Shapiro, C. (2006). Psychomotor disturbance in depression: Assessment using a driving simulator paradigm. *Journal of Affective Disorders*, 93, 213-218.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Cox, D.J., Merkel, R.L., Kovatchev, B. & Seward, R. (2000). Effect of stimulant medication on driving performance of young adults with attention-deficit hyperactivity disorder. *The Journal of Nervous and Mental Disease*, 188(4), 230-234.
- Cremona, A. (1986). Mad drivers: psychiatric illness and driving performance. *British Journal of Hospital Medicine*, 35, 193-195.
- Dobbs, B. M. (2001). *Medical conditions and driving: current knowledge*. Technical Report, National Highway Transportation Safety Administration and The Association for the Advancement of Automotive Medicine Project.
- Dubois, S., Bedard, M., & Weaver, B. (2008). The Impact of Benzodiazepines on Safe Driving. *Traffic Injury Prevention*, 9(5), 404 - 413.
- Dumais, A., Lesage, A., Boyer, R., Lalovic, A., Chawky, N., Ménard-Buteau, C., et al. (2005). Psychiatric risk factors for motor vehicle fatalities in young men. *Canadian Journal of Psychiatry*, 50(13), 838-844.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Edlund, M.J., Conrad, C. & Morris, P. (1989). Crashes among schizophrenic outpatients. *Comprehensive Psychiatry*, 30, 522-526.
- Elwood, P. (1998). Driving, mental illness and the role of the psychiatrist. *Irish Journal of Psychological Medicine*, 15(2), 49-51.
- Eysenck, M. W. (1997). *Anxiety & Cognition: A Unified Theory*. Psychology Press: Hove.
- Gianutsos, R. (1994). *Computer Programs for Cognitive Rehabilitation. Volume 6. Resource Manual: Driving Advisement Seminar*. Life Sciences Associates, Bayport, N.Y., USA.

- Grabe, H., Wolf, T., Gratz, S., & Laux, G. (1998). The influence of polypharmacological antidepressive treatment on central nervous information processing of depressed patients: implications for fitness to drive. *Neuropsychobiology* 37, 200-204.
- Hemmelgarn, B., Suissa, S., Huang, A., Boivin, J.F. & Pinard, G. (1997). Benzodiazepine use and the risk of motor vehicle crash in the elderly. *Journal of the American Medical Association*, 278(2), 27-31.
- Hebert, C., Delaney, J. A., Hemmelgarn, B., Levesque, L. E., Suissa, S., Hebert, C., et al. (2007). Benzodiazepines and elderly drivers: a comparison of pharmacoepidemiological study designs.[see comment]. [Comparative Study]. *Pharmacoepidemiology & Drug Safety*, 16(8), 845-849.
- Iancu, I., Spivak, B., Wiener, A. & Weizman, A. (1996). Psychiatric guidelines for licensing drivers. *International Journal of Risk and Safety in Medicine*, 8, 169-173.
- Iwamoto, K., Masahiro, T., Yukako, N., Yukiko, K., Ryoko, I., Yuji, U., et al. (2008). The effects of acute treatment with paroxetine, amitriptyline, and placebo on driving performance and cognitive function in healthy Japanese subjects: A double-blind crossover trial. *Human Psychopharmacology: Clinical and Experimental*, 23(5), 399-407.
- Jackson, H.J. & Burgess, P.M. (2002). Personality disorders in the community: results from the Australian National Survey of Mental Health and Wellbeing Part II. Relationships between personality disorder, Axis I mental disorders and physical conditions with disability and health consultations. *Social Psychiatry and Psychiatric Epidemiology*, 37(6), 251-260.
- Judd, L.L. (1985). The effect of antipsychotic drugs on driving and related driving psychomotor functions. *Crash Analysis and Prevention*, 17(4), 319-322.
- Kagerer, S., Winter, C., Möller, H., & Soyka, M. (2003). Effects of haloperidol and atypical neuroleptics on psychomotor performance and driving ability in schizophrenic patients. Results from an experimental study. *Neuropsychobiology*, 47(4), 212-218.
- Kolman, P.B.R (1983). The incidence of psychopathology in burned adult patients. A critical review. *Journal of Burn Care & Rehabilitation*, 4, 430-436.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Leveille, S.G., Buchner, D.M., Koepsell, T.D., McCloskey, L.W., Wolf, M.E. & Wagner, E.H. (1994). Psychoactive medications and injurious motor vehicle collisions involving older drivers. *Epidemiology*, 5, 591-598.
- Mathers, C.D., Stein, C., Ma Fat, D., Rao, C., Inoue, M., Tomijima, N, Bernard, C., Lopez, A.D. & Murray, C.J.L. (2002) *Global burden of disease 2000: Version 2. Methods and Results*. Global Programme on Evidence for Health Policy

Discussion Paper No. 50. World Health Organisation. Available at http://www3.who.ch/whosis/discussion_papers/pdf/paper50.pdf

Martin, M. & Sugarman, P. (1997). ABC of mental health: Disorders of personality. *British Medical Journal*, 315, 176-179.

McDonald, A.S. & Davey, G.C.L. (1996). Psychiatric disorders and crash injury. *Clinical Psychology Review*, 16(2), 105-127.

McGwin, G., Sims, R.V., Pulley, L. & Roseman, J.M. (2000). Relations among chronic medical conditions, medications, and automobile crashes in the elderly: A population-based case-control study. *American Journal of Epidemiology*, 152(5), 424-431.

Metzner, J.L., Dentino, A.N., Godard, S.L., Hay, D.P., Hay, L. & Linnoila, M. (1993). *Impairment in driving and psychiatric illnesses*. *Journal of Neuropsychiatry*, 5(2), 211-220.

Murray, C. J., & Lopez, A. D. (1996). *The global burden of disease*. Boston, MA: Harvard School of Public Health.

Nada-Raja, S., Langley, J.D., McGee, R., Williams, S.M., Begg, D.J. & Reeder, A.I. (1997). Inattentive and hyperactive behaviours and driving offences in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 515-522.

Narrow, W.E., Rae, D.S. & Regier, D.A. (1998). *NIMH epidemiology note: prevalence of anxiety disorders. One-year prevalence best estimates calculated from ECA and NCS data. Population estimates based on U.S. Census estimated residential population age 18 to 54 on July 1*, Unpublished.

National Alliance for the Mentally Ill (NAMI, 2003). *Schizophrenia Fact Sheet*. Retrieved on May 2, 2003 from www.nami.org/helpline/schizo.htm

National Institute of Mental Health (NIMH, 1999). *Schizophrenia*. Retrieved on May 2, 2003 from www.nimh.nih.gov/publicat/schizosph.htm

National Institute of Mental Health (NIMH, 2000). *Attention Deficit Hyperactivity Disorder (ADHD) - Questions and Answers*. Retrieved on May 30, 2003 from <http://www.nimh.nih.gov/publicat/adhdqa.cfm>

Niveau, G. & Kelley-Puskas, M. (2001). Psychiatric disorders and fitness to drive. *Journal of Medical Ethics*, 27, 36-39.

Noyes, R. (1985). Motor vehicle crashes related to psychiatric impairment. *Psychosomatics*, 26, 569-580.

O'Hanlon, J., Robbe, H.W.J., Vermeeren, A., VanLeeuwen, C. & Danjou, P.E. (1998). Venlafaxine's effects on healthy volunteers' driving, psychomotor, and vigilance performance during 15-day fixed and incremental dosing regimens. *Journal of Clinical Psychopharmacology*, 18, 212-221.

- O'Hanlon, J.F., Vermeeren, A., Uiterwijk, M.C., Van Veggel Loe, M.A., Swijgman, H.F. (1995). Anxiolytic effects on the actual driving performance of patients and healthy volunteers in a standardized test. *Neuropsychobiology*, 31, 81-88.
- Parker, D. West, R., Stradling, S. & Manstead, A.S.R. (1995). Behavioural characteristics and involvement in different types of traffic crashes. *Crash Analysis Prevention*, 27, 571-581.
- Petch, E. Mental disorder and fitness to drive. *The Journal of Forensic Psychiatry*, 7(3), 607-618.
- Pless, I.B., Taylor, H.G. & Arsenault, L. (1995). The relationship between vigilance deficits and traffic injuries involving children, *Paediatrics*, 95, 219-224.
- Ramaekers, J.G. (2003). Antidepressants and driver impairment: Empirical evidence from a standard on-the-road test. *Journal of Clinical Psychiatry*, 64(1), 20-29.
- Ray, W.A., Fought, R.L. & Decker, M.D. (1992). Psychoactive drugs and the risk of injurious motor vehicle crashes in elderly drivers. *American Journal of Epidemiology*, 136(7), 873-883).
- Ray, W., Purushottam, B.T. & Shorr, R.I. (1993). Medications and the older driver. *Clinics in Geriatric Medicine*, 9(2), 413-438.
- Risser, R., Schmidt, L., Brandstätter, C., Bukasa, B., & Wenninger, U. (1993). *Verkehrspsychologische Testverfahren und Kriterien des Fahrverhaltens*. Wien: Verkehrspsychologisches Institut des Kuratoriums für Verkehrssicherheit;
- Routley, V., Staines, C., Brennan, C., Haworth, N. & Ozanne-Smith, J. (2003) [Suicide and natural deaths in road traffic: Review](#), Monash University Accident Research Centre, Report No. 216
- SANE (2009). *Schizophrenia*. Retrieved 20 July, 2009, from <http://www.sane.org/information/factsheets/schizophrenia.html>.
- Schizophrenia Research Institute (2009). Retrieved 5th October 2009 from <http://www.schizophreniaresearch.org.au/home.php?r=3&menu=About>
- Shallice, T. (1982). Specific impairments of planning. D. E. & W. L. Broadbent (Eds), *The neuropsychology of cognitive function*. (pp. 199-209). London: The Royal Society.
- Silverstone, T. (1988). The influence of psychiatric disease and its treatment of driving performance. *International Clinical Psychopharmacology*, 3(1), 59-66,
- Soyka, M., Kagerer, S., Brunbauer, A., Laux, G., & Moller, H.-J. r. (2005). Driving ability in schizophrenic patients: effects of neuroleptics. *International Journal of Psychiatry in Clinical Practice*, 9(3), 168 - 174.
- Stuss, D. T., & Benson, D. F. (1986). *The Frontal Lobes*. New York: Raven.
- Swedish Road Administration. (1998). *Swedish Road Administration Statute Book*. Sweden.

- Törnros, J., Vikander, B., Ahlner, J. & Jönsson, K.A. (2001). Simulated driving performance of benzodiazepine users. *Journal of Traffic Medicine*, 29(3-4), 4-15.
- Tsuang, M.T., Boor, M., Flemming, & J.A. (1985). Psychiatric aspects of traffic crashes. *American Journal of Psychiatry*, 142, 538-546.
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- van Laar, M.W., van Willigenburg, A.P.P. & Volkerts, E.R. (1995). Acute and subchronic effects of nefazodone and imipramine on highway driving, cognitive functions and daytime sleepiness in healthy adult and elderly subjects. *Journal of Clinical Psychopharmacology*, 15, 30-40.
- Velligan DI, Mahurin RK, Diamond PL; Hazelden BC, Ec Kert SL, & Miller AI. (1997). The functional significance of symptomatology and cognitive function in schizophrenia. *Schizophrenia Research*, 25, 21-31.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C., Suruda, A.J. & Deane, J.M. (2002). Evaluating the crash and citations rates of Utah drivers licensed with medical conditions, 1992-1996. *Accident Analysis & Prevention*, 34, 237-246.
- Webb, M.R., Dietrich, A.J., Wood, R., Katon, W. & Schwenk, T.L. (2000). Diagnosis and Management of Depression. *American Family Physician Monograph 2*. Kansas: American Academy of Family Physicians.
- Wingen, M., Johannes, G. R., & Jeroen, A. J. S. (2006). Driving impairment in depressed patients receiving long-term antidepressant treatment. *Psychopharmacology*, 188(1), 84.
- Woodward, L.J., Fergusson, D.M. & Horwood, L.J. (2000). Driving outcomes of young people with attentional difficulties in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(5), 627-634.
- World Health Organisation. (2010). Depression. Accessed on August 9th, 2010 at http://www.who.int/mental_health/management/depression/definition/en/
- World Health Organisation (WHO, 2001a). *Mental and Neurological Disorders - Fact Sheet N° 265*. Retrieved on June 11, 2003 from <http://www.who.int/inf/fs/en/fact265.html>
- World Health Organisation (WHO, 2001b). Mental health: new understanding – new hope. Retrieved on June 26, 2003 from http://www.euro.who.int/mediacentre/PressBackgrounders/2001/20011128_1
- World Health Organisation (WHO, 2004). *The Global Burden of Disease 2004 Update*. Geneva: WHO.
- World Health Organisation (1992). *The ICD-10 Classification of mental and behavioural disorders. Diagnostic Criteria for research*. Geneva: WHO.

3.10 RESPIRATORY DISORDERS

This section deals with the group of respiratory diseases known collectively as Chronic Obstructive Pulmonary Disorders (COPD) as well as with asthma. A surprising finding was the non-existence of research that evaluated the impact of COPD on driving and the crash risk of those suffering from these diseases. However, some research is presented that deals with the effects of respiratory diseases on other functional abilities. Sleep apnoea is also classified as a respiratory disease however, as it only occurs during sleep, it is more commonly identified as a sleep disorder. Therefore, in the present review, sleep apnoea has been discussed separately (see section 3.11 for sleep apnoea and related conditions).

Definition of respiratory disorders

Chronic obstructive pulmonary disease (COPD), or chronic obstructive lung disease as it is sometimes called, refers to a group of disorders that are characterised by breathing disorders. The three main COPD diseases are emphysema, chronic bronchitis and asthmatic bronchitis. A brief description of each is provided below.

The technical definition of COPD is made by measuring a patient's airflow expressed as FEV₁ (forced expiration volume during the first second) and FVC (forced vital capacity). The British Thoracic Society defines COPD as FEV₁/FVC < 0.7. Smoking is believed to be the main causal factor in 80% of COPD cases (UCDavis, 1999). Lundback et al. (2003) report that the two most important determinants of developing COPD are smoking and age, with 50% of elderly smokers doing so.

Emphysema

Approximately 3% of emphysema cases occur as a result of a rare genetic condition called alpha 1-antitrypsin deficiency (A1AD), which causes inhibition of the production of an enzyme responsible for protecting the cells that line the lungs. This predisposes the person to develop emphysema at a young age. If people with A1AD also smoke, they "have no chance at all for escaping emphysema" (UCDavis, 1999, p3).

Chronic bronchitis

Chronic bronchitis occurs from the inflammation of the bronchi, which are air passages situated inside the lungs. Smoking and passive smoking are the main causes of chronic bronchitis, with the severity of the disease increasing with greater exposure to smoke inhalation. Other factors that exacerbate the condition are air pollution, allergies and infections (Kaufman, 2002).

Asthma

Asthma often begins in childhood and results from irritation and inflammation of the airway passages. During an asthma attack, this inflammation causes the passages to swell and restrict airflow. The frequency and severity of attacks vary but they may occur daily or even hourly (WHO, 2000). Exposure to allergens is thought to be responsible for the onset of asthma. In childhood, these allergens may include mites, cats and cockroaches. Drugs (eg aspirin), workplace chemicals, and cigarette smoke are additional risk factors (WHO, 2000). Asthma can sometimes be confused with sleep apnoea due to the nocturnal aggravation of symptoms that can occur. However, if

coughing is also present and the person feels tight-chested in the morning, then nocturnal asthma rather than sleep apnoea could be the cause (Shneerson, 2002). **Asthmatic bronchitis** occurs when an asthmatic, exposed to aggravating toxins and irritants such as smoking, develops a chronic cough (UCDavis, 1999).

Prevalence of respiratory disorders

For COPD, prevalence estimates are influenced in part by the criteria used to define the disease. One study (Lundback et al., 2003) reports that using the definition of COPD laid down by the Global Initiative for Chronic Obstructive Pulmonary Disease produced incidence rates that were double those found when the criteria provided by the British Thoracic Society were used.

COPD

- Affects 8-14% of those aged over 45 years in Sweden (Lundback et al., 2003)
- Occurrence is on the rise, particularly amongst Caucasian women (GOLD, no date)

Recently the WHO predicted that by 2030, COPD will be the third leading cause of death (WHO, 2008a). The WHO estimates that the prevalence of COPD is approximately 210 million worldwide (WHO, 2008a). In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at 5.9 million or around 2% of the total population. Similarly, the prevalence of this disease in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 7.6 million or around 2% of the total population.

It should also be noted that COPD is often associated with cardiovascular problems and increased mortality.

Asthma

- 3 million asthmatics in Japan (30% with moderate asthma and 7% with severe asthma);
- 8% of the population in Switzerland;
- 1 in 6 children in Australia have asthma; and
- On average, the worldwide incidence of asthma is rising by 50% every decade (WHO, 2000).

The WHO estimates that the prevalence of asthma is approximately 300 million worldwide (WHO, 2008b). In 2000, the prevalence of the disease in Northern American countries (AMROA group which includes US, Canada and Cuba) was estimated at 18.9 million or around 6% of the total population. Similarly, the prevalence of this disease in Western European countries (EUROA group which includes Belgium, Denmark, Finland, France, Germany, Netherlands, Norway, Sweden, UK and others) was estimated by the WHO at 18.9 million or around 5% of the total population.

Functional impairments associated with COPD relevant to driving

Functional impairments associated with COPD include:

- Structural damage to lungs;
- Reduced airflow capacity and progressive exhalation difficulties;
- Dyspnoea;
- Wheezing; and
- Coughing / chronic cough.

Symptoms specific to the three main types of COPD are described below.

Emphysema

The early symptoms of emphysema are: shortness of breath; minor coughing and only small amounts of sputum. However, by the time these early symptoms are noticed, individuals will already have lost an alarming 50-70% of lung tissue. As time passes, symptoms become progressively worse. In the later stages, breathing becomes laboured and rapid even when resting, and the person suffers with constant “air hunger” (UCDavis, 1999, p4).

Chronic Bronchitis (Kaufman, 2002):

- overproduction of bronchial mucus;
- sputum-producing cough for 3 or more months;
- shortness of breath;
- wheezing;
- headaches;
- tiredness;
- swollen ankles, feet and legs (due to right sided heart failure).

Chronic Asthma:

- wheezing;
- breathlessness.

COPD and cognitive impairment

Grant et al. (1987, cited in Dobbs, 2001) compared the neurological performance of three groups of participants – those with mild hypoxia (n = 86), moderate hypoxia (n = 155) or severe hypoxia (n = 61) - with a group of 99 healthy controls who were matched for age and education. It was found that as the severity of hypoxia increased, so too did

the level of neurological deficits, with participants with mild hypoxia experiencing a 27% decline and participants with severe hypoxia exhibiting a 61% decline. The skills particularly affected were perceptual learning and problem solving. Controls and the participants with mild hypoxia, however, performed to a similar standard. Further analysis revealed that age, education and PaO₂ were associated with poorer cognitive performance.

Peruzza et al. (2003), on the other hand, did not find any significant difference between controls and participants with COPD for cognitive impairment, as measured by the Mini Mental State Examination (MMSE).

Another method of categorising respiratory diseases is according to their severity as measured by Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second (FEV₁) readings, which are the basic respiratory function tests (Utah Licensing Guidelines, 1992). This type of classification gives an indication of the extent of cognitive impairment, if any (Grant et al., 1987, cited in Dobbs, 2001). Peruzza et al. (2003) reported that the more severe the COPD, the greater the impairment and reduction in the “functional status” of the individual. Specifically, a comparison of the walking ability of 60 elderly participants with COPD and 58 age-matched healthy controls indicated that the participants with COPD walked much shorter distances in 6 minutes than the controls.

Treatment of respiratory disorders

In this section, treatments for COPD, chronic bronchitis and asthma are listed and research related to the effectiveness of each is cited. The assessment of effectiveness relates only to the treatment of the disease and the alleviation of symptoms since no literature that investigated the impact of these therapies on driving ability could be found.

COPD

COPD can be treated using the following:

- oxygen therapy
- drug therapy including oxitropium bromide (not available in Australia) and theophylline (obsolete in Australia) (Bellia et al., 2002) and
- inhaled beta sympathomimetic or anticholinergic bronchodilators for acute exacerbations of COPD (Cazzola et al., 2003).
- Inhaled or systemic corticosteroids

Crockett, Cranston, Moss and Alpers (2001) undertook a systematic Cochrane review of five randomised controlled trials on the effect of long-term, at-home use of oxygen therapy for COPD. The main outcome measure was survival. Two forms of oxygen therapy were included: continuous and nocturnal only. Significant improvements in mortality were observed over 2 years with continuous oxygen therapy compared to that found with nocturnal therapy only (Peto odds ratio 0.45, 95%CI 0.25 - 0.81). There was also a significant increase in survival after 5 years for those treated with oxygen therapy

compared to those receiving no such treatment (Peto odds ratio 0.42, 95% CI 0.18-0.98). Oxygen therapy only produced improvements in those with severe hypoxia.

Bellia et al. (2002) have also reported that the drugs oxitropium bromide, theophylline, administered singly or in combination produced a decrease in symptoms amongst those with mild to severe COPD during an eight-week period.

Cazzola et al. (2003) found that formoterol was effective in treating people with COPD experiencing the acute phase of the illness.

Chronic Bronchitis

There is no cure for chronic bronchitis, but symptoms can be controlled and complications can be prevented by using various drugs including:

- antibiotics,
- medications that dilate the airways
- corticosteroids.

Severe cases of the disease may require oxygen therapy. Very advanced cases may need lung transplants (Kaufman, 2002).

Asthma

Treatments for Asthma include:

- corticosteroids (taken orally or through inhalation) and
- leukotriene antagonists, short-acting and long-acting [beta]-agonists, cromoglycate, and nedocromil.

Effectiveness of treatment

Niven and Argyros (2003) state that the above drugs usually control asthma satisfactorily, although the long-term use of high doses of corticosteroids may be associated with significant side effects (for example mild weakness in the muscles of the arms or legs or blurred vision).

Pre-May 2003: Relationship between respiratory disorders and road safety outcomes

Crashes

No research papers that explicitly examined the relationship between the relative risk of crashes and specific respiratory disorders were identified in the literature search. This is a little surprising given that reduced blood oxygenation can impair judgement or even cause a loss of consciousness (Doege & Engelberg, 1986). In addition to inducing mental confusion, respiratory diseases can also interfere with driving by the sudden onset of severe fits of coughing and cough syncope (Vernon, Diller, Cook, Reading &

Dean, 2001). The New Zealand and UK licensing guidelines also warn of the possibility of loss of consciousness.

The only study identified was that by Vernon, Diller, Cook, Reading, Suruda and Deane (2001) who conducted a retrospective case control study of crash rates (all crashes and at-fault crashes) and citation rates for 2,688 drivers with 'pulmonary conditions'. These conditions included pulmonary disease or symptoms or impaired function or severe respiratory difficulties (see section 3.1 for a more detailed description of the study methods). Participants were also classified according to their licence status (restricted/no restrictions), with the majority of participants having no restrictions (n = 2437). Drivers with pulmonary conditions with no licence restriction (i.e., the lowest level of impairment) had significantly higher crash rates and at-fault crashes (RR: 1.18, 95%CI 1.03 - 1.34, RR: 1.26, 95%CI 1.06 - 1.50 respectively) than the general population of drivers in the state of Utah, USA. In contrast, the crash rates and at-fault crashes for drivers with pulmonary conditions with restricted licences (i.e., the highest level of impairment) were not significantly different (RR: 0.91, 95%CI 0.40 - 2.09, RR: 1.60, 95%CI 0.69 - 3.71 respectively) than the general population of drivers (see Table 32).

Vernon et al. concluded that unrestricted drivers with pulmonary conditions have a slightly elevated risk of crashing than the general population of drivers. One of the main limitations of this study was that the authors did not control for driver exposure, which assumes that drivers in the pulmonary group and matched controls drive similar distances. As noted by Lings (2001), it is reasonable to assume that medical conditions may influence driving distances. It should also be noted that the pulmonary group comprised drivers with other conditions such as pulmonary disease or symptoms, impaired function or severe respiratory difficulties and therefore it is impossible to isolate the crash risk associated with respiratory difficulties from this study.

Dobbs (2001) states that when determining the road safety risk associated with respiratory disorders, the effect that the disease has on the functional skills required to drive (sensory, cognitive and psychomotor) should be assessed (see previous section for a review of the evidence relating to functional impairments and COPD). In this context, the effect that hypoxaemia (i.e. oxygen deficiency) has on cognitive functioning is of major concern.

Citations

As outlined above, Vernon et al. (2001) compared the relative risk of driving citations of drivers with pulmonary condition with and without licensing restrictions and compared them to drivers without a medical condition. Vernon et al. reported that unrestricted drivers with a pulmonary condition had a significantly lower citation rate than control participants (RR: 0.87, 95%CI 0.79 - 0.97). In contrast, the rate of citations amongst those with pulmonary conditions with a restricted licence did not differ from controls (RR: 0.49, 95%CI: 0.18 - 1.30).

Post May 2003: Relationship between respiratory disorders and road safety outcomes

The review of literature published between May 2003 and mid-2009 revealed only two studies addressing road safety outcomes and respiratory disorders: both focused on

performance measures with one specifically investigating the effects of intranasal oxygen treatment of COPD on driving performance (see Table 36).

Crashes

No research papers assessing crash risk and respiratory disorders were identified in the review of literature published between May 2003 and mid-2009.

Citations

There were no studies in the review period that investigated the association between citations and respiratory disorders.

Driving Performance

Orth and colleagues (2008) assessed driving performance in a simulator using a sample of seventeen patients with COPD (average age 55.2 years), and 10 healthy controls (average age 55.1 years). Among the control group, COPD was excluded clinically, and for both groups, sleep apnoea was excluded polysomnographically. Participants drove on a simulated highway for 60 minutes with a mean speed of 100 km/h. Weather changes, daytime conditions were presented in random order. Results revealed no significant difference between case and control on number of concentration faults. Cases caused significantly more crashes than controls. Multiple regression analysis revealed no predictors for faults and crashes on the basis of disease severity or severity of ventilation while sleeping.

Orth et al (2008) concluded that patients with COPD have increased crash risk in a driving simulator. The authors did not speculate as to how the results of the study translate to real world crash patterns.

Treatment of COPD and road safety outcomes

Pretto and McDonald (2008) assessed driving performance in a simulator using a sample of 33 individuals with COPD. Two participants withdrew from the study due to nausea during the simulator drive, and one participant's data was withdrawn due to a failure in oxygen supply during the testing procedure. Participants underwent a baseline driving simulator assessment for 20 minutes followed by an assessment on psychomotor vigilant tasks (PVT). They were then randomised to receive either intranasal air or intranasal oxygen which was provided in a double-blind fashion for at least 5 minutes prior to and during repeat driving and PVT assessments. Participants received the alternate gas in the cross-over stage of the study. Driving performance was measured by variation in lane position and speed. The results showed a non-significant difference in driving performance when participants were breathing intranasal air compared with intranasal oxygen. There was no significant difference between scores on the psychomotor vigilant task when participants were breathing intranasal air compared with intranasal oxygen.

Pretto and McDonald concluded that the study demonstrated no effect of supplemental oxygen on driving performance using a driving simulator, therefore indicating that driving without supplemental oxygen would not adversely affect driving performance. The authors noted that the findings were consistent with previous research. Pretto and McDonald noted that a limitation of the study was that the simulated drive duration of

20 minutes was potentially too short, given that neurocognitive performance might decline further over longer times periods without supplemental oxygen.

Summary

Despite the high prevalence of COPD, there has been little research that explicitly investigates the road crash risk associated with this disease. The cognitive deficits (eg mental confusion and impaired judgement) that oxygen deprivation can produce, as well as the interference of sudden coughing fits and syncope can, potentially, impair driving ability. There is preliminary evidence that individuals with COPD have increased crash risk based on their driving performance in a simulator. Research has shown that the greater the severity of symptoms, the greater the functional impairment. One study regarding the treatment of COPD suggests that driving without supplemental oxygen does not adversely affect driving performance. The drug therapies used to alleviate COPD can provide satisfactory symptom-relief in many people with respiratory disorders, although the impact of these drugs and their associated side effects on driving ability has yet to be specifically investigated. Notwithstanding this, the licensing guidelines for several countries stipulate that those drivers with COPD who require supplemental oxygen must either undergo an additional road test (private licence holders in Canada) or hold a restricted licence only (commercial licence holders in USA).

Table 36 Summary of studies of risk associated with respiratory disorders

Study: Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Orth et al (2008)	Pop/case-control; Cases n= 17 Controls n = 10 'Cases' =COPD in stable phase of diseases	Performance on driving simulator, lapses in concentration & simulator crashes.	NS difference between case and control on number of concentration faults. Cases caused significantly more crashes than controls. No predictors for faults and crashes on the basis of disease severity or severity of ventilation while sleeping.
Pretto & McDonald (2008)	Randomised, double blind, cross-over protocol	Performance on driving simulator, maintaining lane position & speed variation.	NS difference in driving performance (maintaining lane position, speed variation) when Ss were breathing intranasal air compared with intranasal oxygen. NS differences in psychomotor vigilant task.
Vernon et al (2001)	Pop/case-control; Cases n= 2688 Control n= 20,210 'Cases'= pulmonary conditions (including pulmonary disease or symptoms, impaired function or severe respiratory difficulties)	(i) Crash-all (ii) At-fault crash (iii) Citation Rates per 10,000 lic days	Not restricted (n=2437) 1.18, all crashes* 1.26, at-fault* 0.87, citations Restricted lic (n=69) 0.91, all crashes 1.60, at-fault 0.49, citations

Approaches to management

Assessment of fitness to drive

The licensing guidelines for holders of private licences in the six countries surveyed (see Table 37) generally stipulate that only in cases of severe asthma are drivers required to desist from driving. Utah (USA) requires severe asthmatics to acquire a restricted licence while Australia and New Zealand specify that driving may resume after a suitable time period after the onset of severe symptoms, such as loss of consciousness. Sweden and Canada do not specifically provide guidelines for asthmatics. Likewise, the licence guidelines for COPD specify that drivers with severe symptoms should not drive. Australia and Utah (USA) require that in cases of severe COPD, a restricted licence only may be held.

In Canada, private licence holders who require supplemental oxygen must undergo a road test and be under supervision whilst commercial licence holders in Utah, USA may only hold a licence restricted to intrastate travel.

Self-regulation

Briggs, Patel, Butterfield and Honeybourne (1990) conducted a postal study to ascertain whether participants with moderate to severe respiratory disorders had limited or ceased their driving as a result of their condition. Of the 158 participants who completed the questionnaire, 24.7% had either ceased or reduced their driving for respiratory-related reasons. The particular disabilities that prompted these people into limiting or stopping their driving were difficulty in parking and using seat belts, and the inability to walk to the car. These participants had a significantly lower FEV1 and FEV1% as predicted. As an aside, there was a high death rate amongst respondents, with 46 people not returning surveys due to passing away.

Summary

Approaches to management in terms of fitness to drive advise that only when drivers have severe asthma or COPD they should not drive. Specific guidelines are in place for the management of asthma and driving in Utah, Australia, New Zealand, whereas Sweden and Canada do not specifically provide guidelines. For individuals suffering from COPD Australia and Utah are the only jurisdictions with guidelines. One study has investigated the relationship between self-regulation and individuals with moderate to severe respiratory disorders. Findings indicated that almost a quarter of the sample had ceased or reduced their driving, although it should be noted that there was a high death rate amongst respondents.

Table 37 Private licensing guidelines for drivers with respiratory disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2006)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Asthma	Not specifically addressed.	<p><i>Severe chronic asthma:</i></p> <p>Desist from driving for 2 weeks following an attack that required admission to an ICU or from which loss of consciousness ensued.</p> <p><i>Exception:</i> Specialist clearance is given.</p>	<p>Notification to DVLA not required.</p> <p><i>Exceptions:</i> Asthma causes debilitating dizziness, fainting or loss of consciousness.</p>	<p>No licence restrictions if disease is stable or respiratory symptoms are minimal or occur when activity levels are greater than normal, with or without medication.</p> <p>Annual review required.</p> <p>Licence restrictions apply if PO₂ > 50 or respiratory symptoms occur with normal activity. Speed & area restrictions apply.</p> <p><i>Severe Breathing Difficulties:</i> No driving if severe symptoms occur with any activity or PO₂ < 50 &/or PCO₂ > 50.</p>	<p><i>Severe asthma attacks:</i> Person warned to desist from driving especially if severe emphysema or loss of consciousness may occur.</p>	Not addressed.
COPD (Chronic Obstructive Pulmonary Disease)	<p>Person may drive with impairment levels ranging from none to severe.</p> <p><i>Exception:</i> People with moderate</p>	<p>This disease has a variable effect on driving depending on its “type & phase” (p82).</p> <p><i>Severe:</i> Person may not hold an</p>	<p>Notification to DVLA not required.</p> <p><i>Exceptions:</i> COPD causes debilitating dizziness, fainting or loss of</p>	<p>No licence restrictions if disease is stable or respiratory symptoms are minimal or occur when activity levels are greater than normal, with or without</p>	<p><i>Severe COPD Episodes:</i> Person warned to desist from driving especially if severe emphysema or loss of consciousness may occur.</p>	Not addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2006)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
	to severe impairment who require oxygen therapy must ensure their equipment is stored securely, undergo a road test using supplemental oxygen & be under supervision.	unconditional licence. A conditional licence may be issued depending on treatment response. Periodic review required.	consciousness.	medication. Annual review required. Licence restrictions apply if PO ₂ > 50 or respiratory symptoms occur with normal activity. Speed & area restrictions apply. <i>Severe Breathing Difficulties:</i> No driving if severe symptoms occur with any activity or PO ₂ < 50 &/or PCO ₂ > 50.		
Respiratory Failure	Not specifically addressed.	<i>Severe:</i> Person may not hold an unconditional licence. A conditional licence may be issued depending on treatment response. Periodic review required.	Not specifically addressed.	<i>Severe Dyspnea:</i> No driving if severe symptoms occur with any activity or PO ₂ < 50 &/or PCO ₂ > 50.	<i>Severe & Chronic:</i> No driving.	Not addressed.

References

- Austrorads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia.* Sydney, Australia.
- Bellia, V., Foresi, A., Bianco, S., Grassi, V., Oliveri, D., Bensi, G., & Volonte, M. (2002). Efficacy and safety of oxitropium bromide, theophylline and their combination in COPD patients. *Respiratory Medicine, 96*, 881-889.
- Briggs, J.E., Patel, H., Butterfield, K., & Honeybourne, D. (1990). The effects of chronic obstructive airways disease on the ability to drive and to use a roadside alcometer. *Respiratory Medicine, 84*, 43-46.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers.* 1-13.
- Cazzola, M., Santus, P., Matera, M.G., Carlucci, P., Belloi, E., Marco, & Centanni, S. (2003). A single dose of formoterol is as effective as the same dose administered in a cumulative manner in patients with acute exacerbation of COPD. *Respiratory Medicine, 97*, 458-462.
- Crockett, A.J. Cranston, J.R., Moss, J.R., & Alpers, J.H. (2001). A review of long-term oxygen therapy for chronic obstructive pulmonary disease. *Respiratory Medicine, 95*, 437-443.
- Dobbs, B. M. (2001). *Medical conditions and driving: current knowledge.* Technical Report, National Highway Transportation Safety Administration and The Association for the Advancement of Automotive Medicine Project.
- Doege & Engelberg, (1986). Respiratory Disorders. In *Medical Conditions Affecting Drivers* by T.C. Doege and A.L. Engelberg (Eds). American Medical Association.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive.* Swansea, UK.
- GOLD (no date) Chronic obstructive pulmonary disease (COPD). What is COPD? http://www.goldcopd.com/Gold_guidelines/COPD1.html
- Kaufman, D.A. (2002). Chronic bronchitis. Medline Plus Medical Encyclopedia. **Error! Hyperlink reference not valid.** (Accessed June 2003).
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive.* Wellington, New Zealand.
- Lundback, B., Lindberg, A., Lindstrom, M., Ronmark, E., Jonsson, A.C., Jonsson, E., Larsson, L-G., Andersson, S., Sandstrom, T., & Larsson, K. (2003). Not 15 but 50% of smokers develop COPD? – Report from the obstructive lung disease in Northern Sweden studies. *Respiratory Medicine, 97*, 115-122.
- National Institutes of Health (NIH) (2001). International Guidelines Released on Chronic Obstructive Lung Disease (COPD) Fourth Leading Cause of Death in

- IS and Worldwide (2001). <http://www.nhlbi.nih.gov/new/press/01-04-04.htm> (Accessed March, 2003).
- Niven, A.S., & Argyros, G. (2003). Alternate treatments in asthma. *Chest*, 123 (4), 1254-1266.
- Orth, M., Diekmann, C., Suchan, B., Duchna, H.W., Widdig, W., Shultze-Werninghaus, G., Raasche, K., Kottebra, S. (2008). Driving performance in patients with chronic obstructive pulmonary disease. *Journal of Physiology and Pharmacology*, 59 (Suppl 6), 539-547.
- Peruzza, S., Sergi, G., Vianello, A., Pisent, C., Tiozzo, F., Manzan, A., Coin, A., Inelmen, E.M., & Enzi, G. (2003). Chronic obstructive pulmonary disease (COPD) in elderly subjects: impact on functional status and quality of life. *Respiratory Medicine*, 97, 612-617.
- Pretto, J.J., McDonald, C.F. (2008). Acute oxygen therapy does not improve cognitive and driving performance in hypoxaemic COPD. *Respirology*, 31, 1039-1044.
- Shneerson, J.M. (2002). Asthma or apnoea? *Asthma Journal*, 7(3), 133-135.
- Swedish Road Administration. (1998). *Swedish Road Administration Statute Book*. Sweden.
- UCDavis Health System (no date) How serious is chronic obstructive lung disease? <http://www.ucdmc.ucdavis.edu/health/a-z/70COLungD/doc70severity.html> (Accessed March, 2003).
- UCDavis Health System. The Well-Connected Report, 1999. Chronic Obstructive Lung Disease. What is chronic obstructive lung disease? http://wellness.ucdavis.edu/medical_conditions_az/chroniclung70.html (Accessed March, 2003).
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- Vernon, D.D., Diller, E.M., Cook, L.J., Reading, J.C. & Deane, J. M. (2001). Further analysis of drivers licensed with medical conditions in Utah. National Highway Transport Safety Authority (NHTSA) Technical Report No. DOT HS 809 211. (www.nhtsa.dot.gov/people/injury/olddrive/utah/Utah_index.htm).
- World Health Organisation (WHO), (2008a). COPD. WHO Fact Sheet No. 307, 2008. (Accessed July 2009).
- World Health Organisation (WHO), (2008b). Asthma. WHO Fact Sheet No. 315, 2008. <http://www.who.int/mediacentre/factsheets/fs315/en/index.html>. (Accessed July 2009).
- World Health Organisation (WHO). Bronchial Asthma. Who Fact sheet No. 206, 2000. <http://www.who.int/inf-fs/en/fact206.html>. Revised January 2000. (Accessed March, 2003).

3.11 SLEEP APNOEA AND RELATED DISORDERS

Sleep apnoea is a relatively common sleep disorder that causes major sleep disruption and fragmentation and, as such, presents a serious health hazard to those affected by it. The symptoms that are of most concern in terms of traffic safety are excessive daytime sleepiness, concentration difficulties, and unexpectedly falling asleep whilst driving. These symptoms are also characteristic of narcolepsy and, to a lesser extent, snoring. Sleep apnoea has the potential to adversely affect many different body systems (Al Riyami, Al Rawas & Hassan, 2000) and may place both sufferers and other road users at risk. Fortunately, this condition can be successfully treated

3.11.1 SLEEP APNOEA

Definition of sleep apnoea

People with sleep apnoea stop breathing for 10 seconds or more at regular intervals whilst asleep. This cessation of breathing most often occurs as a result of obstruction of the upper airway (termed obstructive sleep apnoea or OSA). When this occurs, the oxygen level falls. Following an apnoea episode, the person arouses him or herself and breathing begins once again. After arousal, the person subsides back into sleep and the process is repeated many times during the night. Hypopnoea occurs when there is a 50 percent reduction in airflow or breathing, again for 10 seconds or more (Al Riyami, Al Rawas & Hassan, 2000). The severity of sleep apnoea is usually defined in terms of the Apnoea Hypopnoea Index (AHI), which refers to the number of apnoeas and hypopnoeas that the person experiences per hour of sleep (Johal & Battagel, 2001). A person with severe apnoea may have 300 to 500 of these episodes per night. Thus, the sleep pattern of individuals with OSA is completely fragmented (Grunstein, 1994).

A rare form of sleep apnoea is “central sleep apnoea” and this is caused by the intermittent failure of the central nervous system to maintain breathing. It can result from such disorders as cardiac failure and cerebral degeneration (Medical Journals, 2003). “Mixed apnoea” refers to a combination of both OSA and central sleep apnoea (Al Riyami et al., 2000).

In OSA, the obstruction of the upper airway that leads to breathing cessation occurs due to the relaxation of the dilating muscles, which fail to keep the upper airway open. Snoring has been described as an “intermediate stage” between healthy individuals who do not snore and those who are apnoeic (Bearpark, Fell, Grunstein, Leeder, Berthon-Jones & Sullivan, 1990) and is one of the main symptoms of OSA (Grunstein, 1994). Snoring occurs as a result of partial upper airway obstruction. When the person breathes in air through this restricted opening, the soft palate and nearby soft tissues vibrate and the typical sound of snoring results (Grunstein, 1994).

There are four main predisposing factors to OSA: male gender; middle age; obesity; and hereditary causes. It is unclear precisely why men are more likely to have OSA although it has been suggested that they have thicker necks and this may cause greater loading on the pharynx when lying down and thus greater narrowing of the airway (Al Riyami et al., 2000). Likewise, it is not known why middle age is associated with OSA. The effect of obesity, on the other hand, is more obvious with the extra fat around the neck compressing parts of the upper airway when the person is lying down. The hereditary factors associated with OSA may include facial structure, narrower airways

and larger than average uvulae (Al Riyami et al., 2000). Consumption of alcohol and cigarettes are also thought to predispose a person to OSA (Grunstein, 1994).

Diagnostic Tools Used to Assess Sleep Apnoea

Polysomnography

This is an overnight sleep study conducted in a sleep laboratory in which subjects are monitored whilst they are asleep. The physical signs that are measured and recorded are: respiration (that is, mouth and nose airflow), eye movements, heart rate, EEG, blood oxygen levels and movement of the chest and abdominal walls (Desai, Ellis, Wheatley, & Grunstein, 2003). This test is regarded as the “definitive” diagnostic tool for determining OSA (Johal & Battagel, 2001).

Epworth Sleepiness Scale

This test is usually used to assess excessive daytime sleepiness. People are required to subjectively rate their sleepiness. For example, they are asked to indicate the likelihood that they would fall asleep whilst watching TV, at the theatre, whilst a passenger in a car for one hour without a break, or when in a car that is stopped in traffic for a few minutes (Benbadis, Perry, Sundstand, & Wolgamuth, 1999). Those who score over 15 are regarded as having severe OSA (ESS scores range from 0 to 24) (Medical Journals, 2003).

Apnoea-Hypopnea Index (AHI)

This refers to the number of apnoea-hypopnea episodes that a person has per hour of sleep (Horstman, Hess, Bassetti, Gugger & Mathis, 2000). While different researchers use different cut-off points, individuals with an AHI of 5-15 are regarded as having mild apnoea, those with an AHI of 16-30 are said to be suffering from moderate apnoea and those with an AHI of >30 have severe apnoea (Shiomi, Arita, Sasanabe, Banno, Yamakawa, Hasegawa, Ozeki, Okada & Ito, 2002). Others have defined obstructive sleep apnoea per se as $AHI \geq 10$ (eg; Lloberes, Levy, Descals, Sampol, Roca, Sagales & De La Cladaza, 2000; Larsson, Lindberg, Franklin & Lundback, 2001).

Multiple Sleep Latency Test (MLST)

This is an objective assessment tool and measures the length of time (i.e. latency) it takes for a person to fall asleep in a quiet, darkened room whilst lying down. It is based on the premise that sleepy persons will fall asleep faster than less sleepy individuals. For a normal person, mean sleep latency is 10 to 15 minutes (Laube, Seeger, Russi & Bloch, 1998). Falling asleep in under 5 minutes is often associated with impaired performance (in Laube, et al., 1998). MLST is regarded as the “gold standard” but Aldrich (1989) did not find any difference in MLST amongst participants with sleep apnoea and participants with narcolepsy who had traffic crashes and those who did not.

Maintenance of Wakefulness Test (MWT)

In this test, the person is required to remain awake while in a quiet, darkened room. If the person falls asleep in under 15 minutes, it is recommended that they do not drive (in Laube, et al., 1998).

Prevalence of Sleep Apnoea

A large proportion (approximately 80%) of people with sleep apnoea remain undiagnosed and untreated, as many people are unaware that they have this condition (see Findley & Suratt, 2001). It is, therefore, difficult to obtain figures that reflect the true prevalence of this disease in the population. In addition, different studies that obtain data on the frequency with which this condition occurs use different methodologies and populations. The following figures are intended to give an indication only of the estimated prevalence of this condition (specific populations are noted where available):

- 1-2% in the general population and 8% in middle aged men (Medical Standards, 2003);
- Occurs in 2 - 4% of North Americans (see Lertzman, Wali, & Kryger, 1995);
- Ranges from 0.3 - 4% in the Western population. A similar range is believed to exist in the Oriental population (see Douglas, 2002);
- Occurs in 24% of working men and 9% of women aged between 30 and 60 years (see Suratt, & Findley 1999);
- 46% of truck drivers (Medical Standards, 2003);
- 27.5% ESS over 10 and 5% OSA amongst Brazilian interstate bus drivers (de Assis Viegas & de Oliveira, 2006).

Functional impairments associated with sleep apnoea relevant to driving

Most of the symptoms associated with sleep apnoea result from the disruption and fragmentation of sleep. Excessive daytime sleepiness raises the most concern in terms of traffic safety due to the propensity of individuals with sleep apnoea to ‘drop off’ at the wheel whilst driving. The symptoms most commonly experienced by people with sleep apnoea that are likely to have either short-term or long-term effects on driving include:

- Excessive daytime sleepiness;
- Nocturnal shortness of breath or choking;
- Restless or unrefreshing sleep;
- Nocturia.

Other symptoms include (from Douglas, 2002):

- Depression;
- Difficulty concentrating;
- Impaired cognitive ability (Johal & Battagel, 2001).

Less common are the following symptoms:

- Morning headaches;
- Enuresis.

Treatments for sleep apnoea and related problems

There are a number of commonly used treatments to alleviate sleep apnoea or its symptoms:

- CPAP (continuous positive airways pressure);
- Mandibular advancement appliances;
- Uvulo-palato-pharyngoplasty;
- Weight loss;
- Treatment of underlying conditions that may also obstruct the upper airway eg hypothyroidism or acromegaly.

These treatments are outlined in more detail below.

Continuous Positive Airways Pressure (CPAP)

Continuous positive airways pressure (CPAP) is the most common and effective treatment available for this condition. People with sleep apnoea are required to wear a mask every night over their nose whilst sleeping. Air is channelled through this mask and into the pharynx, holding the airway open so that breathing is not obstructed (Suratt & Findley, 1999). This approach was “pioneered” in the Royal Prince Alfred Sleep Disorders Unit, Sydney (Bearpark et al., 1990) and effects a marked alleviation of daytime sleepiness, which is symptomatic of sleep apnoea. CPAP is a treatment for sleep apnoea rather than a cure and the individual needs to wear the mask every night to obtain relief from their condition. However, some people with sleep apnoea are averse to doing this, hence it is estimated that it is often used for only about 5 hours per night (Suratt & Findley, 1999). However, other research has indicated that 90% of those with severe apnoea continue to comply with CPAP treatment even after 5 years (see Douglas, 2002). Due to the obtrusive nature of this treatment, and the greater likelihood of those with milder forms of sleep apnoea to abandon its use, it has been suggested that CPAP may not be the best treatment choice for those with few symptoms (Douglas, 2002).

CPAP appears to be an effective treatment for sleep apnoea (Bearpark et al., 1990). For those with moderate to severe sleep apnoea, CPAP has been described as the “treatment of choice” (George, 2001). Findley, Smith, Hooper, Dineen & Suratt (2000) found that in a group of 36 people diagnosed with sleep apnoea who underwent CPAP treatment for two years there was a significant reduction in the number of apnoea and hypopnea episodes per hour of sleep from a mean of 37 ± 3.8 to 2.6 ± 0 . In contrast, for the group of 14 people diagnosed with sleep apnoea who elected not to undertake the CPAP treatment, the number of apnoea and hypopnea episodes remained unchanged.

Wright, Johns, Watt, Melville & Sheldon (1997) reviewed research that evaluated the effectiveness of CPAP. A total of 44 research papers were identified – 1 small randomised controlled trial, 5 non-randomised controlled trials and 38 uncontrolled trials. Although Wright et al. (1997) conclude that much of the research that has evaluated the effectiveness of CPAP in reducing daytime sleepiness has been “poorly evaluated” they did report that this treatment has consistently been found to exert a small but positive effect on the reduction of objectively measured daytime sleepiness.

Dental devices (mandibular advancement appliances)

Mandibular advancement appliances are dental devices that are placed over the upper and lower teeth and push the lower jaw forward to about 75% of its maximum protrusion (Medical Journals, 2003). This stops the tongue from falling backwards during sleep and thus causing the throat to narrow (Douglas (2002). Radiographs have shown that these devices increase the airway space (Johal & Battagel, 2001).

This technique may be useful for those with mild to moderate sleep apnoea (Suratt & Findley, 1999; Johal & Battagel, 2001). While these devices are not as effective as CPAP treatment, they have been shown to reduce the number of apnoea and hypopnoea episodes per hour of sleep, and reduce daytime sleepiness as well as snoring (see Douglas, 2002). Johal & Battagel, (2001) cite research that reported a 45% reduction in AHI scores following treatment. In addition, mandibular advancement splints are viewed as being less obtrusive than CPAP. Some of the drawbacks associated with mandibular advancement splints include a sore or aching mouth, teeth displacement and the production of excessive amounts of saliva (Douglas, 2002). It has been suggested that mandibular advancement appliances are a good alternative for those who cannot (or will not) undergo CPAP treatment (Johal & Battagel, 2001).

Uvulo-palato-pharyngoplasty & other surgical options

Uvulo-palato-pharyngoplasty (UPPP) is a surgical procedure in which the soft palate and pharynx are removed. Other surgical options include removal of the tonsils if they are enlarged, tracheostomy, epiglottoplasty, or removal of any tumours that may be obstructing airflow.

Surrat & Findley (1999) report that surgically removing the soft palate offers improvement for 50% of people with sleep apnoea. It appears however, that there has not been any trial-based research to evaluate the effectiveness of surgery in the treatment of OSA. Bridgman and Dunn (1997) undertook a review of research that evaluated the effectiveness of surgery in the treatment of OSA. This review resides in the Cochrane Library. A total of 594 relevant articles were identified and assessed according to set inclusion criteria. The inclusion criteria were that subjects have a diagnosis of OSA (defined as more than 5 apnoeas or hypopnoeas per hour of sleep) and had been treated with surgery. Treatment efficacy was to be assessed using either randomised or quasi-randomised comparisons to other treatments or to no interventions. Unfortunately, none of the articles satisfied the inclusion criteria. This finding prompted Bridgman and Dunn (1997) to suggest that surgery for OSA should be conducted as part of clinical trials, or if not, individuals ought to be informed of the “experimental nature” of the surgery. In a subsequent update in 2003, Bridgman, Dunn and Duchrane (2003) report that the situation regarding the dearth of randomised controlled trials evaluating the efficacy of surgery in OSA treatment remains unchanged.

Further, Douglas (2002) cites research that indicates that people with OSA who have had surgery and are then subsequently treated with CPAP may, in fact, suffer detrimental effects. It also appears that in some instances UPPP is associated with peri-operative complications, including death.

Weight loss

For those whose sleep apnoea is mild to moderate and who are also obese, weight loss is another effective treatment. However, Surrat & Findley (1999) state that the size of the weight loss must be fairly substantial for it to have a positive effect on sleep apnoea. In addition, this approach is particularly effective if weight is lost from around the neck.

Sleep positions

In a very few cases, merely changing the position of the body from the supine posture during sleep is sufficient to alleviate any obstruction that may be responsible for OSA (Medical Journals, 2003).

Comorbidity

Sleep apnoea appears to place the person at an increased risk for a wide range of other disorders: cardiovascular, cerebrovascular, endocrine, and psychological. It is also associated with increased mortality (Al Riyami, et al., 2000). Shortened life span (approximately 5 years) has been found amongst people with untreated sleep apnoea compared to those who have OSA who have undergone CPAP or tracheostomy treatment. However, it appears that cardiovascular problems are the predominant reason for increased mortality (see Al Riyami, et al., 2000).

3.11.2 NARCOLEPSY

Definition of narcolepsy

Narcolepsy is a rare, genetically linked sleep disorder, which leaves the individual feeling profoundly sleepy during the day (Grunstein, 1995), even when sufficient sleep has been obtained at night. Individuals with narcolepsy are prone to sudden “sleep

attacks” and fall asleep with or without warning during the day. Other symptoms of narcolepsy are cataplexy, sleep paralysis and vivid hypnagogic hallucinations (Medical Standards, 2003).

Prevalence of narcolepsy

According to the Medical Standards (2003), approximately 0.06% of the population suffers from narcolepsy.

Functional impairments associated with narcolepsy relevant to driving

The major impairment associated with narcolepsy of concern for road safety is the propensity to have a sudden ‘sleep whilst driving. During episodes of cataplexy, the person may experience muscular problems ranging from weakness to complete collapse. No loss of consciousness occurs during these episodes (Sleepnet, 2000).

Pre-May 2003: Relationship between sleep apnoea and related disorders and road safety outcomes

In considering the evidence for rates of crashes and measures of driving performance reviewed in this section, it is important to bear in mind a number of methodological limitations/considerations that may partly explain some of the variation in findings.

Methodological shortcomings of some of the research regarding the risk of car collisions for drivers with sleep apnoea have included small samples, reliance on retrospective self-reports, lack of verification of crashes using independent databases such as police and insurance records, lack of consideration of the possible confounding effects of alcohol and drug consumption and incorrect or inadequate diagnosis of sleep apnoea (not based on a full polysomnography) and a lack of control for severity of OSA (Barbe, Pericas, Munoz, Findley, Anot & Agusti, 1998).

With regard to diagnostic criteria for OSA, many studies use the Epworth Sleepiness Scale to assess daytime sleepiness. This scale requires respondents to subjectively rate their level of sleepiness. However, people with OSA are not particularly proficient at assessing their own sleepiness, compared to healthy controls (see Desai, Ellis, Wheatley & Grunstein, 2003). Therefore, this scale may not be the best predictor to use for daytime sleepiness (Horstman et al., 2000). This may be a factor to consider in some studies that do not find a significant relationship between excessive daytime sleepiness and crashes amongst sleep apnoeics.

As discussed elsewhere in this review, few studies include an adjustment for driving exposure. Horstman et al. (2000) point to the differences in observation periods for road safety outcomes across different studies, which may impact on the results (number of crashes). The authors also comment on the common finding that people with OSA tend to drive more than controls, possibly due to the fact that people with OSA who rely on driving for their employment may be more likely to seek medical help for their disorder in a bid to minimise the possibility of work-related driving crashes.

As discussed in Chapter 2, while studies assessing driving performance are useful in identifying particular aspects of driving that might be negatively affected by sleep apnoea, the question remains as to how such impairments are linked to crash risk. A

summary of the studies investigating sleep apnoea and road safety outcomes is presented in Table 34.

Crashes

Barbe, Pericas, Munoz, Findley, Anot and Agusti (1998) undertook an analysis of the effect of sleep apnoea syndrome (SAS) on the risk of car collisions using 60 participants (59 male, 1 female) recruited from a sleep laboratory and 60 healthy controls matched for sex and age (± 5 years). The mean age of participants with SAS and controls was 47 years (± 1 year). The participants with SAS were selected if they fulfilled the following inclusion criteria: more than 20 apnoeas-hypopnoeas per hour while they were undergoing a full polysomnography; had a valid driver's licence and were permanent residents in Mallorca, Spain. Those who abused drugs, were shift workers, had a psychiatric disorder, had other sleep disorders (eg narcolepsy or periodic leg movement disease), or had epilepsy were excluded. Controls were also chosen using all of the above criteria (apart from the apnoea-hypopnoea episodes). To ensure that the controls did not have undiagnosed OSA, their medical history was examined and, when indicated, a full polysomnography was conducted.

The interesting aspect of this research was that it also assessed the relationship between some of the individual, theoretical risk factors (eg; daytime somnolence, anxiety, depression, the severity of OSA as measured by the number of respiratory events and nocturnal hypoxaemia, and vigilance levels) and the actual risk of car crashes. Exposure data (number of kilometres travelled) was also collected and controlled for. Data were collected using both self-report and standardised clinical questionnaires, self-reported crash rates were verified using insurance databases, and driving performance was assessed using a 30-minute computer simulation. To examine some of the aforementioned theoretical risk factors, the Epworth Scale was used to determine levels of daytime sleepiness, the Beck questionnaire was utilised to assess depression and anxiety and the Psychomotor Vigilance Test was employed to gauge vigilance levels.

On average, participants with SAS exhibited 58 ± 3 apnoeas-hypopnoeas per hour, with a range of 21 to 101. In comparison to controls, the participants with SAS had a higher mean alcohol consumption (with significant differences for weekend alcohol consumption), a higher body-mass index (33 ± 0.8 versus 27 ± 0.8 , $p < 0.001$), higher intake of benzodiazepines (although ingestion of all other prescription medications was similar), higher levels of daytime sleepiness, depression and anxiety, and poorer performance on the computer simulation test with slower reaction times and higher degrees of reaction fatigue.

Looking at automobile crash rates, it was found that in the preceding three years more participants with SAS (33 %) than controls (18%) had experienced a crash (OR: 2.3; 95% CI; 0.97 to 5.33, $p = 0.06$). In addition, participants with SAS had a higher mean number of crashes (0.53 ± 0.1 versus 0.22 ± 0.06 , $p < 0.05$), with participants with apnoea more likely to have been involved in more than one crash (OR: 5.2; 95% CI: 1.07 to 25.29, $p < 0.05$).

Participants with SAS drove more kilometres per year than controls however, even after controlling for this, it was found that those with SAS still displayed increased crash rates of a "similar magnitude", and that the likelihood of having one or more crashes amongst those with SAS increased marginally (OR: 2.6, 95% CI; 1.06 to 6.43, $p < 0.05$). Surprisingly, Barbe et al. (1998) did not find the severity of apnoea to be

associated with crash risk. Neither did they find a relationship between depression, anxiety, daytime sleepiness and automobile crashes. While there was a clear increased risk of crashes amongst participants with SAS with slower reaction times and greater reaction fatigue, these differences were not significant. When commenting on this finding, the authors speculated that, had they used a larger sample, this relationship may well have become significant. Finally, there was no significant correlation between crash rates and performance on the computer simulated driving task.

Horstman, Hess, Basetti, Gugger and Mathis (2000) investigated the frequency of crashes amongst a group of 160 participants with SAS retrospectively recruited from a sleep laboratory. One hundred and sixty healthy controls were also selected from the same out-patient clinic. Crashes were measured using a strictly anonymous questionnaire. The severity of sleep apnoea amongst participants was determined from the results of a polysomnography – those with an AHI \leq 34 were deemed to have mild SAS and those with an AHI \geq 35 were categorised as having moderate to severe SAS. The extent of daytime sleepiness experienced by both participants with SAS and controls was assessed using the Epworth Sleepiness Scale (ESS) and, as expected, was found to be significantly higher in participants than controls and higher in those with moderate to severe SAS compared with mild SAS.

In an effort to overcome the effect of under-reporting of crashes due to legal concerns, Horstman et al. (2000) used a strictly confidential questionnaire to gather information on participants' crashes. However, this approach still does not address the issue of recall bias due to memory lapses. A strength of this study is that it considered exposure data (crashes per million km driven). However, the distinction made between two severity levels of apnoea - mild and moderate/severe - was somewhat different from that made by other researchers. Participants with SAS and controls were matched for age (56.5 years and 56.2 years, respectively) and sex (~90% males), and were also similar in terms of alcohol consumption (participants with SAS = 6.7 glasses per week and controls = 6.5) and holding a driving licence (83% participants with SAS and 87% controls). The control group was not drawn from the general population but from the same out-patient clinic as the participants with SAS. This may have resulted in a group that was not representative of the (Swiss) population as a whole.

Significantly more participants with SAS (12.4%) reported crashes than controls (2.9%). In addition, participants with SAS had a greater frequency of multiple crashes compared to controls. The number of crashes per million kilometres driven was significantly lower for controls (0.78) than the combined groups of participants with SAS (6.8), $p < 0.005$. In addition, those diagnosed with moderate to severe SAS had significantly more crashes per million kilometres driven (13.0) compared with participants with mild SAS (1.1), $p < 0.05$. Horstman et al. (2000) also calculated that those with untreated moderate to severe SAS had a 15.5 fold crash risk compared to healthy controls, although no statistical analysis was presented.

This research did not find a significant association between self-ratings of daytime sleepiness and crashes, either in participants with SAS or controls. In addition, Horstman et al. (2000) report little difference in crashes for participants with mild SAS and controls (1.1 crashes per million km driven and 0.78 crashes per km driven, respectively). They concluded from this that a “diagnosis of SAS as such does not seem to be sufficient to predict driving impairment” (p6). Nevertheless, the results clearly demonstrate that level of SAS severity is a critical variable influencing crash risk.

Masa, Rubio and Findley (2000) interviewed a total of 4,002 randomly selected drivers in a western city in Spain to identify those who habitually experienced sleepiness whilst driving. 145 drivers fit this criterion. An age and gender matched control group was selected at random from the remaining 3,857 non-sleepy drivers. A questionnaire was used to elicit information including MVCs in the last 5 years, driving exposure, sleeping patterns, occupation, height, weight and other body measurements and an index of sleepiness as measured by the Epworth Sleepiness Scale.

The results show that, as a group, the habitually sleepy drivers were predominantly male and middle-aged and exhibited many of the symptoms that are seen in those with respiratory-related sleep disorders: snoring, apnoeic episodes, morning fatigue and higher scores on the Epworth Sleepiness scale. Fifty percent of the habitually sleepy drivers reported excessive daytime sleepiness (a score ≥ 9 on the Epworth scale). Using all nocturnal respiratory events as an index of respiratory sleep disorders (i.e. apnoeas, hypopnoeas, and other arousals caused by “increased respiratory effort” during sleep), Masa et al. (2000) calculated a “total respiratory event index” by adding these other arousals to the AHI index: habitually sleepy drivers had a significantly higher number of nocturnal respiratory events than controls (for sleepy drivers with a total respiratory index of ≥ 15 , the adjusted OR: 6.0, 95%CI 1.1 - 32).

The habitually sleepy drivers reported a significantly higher frequency of crashes than controls, in fact, almost 10 times the number of crashes (adjusted OR was 13.3, 95%CI 3.1 - 43). This result was still significant after the number of hours driven was taken into account. Within the group of habitually sleepy drivers, however, there was no statistical difference in the AHI index for those who had been in car crashes and those who had not. This last finding is at odds with other research, which indicates that there is a higher frequency of crashes amongst participants with sleep apnoea with a high AHI index (indicating severe sleep apnoea) (eg Findley, Fabrizio, George & Suratt, 1989; George & Smiley, 1999).

This was a comprehensive study however, as with many other studies of this kind the soundness of findings relies on the validity of the retrospective self-reporting of crashes.

Shiomi et al. (2002) sought to investigate the relationship between severity of sleep apnoea and automobile crashes and compared the crash frequency of participants with sleep apnoea and participants who snore. A total of 554 participants (mostly male with a mean age 49.2 ± 14.3) were recruited from the Sleep Disorders Centre at a Japanese Medical University Hospital. Of these, 448 were diagnosed with sleep apnoea and 106 were “simple snorers”. Crash data were elicited using questionnaires, sleepiness ratings were obtained using the Epworth Sleepiness Scale (ESS) and AHI was measured using a polysomnography. Mild apnoea was defined as AHI of 5-15; mild to moderate apnoea = AHI of 15-30; and severe apnoea = AHI > 30 . A “simple snorer” was a person with an AHI < 5 . Excessive daytime sleepiness was defined as a score of > 11 on the Epworth Sleepiness scale and/or an AHI > 15 .

Shiomi et al. (2002) reported that the participants with severe sleep apnoea had a significantly higher frequency of car crashes than the “simple snorers”. It is worth noting that the four snorers who had been involved in car crashes had high levels of excessive daytime sleepiness (ESS score ≥ 15). As can be seen from Table 38 the frequency of car crashes increases with the severity of sleep apnoea – a finding that has been demonstrated in other studies. The researchers note that the principal reason for the automobile crashes was falling sleep at the wheel whilst driving.

Table 38 Comparison of MVCs of drivers with apnoea with different severity levels and snorers

Condition	Simple Snorer	Mild Apnoea	Moderate Apnoea	Severe Apnoea	All Apneics
Sample size (n)	106	156	111	182	448
AHI	<5	5-15	15-30	>30	>5
MVC Rate	3.8%	5.8%	9.9%	11.0%	8.9%

One of the strengths of this study was the large sample size. It did, however, rely on the participants' self-report of crashes that had occurred over the last five years.

Aldrich (1989) compared the driving records of 424 participants (279 males and 145 females) who had one of four types of sleeping disorders (apnoea, narcolepsy, other sleep disorders with excessive daytime sleepiness and sleep disorders without excessive daytime sleepiness) and the driving records of 70 control participants (approximately age and gender matched). In addition, the relationship of the severity of sleep apnoea and narcolepsy to the frequency of crashes (across entire driving history) was investigated. Information pertaining to car crashes and near-misses (i.e. driving off the road) was elicited using self-report questionnaires, and sleep disorder identification and severity were measured via nocturnal polysomnography, multiple sleep latency tests and medical records.

The group of sleep disorders with associated excessive daytime sleepiness (EDS) comprised people with periodic leg movements, those with insufficient sleep, and people with sleepiness induced by medication or mental illness or from unknown causes. The group of sleep disorders without EDS contained insomniacs, parasomniacs, those with subjective sleepiness only, sleep disturbance from unknown causes and individuals with "schedule disturbances".

Due to the historically higher crash involvement of males, the frequency of crashes and near-misses for each gender was analysed separately. In this study, 200 males (72%) and 96 females (66%) reported crashes. As can be seen from Table 39, none of the participant groups (except females with other sleep disorders without EDS) had a higher *overall* crash rate than their respective controls. However, when participants were asked to estimate the number of sleep-related crashes with which they had been involved, large differences were apparent between controls and participants with sleep disorders. Sleep-related crashes were also reported and were generally significantly higher in drivers with narcolepsy and drivers with EDS compared with controls. However, this was not of direct relevance to the main consideration of this review.

Table 39 Crash and near crash frequency for males and females

MALES	Apnoea n=181	Narcolepsy n=25	Other- EDS n=35	Other-no EDS n=38	Controls n=35
Mean age	50	42	47	48	43
% participants					

with MVCs with any cause	71%	76%	69%	74%	79%
FEMALES	Apnoea n=47	Narcolepsy n=31	Other- EDS n=26	Other-no EDS n=41	Controls n=35
Mean age	47	31	26	41	35
% participants with MVCs with any cause	68%	48%	62%	80%	74%

EDS = excessive daytime sleepiness, MVCs = motor vehicle crashes

Aldrich (1989) stated that there were no significant differences in mean sleep latency as measured by the MLST between participants who had crashes and those who did not (6.6 minutes vs. 7.3 minutes, respectively). He also points out that some people with sleep disorders may self-regulate their driving and this may be why the participants with EDS did not have higher percentages of crashes from any cause (compared to controls) despite having higher proportions of sleep-related crashes. However, as this study did not gather driving exposure data, it is difficult to estimate the extent of any self-regulation. Other serious limitations of this study include the lack of control for variables such as age and years of driving. This is likely to have lead to a bias in estimates of crash involvement since this measure was based on self-reported frequencies of crashes for drivers' entire driving history.

Bearpark, Fell, Grunstein, Leeder, Berthon-Jones and Sullivan (1990) compared the self-reported driving behaviour of 288 controls with two participant groups recruited from an overnight sleep study in a sleep laboratory: snorers (n = 34) and participants with sleep apnoea (n = 101). Participants were defined as having sleep apnoea if they exhibited an AHI of more than 10 (i.e. more than 10 apnoea episodes per hour of sleep). Snorers either did not have apnoea at all or had fewer than 10 episodes per hour of sleep. Controls were screened for apnoea using two indices that are correlated with its presence – Body Mass Index (BMI) which is used to measure obesity, and scores on a “7 item mini-sleep questionnaire”. Participants were similar in variables that might influence their driving ability or crash propensity: age, driving history, and alcohol consumption. All participants were male, with participants with sleep apnoea having a mean age of 52.6 years, snorers 49.8 years and controls 53.4 years. There was no significant difference between the three groups for job-related driving or being a professional driver.

Participants were asked about the number of sleep-related crashes and near-misses that they had been involved in, whether or not they had fallen asleep at the wheel while driving or at traffic lights, and if they had ever pulled off the road due to sleepiness. There was a significant difference in the number of participants with sleep apnoea reporting crashes (19%) compared to snorers (3%) and controls (8%). In addition, a significantly higher percentage of participants with sleep apnoea (57%) indicated that they pulled off the road because they felt sleepy compared to controls (33%). There was also a significant difference between participants with sleep apnoea and controls for sometimes falling asleep at the wheel whilst waiting for traffic lights (15% vs. 1%, respectively). Falling asleep while driving was also more prevalent amongst participants with sleep apnoea than controls (22% vs. 3%, respectively). Snorers, too, reported a high incidence of falling asleep whilst driving, with 21% of this group doing so. Bearpark et al. (1990) concluded from these findings that participants with sleep apnoea and snorers face a greater risk of having sleep-related crashes due to the high levels of

daytime sleepiness that accompanies these disorders and should, therefore, be considered as “high risk groups”.

Lloberes, Levy, Descals, Sampol, Roca, Sagales and De La Cladaza (2000) also compared the self-reported sleepiness of 122 participant with sleep apnoea ($AHI \geq 10$), 67 snorers and 40 controls. The controls were drawn from hospital staff and were matched for age and gender. Participants with sleep apnoea and snorers had been referred for a sleep study due to suspected OSA and underwent a night polysomnography. Results showed that self-reported sleepiness was significantly higher amongst the OSA group compared to either the snorers or the controls (43%, 34% and 5%). Likewise, participants with OSA reported higher number of sleep-related crashes compared to snorers and controls (9%, 1.5% and 0%). Interestingly, self-reported sleepiness was associated with a higher risk of crashes. Other studies have found only weak associations between sleepiness and other measures of driving performance when assessed using standard objective tests such as the Multiple Sleep Latency Test (eg; George, Boudreau & Smiley, 1996). As with Bearpark et al. (1990) outlined above, this study also included self-reported driving off the road. This data can be likened to “near-miss” types of crashes – information that would not be reported to police or insurance companies. The results indicate that participants with sleep apnoea had a significantly higher number of such incidents than did either snorers or controls. Apart from self-reported sleepiness, other variables found to be associated with an increased risk of crashes were driving cessation due to sleepiness (OR: 3, 95%CI 1.1 - 8.6) and being in employment (OR: 2.8, 95%CI, 1.1 - 7.7).

The limitations of this study concern the usual MVC self-report issues and that controls were not required to undergo a polysomnography to detect the presence of sleep disorders or snoring. More importantly, as with the study by Bearpark et al. (1990), this study examined sleep-related crashes only, and is therefore unlikely to be representative of involvement in all types of crashes.

Citations

No studies reporting rates of citations or violations amongst drivers with sleep disorders were found.

Driving Performance

Using a computer simulator, George, Boudreau and Smiley (1996) compared the driving performance of three groups of people: 21 participants with untreated OSA, 16 people with untreated narcolepsy, and a group of 21 healthy controls. The computer simulation assessed the two primary tasks associated with driving: tracking and visual search.

Tracking error performance was significantly worse in participants with a sleep disorder compared to controls: 228 ± 145 cm for participants with sleep apnoea, 196 ± 146 for participants with narcolepsy, and 71 ± 31 for controls, $p < 0.001$. However, not all participants with a sleep disorder demonstrated worse performance than controls. As with other studies, it was found that sleepiness as measured by the standard Multiple Sleep Latency Test (MSLT) was only weakly associated with tracking performance. Approximately half of the participants with sleep apnoea and half of the participants with narcolepsy returned performances that were as good as, or better than that of controls. Such a finding has been reported in other studies. George et al., (1996) state

that this result raises questions as to which specific groups of sleep apnoeics and narcoleptics are unfit to drive.

In a bid to understand which particular aspect of OSA causes driving impairment, Hack, Choi, Vijayapalan Davies and Stradling (2001) compared the driving performance on a computer simulator of a group of 26 participants with OSA with that of a group of 24 control participants. Control participants were assigned to a condition of either one night's sleep deprivation (12 participants) or alcohol consumption to just below the legal limit in the UK (12 participants). The two conditions for the "normal" group were selected for comparison because alcohol primarily impairs cognitive functions needed for driving whereas sleep deprivation interferes with vigilance.

All participants also served as controls by the following process: participants with OSA were given CPAP treatment, the sleep-deprived group had a normal night's sleep, and the alcohol ingestion group abstained from alcohol. Pseudo-randomisation was used to assign the health control participants to either the control or experimental group (eg 6 participants went without sleep for 24 hours and the remaining 6 participants had a normal first night's sleep. 6 participants in the alcohol group drank grapefruit juice by itself first and the remaining 6 drank it laced with vodka first).

All participants in the "experimental conditions" (i.e. untreated OSA, sleep deprivation and alcohol consumption) returned significantly worse performances on the driving simulator than the controls (i.e. OSA treated with CPAP, a normal night's sleep and grapefruit juice only). The results also show that the driving performance of OSA's lay between that of the two health control groups (i.e. sleep deprived or alcohol-impaired). Analyses of the steering errors committed during the simulation indicated that for the drivers who consumed alcohol, steering was impaired throughout the entire simulation whereas for the sleep-deprived participants steering was normal to begin with and then deteriorated progressively throughout the remainder of the simulation. The steering performance of the OSA group resembled that of the sleep-deprived group. This indicates that the poorer driving performance found in participants with sleep apnoea may be the result of vigilance decrements rather than defects in cognitive or motor skills.

The subjects in the normal group were considerably younger, had lower body weight and had been licensed for a much shorter time than the group of OSA participants. However, in partial refutation to this, Hack et al. (2001) point to the results of another study which showed that an older control group returned steering performance results and reaction times that were similar to the younger control participants in the present study.

A limitation of the studies reviewed above examining driving performance and SAS is the absence of a link with real-world crash risk. Only one study of sleep apnea was found which does provide insight on the question of crashes and driving performance. The study, by Barbe et al. (1998), described in detail above, examined crash rates and driving simulator performance in 60 people with sleep apnoea syndrome (SAS) and 60 healthy controls. Compared with controls, the participants with SAS had a poorer performance on the computer simulation test with slower reaction times and higher degrees of reaction fatigue. However, for this sample, there was no significant correlation between crash rates and performance on the computer simulated driving task.

Treatment of sleep apnoea and related disorders and road safety outcomes

Pre-2003 a number of studies have investigated the relationship between sleep apnoea and crashes with a focus on CPAP treatment. One driving simulator study investigated driving performance and untreated obstructive sleep apnoea.

Crashes

Findley, Smith, Hooper, Dineen & Suratt (2000) investigated the effect of CPAP on the frequency of car crashes in 50 participants diagnosed with sleep apnoea (43 males and 7 females). Participants were recruited from a sleep laboratory in Northern Colorado, USA. Participants were classified as having sleep apnoea if they had 5 or more apnoeas-hypopnoeas per hour of sleep. Thirty-six participants with sleep apnoea used CPAP treatment and 14 participants elected not to use CPAP. Both of these groups of participants were matched in terms of age (mean of 56 ± 2 years), weight (mean of $233\text{lbs} \pm 80\text{lbs}$), number of apnoeas and hypopnoeas per hour of sleep (mean of 37 ± 3.8), and gender.

All participants completed a questionnaire and a telephone interview in which they were asked about their traffic crash history two years prior to diagnosis and also for the ensuing two years when they were either on CPAP treatment or had refused it. Only at-fault crashes that resulted in property damage over \$500 or personal injury and a traffic conviction were included in the analysis. Participants were also asked to give an approximation of the number of kilometres they travelled pre- and post-diagnosis. Unlike any previous study on crash rates and sleep apnoea, Findley et al. (2000) then cross-matched the subjects' self-reported crashes with their official crash records held by the Colorado Department of Motor Vehicles. In addition, crash rates were compared to those for the general population in Colorado as well as for drivers in the general population with the same demographics as the participants with sleep apnoea in this study.

Findley et al., (2000) found that in the two years prior to diagnosis the participants with sleep apnoea had an average rate of 0.07 crashes per person. This was significantly higher than the crash rate in the general population (0.01 crashes per person, $p < 0.02$) and is significantly higher than the demographically adjusted general population group. Participants with sleep apnoea who undertook CPAP treatment experienced no crashes during the 2 years that they were being treated (a significant reduction, $p < 0.03$). In comparison, the number of crashes in the sleep apnoeic group that opted not to undergo CPAP treatment remained unchanged at 0.07 crashes per person. In addition, the number of sleep apnoeas and hypopnoeas per hour of sleep significantly decreased in the group receiving CPAP treatment from 37 ± 3.8 to 2.6 ± 0.8 .

The authors concluded that participants with sleep apnoea on CPAP treatment may not need to have their licence revoked as they do not appear to pose an increased traffic safety risk either to themselves or to others. As an interesting aside, Findley et al. (2000) reported that participants with sleep apnoea under-reported the number of crashes that they had been involved in - they only acknowledged one-third of these. In addition, a further 4 crashes were denied and 2 crash-involved participants declined to answer the question on crashes.

In the study described above, Horstman et al. (2000) also compared the effect of CPAP treatment on a sub-group of 85 participants with a sleep disorder – these participants

completed 2 questionnaires covering the periods before and during treatment. CPAP treatment was efficacious in reducing in both the mean number of crashes and sleepiness ratings as measured by the Epworth Sleepiness Scale. During CPAP treatment, the mean number of crashes per million kilometres driven dropped significantly from 10.6 to 2.7 ($p < 0.05$), representing a reduction of approximately 75%. Sleepiness ratings also displayed a significant reduction from 13.3 to 6.7 ($p < 0.001$). The researchers suggested that, based on this finding, it is entirely appropriate to allow participants with SAS who have undergone CPAP treatment to drive.

While the study by Findley et al. compared crash rates with those of the general population, no control group was used in the experiment and sample sizes were also small. Similarly, Horstman et al. (2000) did not compare crash rates of treated participants over the study period with controls.

George (2001) obtained similar results to Findley et al. (2000) but used larger sample sizes and included a matched control group. The driving records of 210 participants with sleep apnoea identified via an overnight polysomnography with AHI 10 events per hour were compared to those for a control group drawn from the general population and matched for age, gender and class of driver's licence (private or commercial). Driving records for all subjects were obtained from the Ontario Ministry of Transportation. All of the participants with sleep apnoea received CPAP treatment over a period of 3 years. At the time of follow-up, 182 participants with sleep apnoea were still using CPAP and 27 were not (5 others had undergone surgery and the remaining 6 had died). George (2001) reported that, in the 3 years prior to diagnosis, the participants with sleep apnoea had a significantly higher crash rate than the controls during the same time frame (0.18 crashes per person per year vs. 0.06 crashes per person per year, $p < 0.001$). Following the 3-year CPAP treatment, crashes for the participants with sleep apnoea fell to the same level as the controls (i.e. 0.06 crashes per person per year). During treatment, single crashes dropped by approximately 50% and multiple collisions declined even more. For the 27 participants who were not current CPAP users at the time of follow-up, the number of crashes remained high (0.15 vs. 0.14 crashes per person per year).

The self-rated driving exposure of the OSA group was similar pre-and post-treatment. Unfortunately, no driving exposure data were obtained for the control group and no polysomnographic data were available for the OSA group. Notwithstanding these limitations, the central finding that participants with CPAP treated sleep apnoea display a decrease in the number of crashes remains.

As with other studies, George (2001) points out that while participants with sleep apnoea as a group have a higher frequency of crashes, there are many who have no car crashes. As suggested by George, Boudreau and Smiley (1996), this finding raises the question as to whether particular sub-groups of OSA participants are at greater risk.

Driving performance

Findley, Fabrizio, Knight, Norcross, Laforte and Suratt (1989) compared the performance of people with severe, untreated OSA to that of healthy controls using a driving simulator and several films of different types of roads (rural, city and highways). They also measured performance on a computer simulator. In addition, the performance of participants with sleep apnoea prior to and after receiving CPAP treatment was also measured. The participants with OSA were recruited from the

University of Virginia Sleep Disorders Lab and the age and gender matched controls were selected from among university staff and their families. The authors reported that the 6 participants with severe OSA performed significantly more poorly than the 7 controls on all road types. During the highway road film, participants with sleep apnoea recorded 39 ± 5 correct responses compared to the controls who registered $52 \pm 9\%$ correct responses ($p < 0.01$). For the city/rural road films, a similar pattern emerged between participants with sleep apnoea and controls (41 ± 12 vs. $58 \pm 14\%$ correct responses, $p < 0.05$).

The differences between performance levels of the 2 groups were even more pronounced on the computer simulation, which also depicted a highway scenario. Participants with OSA hit almost 5 times the number of road obstacles as the controls (44 ± 52 vs. 9 ± 7 , $p < 0.05$). The authors speculated that a possible explanation for the even worse performance on the computer simulator may be that it is less stimulating than the driving simulator and takes longer to complete. And finally, the six participants with OSA who received CPAP treatment hit fewer obstacles following treatment than they did prior to treatment (29 ± 19 before CPAP vs. 13 ± 8 after CPAP, $p < 0.05$). There was no significant difference between the performances of the OSA sufferer's following CPAP treatment and that of controls.

In addition to confirming the general consensus that participants with sleep apnoea perform more poorly on driving tasks than normal controls, and that CPAP treatment restores driving ability to a level that is similar to that of controls, this study was also interesting in that it provided a comparison between performance on computer simulators and driving simulators. It also contrasted driving performance on different types of (simulated) roads. However, the sample sizes in this study were very small and the researchers did not take account of any other contributory factors that may have impacted on the participants' performance.

Post-May 2003: Relationship between sleep apnoea and related disorders and road safety outcomes

Review of literature between May 2003 and mid 2009 revealed that six studies investigated the relationship between crash risk and sleep apnoea and related disorders. There were no studies conducted using citation rates and six studies conducted which investigated driving performance.

Crashes

The incidence of sleep debt, sleepiness and crashes in a male population including heavy vehicle drivers in Sweden was investigated by Carter et al. (2003). Questionnaires were mailed to drivers recorded on several databases of professional drivers. One thousand and thirty-four (74%) of the drivers responded appropriately and served as the study subjects. Controls were recruited by means of questionnaires sent to 4000 men on the tax register of which 2608 (66%) responded. Of these, 1865 were used as the reference group. The questionnaires were comprehensive and included occupational, demographic and accident data as well as questions related to sleep, snoring and apnoeas. The ESS was included in the study. A random sample of 180 professional drivers was offered OSA screening by overnight oximetry and a static sensitive bed of whom 161 accepted.

The results showed professional drivers to be slightly heavier than controls (BMI 26.9 vs 26, $p < 0.001$) and they had a slightly higher average ESS (7.1 vs. 6.7, $p = 0.02$). Crashes while driving professionally were slightly higher in the study group (36.6% vs. 32.5%, $p = 0.03$) but much more frequent in leisure driving (13.8% vs. 8.6%, $p < 0.0001$). Professional drivers reported more sleep debt than controls ($p < 0.001$) and crashes during leisure driving was increased in relation to sleep debt ($p < 0.001$). Commuting crashes in the control group were also increased with sleep debt ($p = 0.006$). The authors concluded that sleep debt and reporting sleepiness rather unsurprisingly predicted an increased incidence of crashes. The study did not find, however, that sleep disordered breathing was a risk factor.

A similar study was conducted in Australia by Howard et al. (2004). Questionnaires were sent to 3,268 commercial vehicle drivers and responses were received from 2,342 (72%), 99 of whom were male. Another sample of 161 drivers undergoing polysomnography were included in the study. There were few demographic differences between the two groups. There was a high incidence of OSA or other types of sleep disordered breathing in both groups (59.6% in the polysomnography group, 54% in the survey group). Just over one third (35.5%) of the drivers had a total of 1,407 crashes in the previous 3 years with 48.3% having more than one crash. Most crashes were work related. There was an increased risk of a crash with increasing sleepiness. Those with an ESS of 18 to 24 had an Odds Ratio for a crash of 1.91 and 2.67 for multiple crashes. Similar relationships were found for other measures of sleepiness. There was increased risk of a collision in those drivers who admitted to using sedating medications such as narcotic analgesics or anti-histamines. The high prevalence of drowsy driving, obesity and sleep disordered breathing in this population and the associated crash risks were the significant findings in this study.

A survey of recently crashed drivers to determine the role of sleepiness was undertaken by Crummy et al. (2008). A group of 112 drivers admitted to a major trauma centre were approached to participate in the study. After excluding those who were intoxicated, psychiatrically unwell, subject to police investigation, unable to consent or refused, forty drivers were asked to complete a questionnaire regarding sleepiness (acute or chronic), circadian rhythm disturbance, sleep disordered breathing or other sleep disorder. The study group consisted of 25 males and 15 females with an age range of 18 to 81 years. Nineteen were shift workers.

The drivers did not report high levels of sleepiness prior to their crashes. One driver had a prior diagnosis of OSA and one of restless legs syndrome. One driver was taking benzodiazepines regularly. Almost half the drivers had at least one risk factor for a sleep related collision including shift work and prolonged driving periods prior to the crash. The high proportion of shift workers suggests that circadian rhythm disturbance and chronic sleep deprivation may be an important factor in sleep related crash risk.

A similar study was conducted in New Zealand by Kingshott et al. (2004). Sixty drivers who had been involved in a collision in the last 24 months were compared with an age, BMI and gender matched control group. Inclusion criteria were age between 30 and 70 years, blood alcohol below legal limit, driver in single vehicle crash or causative driver in multiple vehicle crash. Drivers with severe medical conditions were excluded. All subjects underwent polysomnography and a battery of other test including Maintenance of Wakefulness (MWT), subjective sleepiness ratings and computerized performance tests.

There were no significant polysomnographic differences between the two groups and no cognitive differences. There was a small difference of borderline significance in mean MWT latency between the groups (cases 17 min, controls 18 min $p = 0.06$). There was a small difference in reaction times ($p = 0.02$). The crash-involved cases reported more subjective sleepiness than controls ($p = 0.003$), although this was not translated to traditional measures such as the ESS. These findings were independent of Sleep Disordered Breathing (SDB) the incidence of which was not significantly different between the two groups. The authors concluded that there was no evidence for identifying at risk drivers in the test population, particularly using SDB as a criterion. However other causes of sleepiness could not be excluded as being significant in crash risk.

A study of hypersomnolence in Brazilian truck drivers was conducted by de Pinho et al. (2006). Three hundred long haul drivers were recruited at a truck stop roadhouse and administered a standard questionnaire regarding demographics, health, driving and sleepiness. Hypersomnolence was defined as having an ESS > 10 and was found in 138 subjects (46%). A history of crashes was found in 102 drivers (35%) and this was strongly associated with a history of excessive sleepiness ($p = 0.005$). Chronic sleep debt (40%) and poor quality sleep (46.3%) were common in this population and reflects poor lifestyles and irregular working hours. Age was negatively correlated with hypersomnolence (Odds Ratio OR = 0.45) while snoring (OR = 1.89) and work overload (more than 10 consecutive hours, OR = 2.07) were positively correlated.

A retrospective case control study of crashes in drivers with OSA was conducted by Mulgrew et al. (2008). The study group was 783 patients referred for polysomnography with 783 age and sex matched controls. Driving and crash histories were obtained by questionnaire and by interrogating insurance records. The study groups were 71% male and had an average age of 49.9 years (± 11.60). The mean (SD) AHI was 22.6 (21.9) events/h, BMI was 31.8 (10.3) and ESS was 10.1 (5.3). Crash data was collected for the 3 year period prior to polysomnography. In that time there were 252 crashes in patients and 123 in controls giving a relative risk for the OSA group depending on severity between 2.6 for patients with mild OSA and 2.0 for patients with severe OSA ($p < 0.005$). When the data was stratified according to crash outcomes the relative risk for crashes causing injury was much higher in the OSA group, rising to 4.8 in patients with mild OSA and 4.3 in patients with severe OSA ($p < 0.001$). The incidence of subjective daytime sleepiness was not a useful predictive factor for OSA crashes. This was the first study which showed an increased risk for personal injury crashes over crashes in general for OSA drivers.

Citations

No studies into citation rates for OSA or narcolepsy drivers were found.

Driving Performance

Boyle et al (2008) conducted a simulation study of 20 drivers (50% gender distribution, mean age 49.6 years for men, 52.1 years for women) with OSA with continuous EEG monitoring to identify micro-sleeps. The patients were recruited from a sleep disorders clinic and had to satisfy criteria for OSA including an ESS over 10, AHI greater than 5/hr and be symptomatic and untreated. Drivers with heavy tobacco or caffeine consumption and those with neurological contra indications were excluded. Micro sleeps were defined as the occurrence of 3-14 seconds of uninterrupted non artefactual

theta waves replacing the usual waking alpha rhythm. Driver performance was assessed with speed, lane keeping and steering control. Subjects drove for 60 minutes in a virtual reality type simulator.

Over 150 microsleeps were identified during the study. Significant decreases in speed were found during microsleeps ($p > 0.05$) which were interpreted as being the result of reduced pressure on the accelerator pedal. This has implications for crash risk in congested traffic situations. Differences were also found in variability of lane position which increased Steering Entropy during microsleeps. This effect increased with the duration of the drive showing that drowsy drivers fatigue as the drive progresses. All the decrements in performance were worse on curved road segments compared to straight drives.

A similar study by the same group investigated heart rate variability in a group of 11 drivers with untreated OSA compared to 12 controls of similar age distribution. The drivers drove on 3 laps of a simulated featureless road with no traffic, which took about an hour. Continuous ECG monitoring was recorded and analysed both in the time and frequency domains.

Time domain analysis revealed increased heart rate variability in the OSA group compared to controls, which became apparent and increased after a period of monotonous driving. This was interpreted to be an effect of increasing fatigue and was postulated as a possible variable to be exploited in drowsiness detection technology.

Many simulator studies of OSA drivers are deliberately designed to make the driving as monotonous as possible in order to maximise the effects of drowsiness and fatigue. A study with more realistic driving conditions was reported by Tassi et al. (2008). Twelve untreated OSA drivers (mean age 51.8years, BMI 31.09, AHI 58.55) and 8 healthy age, sex, driving and education matched controls (49.33 years, BMI 21.5 and no respiratory disorders) participated in the study. Subjects were psychologically screened and spent the night prior to the study in the sleep laboratory. They were awake for 24 hours during the driving simulation sequences. A driving simulator with a medium traffic density scenario was used, EEG and driving parameters were continuously monitored.

Significant differences between the OSA group and controls were found in speed adjustments, inter vehicle distances overtaking parameters and accelerator release before roadworks. OSA drivers were not dramatically impaired and their driving appeared to be more cautious and careful. This is in contrast to previous studies using monotonous driving scenarios. This may be due to the continuous level of stimulus experienced in a dynamic traffic situation as well as their experience of driving while sleep deprived. EEG recording confirmed that there was more alpha (waking) activity during difficult manoeuvres although there was increased theta (sleepy) activity at other times in the OSA group, especially when errors were being made.

The relationship between circadian effects and symptoms of sleepiness in OSA drivers was investigated in a crossover controlled study by Desai et al. (2005). 13 subjects with mild untreated OSA and 16 controls were subjected to neuropsychological testing and a driving simulator test after a normal night's sleep and a night of supervised sleep deprivation. Subjects were recruited at a hospital based sleep clinic and among students. The inclusion criteria were age between 18 and 60, a current licence, no other significant medical conditions and no alcohol or sedative drug use. Driving simulation was undertaken at different times over a 24 hour period and polysomnography was

performed when the study period ended and the subjects slept before going home. Mild OSA was defined as a Respiratory Disturbance Index (RDI) between 5 and 15.

The subjects with OSA were slightly older than controls (mean 45 vs 38 years) and heavier (mean BMI 30.3 vs 25.5). Self reported crash rates and caffeine intake were similar for both groups. The groups drove with more errors in the simulator when they were sleep deprived and there was a pronounced diurnal effect for both groups, with worst performance at 3:00 pm. Both groups reported increased subjective sleepiness when sleep deprived but the extent was less for the OSA subjects. Subjects with OSA performed worse on reaction time tests at all times of the day when sleep deprived compared to the sleep deprived controls. The results suggested that mild OSA patients were not different to controls in their responses to diurnal factors and sleep deprivation. However OSA patients tended to be less aware of daytime sleepiness and performed worse than controls on reaction time testing.

Pichel (2006) et al. conducted a study to identify associations between sleep complaints and performance on simulated driving. One hundred and twenty nine drivers (107 males, 22 females) were recruited consecutively from a hospital sleep clinic waiting list. Thirty six were excluded because of criteria including insufficient driving exposure, other diagnoses, excessive drug & alcohol use and unavailability of driving history. The remaining 93 (78 males, 15 females) underwent polysomnography, a battery of neuropsychological assessments, an ESS rating, a series of driving questionnaires, vigilance tests and a drive in a divided attention computer screen type simulator.

The diagnosis of OSA defined as AHI>10 was confirmed in 77 drivers out of 93 patients (88%). The mean age was 50.8 +/- 10.7 years, the BMI 30.1 +/- 5.3 and the AHI 37.2 +/- 23.4. While some of the neuropsychological parameters were associated with tracking errors on simulated driving, there was no relationship between OSA at any severity any of the simulator performance measures. Poor reaction time on testing was associated with a history of dozing while driving ($p < 0.05$). The tendency to fall asleep while driving was associated with tracking errors ($p < 0.05$). Other non relevant negative associations with driving performance were age, female gender, a history of crashes in the previous year, general quality of life and alcohol consumption. The authors concluded that while some measures of simulated driving performance is associated with sleep complaints in OSA patients, they are not associated with crashes but are associated with other undesirable traffic behaviours such as falling asleep while driving.

A study to compare driving simulator performance (measured by crash rate) and neuropsychological testing in drivers with narcolepsy was conducted by Kotterba and colleagues (2004). The purpose of the study was to evaluate the predictive value of off-road testing in this condition. The study group consisted of 10 men and 3 women with an average age of 40.9 years +/- 12.4. The diagnosis of narcolepsy was confirmed by symptoms, two sleep onset REM periods in the MSLT test and positivity for the HLA DR15/DQ* 0602 gene. Eight were drug free, the remainder took stimulants, tricyclics or both. The control group consisted of 9 men and 1 woman of age 55.1 (+/- 7.8) years. All were active drivers and none had other neurological conditions or sleep disorders. All subjects were given a battery of computerised neuropsychological tests of vigilance, alertness and divided attention. They drove in a simulator for 60 minutes in randomly presented scenarios including poor weather and the presence of obstacles. Simulated driving was observed and crashes and concentration lapses recorded.

The study group had significantly raised ESS compared to controls (16.7 vs. 6.6, $p < 0.01$) and increased crash rates in the simulator (3.2 vs. 1.3, $p < 0.01$). No significant concentration lapses were found and none of the study group fell asleep or experienced cataplexy. There were no significant differences between the study group and controls on any of the neuropsychological tests although there were high inter-individual differences in the study group. One patient experienced cataplexy during the test. This study suggested that neuropsychological testing was not appropriate for drivers with narcolepsy. This is not really a surprising finding as the condition is episodic and subjects may appear normal between attacks, even in the presence of a significantly higher ESS.

Treatment of sleep apnoea and related disorders and road safety outcomes

Crashes

No studies were found into the effect of treatment on real world citation and crash rates.

Citations

No studies were found into the effect of treatment on real world citations.

Driving Performance

A study into the effectiveness of CPAP for OSA patients was conducted by Mazza et al. (2006). Twenty patients with OSA and 20 controls were evaluated with polysomnography and then subjected to testing using a driving reaction time test on a short test platform where drivers had to avoid a water hazard in a real car. They were also subjected to neuropsychological tests of divided attention in a computerised driving simulator, a test of sustained & selective attention, and a Maintenance of Wakefulness test. Ten of the test subjects agreed to be re-tested after being established on CPAP treatment for their condition. The test subjects were recruited from a sleep disorders clinic and were age matched to a random control group. There were significant differences between the groups in ESS, BMI, RDI and nocturnal O₂ desaturation ($p < 0.001$ for most parameters). Exclusion criteria included other neurological or psychiatric illnesses and alcohol or drug use.

In the pre-treatment study there were significant differences between test subjects and controls in MWT errors ($p = 0.009$) and simulator divided attention, reaction time and off road events (p 's < 0.001). In the on-road reaction test there were differences in reaction time, distance to stop ($p < 0.001$), anticipated reaction time ($p = 0.02$) and distance to stop ($p = 0.01$). These differences were completely abolished after treatment with CPAP for the study group with the exception of simulated driving reaction time which remained slightly higher in the study group ($p < 0.01$). Unfortunately, the post treatment ESS scores were not stated and polysomnography was not repeated after treatment. Despite these omissions, this study showed the effectiveness of CPAP in normalising the driving performance which helps confirm its use as a therapeutic measure for OSA drivers.

A study by Orth et al. (2005) investigated the effect of CPAP on simulated driving and neuropsychological testing. The subjects were 31 men (age 55.3 +/- 10.2 years, BMI 29.9 +/- 2.2) with polysomnographically confirmed OSA defined as AHI >5 together with clinical symptoms such as drowsiness. Exclusion criteria were other significant

diagnoses of cerebral disease, alcohol or drug abuse, chronic lung disease and inability to drive. Subjects underwent neuropsychological testing including the ESS and measures of vigilance, alertness and divided attention. They drove for 60 minutes in a simulator under a variety of conditions. Testing was performed twice on each day before, 2 days after and 42 days after initiation of CPAP therapy in polysomnographically proven OSA patients. The timing of the tests were in daytime periods of the circadian rhythm. 42 days was chosen as it was the current recommendation of the German Society of Sleep Research and Sleep Medicine for return to professional driving after initiation of treatment. Twenty four of 31 patients (77%) agreed to CPAP therapy and complete the testing on days 0 and 2. Twenty-one (68%) were tested on day 42. ESS scores improved significantly between days 0 and 2 (10.1 vs 8.9, $p < 0.05$) and improved still further by day 42 (6.1, $p < 0.001$). Sleep architecture was not changed by CPAP with the exception of the arousal index (12.6 to 4.8 to 3.7, $p < 0.05$) but ventilatory parameters such as AHI and minimal oxygen saturation improved greatly ($p < 0.001$).

There was a significant improvement in simulated driving performance after the initiation of CPAP, even after 2 days and the improvement continued at 42 days. Parameters such as accident rate ($p < 0.01$ at 2 days, $p < 0.001$ at 42 days) and concentration faults ($p < 0.001$ at 2 and 42 days). There was no significant association between ESS or neuropsychological test results and simulated driving performance. Neither was there a relationship between polysomnographic parameters and driving performance. This study confirmed the effectiveness of CPAP in improving driving performance of OSA patients and the demonstrated that only a short period of treatment is necessary before driving safety improves.

A similar study with a crossover design was conducted with OSA drivers (Turkington et al., 2004). Eighteen drivers with OSA and eighteen OSA controls were recruited from a hospital sleep disorders clinic. The groups did not differ significantly in age (49.9-51.7 years), sex (predominantly male), BMI (39-36.6), neck circumference (47-45 cm), RDI (59.8-58.3) or Epworth score (15.5-16). Both groups underwent initial testing in a simulator which recorded tracking error, reaction time and the number of off road events during a 20 minute drive. The groups were tested before treatment, and at days 1, 3 and 7 of a two week trial CPAP period. They were tested again 7 days after discontinuation of treatment.

After 7 days of CPAP the performance of the study group increased significantly over the controls (tracking error; $p = 0.004$, reaction time' $p = 0.036$; off road events, $p = 0.05$). Seven days after CPAP was discontinued there were still significant improvements but the size of the effect was less (p 's = 0.025, 0.043, 0.05, respectively). Subjective hypersomnolence improved while on CPAP. The control group maintained constant performance levels over the study time, confirming a lack of a learning effect. This study resulted in similar conclusions to Orth et al and also showed that the benefits of CPAP persisted for at least a few days after treatment ceased which has implications for policies aimed at enforcing compliance.

Summary

From the foregoing research, it is clear that people with OSA face an increased risk of crashes, primarily due to sleepiness or falling asleep at the wheel. The tendency to 'drop off' is probably the result of excessive daytime sleepiness, one of the main symptoms of the sleep fragmentation that occurs in participants with sleep apnoea. In addition, the

evidence also indicates that the more severe the sleep apnoea, the greater the risk of an MVA. Some researchers have challenged this result and claimed that the evidence is inconclusive, while others point to the small samples that have been used in some studies that have shown this effect. However, Masa et al. (2000) demonstrated that this relationship did exist and also used a comparatively large sample (145 subjects). It is also clear that following treatment with CPAP, people with sleep apnoeas' risk of traffic crashes declines to the level of that found amongst healthy controls.

Research reviewed in the current update has continued to demonstrate that sleep disorders are common in the driving population, especially in commercial and bus drivers in some countries. The common factors of poor sleep combined with non physiological working hours, poor general health and use of alcohol and other substances put this group at a high risk of crashes which is a an alarming and very dangerous situation. There is also evidence that drivers with sleep deprivation for whatever reasons are over represented in studies of crashed drivers and that the incidence of crash risk increases with the parameters of sleep disorders such as the AHI measured during polysomnography.

Drivers with OSA continue to demonstrate increased crash rates over controls. A large-scale study by Mulgrew et al. (2008) reported a Relative Risk of 2.6 for less severely affected drivers, paradoxically falling to 2.0 for the most severely affects. The RR was even higher at 4.8 and 4.3 respectively for crashes involving personal injury, where it approaches that of having a blood alcohol at the legal limit in many countries. Other than making the formal diagnosis and determining an AHI rating, attempts to find other reliable correlates of crash risk from simulator studies and neuropsychological testing have met with mixed and generally negative results and there does not seem to any universally applicable predictive test at this stage. This was also the case from the one study of narcolepsy where no neuropsychological tests were found to be of predictive value, a finding not surprising given the episodic nature of the condition.

Research has continued into treatment of OSA with CPAP. Studies have confirmed the efficacy of this treatment and the rapidity with which it becomes effective. The effectiveness was shown to extend for several days after treatment ceased, probably reflecting the time taken to accumulate a sufficiently severe sleep debt. CPAP is now well established as the acute treatment of choice in significant OSA. Long term studies into the effect of lifestyle modification, weight loss and non technological ways to improve sleep hygiene are needed in the future to provide guidance for management of this increasingly common condition.

In terms of vehicle licensing, it is clear that not all people with sleep apnoea have crashes and that identifying those that do, through further research, is imperative for road safety. It is also important that those who do not pose a serious traffic safety risk are not unnecessarily restricted.

Table 40 Summary of studies of risk associated with sleep apnoea

Study: Author/date	Methods	Outcome Measure of Risk	Sub-category	Crash Risk/ Main Finding
Aldrich (1989)	4 xCase-1x control Case 1 n=181 apnoeic Case 2 n=25 narcolep Case 3 n=35 eds Case 4 n=38 non-eds Control n=70	1. Self-report MVCs any cause. 2. Self-report sleep-related MVCs. 3. Near crashes 4. MLST score	Association between OSA severity level & (mild-moderate & severe). Includes Other sleep disorders without EDS & Other sleep disorders with EDS	<u>MVCs - any cause</u> Control higher than case (1 exception) <u>MVCs sleep-related</u> 31% male OSA vs. 11% male controls. 20% female OSA vs. 6% female OSA <u>OSA Severity & sleepy-MVCs</u> 15% male mild-moderate OSA vs. 37% severe OSA. 12% female mild-moderate OSA vs. 20% severe OSA
Barbe, Pericas, Munoz, Findley, Anto, Agusti & De Lluç Joan (1998)	Case-controls Case n=60 Control n=60 Subjects matched for sex (59 males & 1 female) & age (± 5 years)	1. Crashes (self-report & insurance companies) 2. Driving performance. 3. Scores on Epworth Sleepiness Scale, Beck anxiety & depression test & Psychomotor Vigilance Test	Differentiates between degrees of apnoea	Overall, apneics had more MVCs than controls (OR:2.3; 95% CI:0.97 to 5.33 p=0.06) & were more likely to have had more than 1 MVC OR:5.2;95% CI: 1.07 to 25.59 p < 0.05). Even after controlling for exposure, apneics had more MVCs than controls. No significant association between common theoretical risk factors (eg daytime sleepiness, anxiety, depression, OSA severity & vigilance levels) and MVCs.
Bearpark, Fell, Grunstein, Leeder, Berthon-Jones & Sullivan (1990)	2 x Case- 1x control; Case 1 n=101 Case 2 n=34 Control n= 288	1. At-fault crashes 2. Near-misses 3. Falling asleep at the wheel at traffic lights 4. Pulling off the road due to sleepiness	apneics snorers	19% apneics report MVCs vs. 8% controls (significant). 57% apneics pulled off road due to sleepiness vs. 33% controls (significant). Fell asleep whilst driving: 22% apneics, 21% snorers, 3% controls.
Boyle, Tippin, Paul, Rizzo (2008)	Cross sectional study of 20 untreated OSA drivers, 50% male. Simulator study with EEG monitoring to determine effect of microsleeps	Speed, lane position, steering parameters	OSA drivers, ESS>10, AHI>5, symptomatic	Speed reductions during microsleeps (p>0.05), increased steering entropy. Degradation worse on bends and with time into the drive
Boyle, Hill, Tippin, Faber, Rizzo (2007)	11 untreated OSA drivers, 12 controls of similar age. Case controlled simulator study with ECG monitoring	Heart rate variability	OSA drivers	Increased heart rate variability in OSA drivers compared to controls after 25 minutes of driving (p<0.001).
Carter, Ulfberg,	1034 commercial drivers	Detailed questionnaire	Male truck and bus	Increased BMI and ESS rates in study group. Increased

Study: Author/date	Methods	Outcome Measure of Risk	Sub-category	Crash Risk/ Main Finding
Nystrom, Edling (2003)	recruited by mail, 1865 controls. All male.	covering lifestyle, sleep habits, driving hours, experience of drowsiness, sleep debt and effects of drowsy driving (crashes)	drivers , male general population	crashes especially in leisure driving (p<0.0001). Crashes related to sleep debt in both groups (p< 0.0001)
Crummy, Cameron, Swann, Kossmann, Naughton (2008)	40 hospitalised crashed drivers. Alcohol, psychiatric illness and lack of consent excluded. Self reported sleepiness questionnaire.	Risk factors for sleep related crashes including sleep disorders, circadian disturbance, sleep disordered breathing	Drivers hospitalised after crashes,	High prevalence (19/40) of shift workers suggesting circadian disturbance and sleep deprivation is a risk factor for crashes.
De Assis Viegas and de Oliveira (2005)	Cross sectional survey of 262 interstate bus drivers by means of questionnaire and anthropometry.	Self reported drowsiness symptoms, crash history, ESS. Measured body parameters.	Brazilian bus drivers	High incidence of obesity, OSA symptoms, disturbed sleep, crashes and substance use.
De Pinho, Silva-Junior, Bastos, Maia, de Mello, de Bruin, de Bruin (2006)	Crossover study of 300 truck drivers recruited at truck stop. Self reported by questionnaire.	As above. Hypersomnolence defined as ESS > 10	Truck drivers	Crashes associated with hypersomnolence (p=0.005). Negative correlation of hypersomnolence with age but positive correlation with snoring and working more than 10 hours continuously
Desai, Marks, Jankelson, Grunstein (2005)	Case controlled crossover study of 13 subjects with mild OSA, 15 controls. Simulator test at different times of the day after a night's sleep deprivation.	Neuropsychological tests, simulated driving performance, sleep study	Patients with mild OSA (5<RDI<15)	Increased reaction time in sleep deprived OSA drivers compared to controls (p=0.02). Reduced subjective sleepiness in sleep deprived OSA subjects. Driving not significantly different between two groups.
Findley, Fabrizio, Knight, Norcross, LaForte & Suratt (1989)	Case-control Case n=12 Control n=12 Age & gender matched	Response to simulated road obstacles.	Severe untreated OSA. 6 treated with CPAP (before-after) Possible selection bias	<u>Driving simulator</u> OSA drive worse than controls <u>Computer simulator</u> OSA drive worse than controls. OSA drive worse on computer simulator than on driving simulator. <u>CPAP treatment</u> No significant difference between treated OSA & controls
Findley, Smith, Hooper, Dineen & Suratt (2000)	50 OSA cases 2 conditions = 36 CPAP treat vs 14 not CPAP treat	1. Self-report at-fault MVCs 2. At-fault MVCs from	OSA – CPAP treated OSA – not CPAP treated.	<u>Pre-diagnosis</u> OSA significantly higher MVCs vs. general population (0.07 per person per year vs. 0.01, p < 0.02)

Study: Author/date	Methods	Outcome Measure of Risk	Sub-category	Crash Risk/ Main Finding
		Official database		<u>During CPAP</u> OSA no crashes – a significant reduction $p < 0.03$ <u>No CPAP</u> No reduction in MVCs
Gurubhagavatula, Nkwuo, Maislin, Pack (2008)	247 high risk commercial drivers selected from questionnaire survey 159 low risk controls from same survey. Comparison of different methods of screening	AHI > 5/hr, ESS >10. Screening by questionnaire, overnight oximetry and polysomnography	Truck drivers with OSA	Simple screening by questionnaire with or without oximetry found to be a cost effective method of screening taking into account the costs of crashes associated with undetected cases.
George, Boudreau & Smiley (1996)	2x case-1x control Case 1 = 21 (OSA) Case 2 = 16 Control = 21	1. Tracking errors 2. Visual search	Untreated OSA & untreated narcolepsy	Cases significantly worse on tracking. OSA worse than narcoleptics. BUT approx 50% OSA & narcoleptics performed as good as, or better than controls.
George (2001)	Case Control Case n=210 Control n =210 Case=sleep apneics	Crashes (from Transport database)	Sleep apneics AHI>10 182 use CPAP 27 elect not to use CPAP	<u>3 years prior to diagnosis</u> sleep apneics had a significantly higher MVC vs. controls (0.18 MVCs per person per year vs. 0.06 MVCs per person per year, $p < 0.001$). <u>After CPAP treatment</u> MVCs for sleep apneics fell to same level as controls (i.e. 0.06 crashes per person per year).
Hack, Choi, Vijayapalan, Davies & Stradling (2001)	Case-control Case n=26 Control =24 healthy normals	Driving performance i.e. 1. tendency to wander 2. task deterioration 3. no. of off-road events 4. reaction time to peripheral events	Control divided into 2 conditions: alcohol drink (12) or sleep deprived (12). Apneics & normals also acted as their own controls (via CPAP, no drink & full night's sleep).	OSA driving performance similar to alcohol-impaired performance rather than sleep deprived. OSA impaired driving due to vigilance decrements not cognitive impairment. CPAP treatment improved OSA driving.
Horstman, Hess, Basetti, Gugger & Mathis (2000)	Case Control Case n=156 Control n=160 (matched for age & gender)	1. Self-reported crashes. 2. Official Federal statistics of MVCs due to sleepiness. (Only crashes resulting in injury or property	Mild SAS=AHI ≤ 34 ; Mod & severe SAS=AHI ≥ 35 Compares 85 apneics before & after CPAP treatment.	15.5 fold increase of MVCs per km driven for those with moderate to severe SAS. MVCs for severe SAS=13.0 per million km. MVCs for milder SAS=1.1 million per km. MVCs for controls=0.78 per million km. During treatment with CPAP, MVC rates fell from 10.6

Study: Author/date	Methods	Outcome Measure of Risk	Sub-category	Crash Risk/ Main Finding
		damage)		to 2.7 per million km (p < 0.05).
Lloberes, Levy, Descals, Sampol, Roca, Sagales, De La Cladaza (2000)	2x case – 1x control Case 1=122 apnoeics Case 2=67 snorers Control=40 (age & gender matched)	1. Self-reported sleepiness 2. Self-reported MVCs 3. Self-reported driving off road	Apneics Snorers	<u>Self-reported sleepiness</u> Significantly higher in apneics vs. snorers or controls (43%, 34%, 5%). Self-reported sleepiness assoc. with MVCs. <u>Sleep-related MVCs self-report</u> OSA had more MVCs than snorers or controls (9%, 1.5%, 0%). <u>Running off road</u> OSA had significantly more than snorers or controls
Masa, Rubio, Findley (2000)	Case-control Case=145 Control=145 Age& gender matched.	1. MVCs (self-report) 2. Simulated driving performance. 3. Nocturnal respiratory events	Habitually sleepy drivers.	<u>Nocturnal respiratory events</u> significantly more in case vs. controls ((for case with a total respiratory index of ≥15, adjusted OR was 6.0, CI=1.1 to 32). <u>Frequency of MVCs</u> Case significantly more (10X) MVCs vs. controls (adjusted OR was 13.3, CI=3.1 to 4.3).
Shiomi, Arita, Sasanabe, Banno, Yamakawa, Hasegawa, Ozeki, Okada & Ito (2002)	554 cases Apneics=448 Snorers=106	1. Self-report MVCs	Severity of OSA Mild = AHI 5-15; Mild to moderate = AHI 15-30; Severe = AHI>30. Snorer=AHI<5	Severe OSA significantly higher MVCs vs. snorers”.
Tassi, Greneche, Pebayle, Eschenaluer, Hoeft, Bonfond, Rohmer, Muzet (2008)	12 OSA drivers and 8 matched controls. Driving simulation in medium traffic density situation with roadworks and other obstacles.	EEG monitoring and simulator parameters	OSA drivers	OSA drivers drove safely with an increase in cautiousness. No gross deficits. EEG confirmed increased arousal at difficult driving manoeuvres and sleep patterns during driving mishaps.
Howard, Desai, Grunstein, Hukins, Armstrong, Joffe, Swann, Campbell, Pierce (2004)	Questionnaire study of 2342 truck drivers and 161 drivers undergoing polysomnography.	Detailed questionnaire covering lifestyle, sleep habits, driving hours, experience of drowsiness, crashes, drug use and effects of drowsy driving	Truck drivers	High incidence of obesity, sleep disordered breathing and chronic sleepiness. Highest risk group with ESS over 18 had OR for a crash of 1.91 and 2.67 for multiple crashes. Crash risk increased with antihistamine and narcotic analgesic use.
Kinkshott, Cowan, Jones, Flannery, Smith, Herbison, Taylor (2004)	60 crash involved drivers 60 controls matched for age, gender, BMI	Polysomnography, MWT, neuro-psych tests including reaction time	Crash involved drivers	No significant difference in Sleep Disordered Breathing between 2 groups. Crash group had higher incidence of sleepiness (p=0.003), MWT latency (p=0.06) and slower reaction time (p=0.02)

Study: Author/date	Methods	Outcome Measure of Risk	Sub-category	Crash Risk/ Main Finding
Kotterba, Mueller, Leidag, Widdig, Rasche, Malin, Schultze-Weninghaus, Orth (2004)	13 drivers with narcolepsy 10 controls. Case controlled study of simulated driving for 60 minutes and neuropsychological testing	Crash and lapse rates, tests of vigilance, alertness and divided attention	Drivers with narcolepsy	Narcolepsy group had higher ESS and crash rates in simulator than controls. No significant differences on neuropsych testing between the groups. Neuropsych testing not useful for these patients.
Mazza, Pepin, Naegele, Rauch, Deschoux, Ficheux, Levy (2006)	Case controlled study of 20 OSA drivers and 20 controls with polysomnography, neuropsychological test, a simple simulator and a real driving reaction time test. Study repeated after half the study group were treated with CPAP.	Neuropsychological tests of reaction time divided attention, MWT, attention and reflex braking in a real car.	OSA drivers treated with CPAP	Initial test showed significant reduction in test parameters for OSA drives compared to controls. Differences abolished by CPAP treatment, confirming it as an effective treatment for these drivers.
Mulgrew, Nasdvadi, Butt, Cheema, Fox, Fleetham, Ryan, Cooper, Ayas (2008)	Retrospective case controlled study of crashes in 783 OSA patients using polysomnography and insurance company records	Correlation of crash type and severity with OSA parameters from self reported history and polysomnography	OSA drivers	OSA drivers had greater number of crashes and higher crash risk . RR= 2.0-2.6 (p<0.001). Risk for personal injury even higher at 4.3-4.8 (p<0.001).
Orth, Duchna, Leidag, Widdig, Rasche, Bauer, Walther, de Zeeuw, Malin, Schutze-Werninghaus, Kotterba (2005)	31 OSA drivers tested before and after 2 and 42 days of CPAP. Neuropsych tests and simulator	Simulated driving measures of crashes and concentration lapses	OSA drivers on CPAP	Significant improvement in driving performance after 2 days of CPAP, sustained and increased improvement at 42 days
Pichel, Zamarron, Magan, Rodriguez (2006)	93 OSA drivers subjected to polysomnography, neuropsychological testing, driving questionnaires and a simulator test.	Correlation of historic al factors with neuropsych tests and simulated driving performance	OSA drivers	Simulated driving performance associated with sleep symptoms in OSA drivers (p<0.05), no association with crashes. Increased crash risk and poorer driving found for some non-OSA variables including age, gender, alcohol use, crash history and quality of life.

Study: Author/date	Methods	Outcome Measure of Risk	Sub-category	Crash Risk/ Main Finding
Turkington, Siecar, Saralaya, Elliott (2004)	18 OSA drivers, 18 matched controls, mostly male. Driving simulator study before, at days 1, 3, and 7 of a 2 week CPAP trial and again 7 days after it ceased.	Simulator parameters of tracking error, reaction time and off road events	OSA drivers on CPAP	Significant improvement in driving performance after 7 days of CPAP, some residual improvement 7 days after CPAP ceased.
Van den Berg & Landstrom (2006)	154 bus and truck drivers recruited by mail. Self reported questionnaire regarding drowsiness while driving	Detailed questionnaire covering lifestyle, sleep habits, driving hours, experience of drowsiness, countermeasures and effects of drowsy driving	Sample of heavy vehicle drivers	High incidence of experience of drowsiness with 8% reporting nodding off. Limited awareness of early stages of drowsiness. Better sleep before driving and more amenable work schedules identified as the most effective countermeasures.

Approaches to management

Screening for OSA, particularly amongst drivers of heavy vehicles is an area of considerable interest in the management of risk. A study by Gurubhagavatula et al. (2008) addressed the difficulties in diagnosis of OSA in commercial (truck) drivers. This study identified high risk drivers by questionnaires and overnight oximetry screening before proceeding to polysomnography. Questionnaires were mailed to 4410 commercial licence holders in the state of Pennsylvania. Of the 32% who responded (n = 1392), 247 were identified as being at higher risk of OSA. 159 controls were randomly selected from those identified as lower risk. The aim of the study was to compare the cost effectiveness of screening for polysomnography by questionnaire alone to screening by the combination of a questionnaire and oximetry for an intermediate group. The authors used previously determined statistics to assign probabilities of a crash to drivers with and without OSA and to assign costs for a crash. The economic analysis included the costs of polysomnographic screening, oximetry and miscellaneous costs for administrative tasks.

The average age of the drivers was 45.4 +/- 11.0 years, BMI was 29.9 +/- 5.2 kg/m². 8.7% were assessed as having OSA which was defined as the combination of an AHI >5/hr and a ESS score of 10 or more. The accuracy of the various screening and diagnostic methods are shown in Table 41.

Table 41: Screening and diagnostic measures for OSA

	One stage	Two stage	Polysomnography
Sensitivity (%)	70.5	68.8	100
Specificity (%)	71.4	90.9	78.4
Negative predictive value (%)	96.4	96.6	100
Negative likelihood ratio	0.413	0.385	0.000

The authors concluded that a simple one or two stage screening process involving a questionnaire with or without oximetry was a valid strategy to detect OSA in commercial drivers. The costs associated with a missed diagnosis (assumed to result in a crash) were acceptable when compared to the costs (direct and indirect) associated with mass polysomnographic screening.

Limitations of this study included the relatively low participation rate by recipients of the questionnaire, and the use of economic data which may be out of date. The prevalence rate found in this study (8.7%) appears to low when compared to other studies of sleep disorders in commercial drivers. Any increase in prevalence of OSA would make the screening procedure even more economically viable.

Notwithstanding these limitations, this research on alternative methods of diagnosing OSA has provided important information given the gold standard of polysomnography is expensive, time consuming and not universally available. It appears that careful history taking and cheaper investigations such as overnight oxygen saturation recording

may have a place in screening candidates for more intensive investigation and may even be a cost effective method of diagnosis.

Assessing fitness to drive

The following section refers to the Licensing Guidelines for Chronic Illness that are set out in Table 37 for the following six countries: Sweden, Australia, New Zealand, Canada and the USA. General comments are made here and the reader is referred to the tabled guidelines for more detail.

Sleep Apnoea

There appears to be fairly general agreement across the six countries' private licensing guidelines that untreated OSA requires the person to desist from driving. The only exception to this is Australia, where untreated, high-risk people with OSA are required to "restrict" their driving whilst awaiting treatment.

Resumption of driving in all countries usually requires the person to have undergone successful treatment so that the symptoms are controlled and the individual no longer poses a traffic safety risk. While three of the countries mandate periodic review, Australia also requires that the person officially hold a conditional licence rather than an unconditional or unrestricted licence.

Interestingly, only 3 States in the USA make particular mention of sleep apnoea in their guidelines (Utah, Texas, and California). In 1994, another State, Maine, had also proposed the inclusion of sleep apnoea in its guidelines. However, it could be argued that sleep apnoea might possibly be subsumed under regulations relating to loss of consciousness or respiratory dysfunction, and therefore does not require a separate section (Pakola, Dinges and Pack, 1995).

Due to the extra dangers posed with driving commercial vehicles, most of the countries (apart from the USA, whose member States do not deal comprehensively with sleep apnoea) require that licensing requirements be more stringent and stipulate regulations over and above that required for drivers of private vehicles. Sweden specifically states this consideration and New Zealand mentions restriction of driving hours if there is any lingering sleepiness associated with OSA. Australia has also included a provision that if the person receives a score from 16 to 24 on the Epworth Sleepiness Scale s/he is to be barred from holding an unconditional commercial licence.

There appear to be large inconsistencies in the judgements handed down in courts for drivers suffering from OSA who cause fatal crashes. Desai, Ellis, Wheatley and Grunstein (2003) presented a series of 7 case studies in which the drivers had OSA – including those who were diagnosed, undiagnosed or under-treated. Three of these cases were either acquitted or not prosecuted, while the other four were judged guilty (two pleaded guilty and the other two were found guilty). The three cases that were acquitted or not prosecuted utilised the "Jimenez" defence. The "Jimenez defence" arose from a case (Jimenez vs. Queen) in which the High Court in Australia ruled that falling asleep at the wheel was an unexpected event which the driver could not have foreseen.

Not all countries' judiciaries hold the same opinion, however. For example, courts in Canada and Britain hold the view that, prior to nodding off at the wheel, the driver

would have experienced sleepiness and, therefore, should have taken preventative action at this point instead of taking the risk of driving further – this is referred to as the “prior fault principle” (Desai et al., 2003).

To complicate matters further, medical opinion on this matter is also divided. Studies involving healthy individuals (i.e. non-sleep apnoeic) found that there was a “significant awareness” of sleepiness on their part prior to falling asleep at the wheel. However, Desai et al. (2003) point out that people with OSA may not have the same awareness of their sleepy state. On this point, it is interesting to note that when describing the symptoms of OSA, other researchers have listed “daytime *involuntary* sleep spells” (italics added) (Haraldsson, Carenfelt, & Tingvall, 1992 cited in Eby, Trombley, Molnar & Shope, 1998). To provide clear evidence on these issues, Desai et al. (2003) make a call for more research in this area.

Narcolepsy

The licensing guidelines for narcolepsy show a little more variation in comparison to those for sleep apnoea. In the USA, the guidelines for epilepsy apply to narcolepsy while Canada mandates that the person desist from driving for a full year if cataplexy has been experienced. The remaining four countries (Sweden, Australia, New Zealand and the UK) allow a holder of a private licence to drive provided that the symptoms of narcolepsy are treated and satisfactorily controlled, with a requirement of periodic review being stipulated.

Once again, the commercial licence guidelines are more rigorous than those set down for drivers of private vehicles. For example, the New Zealand regulations state that a person who has severe narcolepsy or experiences cataplexy is unfit to drive a commercial vehicle. In Australia, strict criteria (no past cataplexy, 6 months symptom-free, normal sleep latency etc) are imposed before the person may hold a restricted licence.

Self-Regulation

Van den Berg and Landstrom (2006) studied a sample of lorry (truck) and bus drivers with regard to sleepiness while driving. While diagnosed sleep disorders were not the focus of this study, what was of interest is the awareness of sleepiness among drivers who commonly drive long distances with heavy vehicles and for whom awareness of sleepiness would be of prime interest in managing risk. Drivers of heavy vehicles in northern Sweden were identified using various (unspecified) registries and listings. Self assessment questionnaires were sent to 227 drivers and 154 replies (70% participation rate) were used in the study. There were 149 men and 5 women in the group with a mean age of 44.5 (SD 13.00), and 18.9 years of driving experience (SD 12.9). None of the drivers drove for more than 4.5 hours without a break.

The questionnaire was detailed and asked about duration of driving, prevalence of drowsy driving, falling asleep, crashes, countermeasures, sleep quality and lifestyle. Awareness of sleepiness was high as was motivation to deal with the problem. Almost one third of drivers reported having to fight sleepiness with 8% admitting to nodding off while driving. Various countermeasures such as breaks, snacks, playing music, lowering the temperature etc were all employed however there was agreement that better sleep prior to work and more amenable working hours were the most effective. Drivers reported symptoms of advanced sleepiness including yawning, difficulty concentrating

and eye drooping but did not report early awareness of sleepiness. The well known diurnal incidence of sleepiness was also confirmed in this cohort. Factors such as age, type of traffic, experience and state of health did not appear to be significant factors in the incidence of drowsy driving. Poor sleep prior to work was a universal risk factor.

A study into the prevalence of risk factors for OSA in Brazilian interstate bus drivers was conducted by de Assis Viegas and de Oliveira (2006). The study group was 262 male bus drivers employed by an interstate bus company. The average age was 38.1 +/- 5.8 years and the mean BMI was 26.8 (range 19.2 to 40.1) kg/m². Mean neck circumference (NC) was 40.4 with a range from 34 to 48 cm. The subjects completed an anonymous questionnaire regarding driving habits and experience of sleepiness. The ESS and tests of sustained and divided attention were also administered.

Fifty percent of the subjects were overweight with 0.8% in the maximum range for morbid obesity. Excessive daytime sleepiness with an ESS over 10 was found in 27.5% of drivers. The rate was highest in drivers with a BMI over 30. There was no correlation of ESS with NC. Thirty-six percent reported snoring, in 5% this could be heard behind closed doors. Twenty-nine percent reported restless sleep, 12% had woken with a sensation of choking and 5% reported OSA.

A large number of drivers used substances to stay awake, 12% medication, 55% cola based drinks, 65% drank alcohol and 88% drank coffee. Twenty-seven percent were smokers. Forty-eight percent reported sleepiness while driving and 42% had crashed in the past, 8% of which were attributed to sleepiness. There were significant ($p < 0.05$) increases in incidence of drowsy driving for those with a BMI over 30. The study showed that hypersomnolence and its complications were highly prevalent in the study population and that this was a significant risk factor for crashing. The authors suggested that screening procedures for job applicants in the industry should be tightened.

Table 42 Private licensing guidelines for drivers with sleep disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Sleep Apnoea (obstructive sleep apnoea & apnoea syndrome)	<p>All patients to be advised of the risks of driving whilst drowsy.</p> <p>If the condition is severe enough to impair driving ability then the patient should desist from driving until the condition has been adequately treated and controlled.</p>	<p>May not hold an unconditional licence if:</p> <ol style="list-style-type: none"> 1. Diagnosed with OSA via sleep study & have moderate or severe sleepiness & in GP's opinion pose significant driving risk. 2. Frequently feels sleepy or drowsy whilst driving or has MVCs caused by sleepiness or inattention. 3. High-risk OSA that is untreatable or person not compliant with treatment or unwilling to restrict driving whilst waiting for treatment. <p>A conditional licence may be issued if person is compliant with treatment and symptoms are responsive to</p>	<p>Desist from driving until symptoms are satisfactorily controlled. Medical confirmation of this is required.</p>	<p>Only 3 States in the USA specifically mention sleep apnoea in their licensing guidelines (Pakola et al., 1995).</p> <p>May drive unrestricted if mild to moderate problems of alertness and excessive sleepiness (Epworth Sleep Scale [ESS] score 10-12).</p> <p>Review required annually or every 2nd year.</p> <p>Restricted licence may apply with moderate symptoms of hypersomnolence and alertness (ESS score 13 – 15).</p> <p>Six monthly review required.</p> <p>Severe symptoms of</p>	<p>Desist or restrict driving for the following high- risk patients</p> <ol style="list-style-type: none"> 1. Suspect person has OSA with excessive daytime sleepiness whilst driving & awaiting confirmation of diagnosis. 2. Severe daytime sleepiness & history of sleep-related accidents 3. Severe OSA that is untreatable or person not compliant with treatment <p>May resume driving if symptoms satisfactorily controlled under specialist supervision. Periodic medical assessment may be required.</p>	<p>Licence issued if condition successfully treated.</p> <p>Licence denied if alertness is affected to a degree that person poses a road safety risk.</p> <p>Subject to periodic review on a case-by-case basis.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
		treatment. Periodic review required.		inattentiveness or hypersomnolence (ESS score > 15) - Restricted from driving.		
Narcolepsy	Desist from driving if diagnosed with narcolepsy. If they respond favourably to treatment and there are no side effects from medication the may drive after 3 months.	A conditional licence may be granted if person responds to treatment, according to expert opinion. Periodic review required.	Desist from driving upon diagnosis. Driving may be permitted on a 1, 2 or 3 year licence if control of symptoms achieved with regular medical review. Licence up to age 70 may be restored if illness controlled for 7 years.	Only 6 States in the USA specifically mention narcolepsy in their licensing guidelines (Pakola et al., 1995). <i>Utah</i> Narcolepsy falls under the same guidelines set down for epilepsy. An unrestricted licence may be issued if seizure or episode-free for 5 years, without medication. OR seizure-free for 12 months without medication or with medication but no side effects. One or two-yearly review required.	Desist from driving if person is suspected of having narcolepsy that impairs safe driving ability (in medical opinion) & is awaiting confirmation of diagnosis. May resume driving after satisfactory response to treatment or the person does not exhibit cataplexy or other symptoms that pose significant road safety risk. Regular medical assessment may be required.	Licence issued if condition successfully treated. Licence denied if alertness is affected to a degree that person poses a road safety risk. Subject to periodic review on a case-by-case basis.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
				<p>A restricted licence may be issued if seizure or episode-free for 3 to 6 months, without medication or with medication but no side effects.</p> <p>Speed, area & time of day restriction apply, depending on the length of time without seizures.</p> <p>Six-monthly review required.</p> <p>Restricted from driving when episodes are uncontrolled and/or medications affect alertness and coordination.</p>		

References

- Al Riyami, B., Al Rawas, O. & Hassan, M. O. (2000). Pathophysiology of obstructive sleep apnoea syndrome: a review. *Medical Sciences*, 2, 117-124.
- Aldrich, M.S. (1989). Automobile accidents in patients with sleep disorders. *Sleep*, 12(6), 487-494
- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Barbe, F., Pericas, J., Munoz, A., Findley, L., Anto, J.M., Agusti, A.G.N. & De Lluc Joan, M. (1998). Automobile accidents in patients with sleep apnoea syndrome: an epidemiological and mechanistic study. *American Journal of Respiratory Critical Care Medicine*, 158(1), 18-22.
- Bearpark, H., Fell, D., Grunstein, R., Leeder, S., Berthon-Jones, M. & Sullivan, C. (1990). *Road Safety and Pathological Sleepiness: The Role of Sleep Apnoea*. Road Safety Bureau Consultants Report CR 3/90.
- Benbadis, S.R., Perry, M.C., Sundstand, L.S., & Wolgamuth, BR. (1999). Prevalence of daytime sleepiness in a population of drivers. *Neurology*, 52(1), 209-213.
- Boyle, J., Hill, J., Tippen, J., Faber, K., & Rizzo, M. (2007). *The effects of sleep apnea on heart rate variability in a simulator*. Paper presented at the Fourth international driving symposium on human factors in driver assessment, training, and vehicle design.
- Boyle, L. N., Tippin, J., Paul, A., & Rizzo, M. (2007). Driver performance in the moments surrounding microsleep. *Transportation Research Part F: Psychology & Behaviour*, 11(2), 126-132.
- Bridgman, S.A., & Dunn, K.M. (1997). The use of surgery for the treatment of obstructive sleep apnoea (Cochrane Review). In: *The Cochrane Library*. Oxford: Update Software.
- Bridgman, S.A., Dunn, K.M., & Ducharme, F. (2003). Surgery for obstructive sleep apnoea (Cochrane Review). In: *The Cochrane Library*, Issue 1 2003. Oxford: Update Software.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Carter, N., Ulfberg, J., Nyström, B., & Edling, C. (2003). Sleep debt, sleepiness and accidents among males in the general population and male professional drivers. *Accident Analysis & Prevention*, 35(4), 613-617.
- Crummy, F., Cameron, P., Swann, P., Kossmann, T., & Naughton, M. (2008). Prevalence of sleepiness in surviving drivers of motor vehicle collisions. *Internal Medicine Journal*, 38(10), 769-775.

- de Pinho, R. S. N., Silva-Júnior, F. P. d., Bastos, J. o. P. C., Maia, W. S., de Mello, M. T. I., de Bruin, V. M. S., et al. (2006). Hypersomnolence and Accidents in Truck Drivers: A Cross-Sectional Study. *Chronobiology International*, 23(5), 963 - 971.
- Desai, A. V., Marks, G. B., Jankelson, D., & Grunstein, R. R. (2006). Do sleep deprivation and time of day interact with mild obstructive sleep apnea to worsen performance and neurobehavioral function? *Journal of clinical sleep medicine*, 2(1), 63-70.
- Desai, A.V., Ellis, E., Wheatley, J.R., & Grunstein, R.R. (2003). Fatal distraction: a case series of fatal fall-asleep road accidents and their medicolegal outcomes. *Medical Journal of Australia*, 178(8), 396-399.
- Douglas, N.J. (2002). Recent advances in the obstructive sleep apnoea/hypopnoea syndrome. *Annals Academy of Medicine*, 31(6), 697-701).
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Eby, D.W., Trombley, D.A., Molnar, L.J., Shope, J.T. (1998). *The assessment of older drivers' capabilities: A review of the literature*. Report No. UMTRI-98-24, The University of Michigan, Transportation Research Institute, August, 1998.
- Findley, L., Smith, C., Hooper, J., Dineen, M., & Suratt, P.M. (2000). Treatment of nasal CPAP decreases automobile accidents in patients with sleep apnoea. *American Journal of Respiratory Critical Care Medicine*, 161, 857-859.
- Findley, L.J., Fabrizio, M.J., George, T., & Suratt, P.M. (1989). Severity of sleep apnoea and automobile crashes. *New England Journal of Medicine*, 320, 868-869.
- Findley, L.J., Fabrizio, M.J., Knight, H., Norcross, B.B., Laforte, A.J., & Suratt, P.M. (1989). Driving simulator performance in patients with sleep apnoea. *American Review of Respiratory Disease*, 140, 529-530.
- Findley, L.J., & Suratt, P.M. (2001). Serious motor vehicle crashes: the cost of untreated sleep apnoea. *Thorax*, 56, 505.
- George, C.F.P. (2001). Reduction in motor vehicle collisions following treatment of sleep apnoea with nasal CPAP. *Thorax*, 56, 508-512.
- George, C.F.P., & Smiley, A. (1999). Sleep apnoea & automobile crashes. *Sleep*, 22(6), 790-795.
- George, C.F.P., Boudreau, A.C., & Smiley, A. (1996). Comparison of simulated driving performance in narcolepsy and sleep apnoea patients. *Sleep*, 19(9), 711-717.
- Grunstein, R. (1994). *Medical Aspects of Driver Sleep Disorders*. Seminar "Sleep disorders, driver fatigue and safe driving. Held at Parliament House, Sydney, November, 1994.

- Gurubhagavatula, I., Maislin, G., Nkwuo, J. E., & Pack, A. I. (2004). Occupational Screening for Obstructive Sleep Apnea in Commercial Drivers. *Am. J. Respir. Crit. Care Med.*, 170(4), 371-376.
- Hack, M.A., Choi, S.J., Vijayapalan, P., Davies, R.J.O., & Stradling, J.R. (2001). Comparison of the effects of sleep deprivation, alcohol and obstructive sleep apnoea (OSA) on simulated steering performance. *Respiratory Medicine*, 95, 594-601.
- Horstman, S., Hess, C.W., Basetti, C., Gugger, M., & Mathis, J. (2000). Sleepiness-related accidents in sleep apnoea patients. *Sleep*, 23(3), 1-7.
- Howard, M. E., Desai, A. V., Grunstein, R. R., Hukins, C., Armstrong, J. G., Joffe, D., et al. (2004). Sleepiness, Sleep-disordered Breathing, and Accident Risk Factors in Commercial Vehicle Drivers. *Am. J. Respir. Crit. Care Med.*, 170(9), 1014-1021.
- Johal, A., & Battagel, J.M. (2001). Current principles in the management of obstructive sleep apnoea with mandibular advancement appliances. *British Dental Journal*, 190(10), 532-536.
- Kingshott, R. N., Cowan, J. O., Jones, D. R., Flannery, E. M., Smith, A. D., Herbison, G. P., et al. (2004). The Role of Sleep-Disordered Breathing, Daytime Sleepiness, and Impaired Performance in Motor Vehicle Crashes—A Case Control Study. *Sleep and Breathing*, 8(2), 61-72.
- Kotterba, S., Mueller, N., Leidag, M., Widdig, W., Rasche, K., Malin, J.-P., et al. (2004). Comparison of driving simulator performance and neuropsychological testing in Narcolepsy. *Clinical Neurology and Neurosurgery*, 106(4), 275-279.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Larsson, L.G., Lindberg, A., Franklin, K.A., & Lundback, B. (2001). Symptoms related to obstructive sleep apnoea are common in subjects with asthma, chronic bronchitis and rhinitis in a general population. *Respiratory Medicine*, 95, 423-429.
- Laube, I., Seeger, R., Russi, E.W., & Bloch, K.E. (1998). Accidents related to sleepiness: review of medical causes and prevention with special reference to Switzerland. *Schweiz Med Wochenschr*, 128, 1487-1499.
- Lertzman, M., Wali, S.O., & Kryger, M. (1995). Sleep apnoea a risk factor for poor driving. *Canadian Medical Association Journal*, 153(8), 1063.
- Lloberes, P., Levy, G., Descals, C., Sampol, G., Roca, A., Sagales, T., De La Cladaza, M.D. (2000). Self-reported sleepiness while driving as risk factor for traffic accidents in patients with obstructive sleep apnoea syndrome and in non-apnoeic snorers. *Respiratory Medicine*, 94, 971-976.
- Masa, J.F., Rubio, M., Findley, L.J. (2000). Habitually sleepy drivers have a high frequency of automobile crashes associated with respiratory disorder during

- sleep. *American Journal of Respiratory Critical Care Medicine*, 162(4), 1407-1412.
- Mazza, S., Pepin, J. L., Naegele, B., Rauch, E., Deschaux, C., Ficheux, P., et al. (2006). Driving ability in sleep apnoea patients before and after CPAP treatment: evaluation on a road safety platform. *Eur Respir J*, 28(5), 1020-1028.
- Medical Journals (2003). www.priory.com (accessed 14/04/03).
- Medical Standards (2003). www.nrtc.gov.au/publications (accessed 14/04/03).
- Mulgrew, A. T., Nasvadi, G., Butt, A., Cheema, R., Fox, N., Fleetham, J. A., et al. (2008). Risk and severity of motor vehicle crashes in patients with obstructive sleep apnoea/hypopnoea. *Thorax*, 63(6), 536-541.
- Orth, M., Duchna, H. W., Leidag, M., Widdig, W., Rasche, K., Bauer, T. T., et al. (2005). Driving simulator and neuropsychological testing in OSAS before and under CPAP therapy. *Eur Respir J*, 26(5), 898-903.
- Pakola, S.J., Dinges, D.F., & Pack, A.I. (1995). Review of regulations and guidelines for commercial and noncommercial drivers with sleep apnoea and narcolepsy. *Sleep*, 18(9), 787-796.
- Pichel, F., Zamarrón, C., Magán, F., & Rodríguez, J. R. (2006). Sustained attention measurements in obstructive sleep apnea and risk of traffic accidents. *Respiratory Medicine*, 100(6), 1020-1027.
- Shiomi, T., Arita, A. T., Sasanabe, R., Banno, K., Yamakawa, H., Hasegawa, R., Ozeki, K., Okada, M., & Ito, A. (2002). Falling asleep while driving and automobile accidents among patients with obstructive sleep hypopnea-hypopnea syndrome. *Psychiatry and Clinical Neurosciences*, 56, 333-334.
- Sleepnet. (2003). www.sleepnet.com (accessed June 2003).
- Suratt, P.M., & Findley, L.J. (1999). Driving with sleep apnoea. *The New England Journal of Medicine*, 340(11), 881-883.
- Swedish Road Administration. (1998). *Swedish Road Administration Statute Book*. Sweden.
- Tassi, P., Grenèche, J., Pebayle, T., Eschenlauer, A., Hoeft, A., Bonnefond, A., et al. (2008). Are OSAS patients impaired in their driving ability on a circuit with medium traffic density? *Accident analysis and prevention*, 40(4), 1365-1370.
- Turkington, P. M., Sircar, M., Saralaya, D., & Elliott, M. W. (2004). Time course of changes in driving simulator performance with and without treatment in patients with sleep apnoea hypopnoea syndrome (Sleep Disordered Breathing). *Thorax*, 59(1), 56-54.
- Turkington, P.M., Sircar, M., Allgar, V., & Elliott, M.W. (2001). Relationship between obstructive sleep apnoea, driving simulator performance, and risk of road traffic accidents. *Thorax*, 56, 800-805.

- Wright, J., Johns, R., Watt, I., Melville, A., & Sheldon, T. (1997). Health effects of obstructive sleep apnoea and the effectiveness of continuous positive airways pressure: a systematic review of the research evidence. *British Medical Journal*, 314(7084).
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- van den Berg, J., & Landström, U. (2006). Symptoms of sleepiness while driving and their relationship to prior sleep, work and individual characteristics. *Transportation Research Part F: Traffic Psychology and Behaviour*, 9(3), 207-226.
- Viegas, C., & de Oliveira, H. (2006). Prevalence of risk factors for obstructive sleep apnea syndrome in interstate bus drivers. *J Bras Pneumol*, 32, 144-149.

3.12 VESTIBULAR (BALANCE) DISORDERS

Definition of vestibular disorders

Balance disorders refer to any condition that results in vertigo, dizziness or imbalance (NIDCH, 2000). Balance disorders may originate in the vestibular apparatus (located in the inner ear), the brain (termed central vestibular disorder), any other parts of the body (systemic disorder) or due to vascular or blood flow problems (Rosenberg & Gizzi, 2000).

The vestibular apparatus send information to the brain to enable people to accurately perceive their position in space, as well as co-ordinate movement and retain balance, relative to gravity and movement. Information from the vestibular system is integrated with information from vision and from the musculoskeletal system in order to maintain balance and co-ordinate movement (NIDCH, 2000).

Vertigo, the main symptom of vestibular disorders, affects “virtually every aspect of life” because it limits the ability to participate in activity that involves movement. Apart from the false illusion of movement that vertigo induces, it also carries with it the danger of falling and is associated with other symptoms such as nausea and vomiting (Salt, 2003).

The two most common types of balance disorders that will be considered in this section are:

Ménière's disease

Ménière's disease refers to an inner ear disorder in which the pressure of the fluid (endolymph) changes within the inner ear (School of Medicine, 1995) resulting in episodes of vertigo, fullness in the ear, tinnitus (i.e. ringing in the ear), and progressive and fluctuating loss of hearing (particularly for sounds in the lower frequency levels). This hearing loss may eventually become total and permanent for some people (Salt, 2003). In the majority of cases (75%) only one ear is affected by Ménière's disease (VEDA, 2009). Ménière's disease is labelled an idiopathic disease because its underlying cause is unknown (VEDA, 2009). It may, however, occur following other illnesses that interfere with the normal resorption of endolymph such as viral infections, trauma, or other diseases (muscular sclerosis, thyroid disease, transient ischemic attacks) (School of Medicine, 1995). An episode of Ménière's may last anywhere from two to four hours and attacks can be incapacitating. Following the attack, a period of extreme fatigue or exhaustion often occurs, prompting the need for hours of sleep. Episodes may re-occur in clusters with variable and sometimes long periods of remission (EM Guidemap).

Benign Paroxysmal Positional Vertigo (BPPV)

BPPV refers to the occurrence of vertigo following a change in the position of the head or body, relative to gravity (School of Medicine, 1995). For example, a person rolling over in bed or getting up in the morning or even tilting the head to look up at an object on a shelf (School of Medicine, 1995). Typical symptoms of BPPV include vertigo, imbalance, light-headedness and nausea (VEDA, 2009). BPPV is caused by debris, *otoconia* (calcium carbonate crystals), which has collected in the semi-circular canal in the inner ear. When the person moves, so too do these debris thus giving a false

sensation of a head turn (Barton, 2000). In older people, BPPV is thought to be the result of age-related degenerative changes in the vestibular system located in the inner ear whereas in patients under 50 years of age it is more likely to follow head injuries (VEDA, 2009). Approximately 50% of the dizziness experienced by the older population can be attributed to BPPV. Ear infections may also be a casual factor (VEDA, 2009). Symptoms may persist for days or weeks and may recur, although they generally resolve in a matter of months (School of Medicine, 1995).

Prevalence of vestibular disorders

There are limited statistics outlining the prevalence of balance disorders. In part, this is due to the disorders' qualifying criteria which differs across conditions and partly because the symptoms are hard for patients to describe resulting in misdiagnosis. The following section enumerates the limited epidemiological data available regarding vestibular disorders.

Balance disorders

- The prevalence of balance problems at age 70 is reported to be 36% in women and 29% in men (Jonsson, Sixt, Landahl & Rosenhall, 2004);
- It is estimated that approximately 12.5 million Americans who are 65+ years have a significant balance problem that impairs their ability to function (NIDCH, 1997);
- Approximately 50% of the USA population is affected by a balance or vestibular condition at some point in their lives (NIDCH, 1997);
- 50% of the falls that occur in the elderly are due to vestibular problems (Batty, 1998, cited in Neurocom, 2003).

Ménière's Disease

Prevalence figures for Ménière's Disease vary depending on the criteria used to diagnose Ménière's disease. Estimates include:

- The estimated prevalence of Ménière's ranges from 0.2 to 1% of the population;
- NIDCD (2001) estimates that there are currently approximately 615,000 individuals with diagnosed Ménière's disease in the United States and 45,500 newly diagnosed cases each year;
- Havia (2004) contents that the population based prevalence of Ménière's disease may be as high as 513 per 100,000 individuals aged 12 or more. A peak in prevalence was also noted by the author, in the age ranges of 61-70;
- Britain and Sweden have a relatively high incidence 1 % of Ménière's disease;
- Onset is usually middle age (i.e., 40 years to 50 years) (Salt, 2003).

BPPV

- The lifetime prevalence of BPPV is estimated to be 3.2% in females, 1.6% in males and 2.4% overall (von Brevern et al., 2007).

- In Germany, an estimated 1.1 million adults suffer from BPPV each year (von Brevern et al., 2007).
- BPPV is more common amongst women (EM Guidemap).

Functional impairments associated with vestibular disorders relevant to driving

The major symptoms of the above disorders that are significant in terms of functional driving impairments are:

- Vertigo;
- Nystagmus;
- Oscillopsia.

Vertigo “is the illusory sensation of motion” (Rosenberg & Gizzi, 2000, p. 1) and may give the impression of falling (NIDCH, 2002). Vertigo attacks may range from mild to severe. Mild episodes may induce a false impression that the environment is tilting or moving somewhat. A severe episode of vertigo may produce strong spinning sensations with accompanying symptoms of nausea, sweating or vomiting (EM Guidemap). Other symptoms may include fear, anxiety, and heart and blood pressure changes (NIDCH, 2002). Vertigo is particularly incapacitating because it prevents the person from doing anything that involves movement and the spinning sensation carries with it a real threat of falling (Salt, 2003).

Nystagmus is “a rhythmic oscillation of the eyes” (Barton, 2000, p3) and has a large number of possible causes including vestibular disorders. Nystagmus may have a slow-fast rhythm depending on the cause. The eye moves in one direction during the slow phase. The brain senses this and compensates by pulling the eye back in the other direction, in a jerk like motion. The direction of the nystagmus (i.e. right or left nystagmus) is defined by the direction that the eye moves in the fast phase (EM Guidemap). Subjects with nystagmus do not necessarily perceive the environment as jerking however they may feel it is moving.

Oscillopsia refers to the illusion that the environment is moving “to and fro” (Rosenberg & Gizzi, 2000). It indicates a decrease in function in one side of the vestibular apparatus (i.e. bilateral vestibular function.) Patients with oscillopsia may experience blurred vision, disorientation and visual acuity decrements. In the road environment, there may be difficulty in reading signs whilst the person is in motion. Walking on uneven surfaces, such as gravel, may affect balance due to the uneven motion it engenders (EM Guidemap).

Treatment for vestibular conditions

Ménière's Disease

- Dietary recommendations, such as low sodium diets, reductions in the consumption of sugar, MSG (Salt, 2003), alcohol and caffeine consumption (School of Medicine, 1995).

- Medications such as anti-vertigo, anti-nausea and anti-emetic drugs (Salt, 2003), and certain kinds of antibiotics (NIDCH, 2002).
- Vestibular exercises and manoeuvres to position the head and body, particularly the Epley manoeuvre (School of Medicine, 1995). This type of therapy is designed to stimulate the body into compensating for the disorder (NIDCH, 2002).
- Surgery eg labyrinthectomy (School of Medicine, 1995).

Benign Paroxysmal Positional Vertigo (BPPV)

- Dietary recommendations (VEDA, 2009).
- Vestibular exercises and manoeuvres to position the head and body, particularly the Epley manoeuvre (School of Medicine, 1995). This type of therapy is designed to stimulate the body into compensating for the disorder (NIDCH, 2002).
- Medication (VEDA, 2009).
- Surgery is only very rarely conducted and consists of “canal plugging” (School of Medicine, 1995).

Pre-May 2003: Relationship between vestibular conditions and road safety outcomes

Vestibular disorders have not been studied extensively in the context of relative risk for driving (Campbell & Lutsep, 2001). Some clinicians have recommended that commercial drivers with vestibular disorders may need to curtail their driving due to the symptoms of vestibular disease (Salt, 2003). The review identified two studies which indicated that the driving ability of patients with vestibular disease or its symptoms is impaired (see Table 43).

Crashes

There were no studies identified during the review period addressing crash risk associated with vestibular disorders.

Citations

There were no studies identified during the review period addressing citations as an outcome measure of risk associated with vestibular disorders.

Driving Performance

Clarke, Clarke & Scherer (1993) investigated the extent to which involuntary eye movements such as occur with nystagmus (a symptom of vestibular disease), impact on steering a car and driving speed. The driving performance of 30 healthy subjects was tested on a computer simulator. Vestibular imbalance was then induced in these healthy subjects by unilateral caloric stimulation to either the right or left ear and driving ability was re-tested. Unilateral caloric stimulation involves flushing one ear with water, which

stimulates the labyrinth in the vestibular apparatus, and this in turn induces nystagmus. It was found that driving speed was reduced following induced nystagmus and that subjects drove much closer to the centre line. Pronounced deviations in steering behaviour were also observed. When right nystagmus was induced, and subjects were required to turn left, the car was steered first to the right and then an abrupt correction was made to the left. When right nystagmus was induced, and subjects were required to turn right, the car was steered to the right but the trajectory deviated markedly from “normal” steering behaviour. Similar deviations in steering were observed when left nystagmus was induced, except in the opposite direction.

Page & Gresty (1985) presented the driving history of two patients with vestibular disease and made comparisons with four other patients with “minimal neuro-otological disease” who became disorientated in certain driving situations. All of the patients described unusual illusions of movement, that is, that the car was veering off course. However, only those with vestibular disease actually drove off course.

Post-May 2003: Relationship between vestibular conditions and road safety outcomes

The current review found no studies dealing with crashes and vestibular disorders. There was one study identified concerning citations and vestibular conditions and one study relating to driving performance.

Crashes

No studies were identified that deal specifically with crashes and vestibular disorders.

Citations

Cohen, Wells, Kimball and Owsley (2003) used a structured version of the Driving Habits Questionnaire (DHQ) to interview patients with various vestibular disorders [BPPV (n = 34), Chronic vestibulopathy (n = 27), Meniere’s disease (n = 48), PostOp (n = 9)] and 51 controls. Diagnosis of vestibular conditions were determined through hospital and clinic medical reports and patients recruited through the senior researcher’s clinical caseload. Controls were recruited from visitors accompanying patients to the hospital department. Patients and controls’ did not differ across age or gender, though, as expected, significant differences were recorded in patients’ and controls’ history of vertigo ($p = .0001$). Co-morbid medical conditions were not assessed, and controls’ medical status was not reported. Participants were asked to report their rate of citation for moving violations. Fewer patients (6%) than normal subjects (16%) reported receiving fines over for moving violations in the past year, however this was not statistically significant ($p = .072$). Overall, controls reported no significant deficits.

Driving Performance

In the study by Cohen et al. (2003), reviewed above, significant differences were identified between drivers with vestibular disorders and controls for self-reported driving difficulties. Overall, controls reported no significant driving deficits. Patients with BPPV reported little difficulty with aspects of driving e.g. difficulty staying in lane or driving up or down a ramped parking garage. All diagnostic groups found the following driving challenges significantly more difficult compared to controls: driving in rain, driving alone, making left turns across traffic, freeway driving, driving amongst

high traffic on local roads, driving in rush hour, driving at night, driving in parking garages, changing lanes, staying within the lane, traffic checks and driving up or down ramped garages. It was found that the two conditions most problematic for driving were Ménière's and Chronic Vestibulopathies.

No other studies were identified during the review period examining driving performance as an outcome measure of risk associated with vestibular disorders.

Summary

Overall, there was little evidence for the risk associated with drivers with vestibular disorders. No studies were identified which dealt specifically with crash risk. Neither were there studies which distinguished between acute (recent onset) vestibular symptoms and chronic conditions. Vestibular disorders interfere with patients' ability to accurately perceive their position and motion relative to the fixed environment and gravity. Vertigo induces illusions of spinning and rocking such that sufferers may become disorientated, lack co-ordination and lose their balance. Both of these symptoms have been found to impair driving ability, especially in situations of lowered visibility and circumstances where spatial navigation skills are required. They have also been found to produce unwanted deviations in the direction of steering. More research on the impact of these disorders is required, particularly with regards to crash risk.

Table 43 Summary of studies of risk associated with vestibular disorders

Study: Author/date	Methods	Outcome Measure of Risk	Results
Clarke, Clarke & Scherer (1993)	Case-control Control n= 30 Case=30 Same subjects used for control & case groups.	1. Alterations in speed. 2. Deviations in steering behaviour.	Following induced nystagmus: - Reduction in speed. - Drove much closer to the centre line - Pronounced deviations in steering behaviour were also observed – car veered in the direction of the nystagmus and then driver overcompensated by pulling sharply in the opposite direction.
Cohen, Wells, Kimball & Owsley (2003)	Case-control Control (n=51) Case (broken up into vestibular disorders): BPPV (n=34) Chronic vestibulopathy (n=27) Meniere’s disease (n=48) PostOp (n=9)	Self-reported driving challenges (elicited through structured interview of Driving Habits Questionnaire), rate of citations.	Controls reported a higher rate of citations compared to diagnostic groups. BPPV patients reported few problems with particular aspects of driving. All other patient groups reported moderate to extreme problems in situations of lower visibility and where specific path integration or spatial navigations skills were required.
Page & Gresty (1985)	Case reports 6 patients (2 with vestibular disease & 4 with “minimal neuro-tological disease”).	On-road self-reported driving behaviour.	All patients reported illusory sensations that the car was veering off course. However, only those with vestibular disease actually drove off course.

Approaches to management

Fitness to drive

All of the countries surveyed in the Licensing Guideline Tables generally stipulate licensing criteria that indicate the serious nature of Ménière's Disease, although there are some variations in the exact nature of standards. Sweden, New Zealand and the UK specifically require that private licence holders with Ménière's Disease must desist from driving if the symptoms preclude safe driving, particularly if vertigo is of sudden and unexpected onset (see Table 39). The USA guidelines regard vestibular diseases, which have vertigo as a major symptom, to be episodic conditions and therefore the criteria for epilepsy apply. The UK and Australia also mandate that a conditional licence only may be held, although the UK does make provision for the reinstatement of an unrestricted licence if the person remains symptom-free. The licensing criteria for commercial licences are somewhat more severe, with the UK and Australia requiring a one-year period free of symptoms.

Training and rehabilitation

No studies were identified which specifically addressed the impact of therapies on driving capabilities for people with vestibular disorders.

Table 44 Private licensing guidelines for drivers with vestibular disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Meniere's disease	<i>Recurrent attacks of vertigo without warning:</i> Desist from driving until vertigo is controlled.	May not hold an unconditional licence A conditional licence may be issued subject to treatment response & person's functional ability to drive safely. Periodic review required.	<i>Upon diagnosis:</i> Desist from driving. Driving may resume after satisfactory treatment of symptoms. Unrestricted licence will be reinstated if person remains free of symptoms.	An unrestricted licence may be issued if balance problems or episodes are rare, or never incapacitating for driving. Reviews required every 2 – 5 years. Those experiencing recurring or incapacitating episodes, but not in past 1 – 3 months may drive with medical practitioner approval. Reviews required every 6 to 12 months. Restricted from driving if balance problems are chronic and incapacitating.	Desist from driving if vertigo impairs driving ability & occurs suddenly. May resume driving when treated successfully.	Licence denial if vertigo attacks are unexpected & impair safe driving.
Benign Paroxysmal Positional Vertigo	<i>Recurrent attacks of vertigo without warning:</i> Desist from driving until vertigo is controlled.	No licence restrictions if no symptoms are experienced when upright. Desist from driving if symptoms are present in the upright	Not specifically addressed.	An unrestricted licence may be issued if balance problems or episodes are rare, or never incapacitating for driving. Reviews required	Desist from driving if vertigo impairs driving ability & occurs suddenly. May resume driving when treated successfully.	Not specifically addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
		position.		<p>every 2 – 5 years.</p> <p>Those experiencing recurring or incapacitating episodes, but not in past 1 – 3 months may drive with medical practitioner approval. Reviews required every 6 to 12 months.</p> <p>Restricted from driving if balance problems are chronic and incapacitating.</p>	Some people may only be temporarily affected by vertigo & may only need to pull over to the side of the road until sufficiently recovered.	

References

- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Barton, J.J. (2000). *Vertigo and vestibular function*. Review of Ophthalmology. Available at http://www.sightstreet.com/ContentLibrary/neu_vertigo_csro1020.htm . (Accessed 3 July, 2003).
- Campbell, G.H., & Lutsep, H.L. (2001). Driving and neurological disorders. *eMedicine Journal*, 2(11), 1-28.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Clarke, A.H., Clarke, M.C., & Scherer, H. (1993). *The influence of vestibular disorder on driving behaviour*. Conference proceedings from Vision in Vehicles V; Fifth International Conference, Glasgow, 1993, p41-48.
- Cohen, H. S., Wells, J., Kimball, K. T., & Owsley, C. (2003). Driving disability and drowsiness. *Journal of Safety Research*, 34(4), 361-369.
- EM Guidemap (no date) – Vertigo. Available at <http://www.homestead.com/emguidemaps/files/vertigo.htm> (Accessed 3 July, 2003).
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Jönsson, R., Sixt, E., Landahl, S., & Rosenhall, U. (2004). Prevalence of dizziness and vertigo in an urban elderly population. *Journal of vestibular research: equilibrium & orientation*, 14(1), 47-52.
- Havia, M., & Kentala, E. (2004). Progression of symptoms of dizziness in Ménière's disease. *Archives of otolaryngology--head & neck surgery* 130(4), 431-435.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Neurocom (2003). *Vestibular Disorders*. Available at <http://www.onbalance.com//index.cfm?action=vest>. (Accessed July, 2003).
- NIDCH (2009). The National Institute on Deafness and other Communication Disorders. Available at http://www.nidcd.nih.gov/health/balance/balance_disorders.htm (Accessed May, 2009).
- NIDCH (2001). The National Institute on Deafness and other Communication Disorders. Available at <http://www.nidcd.nih.gov/health/balance/meniere> (Accessed May, 2009).
- Page, N.G.R., & Gresty, M.A. (1985). Motorist's vestibular disorientation syndrome. *Journal of Neurology, Neurosurgery, and Psychiatry*, 48, 729-735.

- Rosenberg, M.L., & Gizzi, M.G. (2000). Practical issues in the management of the dizzy and balance disorder patient. *Otolaryngologic Clinics of North America*, 33(3), 1-9.
- Salt, A.N. (2003). *The Ménière's Page*. Washington University School of Medicine. Available at <http://www.oto.wustl.edu/men>. (Accessed July, 2003).
- School of Medicine (1995). *Vestibular Disorders*. Available at <http://www.bme.jhu.edu/labs/chb/disorders/disorder.htm>. (Accessed July, 2003).
- Swedish Road Administration. (1998). *Swedish Road Administration Statute Book*. Sweden.
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- VEDA (2009). Vestibular Disorders Association. Available at <http://www.vestibular.org>. (Accessed July, 2009).
- Von Brevern, M., Radtke, A., Lezius, F., Feldmann, M., Ziese, T., & Lempert, T. (2007). Epidemiology of benign paroxysmal positional vertigo: a population based study. *J Neurol Neurosurg Psychiatry*, 78(7), 710-715.

3.13 VISION DISORDERS

Visual function is fundamental to driving a motor vehicle. Some researchers have suggested that vision may be responsible for up to 95% of the sensory input for drivers (Hills, 1980; Shinar & Scheiber, 1991), although Sivak (1998) has argued that there is a lack of data available to derive accurate estimates. Nevertheless, vision is the principal source of sensory information used when driving and it seems obvious that vision deficits would be related to crash risk. However, most measures of visual ability seem to share only minimal relationships with the perceptual requirements of driving in complex and dynamic traffic conditions (Schiff & Arnone, 1995). The evidence relating crash risk to visual diseases (that may cause multiple impairments) is even more unclear and difficult to evaluate. Biases in sample databases restrict the usefulness of many studies. Another complicating factor is that methods of measuring visual parameters vary in different parts of the world and across studies, making comparisons across studies difficult (for example, acuity is measured differently in the US and other parts of the world and there are also very many ways of measuring visual fields, some of which are more relevant to driving than others).

Many of the eye conditions reported are also directly associated with ageing. Around 82% of all people who are visually impaired are age 50 and older, although they represent only 19% of the world's population (WHO, 2009). This introduces many possible confounds of vision with cognitive and physical limitations of the ageing driver. Therefore, appropriate research protocol and/or appropriate statistical techniques must be implemented to account for the potential for confounding. However, adjusting for comorbid conditions in statistical modelling discounts the crash risk associated with specific eye conditions due to collinearity, that is, they account for some of the same variance in crash risk.

The following discussion reviews and evaluates the evidence associating crash risk with specific visual conditions. The primary focus of this section is on crash risk associated with vision diseases that have a high prevalence and result in serious visual impairments. The crash risk related to other eye conditions that result in visual difficulties (either with or without corrective lenses) is also outlined. Table 45 provides a summary of findings of studies on visual diseases and conditions on crash risk. Table 47 provides a list of abbreviated terms used throughout the chapter.

Eye diseases and conditions

According to the Centre for Eye Research Australia, around three quarters of all vision loss is caused by just five conditions: refractive error, cataract, glaucoma, age-related macular degeneration, and diabetic retinopathy (Taylor, Keeffe & Mitchell, 2004). The most common, refractive error has affordable, cost effective and simple correction. Cataract is common among older adults, but it is also treatable with effective surgical techniques. The visual loss associated with the other three prevalent conditions can usually be managed if detected early enough. However, in many cases some visual deterioration will have occurred and the individual affected by the condition may continue to drive, potentially increasing crash risk. The evidence relating to crash risk and these conditions is reviewed below.

3.13.1 CATARACT

Definition of cataract

Cataract is a condition where the normally clear lens of the eye becomes clouded and opaque. Cataracts restrict the amount of light passing through the lens and also scatter the light resulting in images being poorly focussed on the retina. Vision with cataracts has been likened to looking through a frosted window and the symptoms include blurred vision, glare or light sensitivity, double vision, fading or yellowing of colour vision, poor night vision, and as the condition worsens, halos around lights (Taylor et al., 2004).

Cataract is a degenerative condition that usually develops slowly and as a normal part of the ageing process. However, cataracts can be caused by diabetes, injury to the eye, long-term ultraviolet light exposure, and certain medications, and is also associated with a family history of the condition and smoking. While cataracts can occur in one eye, the condition is typically bilateral in older adults, although the rate of development generally differs in each eye and among individuals. The condition may take several years to worsen to a point where daily activities such as reading and driving are compromised. The only treatment available for cataracts is surgery. In the early stages and with mild symptoms, corrective glasses may be recommended (Taylor et al., 2004).

Prevalence of cataract

The estimates of cataract prevalence vary considerably depending on the source. Large numbers of people with cataracts may not be included in some databases because they do not have significant visual impairments (Lighthouse International, 1998). However, cataracts are thought to affect around half of all adults aged over 75 years, with approximately one quarter having late stage cataract development (Klein, Klein, & Linton, 1992a) or a chronic cataract condition (Centre for Disease Control, 1995). Australian data shows that the ten-year prevalence of cataract increases from just under 5% in the 40-49 year age group to 100% in the 90+ year age group (Taylor, 2001) with surgery required in up to half of these cases (Taylor et al., 2004). Overall, data from Australia, Europe and the US shows that around 17.2% of the population over 40 years of age are affected by cataract (Congdon et al., 2004).

Cataracts are the leading cause of blindness in the world accounting for an estimated 16 million cases of blindness (Lighthouse International, 1998). Cataracts cause approximately one in seven cases of blindness in the US in people aged over 45 years. The only treatment available for cataracts is surgery (Taylor et al., 2004). The procedure removes the clouded lens and replaces it with an artificial or "intraocular" lens and can significantly improve vision (McCarty, Nanjan & Taylor, 2000; Owsley, McGwin, Sloane, Wells, Gauthreaux & Stalvey, 2002; Talbot & Perkins, 1998).

Functional impairments associated with cataracts relevant to driving

Cataracts compromise many aspects of vision including visual acuity (Mäntyjärvi & Tuppurainen, 1999; Owsley, Stalvey, Wells & Sloane, 1999; Rubin, Adamsons & Stark, 1993), contrast sensitivity (Mäntyjärvi & Tuppurainen, 1999; Rubin et al., 1993), and visual field sensitivity (Owsley et al., 1999). Although surgical removal of the cataract is effective with at least 85% of cases reaching 20/40 acuity or better post-surgery (McCarthy, Nanjan & Taylor, 2000; Talbot & Perkins, 1998), surgery is usually only performed when limitations in visual function become serious. Therefore, a large number of older adults may be driving with cataract-affected vision.

Pre-May 2003: Relationship between cataract and road safety outcomes

A number of studies have examined visual functioning and licensing implications of people with cataract, however, despite the prevalence of cataracts, research evaluating the crash risk associated with this condition is limited.

Crashes

The Impact of Cataracts on Mobility (ICOM) project is an ongoing prospective study evaluating data on the effects of cataract surgery on driving mobility in older adults. Recent analyses by Owsley and colleagues (Owsley et al., 1999; Owsley, Stelvey, Wells, Sloane & McGwin, 2001) using the baseline data from the ICOM project have examined the issue of crash risk and cataracts.

Owsley et al. (1999) recruited 279 participants from eye clinics who had vision impairments primarily due to cataracts (97% bilateral) and 105 participants free of identifiable eye disease. The cataract group were all assessed as having visual acuity of 20/40 or worse (best-corrected distance) in at least one eye and the comparison group had acuity of 20/25 or better in each eye (best-corrected distance). All participants were older adults aged between 55 and 85 years, independently living in the community, and legally licensed to drive. Crash risk was determined from crash data for the 5 years prior to enrolment in the ICOM study and was obtained from the Alabama State records. Only crashes where the participant was deemed to be at least partially at-fault were used. Determination of "at-fault" was made retrospectively from details of the crash records and visual function was measured at the end of the crash period surveyed. A Driving Habits Questionnaire (DHQ) was completed by participants to obtain information about current driving status, driving exposure, dependence on others, driving difficulty and self-reported crashes and citations. Participants were also assessed for general health, cognitive status, and depression.

Owsley et al. (1999) found that older drivers with cataract were almost 2.5 times more likely than those without eye disease, to have had an at-fault crash during the previous 5 years even after adjusting for driving exposure (RR: 2.48, 95% CI 1.06 - 6.14). When adjusting for impaired health, the relative crash risk for those with cataracts remained 2.5 times higher. While the number of at-fault crash-involved older drivers was still relatively low (35 for participants with cataract and 6 for no cataract), the authors also reported some findings from the DHQ, which indicate that drivers with cataracts experience difficulties when driving. Compared to drivers without cataract, drivers with cataracts were significantly more likely to report difficulty driving in the rain, driving alone, turning across traffic, driving on interstate roads, driving in heavy traffic, driving in rush hour, and driving at night. Cataract-affected drivers also preferred not to drive long distances and preferred not to drive more than 150 miles per week or more than 5 days per week, and were more likely to have received advice to limit or stop driving (although most participants in both groups did not report any such advice).

An interesting finding reported by Owsley et al. was the self-reported difficulties experienced by drivers with cataract, particularly in relation to night driving. While it is possible that self-regulatory driving practices reduce risk of crashes, there appear to be no studies on crash rates for night and day time driving amongst drivers with cataract. More research is needed to address this issue.

In a more recent study, Owsley et al. (2001) examined the types of visual impairments caused by cataracts that serve as the basis for the elevated risk of motor vehicle crashes. Their sample, comprising participants from the same ICOM project, included 274 older adults with cataract in one or both eyes and 103 older adults without cataracts. One quarter of the cataract group also had a coexisting visual condition, mostly either age-related maculopathy or glaucoma, whereas the comparison group had no evidence of eye disease. This is problematic given that the relatively low numbers of at-fault crash involved drivers could largely consist of older adults with co-existing eye conditions. This is also a limitation of the previous study which was essentially the same sample (Owsley et al. 1999). Crash risk data were compiled in the same manner as described by Owsley et al. (1999).

Owsley et al. (2001) assessed visual acuity, contrast sensitivity and disability glare for each eye while the participant wore their normal lens correction used for driving. Contrast sensitivity (see section 3.13.13) was assessed using the Pelli-Robson test (Clement Clarke International Limited) (Pelli, Robson & Willkins, 1988). This study confirmed the crash risk measures demonstrated in the earlier study with the cataract driver group 2.5 times more likely than drivers without cataract to be involved in an at-fault crash. Among the three types of visual assessment, only the lowest level of contrast sensitivity (1.25 or less) was significantly associated with at-fault crash risk. The odds ratio for low contrast sensitivity (in the better of the participant's two eyes) amongst crash-involved was 2.65 (95% CI: 1.06 - 6.61). After adjusting for demographics, cognitive status, general health, and driving exposure this association increased to 4.97 (1.69 - 14.63). While the confidence interval was large, the same pattern of findings was confirmed in the worst of the two eyes with an adjusted odds ratio of 7.06 (1.88 - 26.52). Other measures of contrast sensitivity deficits such as impairments in both eyes compared to one eye or neither eye, further supported the importance of adequate contrast sensitivity in drivers with cataracts. However, contrast sensitivity impairment is also associated with other conditions such as age-related macular degeneration (Szlyk et al., 1995), glaucoma (Szlyk, Taglia, Paliga, Edward & Wilensky, 2002), and diabetic retinopathy (Sokol, et al., 1985), which was found to co-exist with cataracts in 25% of that group (Owsley et al., 2001). Nevertheless, the effects of cataract surgery on measures other than visual acuity appear to be pertinent determinants of post-surgery crash risk.

Salzberg and Moffat (1998) examined the crash and driving citation records (also see below) of 45 drivers with cataracts who were referred to the Washington State Department of Licensing Special Examination Program. This special exam program included an in-depth interview and an extended on-road driving test typically within a limited range of travel near the driver's residence and routes used by the driver. The most common outcome of the examination process was to restrict the driver's travel to within specific areas and times of day, and require the driver to use corrective lenses or particular vehicle controls (e.g., power steering). However, drivers who failed the exam had their licences cancelled.

The records of the drivers with cataracts who passed the exam were examined over a 5-year period (1.75 years prior to the examination and 3.75 years after) and compared to 449 drivers in a control group of older drivers without medical conditions matched on age, gender and city of residence. The control group had a crash rate of 3.82 per 100 licensed drivers prior to the examination period and 1.17 in the post examination period. This was comparable to crash rates for the population of approximately 4 million licensed drivers in the state of Washington, that recorded a rate of 3.47 collisions per 100 licensed drivers during 1996. The older drivers with cataracts who continued to drive had a pre-exam crash

rate of 5.08 and post-exam rate of 2.05. Thus, while the crash rate reduced substantially in the period after the special exam to a level below the general population, this was also true of the control group that was not part of the special examination program. The authors explained the reduction of crashes in the control group by the normal lowering of driving exposure with increasing age. However, drivers with cataract are also likely to restrict the amount and range of their driving even in the absence of an examination (Owsley et al., 1999). A lack of examination of the control group meant that some of these drivers might have developed cataracts during the 5-year period that may subsequently result in self-imposed driving restrictions. Furthermore, it is unclear how many cataract-affected drivers had cataract surgery to restore impaired vision. An additional limitation of this study was the pooling of data to assess the crash rate per 100 licensed drivers per year. This reduced the ability to assess the range of individual variation in what was a fairly restricted sample of 45 cataract-affected drivers.

Results of the Salzberg and Moffat (1998) study suggested that the special examination program for drivers with cataracts did not appear to reduce the rates of crashes beyond that achieved by normal self-regulatory behaviours undertaken as an individual ages. However, the research compared only those drivers who had *passed* the examination process with a control group of drivers. Crash rates of those drivers with cataract who were not referred into the program or who failed the special exam or who ceased driving voluntarily (rather than take the special examination) would also be of interest in evaluating the overall effectiveness of such programs. This restricted sampling is a serious bias, calling into question the validity of conclusions.

McGwin, Sims, Pulley and Roseman (2000) used a population-based case-control study to examine the relations among medical conditions, medications and crash risk of drivers aged over 65 years. They used the Alabama state records to identify individuals who had been involved in crashes during 1996 (cases) ($n = 901$, including 244 at-fault and 182 not at-fault drivers) and a random sample of controls ($n = 475$) matched on 1-year age groups and gender. Participants were interviewed by telephone and asked to recall the previous 18-24 months and to indicate if a health care professional had told them that they had any medical conditions (from a list including cataracts, glaucoma, and diabetic retinopathy) and whether they were taking medications. Visual functioning and short mental status questionnaires were administered for current deficits, and self-reported driving habits and mileage were also obtained for the previous period. The inconsistency between recalled (and unverified) medical conditions, and visual and cognitive status assessed by telephone interview and up to 12 months beyond the end of the crash record period, were major shortcomings of this study. While a number of associations were found for medical conditions and medications with crash risk, few were statistically reliable. No reliable associations of cataract (nor glaucoma or visual function) with crash risk were established (OR: 1.0, CI: 0.7 – 1.5 for not at-fault compared with non crash-involved).

Earlier research by Foley, Wallace and Eberhard (1995) also used a population-based cohort study to evaluate the role of self-reported physical, mental and sensory factors in vehicle crashes regardless of whether an injury was sustained. In total 1791 drivers aged 68 years and older in the Iowa Established Populations for Epidemiologic Studies of the Elderly cohort were interviewed and police reported crash records were examined. The regression model examined age and gender adjusted odds ratios for the selected risk factors (relative risk for cataract was not significant, RR: 0.9, CI: 0.6 – 1.2). Foley et al. found that gender was a more important factor among this group than age with men exhibiting a 60% increase in crash risk than women. An elevated crash risk was also revealed among drivers

with episodes of back pain, use of anti-inflammatory drugs, and poor memory performance, but not on the visual measures of cataract and glaucoma, and the ability to read newsprint and recognise faces at a distance. However, telephone interviews used in this study and McGwin et al. (2000) are unlikely to be sensitive measures of the visual conditions such as cataract and glaucoma and provide no estimate of the progression of these conditions. The study was also unadjusted for other medical conditions and, in particular, driving exposure was not evaluated. The crash rate of the participants was also 20% less than the average for that age group suggesting that the sample may not adequately represent the older driver cohort.

Using a similar method to Foley et al. (1995), Stewart, Moore, Marks, May and Hale (1993) examined 142 crash involved and 1289 non crash-involved older drivers during a 5-year period prior to interview. Unlike Foley et al., Stewart et al. did not find age, gender, common drug ingredient, or memory loss to be associated with increased crash risk. They also found no association between ocular disease and increased crash risk. This study was limited by its reliance on risk factors including visual disease that were not clinically verified and by recording of crash events that occurred prior to the interview. This discounts the degenerative nature of the condition and may have the effect of underestimating the association of risk factors with crashes.

A study by McCloskey, Koepsell, Wolf and Buchner (1994) employed a matched case-control evaluation of drivers treated for injuries in police-reported crashes during 1987 and 1988. McCloskey was careful to adjust for the amount of driving and for confounding variables among the cases and controls. However, they found that the 234 older drivers involved in injury-crashes were not significantly more likely to have ocular disease (including cataracts) than controls. McCloskey et al. attributed the lack of association to factors such as adoption of self-regulation of driving practices by those experiencing visual difficulties and inclusion of participants with early-stage development of ocular conditions which might result in an inability to differentiate crash and non-crash involved older drivers.

Citations

In their study described above, Salzberg and Moffat (1994) also compared the traffic violation records of 45 drivers with cataracts who passed the Washington state exam to 449 drivers in a control group of older drivers without medical conditions matched on age, gender and city of residence. Pre- and post- exam traffic violation rates were examined over a 5-year period (1.75 years prior to the examination and 3.75 years after). The control group had a violation rate of 7.51 per 100 licensed drivers prior to the examination period and 2.26 in the post examination period. The older drivers with cataracts who continued to drive had a pre-exam violation rate of 15.24 and post-exam rate of 2.05. Generally, these reductions mirrored the pattern of pre- and post exam reductions observed for crashes. However, clearly, there was a dramatic reduction in violation rates for drivers with cataracts compared with those observed over the same period for drivers in the control group. The contribution of self-regulatory practices here is not known, but it is possible that while both groups may have self-regulated their driving behaviours over the 5 year period thus reducing exposure and the opportunity for driving offences, these practices may have been more widespread and/or more effective amongst those with cataracts. As discussed more fully in the previous section, restricted sampling procedures adopted in this study resulted in a serious bias, calling into question the validity of conclusions.

Driving Performance

Studies by Wood and colleagues (Wood & Troutbeck, 1994; Wood & Troutbeck, 1996) on the performance of drivers with cataract-related visual impairments indicate some deficits in driving performance and changes in driving behaviour. Wood and Troutbeck (1994) used specially designed goggles to replicate the visual impairments caused by cataracts, but visual acuity still satisfied the drivers' licence requirements. Driving performance measures including peripheral awareness, manoeuvring, reversing, reaction time, speed estimation, and road position were assessed on a closed road circuit. Drivers with simulated cataracts had poorer awareness of peripheral cues than baseline conditions, but had no other differences indicating drivers were less safe. Reaction times were not delayed for drivers with simulated cataracts, but they took longer to reverse park and complete the driving course. They also completed the course touching significantly fewer cones than in the baseline condition, although this was probably due to driving more slowly through the course. While simulating cataracts was useful to show how visual impairment could affect driving without adaptation and contamination by other factors, it is not representative of how the condition develops and how compensation could occur.

A later study by these authors (Wood & Troutbeck, 1996) examined the same driving performance measures on a road circuit, but included drivers with true visual impairment from cataracts. They found that the impairments from true cataract-impaired drivers supported the findings from simulated cataracts albeit with less marked differences to age-matched control participants. However, the degree to which performance assessed on a driving course without road intersections and other vehicles translates into real world crashes is still uncertain. Driving on a road may overcome some of the limitations of laboratory simulations relating to fidelity and detachment from driving risk, but closed-course measures do not adequately represent performance in the real-world driving environment involving complex and dynamic traffic interactions.

Treatment of cataract and road safety outcomes

Crashes

The surgical removal of the crystalline lens replaced by an intraocular lens can lead to significant improvements in visual acuity and contrast sensitivity, and a reduction in disability glare and visual field sensitivity (Elliott, Patla, & Bullimore, 1997; Rubin et al., 1993; Talbot & Perkins, 1998). However, very few studies have specifically examined the effects of cataract surgery on crash risk. Recently, Owsley and colleagues (Owsley et al., 2002) have published findings on the impact of cataract surgery on crash risk in the years following surgery for a group of cataract-affected drivers in the ICOM study. They compared 174 drivers that had undergone surgery to 103 cataract-affected drivers that had not had surgery. The unadjusted crash risk ratio for the surgery group compared to the no surgery group in the two years following the procedure was 0.64 (95% CI, 0.37-1.13). After adjusting for race, baseline visual acuity, and contrast sensitivity, the crash risk ratio was 0.47 (95% CI, 0.23-0.94), suggesting around a 50% reduction in crash risk. The absolute rate of crashes associated with the cataract surgery group was around 5.8 crashes per million miles of travel compared to around 9 crashes per million miles in the group that declined surgery. Furthermore, compared to the previous five-year period, the no surgery group recorded a significant 72% increase in crash rate whereas the surgery group experienced an increase of 27%, which was not statistically significant.

Notwithstanding the important finding that drivers who elect surgery to improve their cataract-affected vision may have a more favourable crash risk than those who do not elect surgery, there may be a number of variables that could explain some of the benefits

reported. Klein (2002) noted that there may be factors motivating the decision not to have surgery that could influence their crash risk at follow-up. The no surgery group had better vision than the surgery group at baseline (i.e., prior to surgery) and did not appear to deteriorate during follow-up period. However, the no surgery group had a worse crash rate at baseline than the surgery group (though not significant) and a significant increase in crash rates during follow-up. Owsley et al. (2002) were also careful to note that the crash record for both driver groups declined and therefore cataract surgery may not effectively improve their driving performance.

Citations

No studies reporting the relationship between treatment of cataract and driving citations or traffic violations were found.

Driving performance

Improvements on self-reported measures of visual function while driving after a cataract surgery have also been demonstrated (Mönestam & Wachtmeister, 1997). Mönestam & Wachtmeister found that 81% of drivers in their study of cataract surgery participants reported some problems or large problems with visual function while driving prior to surgery. Among the drivers with cataracts, 71% specified driving in darkness as a problem, 37% reported problems with estimating distance, 11% said they experienced difficulty with glare and 7% felt eye fatigue. Post surgery, a greater percentage of participants were driving and only 5% reported problems with visual function (mostly glare disability) *in the operated eye*. The percentage of participants reporting difficulty with estimating distance reduced from 37% to 6% after surgery. Mönestam & Wachtmeister concluded that cataract surgery benefited drivers in terms of subjectively reported visual function difficulties and surgery on the second eye should be operated on, if necessary, to achieve best possible visual function for driving.

Talbot and Perkins (1998) assessed 50 participants aged between 47 and 90 years pre- and post cataract surgery on their second eye to examine whether a second surgical operation is necessary to restore adequate visual function. They found that while 88% of the eyes that had been operated on had visual acuity of 6/12 (20/40) or better, only 52% of individuals that had cataract surgery on one eye passed the driving standards or the UK Driver and Vehicle Licensing Agency (DVLA). Failure to pass the licensing standards was largely due to poor binocular visual functions such as visual field. However, after their second eye operation, 66% of people had improved binocular visual acuity, 54% of people had binocular field of vision improved by 20 degrees or more horizontally, and 36% had improved vertical fields. Importantly, after the second surgery, no participants had a binocular visual acuity worse than 6/12 and only 14% failed the visual field assessment, which improved the rate of drivers passing the DVLA standard from 52% to 86%. Nevertheless, there was only a minor improvement in contrast sensitivity after the second surgery and a small proportion of people experienced a reduction in visual field sensitivity.

Post-May 2003: Relationship between cataract and road safety outcomes

Only one new study was identified in the review period post-May 2003 which addressed the relationship between cataract and road safety outcomes and two studies were identified addressing the effects of treatment on driving performance of drivers with cataract. A summary of the study findings is presented in Table 45.

Crashes

There were no studies identified during the review period that assessed the relationship between cataracts and crash risk.

Citations

No studies were identified addressing the relationship between cataracts and driving citations.

Driving Performance

Carberry, Wood, Watson and King (2006) examined driving performance in 33 Australian drivers with cataracts and 13 controls. Participants completed a driving performance assessment on a closed test track, tests of static acuity, contrast sensitivity and visual field extent and questionnaires about vision and driving activities. The cataract group were significantly worse than the control group on driving performance. Impaired visual acuity and contrast sensitivity were significantly associated with worse driving performance for this group. Participants with cataracts rated their vision more poorly, however they did not report significant differences from the controls in terms of driving exposure, avoidance of night driving or heavy traffic, self-rated driving performance or self-reported crashes (time period not specified).

Treatment of cataract and road safety outcomes

An alternative to surgery is the use of medicated eye drops to reduce lens opacity. Babizhayev found that one formulation of eye drops improved visual acuity and disability glare in older adults with and without cataracts, with the effect being more pronounced for the group with cataracts. However the study did not examine the effect of this treatment on crashes, citations or driving performance.

One study conducted by Mönestam and colleagues (Mönestam et al., 2005; Mönestam and Lundqvist, 2006) was identified in the review period post-May 2003 which examined the effects of cataract surgery on driving performance. The study is described below and summarised in Table 45.

Crashes

There were no studies that assessed the relationship between cataract treatment and crashes post-May 2003.

Citations

There were no studies that assessed the relationship between cataract treatment and driving citations.

Driving Performance

Mönestam and Lundqvist reported on subjective visual and driving difficulties five years after cataract surgery. Of the 810 surgery patients they studied, 204 drove before the surgery; 285 drove a few months after surgery; and 189 drove five years later. Before surgery, 50% of the 222 drivers answering the questionnaire said they encountered visual difficulties with daytime driving; 69% had visual difficulties with night-time driving; 10%

did not drive in darkness. Shortly after surgery, 6% of 281 drivers reported difficulties with daytime driving, 24% reported difficulties with night-time driving, and 10% did not drive in darkness. Five years after surgery, 5% of 188 drivers reported difficulties with daytime driving, 32% reported difficulties with night-time driving, and 12% did not drive in darkness. The percentage of drivers reporting difficulties with night driving increased significantly between the short-term and long-term follow-ups. Those with low contrast visual acuity of 20/50 or worse were more likely to report subjective visual difficulties (OR 2.6, 95%CI 1.1-6.8).

Wood and Carberry compared 29 patients scheduled for bilateral cataract surgery with 18 controls of similar age (though the mean age of controls was somewhat younger) but normal visual acuity. Driving performance was measured on a closed test track. Before surgery, drivers with cataracts were significantly worse than controls for road sign recognition, road hazard recognition, and hazard avoidance. After surgery, drivers with cataracts were significantly improved on each of these measures and performed comparably with controls.

Summary

Cataracts can cause visual impairments such as reduced visual acuity, reduced contrast sensitivity and loss of visual fields. The limited data available indicates that individuals with cataracts may have a greater crash risk than those without cataracts.

Cataract surgery can eliminate cataract-related degeneration of vision and significantly restore some visual function, particularly when surgery is performed on both eyes (where necessary). Surgery can improve driving performance on a closed-road test track back to the level of drivers without cataracts. However in terms of crash data, the post-surgical advantage reported for drivers was only that their crash risk increased at a slower rate than those who elect not to have cataract surgery, and the severity of crashes was unknown. Thus, in addition to general improvements in quality of life, the limited benefit of surgery for improving driving performance should be carefully weighed up for each individual against the risks, costs and inconvenience of surgery. For those who do not elect to have surgery, regular ophthalmic reviews should be conducted, including clinical history and preferably tests of contrast sensitivity, glare and visual field sensitivity, in addition to visual acuity, to provide adequate advice to the driver and for referral to licensing agencies where applicable.

Further research such as the ICOM project will more clearly demonstrate whether cataract surgery can be effective in alleviating visual impairments caused by cataracts and reduce crash risk post-surgery. Information such as this is important in establishing practices for assessing fitness to drive and provides support for vision specialists on when it may be appropriate to recommend earlier surgical removal of cataracts for maintaining safe mobility. Further research is also necessary into the effects of alternative treatments such as the use of medicated eye drops.

3.13.2 GLAUCOMA

Definition of Glaucoma

Glaucoma is the generic name given to a group of eye diseases where the optic nerve becomes damaged. In most cases, this is caused by blockage in the systems that circulate or drain the aqueous fluid from the eye. Damage to the optic nerve can also be caused by a

lack of blood flow to the nerve fibres, or a weakness in the nerve structure. When sufficient optic nerve tissue loss occurs, peripheral vision declines with central vision loss occurring much later (Coleman, 1999). The damage to the optic nerve and resultant loss of vision is permanent, but often occurs gradually and without obvious symptoms until impairments of central vision develop. Even in developed countries with good access to medical practitioners and public education programs, as many as half of the individuals that have glaucoma remain undiagnosed (Tielsch et al., 1991).

The diagnosis of glaucoma usually relies on ophthalmoscopic examination of the optic nerve and on measurements of intra-ocular pressure, but indications for glaucoma-related damaged vision are also provided by assessments of glaucomatous visual field defects (Alward, 1998; Coleman, 1999). There are several variants of glaucoma classified in terms of the aqueous outflow. The most common type of glaucoma is primary open-angle glaucoma (POAG). The specific aetiology of POAG is still unknown, but the damage to the optic nerve is related to high eye pressures or decreased blood flow to the optic nerve head. There is also a genetic link to glaucoma, with relatives of individuals with glaucoma having a substantially elevated risk (Wolfs et al., 1998). A second type of glaucoma, primary angle-closure-glaucoma (PACG), is uncommon and presents a different clinical picture to POAG.

Damaged vision from glaucoma is irreversible so the goal of treatment is to prevent further loss of visual function. This is mainly achieved by lowering the eye pressure to a point deemed safe for the optic nerve. Even when an individual has the target intraocular pressure, they need to be monitored because eye pressure is only a marker for disease progression (Coleman, 1999). Initial treatments begin with topically applied or oral medications used to lower eye pressure. Treatments include medications to lower pressure, laser therapy to increase aqueous outflow, and if necessary, conventional surgery may be used to create a new opening for fluid to leave the eye (Taylor et al., 2004).

Prevalence of Glaucoma

Taylor (2001) estimates that one in eleven Australians will develop glaucoma and of the estimated 210,000 Australians who have glaucoma, around half are undiagnosed. A meta-analysis of data from Australia, the United States and Europe reported that the prevalence of POAG in white people increased from 0.4% in males aged 40-49 years (0.8% for females of the same age) to 5.6% for those in the 90+ age group (6.9%) (Friedman et al., 2004).

Functional impairment associated with glaucoma relevant to driving

The main impairment associated with glaucoma is a reduction in peripheral vision with central vision loss occurring later in the progression of the disease (Coleman, 1999).

Pre-May 2003: Relationship between glaucoma and road safety outcomes

Crashes

People with glaucoma and associated visual field impairment commonly report difficulty with driving (e.g., Gutierrez, Wilson & Johnson, 1997; Parrish et al., 1997), but no studies have specifically analysed the crash risk associated with and without glaucoma. Several studies have conducted case-control analyses with glaucoma (in addition to other medical/physical conditions) included as a risk factor. However, studies are mixed, both in

terms of their crash risk association findings and in terms of the study limitations, making direct comparisons difficult.

Owsley, McGwin and Ball (1998) explored visual risk factors associated with crashes among drivers aged 55-87 years. One group of cases consisted of 78 drivers who had at least one crash between 1985 and 1990 where an injury was sustained to any occupant, and another case group consisting of 101 drivers involved in crashes where no injuries occurred. The authors also studied a control group which comprised 115 drivers who had not been involved in a crash during the 5-year period. All participants underwent a battery of standardised vision and visual processing tests and ophthalmological examination. However, the presence of an eye disease for the purposes of analysis was determined on the basis of eye examination *or* self-report. In the univariate analyses of eye diseases (including cataracts, glaucoma, macular degeneration and diabetic retinopathy) for injurious crashes, glaucoma and macular degeneration had significantly increased risk. Participants involved in injurious crashes were 3.6 times (95% CI, 1.2-10.9) more likely to be diagnosed as having glaucoma and 3.3 times (95% CI, 1.2-9.2) more likely to have macular degeneration. However, participants involved in non-injurious crashes were not significantly more likely to have any of the four eye diseases. In the multivariate analyses, including a range of visual impairment measures, only restricted Useful Field of View and glaucoma remained as significant, independent predictors of injurious crash involvement. However, the number of cases with a primary diagnosis of glaucoma was low (n = 11).

The cohort study conducted by Foley et al. (1995) (reviewed above) revealed an elevated crash risk among older drivers with a self-reported history of glaucoma (RR: 1.5, 95% CI, 1.2-2.1), but this was not significant. However, as noted earlier, the interview data were unlikely to be sensitive to a medical condition that largely goes undiagnosed. The study also had limitations in terms of its representativeness and did not account for other medical conditions or exposure. Stewart et al. (1993) found no association between glaucoma and increased crash risk in a sample of 1431 drivers aged over the age of 65 years. However, this study was also limited by the self-report of medical conditions and crashes that occurred prior to interview. Similarly, the study by McCloskey et al. (1994) (see above) assessed the injury-crash risk of cases against matched controls and found no clear association of ocular disease, including glaucoma, with crash risk independent of the other variables examined.

Hu, Trumble, Foley, Eberhard and Wallace (1998) noted that most studies do not adequately address the contemporaneous relationships among events. This is particularly important when the period of progression from onset of a disease to late stage visual deterioration might be 5 years or more (e.g. cataracts, glaucoma and age-related macular degeneration), and crash records are examined over that time frame. Therefore, Hu et al. attempted to examine the order of events from onset to symptoms and crashes using a panel data analysis approach to identify factors that place older drivers at a greater risk of crashing. They examined independent living adults over the age of 65 years from 1981 to 1993 conducting home interviews every three years and a telephone interview in the intervening years. Crash information was obtained from the state records from 1985 onwards. Records were examined for 1811 participants in 1985, but only 882 participants remained in the study by 1993. While this procedure provided accurate measures for many risk factors, the self-report nature of the health status questions limited the objectivity and verifiability of some measures including history of glaucoma and cataracts in an otherwise comprehensive study. Hu and colleagues found that the factors that place females at a risk of a crash were different from those associated with the crashes of male drivers. When the

analysis controlled for driving exposure, men were found to have a higher crash risk if they had a history of glaucoma, but not women. Hu et al. also noted the importance of annual mileage in the model explaining a significant proportion of the crash risk variance.

Citations

No studies reporting the relationship between glaucoma and driving citations or traffic violations were found.

Driving performance

Some studies have examined the relationship between measures of visual function that may be related to glaucomatous visual decrements and driving performance, however, most have not restricted their sample to drivers with glaucoma. One study by Szlyk et al. (2002) aimed to determine whether damage to visual function caused specifically by glaucoma affected driving-related skills. Szlyk et al. examined driving performance measures in a simulator and assessed measures of visual acuity, visual fields and contrast sensitivity in 25 people with glaucoma and 29 age-equivalent, normal-sighted controls. Participants with glaucoma did not perform more poorly on any of the simulator performance indices including crashes and did not report any more real-world crashes than controls. However, longer braking response times, more lane boundary crossings and slower driving speeds were all associated with poorer contrast sensitivity measures in the better eye of participants in the glaucoma group. The lack of differences in performance measures between participants with glaucoma and normal-sighted controls including real-world crashes provides no indication of possible crash risk among this relatively small sample of people with mild to moderate glaucoma.

Treatment of glaucoma and road safety outcomes

Crashes

An interesting finding reported in the study by Owsley, McGwin and Ball (1998), described above, was that glaucoma was related to crashes independent of the visual deficits that might accompany this disease. However, Owsley and McGwin (1999) noted that the use of topical eye medications for the treatment of glaucoma may constitute an additional risk factor for motor vehicle crashes. In a study of the risk of falling, these medications were found to be the greatest single risk factor among participants with glaucoma, even more than the visual impairments associated with the condition (Glynn et al., 1991). The contribution of topical eye medications to crash risk has not been studied, but is worthy of detailed investigation.

Citations

There were no studies that assessed the relationship between glaucoma treatment and crash risk.

Driving performance

There were no studies that assessed the relationship between glaucoma treatment and driving performance.

Post-May 2003: Relationship between glaucoma and road safety outcomes

During the review period between May 2003 and mid-2009, four studies were identified dealing with glaucoma and crash risk, one of these examined treatment effects. Additionally, three studies were identified dealing with driving performance. These studies are described in the following section and summarised in Table 45

Crashes

McGwin and colleagues examined medical and police-reported crash records for 576 patients with glaucoma (from ophthalmology & optometry clinics specialising in glaucoma) and 115 patients without (from university-affiliated ophthalmology and optometry practices). 66.8% of the glaucoma group and 72.2% of the controls were interviewed by telephone to get further demographic and driving habits data. Answers for the rest of the sample (deceased/uncontactable/refused to be interviewed) were imputed from the available data; analyses were conducted with and without the imputed data but the results were the same.

Although controls were supposedly 'free from any eye disease' according to medical records, 53% of them reported having cataracts; 70% of those with glaucoma also reported cataracts (significantly more). Participants with glaucoma were an average of two years older than controls; age ranges were not reported. Participants with glaucoma were more likely to be African-American, less likely to have a college education, more likely to have high blood pressure and diabetes, and reported consuming less alcohol.

Participants with glaucoma had low acuity in both better and worse eyes (as expected). They also reported higher scores for driving avoidance, i.e. they were more likely to avoid driving than controls were for each of 12 situations. For overall crash rates, patients with glaucoma were less likely to crash per person-mile of travel (RR: 0.64, 95%CI 0.46 - 0.90). After adjusting for age, gender, race, education, smoking, alcohol consumption, 'specific self-reported medical conditions' and cognitive status the relative risk of crashes was 0.67, 95%CI, 0.47 - 0.97. When person-time rather than person-miles was used as the exposure measure, RR (crude and adjusted) was still less than 1.0. For at-fault crash rates, there was no difference between participants with or without glaucoma for crude or adjusted rates using either person-miles or person-time.

A later study by the same authors aimed to determine whether the extent of visual field loss in drivers with glaucoma predicted crashes. They examined police-reported crashes occurring during the period Jan 1994 and June 2000 for 406 drivers with glaucoma. Participants included licensed drivers with glaucoma aged 55 or more who had been seen between January 1994 and December 1995 at one of three ophthalmology/optometry practices specialising in glaucoma, and for whom automated visual field data for both eyes was in their medical record. Telephone interviews conducted in 2000 obtained additional information on demographic variables, driving habits, general health, smoking and alcohol use (40% of cases and 37% of controls did not complete the interview; data for these patients was imputed).

A total of 112 patients had 120 crashes. Each crash was treated as a separate case; controls were selected randomly from the drivers who had not had a crash at the time of each of the paired case crash events. Cases were more likely than controls to be male, have cataract(s), lower driving avoidance scores and lower mileage (exposure) than controls. Mean AGIS scores (a method of rating visual field loss severity, named after the Advanced Glaucoma

Intervention Study in which the score was first derived) were marginally worse in the better eye and highly significantly worse in the worse eye. Odds ratios for defects in the better eye were not significantly different from 1. But odds ratios for moderate (AGIS score 6-11) and severe (AGIS score 12-20) defects in the worse eye were significant: Moderate crude (OR: 3.0, 95%CI 1.3 - 7.1), adjusted for alcohol consumption, cataract, diabetic retinopathy and worse eye visual acuity (OR: 3.6, 95%CI 1.4 - 9.4); severe crude (OR: 4.3, 95% CI 1.8-10.3), adjusted (OR: 4.4, 95% CI 1.6 - 12.4).

Similarly, 84 crashes were determined by police to be fault of the participant; 84 controls were selected randomly from those who had not had an at-fault crash at the time of each of the paired case crash events. At-fault cases were more likely than controls to be male, have ever consumed alcohol, have cataract(s), have diabetic retinopathy, have higher cognitive impairment scores, have worse visual acuity in both eyes, and have worse AGIS scores in both eyes. Odds ratios for the better eye were not significantly different from 1, but odds ratios for moderate and severe defects in the worse eye were higher. For moderate defects, crude (OR: 3.3, 95%CI 1.1 - 9.6), adjusted (OR: 4.2, 95%CI 1.2-15.0); for severe defects, crude (OR: 6.9, 95% CI 2.3 - 20.3), adjusted (OR: 9.0, 95%CI 2.4 - 33.2).

Szlyk and colleagues (Szlyk, Mahler, Seiple, Edward & Wilensky, 2005) examined self-reported crashes in 40 drivers with glaucoma and 17 controls matched for age, sex and driving experience. The matching was performed on a group basis rather than individually as fewer controls were recruited than participants with glaucoma. Five glaucoma participants refused to give a crash history; of the remaining 35, 13 reported crashes within the last five years. None of the controls reported any crashes (all provided data). This difference was significant, although a risk ratio was not able to be calculated.

Haymes, LeBlanc, Nicoleta, Chiasson and Chauhan examined self-reported and police-reported crashes during the last five years for 48 drivers with glaucoma (recruited from an eye clinic) and 47 controls matched at group level for age, sex, medical conditions/medications, functional independence and level of physical activity. Eighty-three percent (40/48) of the participants with glaucoma versus 94% (44/47) of the controls drove. Of the drivers, 27% in the glaucoma group and 7% of the controls self-reported a crash in the past 5 years (OR: 5.18, 95%CI 1.33 - 20.24). Twenty percent of the glaucoma drivers compared to 2% of the control drivers admitted fault for a crash (OR:10.75, 95%CI 1.28 - 90.34). After adjusting for age, gender, number of systemic medications, better eye HFA MD (visual field) and on-road driving exposure, the odds ratios were 6.62 (95% CI, 1.40-31.23) for crash involvement and 12.44 (95%CI 1.08 - 143.99) for fault. Participants with slower processing on the UFOV selective attention task (>350ms) were more likely to report crashes than participants with faster processing speeds, adjusted (OR: 10.29, 95%CI 1.10-96.62).

Police-reported crashes gave similar but weaker results; this may be because there were fewer police-reported crashes (some self-report crashes did not require police report; crashes occurring in other provinces would not be in police records). Two glaucoma participants refused permission for their records to be released.

Citations

There were no studies that assessed the relationship between glaucoma and driving citations.

Driving Performance

Szlyk et al (2005) (see above) in their study of 40 drivers with glaucoma and 17 controls also examined performance in a driving simulator. Four drivers with glaucoma had seven crashes between them, while one control crashed once; this difference was significant. Binocular horizontal visual field extent, better eye horizontal visual field extent and total peripheral extent correlated significantly with simulator crashes. There was no significant correlation between number of simulator crashes and percent visual field sensitivity loss, visual acuity or contrast sensitivity for better or worse eyes. This may be due to the small number of crashes in the simulator, however correlations were also not significant for a variety of other driving performance measures (these include brake response time to stop signs, out-of-lane time, extent of eye movements, rate of braking, number of stop sign/red light violations, and mean speed; this data was collected sixteen times per second).

Carberry et al (2006) (see above in cataract section) studied test-track driving performance for 29 drivers with glaucoma and 13 controls. As for drivers with cataracts, they found that drivers with glaucoma performed more poorly than the controls; there was no difference between the two vision-impaired groups. Poor driving performance correlated with impaired visual acuity, contrast sensitivity and severity of visual field loss. Unlike the drivers with cataracts, those with glaucoma did not report any more visual difficulties than the controls. They also reported no differences for driving exposure, avoidance of night driving or heavy traffic, self-rated driving performance or self-reported crashes (time period not specified).

Haymes and colleagues examined on-road driving performance in 20 drivers with glaucoma (recruited from a glaucoma clinic) and 20 controls. There were no significant differences between the groups on age, gender, number of medical conditions, number of systemic medications, driving exposure. On-road testing was performed by a professional driving instructor and an occupational therapist certified in driver rehabilitation. There was no significant difference in time to complete test route. Median number of manoeuvres completed correctly and median overall driving performance rating were not significantly different. However, the driving instructor needed to intervene to ensure safety for twelve of twenty glaucoma drivers, but only four of twenty controls; this difference was significant ($p=.01$). The only visual performance measure examined that correlated with interventions was visual field extent measured by HFA mean deviation for the worse eye.

Treatment of glaucoma and road safety outcomes

Crashes

Haymes et al. (2007) (see above) found that 56% of the 48 drivers with glaucoma studied had previously undergone glaucoma surgery; these drivers were less likely to have been involved in crashes (adjusted OR: 0.15, 95%CI 0.03 - 0.87) and to have been at fault (adjusted OR: 0.05, 95%CI 0.00 - 0.65).

Citations

There were no studies that assessed the relationship between glaucoma treatment and crash risk.

Driving Performance

There were no studies that assessed the relationship between glaucoma treatment and driving performance.

Summary

Studies on glaucoma demonstrate equivocal outcomes for crash risk associations. While some show strong associations with reasonably high odds ratios, others find no significant relationships between glaucoma and crash risk. Those studies that demonstrate no significant relationships tend to have shortcomings in terms of the diagnosis of glaucoma, low prevalence within the sample, and retrospective crash records, but current visual classifications. These factors will tend to bias the results toward the null hypothesis. Incorporation of objective medical measures, exposure factors and injurious crashes appear to be important criteria in demonstrating and defining the crash risk associated with glaucoma. In recent years, more studies have appeared linking glaucoma with increased crash risk, particularly when visual defects are more severe. The one study that showed a lower crash risk for patients with glaucoma did not examine severity, and a mediating factor may have been that drivers with glaucoma were more likely to avoid difficult driving situations (see section on self-regulation below).

Another important consideration is the effect of complications such as field loss present in some people with glaucoma. If there is no field loss then the individual suffers from ocular hypertension rather than glaucoma. Driving performance studies suggest that impairments in driving safety are particularly associated with visual loss, although impaired visual acuity and contrast sensitivity may also play a part. It is important to note that an individual with impaired visual acuity and contrast sensitivity would be suffering from other pathology rather than just glaucoma.

More research is needed to examine the crash risk associated with co morbidity and particularly to identify the relative contributions of co-existing conditions. More research is also needed on the effects of treatments for glaucoma; the limited data available suggests that surgery may be protective against crashes, but that use of eye medications may actually increase crash risk.

3.13.3 AGE-RELATED MACULAR DEGENERATION (AMD)

Definition of Age-Related Macular Degeneration (AMD)

The macula is an area in the central retina with a high concentration of photoreceptors, responsible for high-resolution visual acuity. Age-related macular degeneration, or age-related maculopathy as it is otherwise known, is a condition where the photoreceptors in the macula degenerate. The loss of central vision associated with this condition can seriously affect quality of life by causing difficulties performing tasks such as reading, driving, and other activities of daily living (Scilley et al., 2002). The progression and severity of the disease depends on the type of AMD. The more common nonexudative or dry AMD is a milder form and accounts for approximately 85% of all cases (Gottlieb, 2002). Vision loss associated with dry AMD is usually gradual with varying degrees of vision loss depending on the progression and this form may or may not develop into the more exudative or wet form of AMD. Wet AMD is usually more severe than dry AMD and can progress rapidly, however in some cases of dry AMD there is extensive loss of central vision and field that can be more extensive than wet AMD. Although wet AMD only accounts for approximately 15% of all AMD cases, it is responsible for the majority of severe vision loss due to AMD.

Prevention of AMD is not possible but new treatments are being used with some success. Laser photocoagulation, an early treatment for AMD, has been replaced by a newer treatment, photodynamic therapy that uses a non-thermal laser with an intravenous, light-sensitive drug which accumulates in the abnormal new vessels (Gottlieb, 2002). Success in preventing progression of vision loss has been seen in the use of anti-VEGF (vascular endothelial growth factors) inhibitors. The anti-VEGF drugs are injected into the eye, in some cases at monthly intervals (Coleman et al., 2008). The Age-Related Eye Disease Study Research Group (2001) found in a randomised, placebo-controlled clinical trial that antioxidants with zinc significantly reduce the likelihood of developing advanced AMD in a small sub-group of patients with moderately advanced disease but this formulation is not recommended for smokers (Coleman et al, 2008). Risk factors are age, smoking and there is a strong genetic influence (Coleman et al, 2008). Cardiovascular disease and increased exposure to sunlight have also been inconsistently linked to AMD (Gottlieb, 2002).

Prevalence of AMD

In the United States and the United Kingdom, AMD accounts for over half of the non-refractive vision loss (Coleman, 1999). The Blue Mountains Eye Study in 1992-93 indicated that approximately 9% of adults aged over 65 years in New South Wales, Australia had AMD. There is a significant amount of early stage AMD reported in Australia, with estimates that nearly two thirds of Australians, who live into their nineties will develop the disease, and one in four will suffer a loss of vision from it (Taylor, 2001). When considering AMD that *results in visual impairment*, prevalence estimates increase from less than 0.1% in the 50-59 year age-group to 13% in Australians aged over 90 years (Taylor et al., 2004). Half of all Australians who are visually impaired from AMD have visual acuity less than 6/60, that is, legally blind (Taylor et al., 2004).

Functional impairment associated with AMD relevant to driving

The primary impairment in AMD is central vision loss and associated loss of vision for fine detail. This has serious implications for detecting important cues in the road environment.

Pre-May 2003: Relationship between AMD and road safety outcomes

Crashes

Despite the prevalence of AMD, there is very little evidence linking this disease with increased crash risk. Two studies by Szlyk and colleagues have examined the effects of macular degeneration on measures of driving performance and self-reported crashes. Szlyk, Fishman, Severing, Alexander and Viana (1993) examined the driving performance in a group of participants (n = 20) with central vision impairment from juvenile forms of macular degeneration and a control group (n = 29) with normal vision (for more information regarding driving performance, see the next section). Self-reported and state recorded crash involvement histories were determined for both groups. Analyses revealed that the overall crash records of drivers with central vision deficits were similar to those without impairment. However, the 13 individuals with central vision loss who did not restrict their driving to daylight hours, were more likely to be involved in night-time crashes than the control group. Measures of visual function were also poor predictors of crash involvement. The low number of participants and crash involvement rates meant that crash risk associations were difficult to establish.

In a later study, Szlyk and colleagues specifically assessed participants with age-related macular degeneration (Szlyk et al., 1995). However, a small sample size, large differences in the average age, and the gender imbalance between the groups were major shortcomings of this study. The 11 control group participants (four females and seven males) aged 71 years ($SD = 8.3$ years) showed no evidence of macular degeneration, but some did have early stage development of lens opacity (cataract) or glaucoma. The AMD participants consisted of ten males with a mean age of 75.7 years ($SD = 4.5$) with a clinical diagnosis of AMD. Szlyk et al. attempted to establish a history of visual acuity during the previous five years when crash records were obtained to gain information on the progression of the disease. However, some histories were unavailable, inconsistent or showed major changes in visual acuity. Comparisons of real-world crash involvement were not possible for state recorded crashes because there were no recorded crashes for either older group. However, there were a total of eight self-reported crashes in the older control group and two crashes in the AMD group (apparently by the same participant). These two crashes occurred at night while the driver was attempting to turn at intersections. There is also a high probability that the exposure of the AMD group was considerably less than the control group accounting for the difference in crash rates. Based on self-report, all participants travelled a minimum of 1600 km per year during the crash data period, but no other useful information was provided on exposure. With these limitations it is very difficult to infer any associations between crash risk and AMD. Nevertheless, the study did suggest that the AMD group may compensate for their impairments by restricting their night-time driving, driving in familiar areas, driving at slower speeds and taking less risks.

Only one other study was found to assess the crash risk associated with AMD. Owsley, McGwin et al. (1998) studied 294 participants who underwent a battery of visual tests and comprehensive eye examinations to determine vision impairment and eye disease. Univariate analyses revealed that macular degeneration was related to injurious crash involvement (OR: 3.3, 95%CI 1.2 - 9.2), but not crashes without injury. However, unlike glaucoma, this variable did not demonstrate significant, independent associations with injurious crash involvement after adjusting for other visual variables in the multivariate analyses. This simply means that other diseases, impairments or exposure variables account for some of the same variability in crash involvement as predicted by AMD.

Citations

No studies reporting the relationship between AMD and driving citations were found.

Driving Performance

As outlined in the previous section, Szlyk et al. (1993) examined the driving performance on an interactive driving simulator in a group of participants ($n = 20$) with central vision impairment from juvenile forms of macular degeneration and a control group ($n = 29$) with normal vision. On the driving simulator, the central vision loss group demonstrated longer braking times and a greater number of lane boundary crossings than the control group, but these measures were not found to be related to crash involvement. Szlyk et al. also suggested that simulator performance may not represent real-world driving skills because compensation factors are not adequately assessed.

The second of Szlyk and colleagues' studies (Szlyk et al., 1995) specifically assessed participants with age-related macular degeneration. Findings from the simulator and self-reported crashes of these participants were also compared to the 29 younger control participants from the Szlyk et al. (1993) study. While the AMD participants performed

more poorly on the simulator and on the on-road test, these measures did not relate to real-world crashes. Considering the very small samples and numbers of crashes, it is not surprising that these findings were not significant.

Post-May 2003: Relationship between AMD and road safety outcomes

There were no studies identified in the post-2003 review period that assessed the relationship between AMD and road safety outcomes including measures of crash involvement, citations or driving performance.

Summary

Adequate central vision is clearly critical for driving a vehicle safely, yet the guidelines for fitness to drive for medical practitioners have no procedures or recommendations for managing individuals with macular degeneration. It is important that research with appropriate control measures and sufficiently large sample size is conducted to gain a greater understanding of the crash risk associated with AMD.

3.13.4 DIABETIC RETINOPATHY

Definition of Diabetic Retinopathy (DR)

Diabetic retinopathy (DR) is an eye disease caused by specific vascular complications from diabetes mellitus where the blood vessels that supply the retina are damaged. The longer a person has diabetes, the greater the likelihood of developing DR with greater damage and vision loss caused by poor management of the underlying condition. There are two ways in which vision impairment occurs in DR: (i) proliferative retinopathy, involving blockage of many blood vessels in the retina which may result in blurred vision and blindness; and (ii) macula oedema, in which fluid leaks into the macula resulting in swelling and blurred vision (Taylor et al. 2004). Both conditions may be detected on an eye exam involving an acuity test and an ophthalmoscopic examination of the retina or retinal photograph. Treatment is usually with laser surgery and management of the primary condition of diabetes mellitus.

Prevalence of DR

There are around half a million Australians over the age of 40 with confirmed diabetes mellitus and similar numbers of undiagnosed cases are estimated (Taylor et al., 2004). Tapp et al. (2003; cited in Taylor et al, 2004) estimated that about 15.3% of Australians with diabetes also have some DR. In a Victorian study (Melbourne Visual Impairment Project), McKay, McCarty and Taylor (2000) found that 29.1% of self-reported people with diabetes in the sample could be clinically diagnosed with some DR, and 2.8% had untreated vision-threatening retinopathy. In two other studies of middle-aged to older participants, the prevalence of retinopathy among people with diabetes was 25% (Nathan, Singer, Godine, Harrington & Perlmutter, 1986) and 20.5% (Phillipov, Alimat, Phillips & Drew, 1995). The overall rate of DR in the older Australian population was between 1.1 and 2.2% (National Health and Medical Research Council [NHMRC], 1997). More recent data reported that a minimum 1.6% of Australians with a diagnosis of DM had vision-impairing DR (Taylor, Keeffe & Mitchell, 2004). However, rates across the world vary depending on race, gender and the prevalence of diabetes.

Functional impairments associated with DR relevant to driving

DR is associated with loss of visual acuity. The extent of vision loss varies across individuals and is more severe in those with long-term, poorly controlled diabetes. In cases where the macula is affected, central vision loss is found. These impairments have obvious consequences for safe driving.

Pre-May 2003: Relationship between DR and road safety outcomes

Crashes

Despite the serious implications DR can have for visual function, there are no studies of sufficient scale to adequately estimate the crash risk associated with the disease. A number of studies do examine crash risk in relation to diabetes. However, most of these studies do not separate out the contribution of diabetes-related visual disease. An exception is the study by Salzberg and Moffat (1998), which specifically examined the driving records of 14 older drivers with DR who were referred to the Washington State Special Examination Program and passed (although most had restrictions imposed on their driving). State crash records were examined over a 5-year period including 1.75 years prior to the exam and 3.25 years after. Older drivers with DR were found to have a crash rate prior to the exam of 12.2 collisions per 100 licensed drivers in a year. This pre-exam crash risk was 1.7 times higher than participants with diabetes mellitus, 3.2 times higher than age-matched control participants without medical conditions, and 3.5 times higher than the Washington State population. After the special exam, the rate of crashes in the DR group dropped to zero. However, as noted earlier this study could be criticised because of its use of an aggregate crash outcome measure, which tends to mask the influence of one or two high-risk participants having multiple crashes. The post-exam zero crash rate could also be explained by a large reduction in exposure by the few participants with DR. It is also unclear what type of DR was studied, the severity of the vision loss, and whether treatment was being sought. Other selection biases discussed in more detail in previous sections also call into question the validity of the conclusions.

Three other case-control studies have included DR as a variable in logistic regression analyses of crash risk factors. Owsley, McGwin et al. (1998) and McCloskey et al. (1994) and McGwin et al. found no association with crash risk or even a slight reduction in crash risk associated with DR. However, the prevalence of DR in both crash-involved cases and non-crash involved controls was extremely low for both studies and any associations would not have been reliable. Therefore the role of diabetic eye disease and in particular DR and driving requires further investigation before any conclusions can be drawn about crash risk associations.

Citations

As outlined above, Salzberg and Moffat (1998) specifically examined the citation records of 14 older drivers with DR who were referred to the Washington State Special Examination Program and passed (although most had restrictions imposed on their driving). State citations records were examined over a 5-year period including 1.75 years prior to the exam and 3.25 years after. Older drivers with DR were found to have a citation rate prior to the exam of 8.16 collisions per 100 licensed drivers in a year. This pre-exam citation risk was comparable to that of participants with diabetes mellitus and age-matched control participants without medical conditions. After the special exam, the rate of citations in the DR group dropped to about one quarter of the pre-exam rate (2.20) and in line with

control participants. As described in previous sections, the study had a number of methodological flaws, which bias the conclusions that can be drawn.

Driving Performance

There were no studies that assessed the relationship between DR and driving performance.

Post-May 2003: Relationship between DR and road safety outcomes

In the review period post-May 2003, only one study addressing crash risk and driving performance amongst drivers with DR was identified. One additional study dealing with post-surgery driving performance was also identified. These studies are reviewed below and summarised in Table 45.

Crashes

Szlyk and colleagues studied self-reported crash histories of 25 participants with DR. Crashes were significantly associated with higher levels of glycosylated haemoglobin, which is an integrated measure of diabetic control and indicates an increased risk of progression of DR. Correlations of visual acuity, letter contrast sensitivity, and overall visual field mean deviation with crash history are not reported.

Citations

There were no studies that assessed the relationship between DR and driving citations post 2004.

Driving Performance

Szlyk and colleagues (see above) examined correlations between visual and driving performance measures in 25 participants with DR. Visual acuity, letter contrast sensitivity, and overall visual field mean deviation did not correlate with any performance variables. Retinopathy scores did not correlate with any performance variables. Retinal thickness in (temporal) areas 4 and 7 of the visual field correlated with time off road, simulator accidents and near accidents. Retinal thickness in the central 20 degrees also correlated with accidents and near accidents. Laser scars in the central 20 degrees, and particularly focal scarring, were associated with later, more intense braking. This braking pattern in the simulator was correlated with (self-reported) real-world crashes within the past 5 years.

Treatment of DR and road safety outcomes

Barsam and Laidlaw examined visual performance after vitrectomy in 20 surgery patients with complications of DR, and 15 age and gender matched controls. Visual acuity was tested with the ETDRS chart; visual fields were tested with monocular Goldman perimeter test, monocular Humphrey field analyser central 24-2 test, and binocular Esterman fields. After surgery, patients' Goldman III4e isopter averaged 38% of that of healthy controls, while the V4e isopter was 49% of controls. Patients missed 71% of the points in the central 24 degrees of vision. 70% of patients had sufficient binocular acuity to drive after surgery, but of these 71% would fail the UK visual criteria for licensing based on the Esterman field analysis.

This result, together with the study by Szlyk et al (2004) (above), suggests that patients undergoing surgery for diabetic retinopathy should be informed that while some aspects of

vision are improved by surgery, other aspects may be impaired and that this may affect their ability to drive safely.

Summary

While DR is a leading cause of new cases of legal blindness and affects a large proportion of diabetics, evidence of associations between crash involvement or crash risk and DR is scant. However, while there is a great deal of evidence relating to diabetes mellitus and crash risk, few have controlled for vision complications such as DR. Hence it is difficult to ascertain the contribution of DR to overall risk outcomes for this condition (see section 3.5). Nonetheless, it does highlight the importance of careful diabetes management for limiting crash risk and limiting the progression of DR. If DR does develop, it is treatable with laser therapy and surgery, but regular eye examinations are necessary to prevent severe and permanent vision loss that could compromise driver safety. It is also concerning that laser treatment itself results in scarring that can affect vision and therefore driving performance. However, greater understanding of the role of DR in the crash risk of diabetics, and the potential benefits of DR treatments for reducing crash risk are needed before policy recommendations can be proposed.

3.13.5 REFRACTIVE ERRORS

Definition of refractive errors

To view images with high definition, light must be refracted by the cornea and the lens, and focussed sharply on the retina. If this is not done precisely, blurred vision will result. These refractive errors are the most common eye disorders (Taylor et al., 2004). Most people have some level of refractive error, but normally too slight to noticeably affect their vision. The four common types of refractive error are myopia, hyperopia, astigmatism and presbyopia.

Myopia or near-sightedness, refers to difficulty focussing on distant objects such as road signs or number plates. Myopia is caused by either excessive curvature of the cornea or length of the eye or both so that light focuses in front of the retina.

Hyperopia or long-sightedness, (sometimes termed hypermetropia) is a refractive error where the eye is shorter than normal or the cornea is too flat. This causes light to be focussed behind the retina rather than directly on it, resulting in close objects appearing blurred.

Astigmatism is caused by changes in the curvature of the cornea, which distorts the light entering the eye and prevents it from focusing clearly. This results in a focussing error that causes asymmetric blur at all distances.

Presbyopia is a disorder related to ageing, caused by the crystalline lens losing its flexibility and becoming less able to change its shape (accommodate) to sharply focus the light on the retina. The condition results in difficulty in seeing things at normal reading distance. The condition is usually first noticed around 40-45 years and continues to about age 65 (Taylor et al., 2004).

All of these conditions can be treated or corrected by using prescription glasses or contact lenses.

Prevalence of refractive errors

Worldwide, uncorrected refractive errors are the second leading cause of blindness after cataract, and the cause of almost half of all visual impairment (Resnikoff, Pascolini, Mariotti & Pokharel, 2008). In Australia, the prevalence of refractive error is around 22% of the population aged over 40 years (Taylor, Keefe & Mitchell, 2004) which is lower than the United States or Western Europe where refractive error affects around one third of people over 40 years (Kempen et al., 2004). Taylor et al. (2004) describe the age-related increases in vision impairment from refractive error as:

- 0.5% in 40-49 year age group;
- 1.8% in 50-59 year age group;
- 3.9% in 60-69 year age group;
- 7.8% in 70-79 year age group;
- 13% in the 80-89 year age group;
- 7.9% in 90+ year age group.

It is important to know the prevalence of low visual acuity among drivers as well as in the general population. A study in South Wales (UK) surveyed 298 drivers of private vehicles who passed the on-road study location (Anuradha, Potter & Fernquest, 2007). Drivers completed a questionnaire that recorded gender, age, approximate number of years that they had held the licence, if they suffered from any eye disease, if they wore glasses/contact lenses for driving, and approximate time of last eyesight test. Drivers were then asked which was the furthest vehicle registration plate they could read out of a set of four plates mounted 25m, 20m (the legal limit), 15m and 10m from the driver. Of the 298 drivers, 90% had no known eye disease; 45.2% wore glasses or contact lenses for driving; 94% read the number plate at 25 m with ease and 4.3% read the 20m number plate, giving an overall pass rate of 98.3%. Five drivers failed to meet the legal requirement of ability to read a numberplate at 20m. Of these, three were supposed to be wearing corrective lenses: two had glasses in the car and passed once glasses were worn, while one had forgotten to bring their glasses. Two drivers were unaware that their eyesight failed to meet legal requirements. Two drivers were aged 40-49, three aged over 70 years. That three drivers were aware of their visual impairment but failed to wear corrective lenses while driving is concerning. However, this study did not assess how driving safety might be affected by low visual acuity.

Functional impairments associated with refractive errors relevant to driving

Impairments associated with the four main types of refractive error are described above. Difficulties relevant to driving include focussing on distant objects (e.g. on-coming vehicles or traffic lights in the distance), near objects (e.g. speedometer), distortion of focus on near and far objects and adjusting focal length between objects in the near and far field of view. In addition, dynamic visual acuity affects the ability to perceive movement-related information. This is likely to influence judgements about speed of other vehicles and will also have important consequences for gap selection and making turns across traffic.

Pre-May 2003: Relationship between refractive errors and road safety outcomes

Despite the impairments caused by refractive error and its high prevalence, associations of refractive error with crash involvement are largely unknown. However, the visual impairments caused by refractive error are usually assessed in terms of visual acuity. A considerable body of literature has examined the effects of visual acuity on driving. Evidence for the relationship between visual acuity and road safety outcomes is reviewed later in this section (see section 3.13.11).

Post-May 2003: Relationship between refractive errors and road safety outcomes

Evidence for the relationship between visual acuity and crash risk identified in the review period post-May 2003 is presented in section 3.13.11. Overall, the evidence reveals no consistent link between crashes and visual acuity. It is important that research with appropriate control measures and a sufficiently large sample size is conducted to gain a greater understanding of the crash risk associated with visual acuity.

OTHER VISUAL CONDITIONS

In addition to the conditions described above, there are a number of other less common visual conditions which have significant impairments. However, the scarcity of research relating these conditions to driving means that the crash risk is difficult to establish in most cases.

3.13.6 *RETINITIS PIGMENTOSA*

Definition of Retinitis Pigmentosa

Retinitis Pigmentosa (RP) is a congenital degeneration of the pigmented layer of the retina that can lead to severe visual field loss. In addition, due to loss of rods in this condition, one of the early problems is night blindness. Initially, loss occurs in the mid-peripheral visual field, and as the condition progresses, the far peripheral field deteriorates and eventually central vision loss occurs. The vision impairment with RP is similar to glaucoma in that central vision may remain functional until the condition is advanced. Some people with RP become blind as young as 30 years, but the majority become legally blind by the age of 60 (Berson, 1996). Fortunately, RP is a relatively rare condition.

Prevalence of Retinitis Pigmentosa

Prevalence estimates of RP worldwide are around 1 in 5000, but range from as high as 1 in 3000 individuals to as low as 1 in 7000 individuals (Switzerland). It is estimated that RP affects between 50,000 to 100,000 people in the United States (around 1 in 4000) and approximately 1.5 million people worldwide (Vision Channel, 2003b).

Functional impairments associated with Retinitis Pigmentosa relevant to driving

The major impairments associated with RP are a restricted field of vision and night blindness. This is likely to result in a limited ability to detect important stimuli or events in the road environment whilst driving, particularly at night.

Relationship between RP and road safety outcomes

Evidence identified in the pre- and post-May 2003 review periods relating to the relationship between RP and road safety outcome is presented in the section on visual field loss (Section 3.13.12).

3.13.7 HEMIANOPIA (HEMIANOPSIA)

Definition of hemianopia

Hemianopia is a condition resulting in visual field loss caused by damage to the optic pathways in the brain. This can result from acquired brain injuries due to stroke, tumour or trauma, and causes vision loss in the visual field. If there is total loss in one half of the visual field it is termed homonymous hemianopia (HH). This can occur in:

- corresponding halves of the left or right field of vision in both eyes;
- but it can also occur in the upper half of the field (superior hemianopia, i.e. tunnel vision);
- the lower half (inferior hemianopia), or
- both outer halves of the field (bi-temporal hemianopia).

Driver licensing agencies commonly administer visual field assessments as part of screening procedures or in the re-assessment of referred drivers. Approximately half of the states in the US have visual field requirements for driving but the criteria are highly variable (Owsley & McGwin, 1999). The effectiveness of visual field measures have been questioned because effective visual search strategies incorporating eye and head movements may be used to minimise blind angles and centrally fixate on important information (Isler, Parsonson, & Hansson, 1997; Lövsund, Hedin, & Törnros, 1991). It has also been shown that scanning patterns may be used to compensate (to some degree) for some field defects (e.g., Pambakian, et al., 2000). However, these suggested compensatory strategies remain untested in respect of reduction in crash risk.

Prevalence of hemianopia

One study investigated the prevalence of homonymous visual field loss and stroke in an older community dwelling population (49 years and over) (Gilhotra et al, 2002). The prevalence of homonymous field loss was found to be 0.8%, a similar rate to the 0.5% reported for hemianopic and quadrantanopic field loss in a population based study of all ages in Melbourne, Australia (Taylor et al, 1997). Prevalence rates rose with increasing age from 0.4% in those less than 60 years to 1.1% in those over 70 years, although this trend did not reach statistical significance (Gilhotra et al, 2002).

Functional impairments associated with hemianopia relevant to driving

As noted above, the major impairment associated with hemianopia that is likely to impact on driving is a loss of vision in one half of the visual field in either one or both eyes. As with other conditions resulting in visual field loss, hemianopia is likely to reduce the ability to detect important stimuli or events in the road environment whilst driving. In the

particular case of bitemporal hemianopia, it is possible that the driver, when focussing on near objects (e.g. speedometer), may lose all distance vision in the periphery.

Hemianopia is frequently associated with other cognitive or functional impairments that may result in additional difficulties with driving (see sections 3.3 and 3.4 for further information on impairments related to stroke and traumatic brain injury respectively).

Pre-May 2003: Relationship between hemianopia and road safety outcomes

No research to date has specifically examined the association between hemianopia and crash involvement or citation rate, however some evidence on driving performance and hemianopia is discussed below (also see visual field loss section 3.13.12). A legal case involving hemianopia also provides an interesting medico-legal perspective on the question of licence restrictions for this group (see Chapter 1).

Crashes

No studies were found relating to crashes and hemianopia.

Citations

No studies were found relating to citations and hemianopia.

Driving Performance

In one study, Tant and colleagues tested the on-road driving performance of 28 participants with homonymous hemianopia (Tant et al., 2002). The drivers were formally assessed by a qualified driving examiner, who rated 55 aspects of their driving behaviours, in addition to their usual test protocol. A small proportion of the hemianopic cases passed the on-road test, and the most frequently reported driving problem in this group was lack of stability in steering. The remaining participants failed the on-road component, on a range of measures. Despite the small sample size, it was an interesting observation that some participants with hemianopia passed a standardised assessment. The findings suggest that at least some drivers with hemianopia were able to compensate for their field loss, most likely by using visual scanning and/or head movements. The ability to compensate for field loss is an important consideration in licensing and there is no satisfactory means for pre-screening those who might be able to compensate and those who cannot and this is likely to be influenced by the presence of other functional impairments. This issue is considered further in section 3.13.12 relating to other conditions which affect visual fields.

Post-May 2003: Relationship between hemianopia and road safety outcomes

In the review period post-May 2003, two studies were identified dealing with hemianopia and driving performance including one study addressing the treatment effects with the use of prisms while driving. These studies are reviewed below and results are summarised in Table 45.

Crashes

No studies were found relating to crashes and hemianopia.

Citations

No studies were found relating to citations and hemianopia.

Driving Performance

Wood and colleagues (2009) investigated on-road driving performance in a group of participants with hemianopia or quadrantanopia compared to controls. Cases were selected through an Ophthalmology clinic in Birmingham, USA (n = 30). Participants were excluded on the basis of visual neglect, visual acuity less than 20/60, an expired driving licence, and a range of neurological or ophthalmic disorders. Controls were recruited through an existing database and matched on age (± 2 years). Participants completed a general health questionnaire, a driving questionnaire, a vision assessment and a cognitive assessment. A medical record review was also completed. The on-road driving component consisted of a standardised 14.1 mile (22.7 kilometre) route covering residential, commercial and inter-state roads. Performance was assessed by two independent evaluators masked to the driver's clinical characteristics. Specific driving behaviours were rated, and a 5-point global driving score was calculated at the end of the drive (unsafe and drive terminated, unsafe but drive completed, unsatisfactory but not unsafe, safe but several minor flaws, and safe with near flawless driving performance).

On the five-point global rating scale, all drivers with normal visual fields, 73% of the hemianopes and 88% of the quadrantanopes were rated as 'safe' on the non-interstate drive. On the inter-state drive, 97% of drivers with normal fields, 83% of the hemianopes and 100% of quadrantanopes were considered 'safe'. Univariate comparisons between the hemianopes and quadrantanopes indicated that unsafe behaviours were more likely to be associated with slower visual processing speed, poorer executive function and reduced contrast sensitivity. Overall, in this small sample the study demonstrated that some drivers with hemianopic or quadrantanopic visual fields were rated by independent evaluators as 'safe' drivers.

One approach to licensing in hemianopic field loss is to allow the use of assistive devices. Szlyk and colleagues examined driving performance when people with hemianopia were trained to drive using prisms. Ten males with homonymous hemianopia in either right or left visual field were trained in the use of two prism systems (Gottlieb or Fresnel) to enhance vision in the peripheral fields. All participants received training using both prism systems, with the order of training in each system being counterbalanced across participants. Each person was assessed for visual skill on the first day of the study and one month later (to ascertain test-retest validity) before being provided with the prism systems and training in how to use them. The assessments included clinical vision tests, a set of functional tasks in an indoor/laboratory and outdoor setting, driving in a simulator and on a road course, and psychophysical tests for peripheral visual function. After approximately 25 hours of training over 6 months in general use and use during driving on a road course, participants were re-tested. If the improvement with prisms after the training was greater than the improvement between the first two baseline tests, the task was coded as improved. Tasks were sorted in visual categories and a percentage improvement was calculated based on the number of tasks within each category that were improved for each participant.

The average improvement across visual skills categories was approximately 20%, with the range being between 13 and 36% improvement depending on the particular skill and the type of prism used in the assessment. Two years later the participants were contacted for a follow-up questionnaire. Only seven of ten participants could be contacted; four of these

were still using the prisms frequently, and two occasionally. Three drove, two with the prisms and one without.

Summary

There has been no research to date that has specifically examined the relationship between hemianopic field loss and crash risk. Given that this group tend to have a range of functional impairments associated with their brain injury, it can be difficult to tease out the effects of visual impairment and cognitive impairment. The issue of individual compensation for the visual deficit is likely to be a key factor in this group. However, the limited driving performance data available does show (albeit in small samples) that some drivers with hemianopic field loss can pass on-road driving assessments with trained assessors. There has been some recent interest in assistive devices for driving with hemianopic field loss, particularly the use of prisms. Some studies have shown initial improvement using prisms, however no long-term data regarding safety or acceptability is available.

3.13.8 COLOUR VISION DISORDERS

Definition of colour vision disorders

Abnormalities of colour vision are inherited traits that almost exclusively affect males. These defects usually manifest in a difficulty distinguishing red from green, with blue deficiencies occurring very rarely. The different types of colour vision defects include:

- Deutan Defects (altered green sensitivity);
- Protan Defects (altered red sensitivity);
- Tritan (Blue and yellow confusion);
- Monochromats (unable to see colour).

Prevalence of colour vision disorders

Colour blindness is a fairly common disorder, but varies among different groups. In the US, around 10.5 million males (7% of the male population) and 0.4% of the female population are affected by a red/green colour vision disorder. In Australia, around 8% of the male population have difficulty distinguishing red from green, but only 0.4% of women are affected by colour blindness (Montgomery, 2003; Sewell, 1983).

Functional impairments associated with colour vision related to driving

The most obvious impairment associated with colour vision disorders that is likely to impact on driving is difficulty with distinguishing colours of traffic lights and vehicles lights and in using colour to distinguishing between various stimuli in the road environment.

Pre-May 2003: Relationship between colour vision disorders and road safety outcomes

Crashes

While colour vision is tested in many licensing jurisdictions (Owsley & McGwin, 1999), it does not appear to represent a major crash risk because other information such as luminance, position and pattern allow drivers with colour vision defects to recognise traffic signs and signals. The majority of research suggests that there is no association between crash risk and colour vision abnormalities (Vingrys & Cole, 1988). However, Verriest, Naubauer, Marre, and Uvijls (1980) reported that individuals with protan colour defects (an insensitivity to red light) were over-represented in rear-end collisions relative to normal vision drivers, although protans were not over-represented among the crash-involved group overall. Others have also found self-reported driving difficulties relating to colour vision defects such as distinguishing traffic signals, confusing traffic lights with street lights, and detecting brake lights, but this has not been related to crash rates (Steward & Cole, 1989). However, Wolfe (2002) questions whether reported visual difficulties during driving translate directly into crash involvement and expressed reservations about research that cites this as evidence of crash risk among individuals with protan colour defects. More research is warranted on the crash risk associated with protan drivers given the recent debate about visual standards barring these drivers from holding commercial vehicle licences (see Vingrys, 2002).

Citations

No studies were found which addressed the relationship between colour vision disorders and driving citations or violations.

Driving performance

No studies were found which addressed the relationship between colour vision disorders and driving performance.

Post-May 2003: Relationship between colour vision disorders and road safety outcomes

In the review period post-May 2003, only one study addressing crash risk and colour vision was identified. The study is described below and summarised in Table 45.

Crashes

One study was found examining self-reported crash history and driving/visual difficulties for defective colour vision and non-defective colour vision young males. Vision screening was conducted on 4194 male schoolchildren aged 11-14 between 1987 and 1991 in the province of Cosenza. 6% of the sample (268 participants) had defective colour vision. In 2001, the researchers tracked down the now adult screened participants, and contacted 151 of the defective colour vision participants. For each of these, two random (non-colourblind) classmates from the same town were selected to be control participants. A questionnaire was administered by telephone interview for each participant. This included questions on difficulties with colour in everyday life and in driving, including whether the participants had ever been involved in a road accident. Although there was no significant difference between groups in the proportion of participants with driving licences, fewer defective colour vision participants said that they drove. There was no significant difference in the proportion of participants who reported crashes between the two groups. There were also no significant differences reported for difficulties identifying traffic signal colours, or seeing lights and reflectors on the road/guard rails and other vehicles. More defective colour participants said that they preferred daytime driving over driving at night.

Citations

There were no studies that assessed the relationship between colour vision disorders and driving citations.

Driving performance

There were no studies that assessed the relationship between colour vision disorders and driving performance.

Summary

The majority of research suggests that there is no association between colour vision abnormalities and crash risk. However, some studies have found self-reported driving difficulties (for example detecting traffic or brake lights), but these have not been associated with increased crash involvement. Some research suggests that people with colour vision disorders may self-regulate, with a preference for day time driving.

3.13.8 MONOCULAR VISION

Definition of monocular vision

Monocular vision refers to blindness in one eye.

Prevalence of monocular vision

One Italian study investigated the prevalence of a range of eye diseases in adults aged over 40 years (Cedrone et al., 2006). The best-corrected prevalence rate for monocular blindness was 1.8%, and for monocular low vision was 5%. These estimates are consistent with prevalence data from other European countries with similar socio-economic conditions and public healthcare systems (Cedrone et al., 2006).

Functional impairment associated with monocular vision relevant to driving

Research by McKnight, Shinar and Hilburn (1991) has identified impairments associated with monocular vision in the following areas:

- Binocular depth perception;
- Contrast sensitivity;
- Visual acuity under low illumination and glare.

Pre-May 2003: Relationship between monocular vision and crashes

Crashes

There is currently a paucity of research examining the crash risk associated with blindness in one eye (monocular vision). The relevant research was conducted mostly prior to the mid 1970's, considerably outside our literature search limits.

Dionne, Desjardins, Laberge-Nadeau and Maag (1993) included monocular vision among other medical conditions in regression models predicting the occurrence of crash

involvement. Models controlled for exposure factors, age, and other characteristics of the truck drivers examined in the study, but the sample of monocular vision drivers was limited. Diabetes was the only medical factor that was found to be associated with crash involvement. However, a much older study examining the driving records of 52 monocular drivers in Kentucky during the late 1970s (Keeney, Garvey, & Brunker, 1981) found that they were over-involved in crashes. Keeney et al. found that monocular drivers had almost twice the rate of crashes as the general motoring public. However, this study did not control for exposure, behavioural, or demographic factors that could make this group more vulnerable.

The lack of recent research limits any basis for conclusions regarding whether monocular vision drivers represent an at-risk group of drivers. However, the Canadian Ophthalmological Society (2000) considers that a driver who has recently lost the sight of an eye or stereopsis may require a few months to adapt to the condition and recover the ability to judge distance accurately. This has been acknowledged in the guidelines on fitness to drive in Canada and the UK.

Citations

Only one study was found addressing the relationship between monocular vision and driving citations. Keeney (1981) (reviewed above) reported that drivers with monocular vision had 50% more citation rates than the general population.

Driving performance

Evidence generally indicates that the performance of drivers is not adversely affected by monocular vision. Troutbeck and Wood (1994) examined the effect of restricting visual fields including a monocular vision condition on driving performance in a private closed road. They found that imposing monocular conditions did not significantly affect performance on any of the driving assessments. The research conducted by McKnight, Shinar, and Hilburn (1991) compared visual and driving performance measures of 40 monocular and 40 binocular tractor-trailer drivers. As noted above, monocular drivers were found to be deficient in some of the visual performance measures such as binocular depth perception, contrast sensitivity, and visual acuity under low illumination and glare. However, no differences between monocular and binocular drivers were revealed for the driving measures of visual search, lane keeping, clearance judgement, gap judgement, hazard detection, and information recognition, although binocular drivers read signs at greater distances. McKnight et al. concluded that monocular drivers had some limitations in selected visual capabilities and driving functions dependent on those abilities compared with binocular drivers, but were not deficient in most measures of driving safety.

Post-May 2003: Relationship between monocular vision and crashes

No studies were identified in the review period post-May 2003 specifically addressing monocular vision and road safety outcomes.

3.13.9 CORNEAL PATHOLOGY

Definition of corneal pathology

Corneal pathology results from injury or damage to the cornea.

Prevalence of corneal pathology

A prevalence survey of blindness and low vision was conducted in Oman in 2005 (Khandekar, Mohammed & Raisi, 2007). This study compared survey data from 1997 and 2005, and reported that the prevalence of blindness due to corneal pathology was between 1.1% and 1.9% of the population aged over 40 years (Khandekar, Mohammed & Raisi, 2007).

Functional impairment associated with corneal pathology relevant to driving

Corneal pathology results in a distorted or clouded image and increased glare sensitivity similar to cataracts. Visual detail is no longer discernible, but field of vision remains intact.

Pre-May 2003: Relationship between corneal pathology and road safety outcomes

Assessing the crash risk for this kind of damage is very difficult since the extent and severity of the condition can vary dramatically. No specific information on crash risk associated with corneal pathology was found.

Post-May 2003: Relationship between corneal pathology and road safety outcomes

No research on corneal pathology and road safety outcomes was identified in the review period from May 2003 to mid-2009.

3.13.10 NYSTAGMUS

Definition of nystagmus

Nystagmus is an involuntary and rapid movement of the eyes, usually in a horizontal manner, but sometimes the eyes oscillate vertically or in a circular motion. Nystagmus that appears before six months of age is called congenital, infantile or early onset nystagmus. Nystagmus that develops after this period is termed acquired nystagmus. Cases of early onset nystagmus are normally inherited defects in the eye or the visual pathway to the brain (Royal College of Ophthalmologists, 2003). There are many possible causes for acquired nystagmus, but it is often a symptom of another neurological condition such as a stroke, multiple sclerosis or traumatic brain injury. Most people with nystagmus have significant impairments of vision. The degree of impairment varies from person to person and is also related to the underlying condition. Visual impairment may also vary across the day and is likely to be affected by emotional and physical factors such as stress, tiredness, nervousness or unfamiliar surroundings (Royal College of Ophthalmologists, 2003). Therefore, the effects of nystagmus on driving performance are difficult to determine and little research has been conducted. Considering the variability in the degree of impairment associated with this condition, fitness to drive should be determined on an individual basis. However, criteria for assessing drivers with nystagmus are inadequately addressed in the guidelines provided by most jurisdictions.

Prevalence of nystagmus

Dobbs (2001) cites sources suggesting that the condition affects approximately 1 in 1000 individuals.

Functional impairments associated with nystagmus relevant to driving

No evidence on functional impairments associated with nystagmus relevant to driving was found.

Pre-May 2003: Relationship between nystagmus and road safety outcomes

No evidence linking nystagmus with road safety outcomes was found.

Pre-May 2003: Relationship between nystagmus and road safety outcomes

No evidence linking nystagmus with road safety outcomes was found.

Summary of eye conditions and diseases

The crash risk related to eye disease and visual conditions on the whole, is very difficult to establish given the various methodological shortcomings of the research. The findings of case-control studies appear to vary greatly depending on the definition of a case. Some studies select cases on the basis of the outcome measure (i.e., crash involvement) whereas others select a case based on the impairment or condition. The studies based on cases selected for individual conditions are advantageous given that they are hypothesis driven rather than exploratory and typically involve much larger numbers of drivers with the specific condition of interest. Exploratory studies of multiple risk factors may involve only a few crash-involved drivers with each condition, depending on the prevalence of the condition. While this may be important for road safety practitioners developing cost effective management strategies, the low power of these studies means that they may not provide a sensitive measure of crash risk for medical disease factors. Alternatively, many studies are limited by inadequately defined or subjective measures of conditions and even self-report of crash involvement. Some studies also provide no indication of the severity or stage of development of the condition, for example, "history of glaucoma" might constitute the variable in the risk factor analyses. Another major shortcoming common among the research is not adequately controlling for comorbidity. This is extremely important considering that the major eye diseases are largely related to age, as are many cognitive and physical limitations. Thus, while most studies have matched for age in their case-control methodology, clinically verified medical and physical conditions, as well as driving practices, need to be accounted for in order to establish reliable associations with crash risk. Contemporaneous measurements of crash risk, eye conditions, and other risk factors are also particularly important for the older cohort where medical conditions can progress rapidly.

While variability in crash risk findings is partly related to methodological differences, a considerable proportion of the variability is likely to relate to the stage of progression of the condition. Severe visual impairment associated with some of these conditions does appear to be related to increased crash risk after accounting for exposure. However, the most critical safety aspect is how well a person with these functional limitations self-regulate their driving. This is where the role of a health professional becomes critical. Those aware of difficulties and the risks they may be posing to themselves and their loved ones may be more willing to adopt alternative methods of transport. Overall, further research is warranted for prevalent conditions with significant visual impairments so that licensing agencies and health professionals have a greater understanding of the risks, and can inform or test drivers accordingly.

FUNCTIONAL VISION IMPAIRMENTS

In the review presented above, crash risk is considered in relation to specific vision conditions. In the majority of conditions, crash risk is likely to be attributed to one or more underlying impairments associated with the condition. For example, glaucoma may result in a reduction in various measures of visual acuity, visual field and contrast sensitivity. It is likely that crash risk cannot be determined simply by summing the risks associated with any or all of these impairments.

It is also instructive to consider crash risk associated with vision impairments. This subsection provides an overview of types of visual impairments, methods of assessment and findings in relation vehicle crash risk.

3.13.11 VISUAL ACUITY

Definition of visual acuity

Static visual acuity

Normal visual acuity is defined as 6/6 (metric) or 20/20 vision. Visual impairment, as defined by WHO, refers to a visual acuity of worse than 20/60-20/400 (6/18 - 6/120) in the better eye. Those with acuity ratings of 6/60 (metric) or 20/200 (imperial) or less are classified as legally blind.

Visual acuity is described by Owsley and McGwin (1999) as "perhaps the most ubiquitous visual screening test used by licensing agencies for the determination of driving fitness" (p. 538). However, the use of this measure may not be related to its effectiveness for identifying at-risk drivers. Snellen acuity may have been adopted as a licensing requirement because it was simple to administer and common in a clinical setting for diagnosing eye disease, but it has been criticised for not adequately reflecting the visual requirements of complex traffic situations (Owsley & McGwin, 1999; Schiff & Arnone, 1995). Furthermore, the driving environment surveyed by the driver is typically in motion and cannot be represented by static tests of acuity. It is not surprising then that the relationship between static visual acuity and crash risk has been found to be weak, at best.

Dynamic visual acuity

Dynamic visual acuity (DVA) represents the ability to perceive details of an object when there is relative motion between the object and the observer (Burg, 1968). This aspect of vision may deteriorate more rapidly with age and appears to be more relevant than static acuity for predicting visual difficulties while driving (Shinar & Schieber, 1991).

Prevalence of loss of visual acuity

Prevalence of low vision, as defined above, has been estimated at 2.6% in persons aged 72-74 years and 4.8% in persons aged 75-80 years. The age-adjusted relative prevalence is 1.58% (Buch, Vinding & Nielson, 2001). Estimates of prevalence of visual impairment of 6/12 or worse for bilateral and unilateral impairment in a representative Australian population (n=3654) (Wang, Forans & Mitchell, 2000) were reported as:

- 49-59 years: 0.6% - 3.6%;

- 60-69 years: 1.1% - 8.2%;
- 70-79 years: 5.4%- 20.1%;
- 80+ years: 26.3% - 52.2%.

It is important to know the prevalence of low visual acuity among drivers as well as in the general population. A study in South Wales (UK) surveyed 298 drivers of private vehicles who passed the on-road study location (Anuradha, Potter & Fernquest, 2007). Drivers completed a questionnaire that recorded gender, age, approximate number of years that they had held the licence, if they suffered from any eye disease, if they wore glasses/contact lenses for driving, and approximate time of last eyesight test. Drivers were then asked which was the furthest number plate they could read out of a set of four plates mounted 25m, 20m (the legal limit), 15m and 10m from the driver. Of the 298 drivers, 90% had no known eye disease; 45.2% wore glasses or contact lenses for driving; 94% read the number plate at 25 m with ease and 4.3% read the 20m number plate, giving an overall pass rate of 98.3%. Five drivers failed to meet the legal requirement of ability to read a number plate at 20m. Of these, three were supposed to be wearing corrective lenses: two had glasses in car and passed once glasses were worn, while one had forgotten to bring their glasses. Two drivers were unaware that their eyesight failed to meet legal requirements. Two drivers were aged 40-49, three aged over 70 years. That three drivers were aware of their visual impairment but failed to wear corrective lenses while driving is concerning. However, this study did not assess how driving safety might be affected by low visual acuity.

Functional impairments associated with loss of visual acuity relevant to driving

Impairments associated with loss of visual acuity are outlined in the section on refractive errors (see section 3.13.5). In addition, dynamic visual acuity affects the ability to perceive movement-related information. This is likely to influence judgements about speed of other vehicles and will also have important consequences for gap selection and making turns across traffic.

Pre-May 2003: Relationship between visual acuity and road safety outcomes

A considerable body of literature has examined the effects of visual acuity on driving. Evidence for the relationship between visual acuity and crash risk, citations and driving performance is reviewed below and summarised in Table 45. Almost all research on driving and visual acuity has used static visual acuity.

Crashes

Influential research by Burg (1967, 1968; Hills & Burg, 1977) analysing a very large sample of 17,500 drivers in California found that there was no relationship between acuity and crash involvement for young and middle-aged drivers. A significant relationship between poor visual acuity and crashes was demonstrated for older drivers, but the correlation was low and the authors cautioned that it should not be considered a causal relationship.

Several recent studies have examined visual function in relation to driving with some research indicating small but consistent associations between static visual acuity and crash risk, while others have found no statistically reliable associations. Owsley and colleagues

have conducted several studies since the early 1990s on the relationship between measures of visual function and crash risk, particularly focussing on older driver groups. Owsley, Ball, Sloane, Roenker, and Bruni (1991) conducted a comprehensive evaluation of visual/cognitive factors on 53 drivers in Alabama aged 57-83 years. They found that neither static visual acuity or night acuity was significantly related to crashes or citations with correlations under 0.15.

More recently, in a study of 294 older drivers, Owsley, Ball et al. (1998) found that drivers with static visual acuity worse than 20/40 had nearly 1.5 times the crash involvement rate of drivers with better than 20/40 vision. However, as is common with visual acuity, this association was not significant (RR: 1.45, 95%CI 0.58 - 3.64).

Similarly, Sims et al. (2000) revealed that drivers with visual acuity less than 20/40 had a slightly, but not significantly elevated crash risk (RR: 1.07, 95%CI 0.26 - 4.47). In a large Australian population base of 2,594 participants, Keeffe, Jin, Weih, McCarty and Taylor (2002) found that drivers with a visual acuity below 6/12 were no more likely to have a (self-reported) crash than those with a better acuity.

Gresset and Meyer (1994) examined 30,000 Quebec drivers aged over 70 years and found that those with static acuity of 6/12 to 6/15 had the same crash risk as age-matched controls with better acuity. However, crash risk increased moderately among drivers with poor acuity combined with a lack of binocular vision.

On the basis of a lack of statistically significant associations, a number of other authors (e.g., Brabyn, Schneck, Hagerstrom-Portnov & Steinman, 1994; Decina & Staplin, 1993; McCloskey, Koepsell, Wolf & Buchner, 1994) have concluded that mild reductions in static visual acuity have little relationship to the risk of collisions for older drivers. On the other hand, investigating crash involvement as a part of the Blue Mountains Eye Study, Ivers, Mitchell, and Cumming (1999) found that visual acuity worse than 20/60 in the right eye only was associated with crashes (RR: 2.2, 95%CI, 1.3 - 3.5, age and sex adjusted). Davison (1985) found a comparable relationship among British drivers, although that was derived using Chi-square statistics with relatively low numbers of crash involved drivers which provided little indication of the strength of the association.

Humphriss (1987) examined a considerably larger sample of South African drivers and showed that a number of visual measures including binocular visual acuity, right and left eye monocular visual acuity, and a difference in visual acuity between the two eyes, predicted whether a driver was more likely to be crash involved. However, again the magnitude of the effect was small and not larger than that originally found by Burg (1967, 1968).

One reason why it may be difficult to establish crash risk relationships with visual acuity is because drivers with severe acuity impairments may not be driving. Mandatory licensing re-assessments may have identified those most at-risk and eliminated them from the databases being evaluated. Alternatively, knowledge of vision difficulties including poor visual acuity often leads to self-imposed driving restrictions among older drivers (Ball, Owsley & Stalvey, 1998; Stutts, 1998). These factors would act to weaken any relationship between acuity measures and crash risk. However, several studies including Owsley, Ball et al. (1998), Sims (2000), and Gresset and Meyer (1994) included drivers with visual acuity impairments worse than 20/40 and demonstrated no reliable associations with crash risk.

Early research demonstrated that dynamic visual acuity (DVA) has shown a comparatively stronger and more reliable relationship with crash and conviction rates than static acuity (e.g., Burg, 1967, 1968; Hills & Burg, 1977). However, it was only for the older age groups that a systematic relationship between DVA and crash rates emerged and like static acuity, the predictive value of DVA was low (correlations less than 0.1). The relevance of Burg's research conducted in the 1960s and 1970s to more contemporary driving conditions could be questioned, but there is a paucity of rigorous research that contradicts Burg's conclusion. More up-to-date research with reliable and validated measures of DVA is required to assess its value as a predictor of crash involvement

Citations

No studies were found linking visual acuity with citations.

Driving performance

A number of studies have examined various aspects of driving performance in relation to measures of visual acuity, however, research has typically focussed on crash involvement as a more objective measure of driving risk. The effect of degraded visual acuity on driving performance has been examined in several recent studies. Higgins, Wood and Tait (1998) used modified swimmers goggles to simulate blurred vision equivalent to visual acuity levels of 20/40, 20/100, and 20/200. Degraded visual acuity did not significantly affect manoeuvring ability or gap clearance tasks, but progressive levels of acuity degradation produced significantly lower levels of sign recognition and hazard avoidance. These findings are consistent with deterioration in sign recognition and reaction time among drivers with true visual impairment assessed by Wood (1999) in a closed road circuit. Lamble, Summala and Hyvärinen (2002) examined the performance of experienced drivers with impaired visual acuity (equivalent to 20/100), and found no apparent differences in driving behaviour in normal traffic, although drivers with impaired vision were significantly slower in responding to a lead vehicle's brake lights in a car-following task. Wood and Mallon (2001) also examined in-traffic driving performance of younger and middle-aged drivers and older drivers with and without visual impairments. A driving instructor and an occupational therapist rated both visually impaired and normally sighted older drivers as having significantly poorer driving performance on a wide range of skills. The driving instructor had to intervene to avoid a collision for 12 older drivers (9 with vision impairments). Those with poorer visual acuity were particularly likely to fail to observe other road users, signs, and signals. However, most of the vision-impaired drivers had a visual condition that was likely to result in various deficits of visual function, not just acuity. Also, all acuity measures were above legal requirements, again suggesting that visual acuity may not be sufficiently sensitive to identify at-risk drivers.

Post-May 2003: Relationship between visual acuity and road safety outcomes

No studies were identified in the post-May 2003 review period dealing with crash risk or citation rates of drivers with impaired acuity. Two studies were identified in review period addressing driving performance of people with impaired visual acuity. The studies are reviewed below and summarised in Table 45.

Crashes

There were no studies that assessed the relationship between visual acuity and crash risk post 2004.

Citations

There were no studies that assessed the relationship between visual acuity and driving citations.

Driving Performance

A study of 24 drivers examined whether standard visual acuity and contrast sensitivity measures predict drivers' ability to report relevant targets while driving on a closed-road circuit (Wood & Owens, 2005). There were 3 groups of participants, younger (mean age 21.5), middle-aged (mean age 46.6) and older (mean age 71.9 years), with an equal number of males and females in each group. All participants passed the minimum visual acuity criterion of 20/40. Visual acuity (standard logMAR chart) and contrast sensitivity (Pelli-Robson) were binocularly assessed under different luminance conditions (simulated by goggles with different filter densities). Participants then drove 1.8km on a closed road circuit, once during the day, and four times at night with different filters mounted on the headlights. They were asked to report relevant targets including road signs, large low contrast road obstacles and pedestrians who wore reflective markings creating "biological motion".

As expected, recognition performance across all groups was significantly degraded under low light conditions; the degradation was greater for the older group. Contrast sensitivity was a better predictor than visual acuity measured under standard photopic conditions. However contrast sensitivity was highly correlated with visual acuity measured under low-luminance conditions. Recognition performance is best predicted by a combination of two tests; either 1. photopic visual acuity and photopic contrast sensitivity or 2. photopic and mesopic visual acuity.

In an update of an earlier study, Higgins and Wood (2005) compared driving performance in normally-sighted individuals wearing goggles simulating three levels of optical blur, or mild cataracts. The 24 participants had an average visual acuity of 6/4.5 (20/15 in US notation) when not wearing goggles, and the goggles were adjusted to reduce each participant's acuity to 6/12 (20/40), 6/30 (20/100) and 6/60 (20/200) levels. The simulated cataract goggles used frosted lenses and reduced acuity to a mean of 6/12. All participants drove test circuits under all conditions, with order balanced with a Latin square design except that normal acuity was always last. The closed-road circuit driving task included sign recognition, road hazard avoidance, gap clearance, and manoeuvring through a series of grey traffic cones. Participants also were assessed for contrast sensitivity (Pelli-Robson), low-contrast acuity (SKILL card), and glare sensitivity (Berkeley Glare Test).

Reduced acuity significantly increased time taken to complete the course ($p < .001$), and reduced sign recognition performance ($p < .001$) and hazard avoidance ($p < .001$), with the cataract goggles producing an effect similar to the 6/60 blur goggles. There was a significant trend towards reduced manoeuvring performance for the three optical blur conditions ($p < .033$). Results of the visual tests showed that the optical blur conditions lowered contrast sensitivity, and the simulated cataract goggles lowered contrast sensitivity even more than the 6/60 blur goggles. Scores on the SKILL and glare tests were impaired by the cataract goggles but not by the blur goggles. Cataracts that impair acuity to a level at which licencing is still permitted may affect driving performance more than acuity degradation caused by uncorrected refractive error alone. Including a test of contrast sensitivity could pick up drivers whose vision is not sufficient for safe driving.

Summary

The research is inconsistent regarding crash risk and visual acuity. In a recent review by Van Rijn and Volker-Dieben published in *New Standards for the Visual Functions of Drivers* (The Eyesight Working Group, 2005), the authors summarised the evidence for crash risk related to visual acuity and reported elevated risk ranging from 1.17 to 7.6.

One reason why it may be difficult to establish crash risk relationships with visual acuity is because drivers with severe acuity impairments may not be driving. Mandatory licensing re-assessments may have identified those most at-risk and eliminated them from the databases being evaluated. Alternatively, knowledge of vision difficulties including poor visual acuity may lead to self-regulation among older drivers. These factors would act to weaken any relationship between acuity measures and crash risk. Other studies have demonstrated no reliable associations with crash risk. Therefore, the relationship between visual acuity and crash risk at this stage is unclear.

3.13.12 VISUAL FIELD LOSS

Definition of visual field loss

Visual field loss is characterised by a functional restriction in an individual's field of vision. The condition may occur as a result of disease or trauma at the level of the eye or the brain. For example, loss of visual field is a major symptom associated with AMD, retinitis pigmentosa (RP), glaucoma and specific neurological disorders (e.g. hemianopia).

Prevalence of visual field loss

The prevalence of visual field loss has been estimated at around 3% for drivers between 16 and 60 years of age, around 7% for those 60-65 years and 13% for individuals over 65 years (Johnson & Keltner, 1983). Ramrattan et al. (2001) estimated the overall prevalence in community-dwelling residents (n = 6250) in the Netherlands as 5.6% (3.0% in those aged 50-64 years to 17% in those aged 85 years and older).

Functional impairments associated with visual field loss relevant to driving

As noted above for specific medical conditions, visual field loss is likely to limit the driver's ability to detect relevant cues or events in the driving environment. Vision loss may affect the central field (central scotoma), peripheral fields (tunnel vision) or one half (hemianopia) or one quarter (upper or lower) (quadrantanopia) of the visual field. Normally, the binocular visual fields subtend more than 180° laterally. In the central field, the fovea, capable of the sharpest visual acuity, spans about 3° and surrounding this to about 10° is the macula, also capable of fine visual discrimination. Beyond this central area lie the peripheral fields which play a critical role in detecting motion. Depending on the extent and type of pathology, varying levels of reduced visual acuity and other decrements in visual function may coexist with visual field loss.

Pre-May 2003: Relationship between visual field defects and road safety outcomes

Crashes

Influential early studies by Johnson and Keltner (Johnson & Keltner, 1983; Keltner & Johnson, 1980, 1987) examined the relationship between visual fields and safety among

older drivers. They assessed visual field loss in 10,000 Californian driving licence applicants using automated visual field tests. The majority of these individuals were unaware of their peripheral vision deficits. The vehicle crash and citation rates in drivers with binocular field defects were found to be twice that of control participants matched by age and sex with normal vision. However, drivers with field loss in one eye were not more crash involved and had no more convictions than drivers with normal visual fields.

Johnson and Keltner noted the importance of exposure measures and accounted for this in their analyses, however, other studies that have accounted for exposure have not supported a relationship between crash risk and visual field impairments (Decina & Staplin, 1993; Owsley, Ball et al., 1998). Decina and Staplin (1993) found no significant relationship between horizontal field assessment and state crash records. However, the results may understate the associations because of the retrospective crash analysis and the likelihood that drivers with crashes and poor vision may opt out of the renewal process. They also found that a combined assessment of horizontal field, acuity, and contrast sensitivity provided the strongest relationship between poor vision and crash involvement.

Two small-scale studies have examined the crash risk associated with RP. Fishman, Anderson, Stinson and Haque (1981) examined the driving performance of 42 participants with RP compared to 87 control participants using self-reported crash histories. They found that participants with RP were involved in more crashes than controls, but only around half were involved in a crash during the previous 5-year driving period. When driving hours per week and driving years were taken into account, the crash rate between the two groups was significantly different, but appeared to be related to the subgroup of female participants with RP. Participants with lower central or peripheral field efficiency were not more likely to be involved in a crash. Similarly, a study by Szlyk, Alexander, Severing and Fishman (1992) of 21 participants with RP and 31 normal sighted control participants roughly matched by age, gender and years of driving, found a greater likelihood of self-reported crash involvement in the RP group. Unlike Fishman et al., they reported an elevated crash risk among participants with restrictions in the horizontal visual field. However, participants with retinal degeneration affecting central visual field did not have elevated rates of both self-reported and state recorded crashes. Driver's awareness of their deficit may have led them to develop adequate compensatory strategies. The limited samples of participants and low crash involvement mean that it would be inappropriate to draw conclusions regarding the crash risk of drivers with RP based on this research.

Citations

Johnson and Keltner (1983) (reviewed above) reported evidence for twice as many convictions amongst drivers with binocular field loss, compared to controls, but no difference for those with field loss in one eye.

Driving Performance

Several authors have addressed other driving performance measures in relation to visual fields. One approach, adopted by Wood and colleagues, is to examine the effect of artificially restricting visual fields in drivers completing an on-road driving circuit. (Troutbeck, & Wood, 1994; Wood, Dique, & Troutbeck, 1993; Wood, & Troutbeck, 1994; Wood, & Troutbeck, 1996). The results of this work indicated that simulated field deficits compromised aspects of driving performance such as identifying road signs and vehicles in

the periphery, avoiding obstacles, and reversing, but speed estimation and emergency stopping abilities were less affected.

A more direct approach to studying the effect of visual field loss on driving performance is to study drivers with clinical conditions resulting in field loss. For example, Lövsund et al. (1991) examined the performance of 31 drivers with visual field defects of different size and location and compared them against 20 normally sighted controls. All participants demonstrated good skill for maintaining speed and remaining within the lane, however, some drivers with field defects had substantial increases in reaction time to stimuli presented in the affected visual field areas. Four of the participants with field defects did not have increased reaction times demonstrating an ability to compensate for deficiencies in their visual field. A second experiment by Lövsund et al. (1991) examined the visual scanning behaviour of two of the drivers with field defects that displayed evidence of compensation and two drivers with comparable conditions who did not exhibit compensation. The driver that showed the best ability to compensate concentrated visual fixations on the affected side of the visual field to a much greater extent than the non-compensating driver with similar field restriction did. The degree to which this compensating behaviour ameliorates crash risk is undetermined.

Studies of driving performance in specific eye pathologies resulting in field loss have shown performance decrements in drivers with AMD, RP and glaucoma (Coeckelbergh, Brouwer, Cornelissen, van Woffelaar & Kooijman, 2002; Szlyk, Alexander, Severing & Fishman, 1992; Szlyk, Fishman, Severing, Alexander & Viana, 1993; Szlyk, Pizzimenti, Fishman, Kelsch, Wetzel, Kagan & Ho, 1995). For example, Coeckelberg et al. (2002) examined simulator and on-road driving performance of participants with age-related MD, glaucoma and RP. On the simulator tasks, drivers with central field loss drove slower than other groups and had smaller safety margins. In contrast, drivers with peripheral visual field deficits showed increased deviations in lateral position and made more lane boundary crossings. In on-road driving performance, official driving examiners considered reduced speed and increased scanning to be effective compensatory strategies for drivers with central and peripheral visual field deficits, respectively. Other studies have also reported longer braking times and more lane boundary crossings in drivers with central vision loss due to juvenile forms of macular degeneration as well as in AMD compared with control groups (Szlyk et al., 1993; Szlyk et al., 1995). These studies are reviewed in Section 3.13.3.

Despite the significant impairment associated with hemianopia, some research has suggested that this condition should not be considered a definite contraindication for holding a drivers' licence (Tant, Brouwer, Cornelissen, & Kooijman, 2002). Tant et al. examined safety of drivers with homonymous hemianopia using a practical driving test and a structured scoring protocol. They found that a minority of drivers (4 out of 28 drivers with HH) passed the test. Other studies have also revealed that over time, people with HH develop visual scanning behaviours to compensate for visual limitations (Pambakian, et al., 2000), or can be trained to improve visual search to adapt to the lost visual hemifield (Zihl, 1995).

When considering research on visual fields and driving performance, it is important to acknowledge differences in assessment and definitions of field loss. Some research simply classifies drivers by state driving regulations (i.e., pass/fail), while others employ measures of severity. However, few studies provide information on the *type* of visual field impairment. Simulation studies can also be questioned on the correspondence between

artificial inducement of field loss and field loss with a clinical cause. Furthermore, artificially inducing impairment discounts the effect of adaptation and compensation for disability. In a comprehensive review of the early visual field literature, North (1985) concluded that inconsistencies can be attributed to differences in the procedures used to measure visual fields or compensation and self-regulation by drivers with vision loss, or both. Owsley and McGwin (1999) suggested that this also reflected the current state of knowledge. Owsley and McGwin suggested that a judicious appraisal of the research would suggest that severe binocular field defects are related to crash involvement, but less significant field impairments are unlikely to adversely affect driving performance.

Post-May 2003: Relationship between visual field defects and road safety outcomes

Two studies relating to driving performance associated with visual field loss were identified in the review period between May 2003 and mid-2009. These studies are reviewed below and summarised in Table 45. Other studies addressing field loss associated with glaucoma are reviewed in section 3.13.2.

Crashes

There were no studies that assessed the relationship between restricted visual fields and crash risk post 2004.

Citations

There were no studies that assessed the relationship between restricted visual fields and driving citations post 2004.

Driving Performance

A study conducted by Bowers and colleagues (Bowers, Peli, Elgin, McGwin & Owsley, 2005) investigated on-road driving performance with moderate visual field loss. Drivers with restricted visual fields ($n = 28$) aged 33-84 were recruited through ophthalmology clinics in Alabama, USA. Most of the participants had glaucoma (96%) while the remainder had RP. Participants were functionally assessed for both vision and cognitive status in an eye clinic. The on-road driving component consisted of a 14 mile (22.5 kilometre) course which incorporated a variety of driving manoeuvres, road types and levels of traffic flow. Two evaluators rated specific elements of driving performance on a 5-point scale. Global driving performance scores were calculated at the end of the drive using both sets of ratings.

The results suggested that a more restricted binocular horizontal field, vertical field and total field were significantly associated with poorer performance in speed matching when changing lanes, path-keeping, and lane positioning during curve taking ($p \leq 0.05$). Restricted horizontal and total fields were also significantly associated with poorer performance in maintaining an appropriate following distance during curve taking. A more restricted vertical field was related to poorer performance in path keeping when turning, and a smaller total field was associated with poorer performance in lane positioning when exiting the interstate. Finally, poorer contrast sensitivity in the better eye was associated with several of the global driving scores, including interaction with other traffic, anticipatory skills, vehicle control skills and overall driving.

Racette and Casson (2005) conducted a retrospective review of clinical notes of patients with visual field loss assessed through a driving rehabilitation programme in Toronto,

Canada. Cases were identified as patients with visual field loss (hemianopia, quadrantanopia, monocular vision, mild peripheral field loss or moderate peripheral field loss) who had undergone an on-road assessment (n = 131) and did not display hemi-neglect or gross cognitive impairment. Participants were interviewed by an OT and completed a vision assessment. The on-road drive was completed by OTs as part of a rehabilitation assessment, and the researchers merged these into three categories: safe; unsafe; and unable to determine.

The results indicated no significant differences in driving assessment outcomes between all five categories of field loss ($\chi^2 = 4.37, p = 0.358$). No significant differences were observed between patients with localised field loss only (hemianopia versus quadrantanopia) ($\chi^2 = 3.33, p = 0.068$). In the monocular category, 79% of drivers obtained a 'safe' outcome on the on-road assessment. No patients with loss localised in the left hemi-field (hemianopia or quadrantanopia) were rated as 'safe', and conversely no patients with loss localised in the right hemi-field were rated as 'unsafe' ($\chi^2 = 9.561, p = 0.002$). However, there are several limitations to this study, including no control group for comparison, no reporting of the method of visual field assessment, and no independent assessors masked to clinical condition. This study suggests that although the extent of visual field defects appears to be related to driving performance, large individual differences indicate that individualised on-road assessments for patients with visual field defects are required.

Summary

The research regarding crash risk and visual fields is inconsistent. Some studies have found that vehicle crash and citation rates in drivers with binocular field defects were higher than those of control participants matched by age and sex with normal vision. However, other studies have not found a significant relationship between horizontal field assessment and crash records. One difficulty in assessing visual fields and driving safety is the varied aetiologies underlying the visual field loss which can mask the relationship. Based on their review of the relevant literature, the EC Driving Licence Committee, Eyesight Working Group noted that "it is evident that an adequate visual field is of utmost importance for the ability to drive safely. However, the actual cut-off value that should be set in the standards is as yet unclear. Further research is needed" (p. 9, 2005).

3.13.13 CONTRAST SENSITIVITY

Definition of contrast sensitivity

Contrast sensitivity refers to the ability to perceive visual stimuli differing in contrast and spatial frequency. Luminance, colour, motion, texture and disparity are all forms of contrast sensitivity. In a practical sense, contrast sensitivity encompasses the ability to detect sharp boundaries of objects and to detect slight changes in luminance at regions without distinct contours. Damage caused by cataracts, glaucoma and macular degeneration all affect some type of contrast sensitivity. Decreased contrast sensitivity is also correlated with age (Owsley et al., 1991; Regan, 1993).

A number of tests have been devised to assess these contrast sensitivities in clinical settings. The most well known contrast sensitivity test is the Pelli-Robson (Clement Clarke International Limited). The Pelli-Robson low-contrast acuity test requires the examinee to read from a distance of 2 metres a letter chart on which the letters from left to right and

from top to bottom progressively fade out (Pelli, Robson & Willkins, 1988). Contrast sensitivity is defined by the minimum contrast required to distinguish between a bar pattern and a uniform background.

Contrast sensitivity testing is not conducted in licensing examinations nor is it addressed in most medical guidelines for fitness to drive.

Prevalence of contrast sensitivity difficulties

No reliable data on prevalence of contrast sensitivity could be found.

Functional impairments associated with contrast sensitivity difficulties relevant to driving

Contrast sensitivity affects the ability to distinguish objects from their background. In driving, this is likely to influence the ability to detect important cues in the road environment under low light conditions such as dark-clothed pedestrians at dusk.

Pre-May 2003: Relationship between contrast sensitivity and road safety outcomes

Crashes

Results from several studies indicate that contrast sensitivity might be a more sensitive predictor of crash risk than simple measures of visual acuity. Brown, Greaney, Mitchell and Lee (1993, cited in Janke, 1994) found Pelli-Robson contrast sensitivity to be the best predictor of crashes among a battery of visual, perceptual and cognitive tests for a group of 1,447 insurance policy holders aged over 50 years. However, the correlation between the test and crashes was still relatively low. Owsley et al. (1991) found a similar correlation of contrast sensitivity with crashes, but in a small sample was not significantly related. A more recent study by Owsley et al. (2001) assessed contrast sensitivity in participants with cataracts (see above). They found that the lowest level of contrast sensitivity (1.25 or less) was significantly associated with at-fault crash risk. The odds ratio for crash risk in the better of the participant's two eyes was 2.65 (95%CI 1.06 - 6.61) and 4.97 (95%CI 1.96 - 14.93) after adjusting for other demographic and health factors. In the worse of the two eyes, the adjusted risk ratio increased to 7.06 (95% CI 1.88 - 26.52). It is important to note, however, that the participants also had difficulties with glare and so it is possible that glare contributed to the odds ratios for crash risk. Nevertheless, the study highlights the important role of adequate contrast sensitivity in preventing crashes for drivers with cataracts.

In the study by Decina and Staplin (1993), several visual measures and crash records were obtained from 12,400 Pennsylvanian drivers. Broad contrast sensitivity measures were not individually associated with crash risk, but when used in conjunction with visual acuity and horizontal visual fields predicted crash involvement by drivers aged 65 and older.

Brabyn, Schneck, Haegerstrom-Portnoy and Steinman (1994) found no relationships between their vision tests including measures of acuity, glare, contrast sensitivity, visual fields and visual-attention, and self-reported crash involvement. Using a derived measure of crash proneness that takes into account the extent to which the participant was at-fault, significant associations were found for Pelli-Robson contrast sensitivity as well as contrast thresholds, glare, visual fields and attentional fields. However, deriving a measure from self-reports has inherent problems with validity.

Citations

No studies linking contrast sensitivity and citations were found.

Driving performance

Measures of contrast sensitivity have also been related to proxy measures of driving performance and driving difficulty. Rubin, Roche, Prasada-Rao and Fried (1994) found that older drivers who reported difficulty driving during both the day and night were also more likely to have poorer Pelli-Robson contrast sensitivity scores. Wood and Troutbeck (1996) also studied the effect of inducing visual impairment by wearing specially designed goggles on several measures of driving performance in a closed-road driving circuit free of other traffic. They found that a significant correlation between Pelli-Robson contrast sensitivity and overall driving score in addition to a manoeuvring score.

While the evidence of crash prediction based on contrast sensitivity is limited, it does appear to be at least as sensitive as visual acuity. Although further research is warranted, contrast sensitivity is a promising screening test, particularly in combination with other measures. However, it is important to note that currently there is a lack of sufficient information on the variety of ways to assess contrast sensitivity and to validate the grades of degradation of this response.

Post-May 2003: Relationship between contrast sensitivity and road safety outcomes

Only one study was identified in the post-May 2003 review period addressing the effects of contrast sensitivity on driving. The study is reviewed below.

Crashes

There were no studies that assessed the relationship between contrast sensitivity and crash risk post 2004.

Citations

There were no studies that assessed the relationship between contrast sensitivity and driving citations.

Driving Performance

Marrington, Horswill and Wood (2008) used goggles to examine the effects of impaired contrast sensitivity while driving. Participants were randomly assigned to no, mild or moderate 'simulated cataract' groups. Two simulated driving performance measures were used: hazard perception in videos of road scenes, and hazard change detection in photographs of road scenes.

For the hazard perception task, there was no significant difference between the mild cataract and no cataract groups; but the moderate cataract group were significantly slower than the no cataract group. For the change detection task, both simulated cataract groups were significantly slower than the no cataract group. The moderate cataract group also missed significantly more hazardous changes (i.e. did not perceive them within the 32 seconds allowed for inspection of each scene). These two tasks correlate with crash involvement, so contrast sensitivity is likely to be a mediating variable between presence of cataracts and higher crash involvement.

3.13.14 VISION DISORDERS – GENERAL

Pre-May 2003: Relationship between vision disorders (considered as a group) and road safety outcomes

Crashes

In a recent study, Vernon, Diller, Cook, Reading, Suruda and Dean (2002) compared the rates of adverse driving events (crash, at-fault crash and citations per 10,000 licence days) experienced by drivers licensed with eye conditions that affect visual acuity with a control group of drivers without medical conditions who were matched by age, sex and place of residence (for more information regarding the study design see section 3.1). The study used a retrospective case-control design, with cases defined as those who had a “history of eye conditions that may affect vision function” (p238) totalled 11,683. The majority of these cases (n=10,116) had no licensing restrictions. According to official driving records, drivers with eye conditions with no licence restriction (i.e., the lowest level of impairment) had significantly higher crash rates and at-fault crashes (RR: 1.35, CI, 1.25-1.46; RR: 1.52, CI, 1.38-1.68, respectively) than the control group. Similarly, drivers with eye conditions with restricted licences (i.e., the highest level of impairment) had significantly higher crash rates and at-fault crashes (RR: 1.27, 95%CI 1.04 - 1.55; RR: 1.56, 95%CI 1.25-1.94, respectively) than the control group.

A major shortcoming of this study is that the inclusion criteria for cases were non-specific (i.e., eye conditions that may affect vision function). Consequently, it is not possible to ascertain the relative risk associated with specific visual acuity deficits from this study. In addition, there is no control for exposure rates, which assumes that the matched controls drive similar distances to those with an eye condition, which may or may not be the case.

Citations

As outlined above, Vernon et al. (2002) compared the relative risk of driving citations of drivers with eye conditions with and without licensing restrictions and compared them to drivers without a medical condition. Overall, Vernon et al. reported that the rate of citations amongst both unrestricted and restricted drivers with eye conditions was significantly higher than the general driving population (RR unrestricted: 1.35, 95%CI 1.27 - 1.43; RR restricted: 1.31, 95%CI 1.10 - 1.56).

Driving performance

No studies which examined the relationship between driving performance and vision disorders (considered as a group) were found.

Post-May 2003: Relationship between vision disorders (considered as a group) and road safety outcomes

Crashes

In one study, 1801 community-dwelling drivers were followed over 6 years to determine the relationship between crash rates and visual function (Rubin et al., 2007). Visual function tests were performed at baseline and included assessments of monocular and binocular visual acuity (ETDRS); contrast sensitivity (Pelli-Robson); glare sensitivity (Brightness Acuity Tester); stereoacuity (Randot Circles test); visual fields (HFA 81 points

over 60 degree radius, monocular; binocular from composite fields). Half the drivers also did a UFOV prototype test.

During the follow-up period, 204 stopped driving, died, or were admitted to a nursing home; 120 had a crash and 1477 did not. Glare sensitivity and binocular visual fields were associated with crash risk. Interestingly, for those with good vision at baseline, worsening of these factors was associated with reduced crash risk (HR=0.46 for glare sensitivity, 0.60 for field loss), while for those with poor vision, worsening was associated with increased crash risk (HR = 2.18 for glare sensitivity, 1.29 for field loss). Cut-offs for good vision were <3 letters for glare sensitivity and <20 points missed for binocular visual fields. Visual field was divided into central (≤ 20 deg), upper and lower fields. For lower fields only, crash risk reduced with field loss <10 points and increased with field loss >10 points. The authors examined whether these differences correlated with changes in driving restriction, but adding variables for reduced mileage (>3000m/y to <3000m/y), cessation of night driving, or cessation of driving in unfamiliar areas did not change the results. UFOV data was available for 857 drivers: hazard ratio for loss of 40 points = 2.12, $p=.002$. Of the subtests, the strongest association was for divided attention (HR = 1.47).

Citations

There were no studies that assessed the relationship between vision disorders (considered as a group) and driving citations post 2004.

Driving performance

There were no studies that assessed the relationship between vision disorders (considered as a group) and driving performance.

Summary

There is limited evidence regarding driving safety in people with vision disorders considered as a group. However, the evidence that does exist suggests the relative risk for crashes in people with vision disorders is higher than the general driving population.

Owsley and McGwin (1999) noted that there is increasing agreement among various road safety practitioners that simple tests of vision such as those used at driver licensing agencies do not effectively identify high-risk drivers. Sims, McGwin, Allman, Ball and Owsley (2000) reported that successful identification of unsafe drivers requires multifactorial assessments related to function, medication, affect, neurology, and visuo-cognitive skills. Therefore, while the goal of developing driver screening test with high sensitivity and specificity may be attainable, these tests may not be cost effective or acceptable to the public. For vision related concerns, it seems necessary to develop assessments that will identify a broad range of vision impairments related to visual diseases and conditions that are also related to driving safety. This is difficult given that driving is a complex task where visual limitations may be overcome by cognitive strategies and crash risk might be mitigated by restriction of driving. It appears necessary to develop a battery of brief tests assessing multiple functions. Indeed, several authors have indicated that this approach is more likely to predict crash involvement (e.g., Decina and Staplin, 1993; Sims et al., 2000). Alternatively, assessments such as the Useful Field of View (UFOV) provide a promising approach in testing multiple impairments in a single test. The UFOV is a visuo-cognitive test that examines visual processing and attentional control functions that may be symptomatic of numerous neurological and visual disorders.

Assessing multiple deficits from comorbid conditions is an important advantage of the UFOV test over other individual measures (Myers, Ball, Kalina, Roth & Goode, 2000). The latest research on UFOV indicates that it is consistently and significantly associated with crash risk even after adjusting for other factors (Myers et al., 2000; Owsley, Ball et al., 1998; Sims et al., 2000).

Table 45 Summary of studies of crash risk associated with visual disorders

Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Cataract Studies			
Foley, Wallace, & Eberhard (1995)	Population-based cohort study 1791 drivers over 68 years; 206 crash-involved	Police-reported crashes	Higher crash risk for men RR: 1.6* (95% CI, 1.2, 2.1); Cataracts no increased crash risk RR: 0.9.
McCloskey et al. (1994)	Population-based matched case-control study; 234 crash involved cases, 447 controls	State records of injurious crashes	No clear associations of crash risk with cataracts
McGwin et al. (2000)	Pop-based Case-Control: Cases crash involved (n = 447), controls no crashes (n = 454)	State recorded crashes during 1996.	No significant associations of crashes with rate of any eye disease.
Owsley et al. (2002)	Case-control	Crash Risk: Pre-surgery; Post-surgery.	Adjusted RR: 0.47* (CI, 0.23-0.94) surgery cf. no surgery. 27% increased risk after surgery, 72% increase for no surgery.
Owsley et al. (1999) ICOM Study	Case-control; Cases = 279 p with cataract cases; Controls = 105 p (no eye disease)	At-fault crash risk; Questionnaire data	RR: 2.48*, (95% CI, 1.06 - 6.14)
Owsley et al. (2001) ICOM Study	Case-control; Cases = 279 p with cataract cases; Controls = 105 p (no eye disease)	At-fault crash risk	Cataracts 2.5 times crash risk; Only CS significant; RR: 4.96* best eye, 7.06* worst eye
Owsley, McGwin, & Ball (1998)	Case-control: Cases of injurious crashes (n = 78), non-injurious crashes (n = 101), non-crash controls (n = 115).	Crash Risk; Injurious and non-injurious crashes.	No association of crash risk with diagnosis of cataracts.
Salzberg & Moffat (1998)	Case-control; Cases with cataract (n= 45); Age-matched controls (n= 449).	Crash rate per 100 licensed drivers; State crash records were examined pre and post exam	Crash risk 1.33 times controls, 1.46 times population Post exam 1.76 times control
Stewart et al. (1993)	Older adult cohort study. 142 crash involved, 1289 no crashes	Self-reported crashes and medical/physical/ mental status	No association of visual disorders with crash risk
Glaucoma Studies			
Foley et al. (1995)	See above		Glaucoma RR: 1.5 (CI, 0.9, 2.7)
Hu et al. (1998)	Panel data analysis; 1811 participants 1985;	State recorded crashes	Males with a history of glaucoma RR: 1.7. Females not significant

Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
	882 participants by 1993		
McGwin et al. (2004)	Cohort study 576 glaucoma patients 115 controls	Police reported crashes	Patients with glaucoma less likely to crash: RR = 0.64, 95% CI = 0.46-0.90 (per person-mile of travel).
McGwin et al (2005)	Case-control study 120 glaucoma patients who had crashed 120 (non crash) controls selected from other 294 glaucoma patients	Police reported crashes	Patients with more severe defects more likely to crash: OR for moderate defect in worse eye = 3.0, OR for severe defect in worse eye = 4.3.
Haymes et al. (2007)	Cohort study 48 glaucoma patients 47 controls	Self-reported and police-reported crashes in last 5 years	Glaucoma patients more likely to crash: OR (adjusted) = 6.62 (1.40-31.23); and more likely to admit fault: OR (adj) = 12.44 (1.08-143.99)
Owsley, McGwin, & Ball (1998)	See above		Injury crash associated with glaucoma 3.6 (CI, 1.2-10.9)
Stewart et al. (1993)	See above		No association of glaucoma with crash risk
Szlyk et al. (2005)	Cohort study 40 glaucoma patients 17 controls	Self-reported crashes in last 5 years	Glaucoma patients significantly more likely to report crashes
Age-Related Macular Degeneration Studies			
Owsley, McGwin, & Ball (1998)	See above		Injury crashes with AMD, unadjusted RR: 3.3 (CI, 1.2-9.2). Not sig for non-injury crashes or when adjusted
Szlyk et al. (1993)	Juvenile macular degeneration (n = 20) Control group (n = 29)	Self-reported and state recorded crashes. Simulator performance measures	Macular degeneration group had more night-time crashes
Szlyk et al. (1995)	AMD group of 10 males ave age 76 years Control group 7 males, 4 females ave. age 71 years	Self-reported and state recorded crashes. Simulator performance measures	No significant associations of AMD with crash risk
Diabetic Retinopathy Studies			
McCloskey et al. (1994)	see above		Non-sig reduction in crash risk associated with DR.
Owsley, McGwin et al. (1998)	see above		Found no association with crash risk
Salzberg & Moffat (1998)	14 older drivers with DR who were referred for Special Examination.	Crash rate per 100 licensed drivers; State crash records were examined pre and post exam	Pre exam crash risk 3.2 times controls, 3.5 times population No crashes post exam.
Szlyk et al. (2004)	Cross-sectional study 25 drivers with diabetic retinopathy	Self-reported crashes	Higher crash risk among those with higher levels of glycosylated haemoglobin.

Author/date	Methods	Outcome Measure of Risk	Crash Risk/ Main Finding
Retinopathy Pigmentosa Studies			
Fishman et al. (1981)	42 p with RP; 87 control group participants	Self-reported crash history - crash rates	RP sig more crashes than controls (adjusted for exposure)
Szlyk et al. (1992)	21 RP p; 31 normal-sighted controls	self-reported and state recorded crash involvement	RP sig more crashes than controls
Colour Vision Studies			
Tagarelli et al. (2004)	Cohort study 151 colourblind participants 302 age-matched controls	Self-reported crash history	No significant difference

signif diff from control, $p < .05$

Approaches to management

Assessing fitness to drive

Many licensing jurisdictions have produced guidelines for assessing drivers' fitness to drive including medical standards for licensing and clinical management guidelines. The guidelines used in Canada, Australia, UK, USA, New Zealand, and Sweden for assessing fitness to drive relating to visual conditions are shown in Table 41. The guidelines addressing the major degenerative diseases such as glaucoma, cataracts, macular degeneration and diabetic retinopathy tend to be very non-specific and do not appear to adequately reflect the scientific evidence on crash risk. Generally, people with these conditions must meet the visual acuity requirements and in some cases visual field requirements. However, these assessments may not satisfactorily define the visual impairment associated with these conditions and, as noted above, are unlikely to effectively identify an unsafe driver. However, some of the areas recommend regular monitoring of the conditions and Canadian guidelines suggest that referral for assessment by ophthalmologists or optometrists may be required when visual impairments are suspected.

Guidelines for visual field defects are quite diverse across each of the jurisdictions reflecting the lack of detailed knowledge on what severity of impairment constitutes an unacceptable crash risk. Conditional licensing procedures are not specified in most jurisdictions except for the requirement of corrective lenses to be worn where uncorrected vision does not meet the visual acuity standards. The guidelines for the Utah Driver Licence Division allow for speed and/or area restrictions when visual acuity is 20/50 to 20/70 in the better eye, but greater restrictions and medical approval are required for poorer visual acuity. Night-time licence restrictions are also typically applied in most areas to drivers with night vision impairments. For example, in Sweden, daytime driving may be permitted for those with night blindness. No restrictions are placed on drivers with colour vision defects.

In the post-May 2003 review period, several updates have been identified in the fitness to drive guidelines relating to vision. In 2007, driving with the use of a bioptic telescope to improve visual acuity was legal in many USA states but not in Europe. A demonstration project in the Netherlands showed that after training first with the telescope, and then training to use the telescope while driving, 9 of 14 participants could successfully complete a fitness-to-drive assessment. This project has led to changes in Netherlands guidelines about acceptable aids to driving.

In Australia, Austroads guidelines require an intact field 10 degrees above and below the midline extending 120 degrees (2006). Silveira and colleagues examined visual field extent (measured binocularly with Humphrey Field Analyser Esterman program and Goldmann Perimeter with III4e and IV4e targets) and on-road driving performance in 100 older drivers to determine whether this guideline predicts ability to drive safely. Twelve drivers had visual field defects that 'raised an issue about their ability to drive'; half of these participants failed the driving assessment, while the other half passed. Total field sizes measured by all three tests were significantly correlated with driving performance (Spearman correlations ranged from 0.42 for Goldman IV4e score to 0.47 for Esterman score); the sensitivity of this test is 0.73 for Esterman and 0.46 for Goldman methods, while specificity was 0.89 for Esterman and 0.94 for Goldmann. In other words, failing the visual field criterion does not predict failing the driving assessment, while passing the

visual criterion does not necessarily mean the driving assessment will also be passed. (As participants were all current drivers the effect of driving experience should not have been an issue in passing the assessment.) Total field sizes and error scores for the 120 degree box measured with each method were entered into a logistic regression, but no predictor or combination of predictors was significant. This may be due to the small numbers of participants with visual field defects and/or who failed the driving assessment. Nonetheless, the results suggest that current licensing restrictions fail to prevent those who are unsafe from driving, while unnecessarily restricting some people with vision impairments who may be able to compensate and drive safely.

Given the potential problems associated with driving with visual field loss, there has been considerable interest in identifying an appropriate means to assess adequate visual field loss for fitness to drive. Two recent studies have compared the current UK standard for assessing fulfilment of driver's licence visual field criteria, the binocular Esterman visual field test, with merged monocular visual field tests (the integrated visual field) and the UFOV test. The original study (Crabb et al 2004) was of 65 participants with primary open-angle glaucoma. This found that the EVFT and IVF mostly agreed on the classification of participants, with 44 passing both tests and 13 failing both tests. No participants passed the IVF but failed the EVFT, while 8 failed the IVF but passed the EVFT. UFOV results suggested that these participants were more like those who failed both tests than those who passed both tests.

A second study examined 60 participants with normal visual acuity but paracentral visual field loss in both eyes that was either homonymous or overlapping to produce binocular paracentral scotoma (Chisolm et al, 2008). Again, the IVF and EVFT agreed for most participants (93% agreement, with 59% of participants passing both tests). One participant passed the EVFT but failed the IVF; three passed the IVF but failed the EVFT; all four of these exhibited binocular scotomata of neurological origin, and all four passed the UFOV. There was no difference in this study between UFOV scores for the group who failed both IVF and EVFT, and the group who passed both these tests; rather UFOV results were related to age. The authors suggest this implies that the UFOV is not an appropriate surrogate test for assessing visual fields for fitness to drive. While the IVF is better for assessing central visual field loss, particularly in drivers with glaucoma, the authors concluded that the EVFT remains the most appropriate single test of visual fields.

Self-regulation

Many of the studies on the relationship between crash risk and eye disease or vision impairment may have understated the association because either drivers with major visual deficiencies have been identified and already removed from the driving environment or self-regulated their own driving. In fact, there is good evidence to suggest that drivers with known visual impairments do restrict their driving to some degree and are more likely to give up driving (Ball et al., 1998; Lyman et al., 2001, Owsley et al., 1999). However, in a sample of 402 visually impaired drivers Stalvey and Owsley (2000) found that over half believed that their vision did not make them more likely to crash. While 80% felt safer avoiding certain driving situations such as turning across traffic and interstate highways, relatively few reported actively avoiding these situations. Stalvey and Owsley concluded that many drivers with visual impairments would benefit from behavioural interventions promoting self-regulation and alternative transportation. Indeed, a very recent follow-up study by Owsley, Stalvey, and Phillips (2003) demonstrated increased self-regulatory practices among a group of visually impaired drivers in an educational intervention. These

types of programs may represent effective supplements to mass visual screening programs conducted by licensing authorities. It also indicates that vision specialists play a critical role in educating and advising their participants on the risks of driving with vision impairments.

In the post-May 2003 review period, one study was identified addressing the effects of driver training in drivers with vision impairments. Owsley and colleagues (2004) followed up their previous studies in the area by examining whether the educational program had any effect on crash rates. All licensed drivers in a county aged 60 and above who had been driver in a crash in previous year were invited to participate. After excluding those with low driving exposure and/or cognitive impairment, drivers were screened for visual impairment (acuity between 20/30 & 20/60, or $\geq 40\%$ score reduction on useful field of view). A total of 403 drivers with visual impairments were then randomly assigned to either an individually tailored education program ($n = 227$) or a control group who received no further intervention ($n = 176$). Both groups were followed up by telephone interview at 6 months, 12 months, 18 months and 2 years to collect data on driving behaviour. State-recorded crashes over the two year period were examined, and crash rates calculated per person-mile and person-year. While the drivers in the intervention group did reduce their mileage and avoid certain situations more than the non-intervention group, this did not result in a reduced crash rate: the relative risk was 1.08 (95%CI 0.71 - 1.64) per person-year and 1.40 (95%CI 0.92 - 2.12) per person-mile.

A large sample of Californian drivers aged over 55 reported visual problems and driving avoidance. Forty-seven percent of the sample reported limiting/avoiding driving. Problems with vision were the leading cause. This was more common among females and older drivers (over 75 compared to 55-64).

In terms of specific (self-reported) visual conditions, the risk ratio for reporting driving limitation/avoidance for glaucoma was 1.9, for cataracts was 2.1, and for macular degeneration was 2.5 (all significant at $p < .001$). There were also significant risk ratios ranging from 2.2 to 3.4 for visual symptoms such as having difficulty focusing, judging distance, reading street signs at night, problems with glare from sun or lights, and avoiding physical activity due to vision. The SKILL test (Smith-Kettlewell Institute Low Luminance card - scores high-contrast & low-contrast visual acuity as within or outside normal limits for age norms) was associated with a small but significant risk ratio of 1.2, and needing to wear glasses or contacts for driving had a significant risk ratio of 2.0.

Massof and colleagues (2007) report a large study of perceptions about driving in a low-vision sample, where 851 low-vision patients attending a rehabilitation clinic underwent assessment of visual acuity and contrast sensitivity, and completed an interview with questions about the importance and difficulty of various tasks.

Participants with low visual acuity and/or low contrast sensitivity were more likely to rate driving as not important. Participants who rated driving as important were asked to provide difficulty ratings; those who rated driving as impossible had worse contrast sensitivity and/or visual acuity than those who rated driving as not difficult. Current drivers rated their own driving ability significantly higher than those who no longer drove. These results imply that participants with lower visual function stop driving due to the perceived inability to safely complete driving tasks (although no test is reported as to the visual function of participants who still drove vs those who did not drive).

Self-rated driving ability did not depend on perceived importance of driving. However, participants who had not driven for more than 2 years, or who had never driven, were significantly more likely to rate driving as not important than drivers who were still driving. Those who ceased driving more recently were more likely to rate driving as unimportant, but not significantly so. This suggests that it takes a great deal of time for people with vision problems that prevent driving to find satisfactory alternative mobility strategies.

Two studies of 1309 community-dwelling older adults in Maryland, USA, examined driving habits and visual function over two years (Freeman et al., 2005; Freeman et al., 2006). Driving habits were assessed in an interview completed by 76% of the participants, and by proxy for the other 24%. Tests of visual function were conducted in a clinic at baseline and follow-up, and included visual acuity (Early Treatment of Diabetic Retinopathy Study charts and protocols), contrast sensitivity (Pelli-Robson), visual field (Humphrey VFA monocular fields – binocular fields estimated from composite), and glare sensitivity (Brightness Acuity Tester).

Characteristics of 206 drivers who did not return for follow-up were compared with those who did. These drivers were more likely to be older, male, to have reported more comorbidities, or a worse general health status, and to have had a history of diabetes and stroke than those who did return; these differences may affect the associations reported below.

The first study reports on associations with ceasing driving entirely, which occurred for 386 participants (Freeman et al., 2005). Those who stopped driving were more likely to be older, female, and have poorer mental and physical health at baseline. Measures of visual function that were associated with driving cessation at follow up included: baseline acuity, contrast sensitivity, central and lower peripheral visual fields (age-adjusted linear trend $p < .05$), and 2 year glare sensitivity loss. A regression analysis with all vision variables found that baseline contrast sensitivity, baseline central and lower peripheral visual fields (linear trend p value $< .05$), 2 year contrast sensitivity loss, and 2 year lower peripheral visual field loss (linear trend p value $< .05$) predicted driving cessation. Older adults with worse scores across multiple measures of vision are more likely to stop driving.

The second study reports other changes in driving habits. Those who reduced their mileage from over 3,000 miles/year to under 3,000 miles/year were more likely to be older, female and African America ($p < 0.05$), and more likely to be cognitively impaired and in fair/worse reported health ($p < 0.01$) (Freeman et al, 2006). Worse baseline acuity, contrast sensitivity and central and peripheral visual field scores were also associated with reduced mileage (age adjusted linear trend $p < 0.05$). Participants with worse visual acuity were likely to reduce mileage (OR: 2.76, 95%CI 1.25 – 8.16) whether or not there were other drivers present in the house. Cessation of night driving was associated with age (older) and sex (female) ($p < .05$), diabetes and worse reported health ($p < 0.1$), as well as worse baseline contrast sensitivity and central and peripheral visual field loss (age adjusted linear trend $p < .05$; loss of peripheral visual fields OR: 2.15, 95%CI 1.03 – 4.52). Participants who reported moderate or extreme difficulty with oncoming headlights during night driving were more likely to report cessation of night driving (OR: 2.0, 95%CI 1.3 – 3.0). Those who stopped driving in unfamiliar areas were more likely to be female ($p < .05$) and to have worse baseline acuity (age adjusted linear trend $p < .05$). Oddly, driving habits at follow-up seemed to be more closely correlated with visual function at baseline than visual

function at follow-up, although the two sets of visual function measures were moderately correlated.

Self-regulation may be affected by factors other than visual function. A study of 900 community dwelling individuals aged 58 years and older examined gender differences in visual function and driving self-restriction (Brabyn et al., 2005). Participants reported their driving habits and medical conditions and completed tests of mental status and depression, contrast sensitivity, high and low contrast acuity, high contrast and low contrast low luminance acuity, low contrast acuity with and without glare, glare recovery time, and visual fields.

Of the 900 participants, 148 reported that they did not drive. Of the 752 drivers, those who restricted their driving to daylight hours scored significantly lower ($p < .001$) on all visual measures. A stepwise regression for each gender showed that contrast sensitivity had strongest independent association with night time driving restriction (OR: 2.72) for men, while for women low contrast acuity in glare had the strongest association (OR: 1.84). Depressive symptoms were significantly related to men's but not women's night-driving avoidance. Age was a significant predictor for women but not for men.

Women had slightly better vision than men: small but statistically significant differences between men and women were found for contrast acuity (0.03 log unit difference, $p < .005$), contrast sensitivity (0.05 log unit difference, $p < .001$), low-contrast acuity in glare (0.06 log unit difference, $p < .005$), low contrast, low-luminance acuity (0.07 log unit difference, $p < .001$) and glare recovery (0.05 log unit difference, $p < .05$). However, women were more likely than men to restrict their driving at all levels of visual function. Overall, of men who currently drove 13.6% restrict their driving to daytime, while 27.7% of women drove only during daytime. Among those who ever drove (ex and current drivers) only 6.6% of women and 12.3% of men still drive at night. Far more men than women with vision function poor enough to fail standard visual acuity criteria (20/40) continue to drive at night.

An Australian study of 90 drivers aged 60-91 aimed to determine if functional test scores that were positively correlated with errors on an on-road driving test were also positively correlated with greater avoidance of difficult driving situations (Baldock et al., 2006). The tests included measures of depression, anxiety, mental status (cognitive impairment), physical functioning, visual acuity (Snellen static), contrast sensitivity (Pelli-Robson), horizontal visual field, speed of information processing, visuo-spatial memory, visual selective and divided attention. Drivers also completed a questionnaire on driving habits and attitudes. The 40-60 minute on-road driving assessment was conducted by a professional driving instructor and scored by an occupational therapist; drivers had to perform a number of manoeuvres in situations that became progressively more demanding.

Error scores were weighted by the severity of the error (habitual, hazardous, or requiring intervention by driving instructor). Weighted error scores were correlated with contrast sensitivity, speed of information processing, visuospatial memory, and various measures of visual attention. Avoidance of difficult driving situations significantly correlated with general health, medication use, visual acuity in right eye and various measures of visual attention. Visual attention was thus related to both driving performance and self-regulation: those whose reaction time to visually presented targets was longer or who were more likely to fail to detect targets were more likely to perform poorly on the on-road driving test, but also more likely to avoid driving in difficult situations. In contrast, speed of information processing, visuospatial memory and contrast sensitivity were related to driving

performance, but not to self-regulation; drivers with impairments in these areas may be unaware of the effect on their driving. It should be noted that this study did not include participants who were at all depressed, anxious or cognitively impaired. These three common mental states affect many older drivers and are known to affect driving ability.

Finally, a recent study of 1,202 community dwelling older adults (aged 67-87) in Maryland examined factors predicting driving cessation or restriction after one year (Keay et al., 2009). On enrolment in the study, drivers completed a battery of visual and cognitive tests, and a questionnaire about driving habits, medical conditions and medication. Driving habits were checked by installing a Driver Monitoring System in the participants' car for 5 days at baseline and again at follow up; this recorded mileage and location of driving, as well as video of the driver to check identity. Of the 1,202 participants who were driving at baseline, 18 had stopped driving at follow up (9 men, 9 women) and 41 had restricted driving to local neighbourhood (88% women). Logistic regression models were created to examine which factors predicted driving cessation/restriction. After adjusting for age and sex, all baseline measures of visual function were significant at $p < .05$ (VA $p = .0006$, CS better eye $p < .001$, bilateral VF $p = .001$). Visual attention extent ($p = .02$) and visuomotor integration ($p < .001$) were also significant. Preference for driving mediated the effect of contrast sensitivity on driving cessation/restriction, while depression mediated the effect of visual field loss. In contrast to previous studies, driving experience and availability of alternative transport did not predict driving cessation (perhaps as the area studied was relatively homogenous and lacking in public transport options).

Table 46 Private licensing guidelines for drivers with visual conditions

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Visual Problems	CCMTA (2009)	Austroroads (2006)	Drivers Medical Group, Swansea (2008)	Utah Driver Licence Division (2006)	Land Transport Safety Authority (2002)	Swedish Road Administration (1999)
Acuity (assessed using Snellen chart or similar)	Minimum visual acuity of 6/15 (metric) with both eyes open and examined together.	Minimum visual acuity of 6/12 (metric) required using both eyes together or in the best eye. More than 2 errors on any line of the chart is a fail. Conditional licence may be issued if the person: 1. Meets the standard with use of corrective lenses. 2. Undergoes periodic reviews.	Drivers should be able to read in good light (with the aid of glasses or contact lenses if worn) a registration mark fixed to a motor vehicle and containing the letters and figures 79 millimetres high and 50 millimetres wide (i.e. post 1.9.2001 font) at a distance of 20 metres, or at a distance of 20.5 metres where the characters are 79 millimetres high and 57 millimetres wide (i.e. pre 1.9.2001 font)	Unrestricted licence issued if the person has 20/40 in the stronger eye. A restricted licence may be issued if the better eye has 20/50 to 20/70 (speed and/or area restrictions apply). OR If the better eye has 20/80 to 20/100, speed, area & time of day restrictions apply & Medical Advisory Board approval required Restricted from driving if better eye is 20/200 or worse.	Minimum visual acuity in both eyes together of 6/12 (metric), with or without corrective lenses.	Minimum binocular visual acuity of 0.5 required (with or without corrective lenses). Desist from driving for 6 months if visual acuity is less than 0.3 in one eye & onset was sudden.

<p>Visual Field Defect</p>	<p>Visual field defects must be fully assessed by an optometrist or ophthalmologist.</p> <p>“120 continuous degrees along the horizontal meridian & 15 continuous degrees above & below fixation with both eyes open”</p>	<p>A conditional licence may be issued</p>	<p>Desist from driving if person cannot meet national visual field requirements of at least 120 degrees on the horizontal using a target equivalent to the white Goldman III4e settings. There should be no significant defect within 20 degrees fixation above or below the horizontal meridian.</p>	<p>Unrestricted licence issued if the person has:</p> <ol style="list-style-type: none"> 1. “Monocular visual fields 120 degrees in each eye”. (p29) 2. “Binocular visual fields 70 degrees to the right & left in the horizontal meridian”. (p51) 3. “At least 90 degrees in each eye; acuity 20/40 or better in better eye” (p51). 4. “At least 120 degrees total for both eyes” (p51). <p>A restricted licence may be issued if the person has “at least 90 degrees for both eyes.” (p51).</p> <p>Speed, area & time of day restrictions apply & approval from Medical Advisory Board required.</p>	<p>Minimum visual field requirement must be met – i.e. “a binocular horizontal field of 140 degrees” with “no significant pathological defect encroaching within 20 degrees of the point of fixation”.</p>	<p>Minimum binocular field of vision to be equal to that of 1 good eye.</p> <p>Visual field defects that occur in both eyes are acceptable if the defect is on the periphery of the eye and has limited extent & depth</p> <p>SNRA to be consulted where doubt exists.</p>
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<p>Monocular Vision (loss of vision in one eye)</p>	<p>Recent loss of sight in one eye may require a few months for adaptation to occur in order to adequately judge distance.</p>	<p>Requirements are the same as for visual acuity (above).</p>	<p>May drive if in medical opinion the person has:</p> <ol style="list-style-type: none"> 1. Adapted to the condition. 2. Remaining eye meets eyesight requirements in preamble. 3. Remaining eye has a normal field of vision. <p>People with light perception in the impaired eye are not considered monocular.</p>	<p>May be licensed if vision in one eye only or if vision in one eye is “correctable” to 20/40.</p>	<p>Vision in the good eye must meet the combined visual acuity & visual fields test standards as above. Good eye must be free of disease which impairs driving ability. Probable licence condition requiring external rear vision mirrors on both sides of vehicle.</p> <p>May be required to undergo a practical driving test.</p>	<p>Minimum monocular visual acuity of 0.6 required (with or without corrective lenses).</p>
<p>Diplopia (Double vision)</p>	<p>Diplopia which can be corrected is through the use of a device to obscure one eye does not resist the driver from driving after a 3 month period.</p> <p>However the driver must meet the standards for monocular vision.</p>	<p>Refrain from driving if diplopia occurs when gazing at “objects within 20 degrees of the primary direction of gaze”.</p> <p>Conditional licence may be issued if an occluder is used. Periodic review required.</p>	<p>Desist from driving when condition is diagnosed.</p> <p>May resume driving when DLA notified that condition is controlled using glasses or a patch, which must be worn whilst driving. A person with a stable uncorrected diplopia of 6 months or more may be considered for driving if there is medical support indicating functional adaptation.</p>	<p>May only be licensed if medical recommendation obtained.</p>	<p>Refrain from driving until assessed and treated satisfactorily.</p> <p>May resume driving if diplopia can be treated with prisms or occluders & the visual acuity & visual field test standards (above) are met & adaptation to the condition has occurred.</p>	<p>Diplopia that occurs in any direction whilst eyes move 30 degrees to the left or right with the head facing to the front is unacceptable.</p>

Night Blindness	No required standard.	No specific standard.	Cases will be considered individually. Acuity and visual field requirements must be met.	No specific standard. However, some cases may be recommended to drive during daylight only.	May be issued with conditional licence restricting driving to daylight hours only.	Licence disqualification or denial if person has total night blindness or night vision is seriously limited
Colour Vision Defects	No required standard. Driver must be able to discriminate among traffic lights.	No restrictions. Doctors should counsel drivers of difficulties in detecting red lights eg brake & traffic lights.	No restrictions. DVLA notification not required.	Colour vision not considered necessary for private licences.	No restrictions.	Not addressed.
Cataracts	Assessment by an ophthalmologist or optometrist recommended, if cataracts are suspected.	Regular monitoring of vision required. Must meet visual acuity & visual field standards.	Must satisfy visual acuity and visual field standards (above) & be able to read car number plates in the presence of glare.	Must meet visual acuity & visual fields standards.	Restrictions may be necessary due to glare or vision difficulties eg driving restricted to daylight hours only.	Not specifically addressed.
Glaucoma	Assessment by an ophthalmologist or optometrist recommended, if glaucoma is suspected.	Regular monitoring of vision required. Must meet visual acuity & visual field standards.	For severe bilateral glaucoma: Desist from driving until person can meet visual field criteria (as above).	Must meet visual acuity & visual fields standards.	Must meet visual field requirements.	Not specifically addressed.

Table 47: List of abbreviated terms

AGIS	Advanced Glaucoma Intervention Study
AMD	Age-related macular degeneration
CDRS	Certified Driving Rehabilitation Specialist
CS	Contrast sensitivity
DM	Diabetes Mellitus
DR	Diabetic Retinopathy
ETDRS	Early Treatment of Diabetic Retinopathy Study
EVFT	Esterman Visual Field Test
HFA	Humphrey Field Analyzer
IVF	Integrated Visual Fields
MMSE	Mini-Mental State Examination
OT	Occupational Therapist
PACG	Primary Angle Closure Glaucoma
POAG	Primary Open Angle Glaucoma
RP	Retinitis Pigmentosa
UFOV	Useful Field of View test
VA	Visual acuity
VF	Visual fields
VFL	Visual field loss

References

- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia.* Sydney, Australia.
- Alward, W. L. M. (1998). Drug therapy: medical management of glaucoma. *The New England Journal of Medicine*, 339(18), 1298-1307.
- Age-Related Eye Disease Study Research Group (2001). A randomized, placebo-controlled trial of high dose supplementation with vitamins C and E, beta carotene, and zinc for age-related macular degeneration and visual loss. *Archives of Ophthalmology*, 119(10), 1417-1436.
- Anuradha, S., Potter, C. & Fernquest, G. (2007) Vision and drivers: A South Wales survey, *Journal of Public Health*, 29(3) 230-235.
- Australian Diabetes Educators Association (ADEA). (2002, September). *Draft guidelines for the management and care of diabetes in the elderly.* Consultation Draft retrieved June 3, 2003, from <http://www.adea.com.au>
- Babizhayev, M. A. (2004) Rejuvenation of Visual Functions in Older Adult Drivers and Drivers with Cataract During a Short-Term Administration of N-Acetylcarnosine Lubricant Eye Drops. *Rejuvenation Research*, 7, 186-198.
- Baldock, M. R., Mathias, J. L., McLean, J. & Berndt, A. (2006) Self-regulation of driving and older drivers' functional abilities, *Clinical Gerontologist*, 30(1) 53-70.
- Ball, K., Owsley, C., Sloane, M. E., Roenker, D. L. & Bruni, J. R. (1993). Visual attention problems as a predictor of vehicle crashes in older drivers. *Investigative Ophthalmology & Visual Science*, 34, 3110-3123.
- Ball, K., Owsley, C., Stalvey, B., Roenker, D. L., Sloane, M. E. & Graves, M. (1998). Driving avoidance and functional impairment in older drivers. *Accident Analysis & Prevention*, 30(3), 313-322.
- Barsam, A. & Laidlaw, A. (2006) Visual fields in patients who have undergone vitrectomy for complications of diabetic retinopathy. A prospective study. *BMC Ophthalmology*, 6, 5.
- Berson, E. L. (1996). Retinitis pigmentosa: Unfolding its mystery. *Proceedings of the National Academy of Sciences, USA*, 93(10), 4526-4528.
- Bowers, A., Peli, E., Elgin, J., McGwin, G., & Owsley, C. (2005). On-Road Driving with Moderate Visual Field Loss. *Optometry and Vision Science*, 82(8), 657-667.
- Brabyn, J., Schneck, M. E., Haegerstrom-Portnoy, G. & Steinman, B. (1994). Vision test performance and accident proneness in drivers over the age of 55. *Vision Science and its Applications*, 2, 210-213.

- Brabyn, J., Schneck, M. E., Haegerstrom-Portnoy, G. & Steinman, B. (2005) Night driving self-restriction: vision function and gender differences, *Optometry and Vision Science*, 82(8) 755-764.
- Buch, H., Vinding, T. & Nielson, N. (2001). Prevalence and causes of visual impairment according to World health Organisation and United States Criteria in an aged, urban Scandinavian population. *Ophthalmology*, 108(12) 2347-2357.
- Burg, A. (1967). *The relationship between vision test scores and driving record: general findings*. Report No. 67-24. Department of Engineering, UCLA, Los Angeles.
- Burg, A. (1968). *The relationship between vision test scores and driving record: general findings*. Report No. 68-27. Department of Engineering, UCLA, Los Angeles.
- Canadian Council of Motor Transport Administrators (CCMTA) (2009). *CCMTA medical standards for drivers*. 1-13.
- Canadian Ophthalmological Society (2000). Canadian Ophthalmological Society recommendations: Vision standards for driving in Canada. *Canadian Journal of Ophthalmology*, 35(4), 187–91.
- Carberry, T. P., Wood, J., Watson, B. & King, M. (2006) Self-Awareness of Driving Impairment in Patients with Cataract or Glaucoma. *Proceedings 2006 Australasian Road Safety Research, Policing and Education Conference*. Surfers Paradise, Queensland.
- Cedrone, C., Nucci, C., Scuderi, G., Ricci, F., Cerulli, A. & Culasso, F. (2006) Prevalence of blindness and low vision in an Italian population: a comparison with other European studies, *Eye*, 20, 661-667.
- Center for Disease Control (1995). Number of selected reported chronic conditions per 1,000 persons, *by age: United States, 1995*. Retrieved May 22, 2003, from http://www.cdc.gov/nchswww/data/10_199_1
- Centre for Eye Research Australia (CERA), University of Melbourne Department of Ophthalmology (2003a, Updated February 20). *About cataracts*. Retrieved May 23, 2003, from <http://cera.unimelb.edu.au/new/eyecare/ataract.html>
- Centre for Eye Research Australia (CERA), University of Melbourne Department of Ophthalmology (2003a, Updated February 20). *Diabetes and your eyes*. Retrieved May 23, 2003, from <http://cera.unimelb.edu.au/new/eyecare/diabetes.html>
- Centre for Eye Research Australia (CERA), University of Melbourne Department of Ophthalmology (2003a, Updated February 20). *Refractive Error*. Retrieved May 28, 2003, from <http://cera.unimelb.edu.au/new/eyecare/refractive.html>
- Chisholm, C. M., Rauscher, F. G., Crabb, D. C., Davies, L. N., Dunne, M. C., Edgar, D. F., Harlow, J. A., James-Galton, M., Petzold, A., Plant, G. T., Viswanathan, A. C., Underwood, G. J., Barbur, J. L. (2008) Assessing visual fields for driving in

- patients with paracentral scotomata, *British Journal of Ophthalmology*, 92, 225-230.
- Coeckelbergh, T.R.M., Brouwer, W.H., Cornelissen, F.W., van Woffelaar, P. & Kooijman, A.C. (2002). The effects of visual field defects on driving performance: A driving simulator study. *Archives of Ophthalmology*, 120, 1509-1516.
- Coleman, A.L. (1999). Glaucoma. *The Lancet*, 354(9192), 1803-1810.
- Congdon, N., West, S., Friedman, D.S., Kempen, J. H., Vingerling, J. R., Klein, B. E., O'Colmain, B. J., Wu, S. Y., Taylor, H. R. & Wang, J. J. (2004) Prevalence of cataract and pseudophakia/aphakia among adults in the United States, *Archives of Ophthalmology*, 122, 487-494.
- Crabb, D. P., Viswanthan, A. C., McNaught, A., Poinoosawmy, D., Fitzke, F. W. & Hitchings, B. (1998) Simulating binocular visual field status in glaucoma, *British Journal of Ophthalmology*, 82, 1236-1241.
- Davison, P. A. (1985). Inter-relationships between British drivers' visual abilities, age, and road accident histories. *Ophthalmic & Physiological Optics*, 5, 195-204.
- Decina, L. E. & Staplin, L. (1993). Retrospective evaluation of alternative vision screening criteria for older and younger drivers. *Accident Analysis & Prevention*, 25(3), 267-275.
- Dielemans, I., Vingerling, J. R., Wolfs, R. C., Hofman, A., Grobbee, D. E. & de Jong, P. T. (1994). The prevalence of a primary open-angle glaucoma in a population-based study in The Netherlands. *Ophthalmology*, 101(11), 1851-1855.
- Dionne, G., Desjardins, D., Laberge-Nadeau, C., & Maag, U. (1993). *Medical conditions, risk exposure and truck drivers' accidents: and analysis with count data regression models*. Centre for Research on Transportation (Report No. CRT-929), Université de Montréal, Quebec, Canada.
- Disability Online, State Government of Victoria (2002a, Updated November 29). *Cataracts explained*. Retrieved May 23, 2003, from http://www.disability.vic.gov.au/dsonline/dsarticles.nsf/pages/Cataracts_explained?
- Disability Online, State Government of Victoria (2003, Updated March 31). *Diabetic Retinopathy*. Retrieved May 23, 2003, from http://www.disability.vic.gov.au/dsonline/dsarticles.nsf/pages/Diabetic_retinopathy?
- Disability Online, State Government of Victoria (2002b, Updated July 31). *Eye disorders - common focussing problems*. Retrieved May 26, 2003, from http://www.disability.vic.gov.au/dsonline/dsarticles.nsf/pages/Eye_disorders_common_focussing_problems?OpenDocument

- Dobbs, B. M. (2001). *Medical conditions and driving: current knowledge*. Technical Report, National Highway Transportation Safety Administration and The Association for the Advancement of Automotive Medicine Project.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- EC Driving Licence Committee, Eyesight Working Group (2005). *New Standards for the Visual Function of Drivers*, Brussels.
- Ekström, C. & Haglund, B. (1991). Chronic open-angle glaucoma and advanced visual field defects in a defined population. *Acta Ophthalmologica*, 69(5), 574-580.
- Elliott, D. B., Patla, A. & Bullimore, M. A. (1997). Improvements in clinical and functional vision and perceived visual disability after first and second cataract eye surgery. *British Journal of Ophthalmology*, 81, 889-895.
- Fishman, G. A., Anderson, R. J., Stinson, L. & Haque, A. (1981). Driving performance of retinitis pigmentosa participants. *British Journal of Ophthalmology*, 65(2), 122-126.
- Foley, D. J., Wallace, R. B. & Eberhard, J. (1993). Risk factors for motor vehicle crashes among older drivers in a rural community. *Journal of the American Geriatrics Society*, 43(7), 776-781.
- Freeman E. E, Munoz B, Turano K. A, West S. K. (2005) Measures of visual function and time to driving cessation in older adults. *Optometry and Vision Science* (2005) 82, 765–773.
- Freeman E. E, Munoz B, Turano K. A, West S. K. (2006) Measures of visual function and their association with driving modification in older adults. *Investigative Ophthalmology and Visual Science*, 47, 514–520
- Friedman D. S., Munoz, B., Kempen, J. H., Congdon, N., O'Colmain, B. J., Tomany, S. C., McCarty, C. A., DeJong, P., Nemesure, B. & Mitchell, P. (2004) The prevalence of age-related macular degeneration in the United States, *Archives of Ophthalmology*, 122, 564-572.
- Gilhotra, J. S., Mitchell, P., Healey, P. R., Cumming, R. G., & Currie, J. (2002). Homonymous Visual Field Defects and Stroke in an Older Population. *Stroke*, 33(10), 2417-2420.
- Glynn, R. J., Seddon, J. M., Krug, J. H. Jr., Sahagian, C. R, Chiavelli, M. E. & Campion E. W. (1991). Falls in elderly participants with glaucoma. *Archives of Ophthalmology*, 109(2), 205-210.
- Gottlieb, J. L. (2002). Age-related macular degeneration. *JAMA*, 288(18), 2233-2236.
- Gresset, J. A., & Meyer, F. M. (1994). Risk of accidents among elderly car drivers with visual acuity equal to 6/12 or 6/15 and lack of binocular vision. *Ophthalmic & Physiological Optics*, 14, 33-37.

- Gutierrez, P., Wilson, M. R. & Johnson, C. (1997). Influence of glaucomatous visual field loss on health-related quality of life. *Archives of Ophthalmology*, 115, 777-784.
- Hakamies-Blomqvist, L.E. (1993). Fatal accidents of older drivers. *Accident Analysis and Prevention*, 25(1), 19-27.
- Haymes, S. A., LeBlanc, R. P., Nicolela, M. T., Chiasson, L. A. & Chauhan, B. C. (2007) Risk of Falls and Motor Vehicle Collisions in Glaucoma. *Investigative Ophthalmology and Vision Science*, 48, 1149-1155.
- Haymes, S. A., LeBlanc, R. P., Nicolela, M. T., Chiasson, L. A. & Chauhan, B. C. (2008) Glaucoma and On-Road Driving Performance. *Investigative Ophthalmology and Visual Science*, 49, 3035-3041.
- Higgins, K. E. & Wood, J. M. (2005) Predicting components of closed road driving performance from vision tests. *Optometry and Visual Science*, 82, 647-56.
- Higgins, E., Wood, J., & Tait, A. (1998). Vision and driving: selective effect of optical blur on different driving tasks. *Human Factors*, 40(2), 224-233.
- Hills, B. L. (1980). Vision, visibility, and perception in driving. *Perception*, 9, 183-216.
- Hills, B. L. & Burg, A. (1977). *A reanalysis of California driver vision data: general findings. Report No. LR 768.* Transport and Road Research Laboratories, Crowthorne, Berkshire.
- Hu, P. S., Trumble, D. A., Foley, D. J., Eberhard, J. W. & Wallace, R. B. (1998). Crash risks of older drivers: A panel data analysis. *Accident Analysis & Prevention*, 30(5), 569-582.
- Humphriss, D. (1987). Three South African studies on the relation between road accidents and drivers' vision. *Ophthalmic & Physiological Optics*, 7(1), 73-79.
- Isler, R. B., Parsonson, B. S., & Hansson, G. J. (1997). Age related effects of restricted head movements on the useful field of view of drivers. *Accident Analysis & Prevention*, 29(6), 793-801.
- Ivers, R. Q., Mitchell, P. & Cumming, R. G. (1999). Sensory impairment and driving: The Blue Mountains Eye Study. *American Journal of Public Health*, 89, 85-87.
- Janke, M., (1994). *Age-related disabilities that may impair driving and their assessment. A literature review.* Sacramento: California Department of Motor vehicles. Report RSS-94-156.
- Javitt, J. C. & Aiello, L. P. (1996). Cost-effectiveness of detecting and treating diabetic retinopathy. *Annals of Internal Medicine*, 124, 164-169.
- Johansson, K., Bronge, L. & Lundberg, C. (1996). Can a physician recognize an older driver with increased crash risk potential? *Journal of the American Geriatrics Society*, 44, 1198-1204.

- Johnson, C. A. (2005) Vision requirements for driver's license examiners. *Optometry and Vision Science*, 82, 779-89.
- Johnson, C. A. & Keltner, J. L. (1983). Incidence of visual field loss in 20,000 eyes and its relationship to driving performance. *Archives of Ophthalmology*, 101, 371-375.
- Kahn, H. A., Leibowitz, H. M. & Ganley, J. P. (1977). The Framingham Eye Study. I. Outline and major prevalence findings. *American Journal of Epidemiology*, 106, 17-32.
- Keay, L., Munoz, B., Turano, K. A., Hassan, S. E., Munro, C. A., Duncan, D. D., Baldwin, K., Jasti, S., Gower, E. W. & West, S. K. (2009) Visual and cognitive deficits predict stopping or restricting driving: the Salisbury Eye Evaluation Driving Study (SEEDS). *Investigative Ophthalmology & Visual Science*, 50, 107-13.
- Keeffe, J. E., Jin, C., Weih, L. M., McCarty, C. A. & Taylor H. R. (2002). Vision impairment and older drivers: who's driving. *British Journal of Ophthalmology*, 86, 1118-1121.
- Keeney, A. H., Garvey, J. L., & Brunner, G. F. (1981). *Current experience with monocular drivers of Kentucky*. Proceedings of the 25th conference of the American Association for the Advancement of Automotive Medicine (pp. 215-221), October 1-3, San Francisco.
- Keltner, J. L. & Johnson, C. A. (1987). Visual function, driving safety, and the elderly. *Ophthalmology*, 94(9), 1180-1188.
- Keltner, J. L. & Johnson, C. A. (1980). Mass visual screening in a driving population. *Ophthalmology*, 87, 785-790.
- Khandekar, R., Mohammed, A. J. & Raisi, A. A. (2007) Prevalence and causes of blindness and low vision; Before and five years after VISION 2020 initiatives in Oman: A review, *Ophthalmic Epidemiology*, 14(1), 9-15.
- Klaver, C. C. W., Wolfs, R. C. W., Vingerling, J. R., Hofman, A. & de Jong, P. T. V. M. (1998). Age-specific prevalence and causes of blindness and visual impairment in an older population: The Rotterdam Study. *Archives of Ophthalmology*, 116, 653-658.
- Klaver, C. C. W., Wolfs, R. C. W., Assink, J. J. M., van Duijn, C. M., Hofman, A. & de Jong, P. T. V. M. (1998). Genetic risk of age-related maculopathy: Population-based familial aggregation study. *Archives of Ophthalmology*, 116(12), 1646-1651.
- Klein, B. E. K. (2002). Cataract surgery and motor vehicle crashes-proceed with caution. *JAMA*, 288(7), 885-886.
- Klein, R., Klein, B. E. K. & Cruikshanks, K. J. (1999). The prevalence of age-related maculopathy by geographic region and ethnicity. *Progress in Retinal and Eye Research*, 18(3), 371-389.

- Klein, B. E. K., Klein, R. & Linton, K. L. P. (1992a). Prevalence of age-related lens opacities in a population. *Ophthalmology*, *99*, 546-552.
- Klein, B. E. K., Klein, R. & Linton, K. L. P. (1992b). Prevalence of age-related maculopathy: The Beaver Dam Eye Study. *Ophthalmology*, *99*, 933-943.
- Klein, R., Klein, B. E. K. & Moss, S. E. (1989). The Wisconsin Epidemiological Study of Diabetic Retinopathy: a review. *Diabetes & Metabolism Reviews*, *5*, 559-570.
- Kooijman, A., Melis-Dankers, B., Peli, E., Brouwer, W., Pijnakker, P., Van Delden, G., Van Pluuren, E., Van Iddekinge, B., Derksen, P., Busscher, R., Bredewoud, R., Van Rosmalen, J., Postema, F., Wanders, I., De Vries, J. & Witvliet, J. (2008) The Introduction of Bioptic Driving in The Netherlands. *Visual Impairment Research*, *10*, 1-16.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Lamble, D., Summala, H., & Hyvärinen, L. (2002). Driving performance of drivers with impaired central visual field acuity. *Accident Analysis & Prevention*, *34*, 711-716.
- Lighthouse International (1998). *Causes of vision impairment*. Retrieved May 13, 2003, from http://www.lighthouse.org/vision_impairment_causes.htm
- Lövsund, P., Hedin, A., & Törnros, J. (1991). Effects of driving performance of visual field defects: a driving simulator study. *Accident Analysis & Prevention*, *23*(4), 331-342.
- Lyman, J. M., McGwin, G. Jr. & Sims, R. V. (2001). Factors related to driving difficulty and habits in older drivers. *Accident Analysis & Prevention*, *33*, 413-421.
- Mäntyjärvi, M. & Tuppurainen, K. (1999). Cataract in traffic. *Graefe's Archive of Clinical & Experimental Ophthalmology*, *237*, 278-282.
- Marottoli, R. A., Richardson, E. D., Stowe, M. H., Miller, E. G., Brass, L. M., Cooney, L. M. & Tinetti, M. E. (1998). Development of a test battery to identify older drivers at-risk for self-reported adverse driving events. *Journal of the American Geriatrics Society*, *46*(5), 562-568.
- Marrington, S. A., Horswill, M. S. & Wood, J. M. (2008) The effect of simulated cataracts on drivers' hazard perception ability. *Optometry and Vision Science*, *85*, 1121-7.
- Massof, R. W., Deremeik, J. T., Park, W. L. & Grover, L. L. (2007) Self-reported importance and difficulty of driving in a low-vision clinic population. *Investigative ophthalmology & visual science*, *48*, 4955-62.
- McCarty, C. A., Nanjan, M. B. & Taylor, H. R. (2000). Operated and unoperated cataract in Australia. *Clinical and Experimental Ophthalmology*, *28*, 77-82.

- McCloskey, L. W., Koepsell, T. D., Wolf, M. E. & Buchner, D. M. (1994). Motor vehicle collision injuries and sensory impairments of older drivers. *Age & Aging*, 23(4), 267-273.
- McGwin, G., Jr., Mays, A., Joiner, W., DeCarlo, D. K., McNeal, S. & Owsley, C. (2004) Is Glaucoma Associated with Motor Vehicle Collision Involvement and Driving Avoidance? *Investigative Ophthalmology and Visual Science*, 45, 3934-3939.
- McGwin, G. Jr., Sims, R. V., Pulley, L. & Roseman, J. M. (2000). Relations among chronic medical conditions, medications, and automobile crashes in the elderly: A population-based case-control study. *American Journal of Epidemiology*, 152(5), 424-431.
- McGwin, G., Jr., Xie, A., Mays, A., Joiner, W., DeCarlo, D. K., Hall, T. A. & Owsley, C. (2005) Visual Field Defects and the Risk of Motor Vehicle Collisions among Patients with Glaucoma. *Investigative Ophthalmology and Visual Science*, 46, 4437-4441.
- McKay, R., McCarty, C. A. & Taylor, H. R. (2000). Diabetic retinopathy in Victoria, Australia: the Visual Impairment Project. *British Journal of Ophthalmology*, 84, 865-870.
- McKnight A. J, Shinar D, & Hilburn B. (1991). The visual and driving performance of monocular and binocular heavy-duty truck drivers. *Accident Analysis & Prevention*, 23(4), 225-227.
- Melis-Dankers, B. J. M., Kooijman, A. C., Brouwer, W. H., Busscher, R. B., Bredewoud, R. A., Derksen, P. H., Amersfoort, A., Ijsseldijk, M. A. M., van Delden, G. W., Grotenhuis, T. H. P. A. & Witvliet, J. M. D. (2008) A Demonstration Project on Driving with Reduced Visual Acuity and a Bioptic Telescope System in the Netherlands. *Visual Impairment Research*, 10, 7-22.
- Mitchell, P., Smith, W., Attebo, K. & Wang, J. J. (1995). Prevalence of age-related maculopathy in Australia: The Blue Mountains Eye Study. *Ophthalmology*, 102, 205-210.
- Mitchell, P., Smith, W., Wang, J. J. & Attebo, K. (1998). Prevalence of diabetic retinopathy in an older community. *Ophthalmology*, 105, 406-411.
- Mönestam, E., Lundquist, B. & Wachtmeister, L. (2005) Visual function and car driving: longitudinal results 5 years after cataract surgery in a population. *British Journal of Ophthalmology*, 89, 459-463.
- Mönestam, E. & Lundqvist, B. (2006) Long-time results and associations between subjective visual difficulties with car driving and objective visual function 5 years after cataract surgery. *Journal of Cataract & Refractive Surgery*, 32, 50-55.
- Mönestam, E. & Wachtmeister, L. (1997). Impact of cataract surgery on car driving: a population based study in Sweden. *British Journal of Ophthalmology*, 81(1), 16-22.

- Montgomery, G. (2003). *Color blindness: more prevalent among males*. A report from the Howard Hughes Medical Institute. Retrieved May 29, 2003, from <http://www.hhmi.org/senses/b130.html>
- Myers, R. S., Ball, K., Kalina, T. D., Roth, D. L. & Goode, K. T. (2000). Relation of useful field of view and other screening tests to on-road driving performance. *Perceptual & Motor Skills*, 91(1), 279-290.
- Nathan, D. M., Singer, D. E., Godine, J. E., Harrington, C. H. & Perlmutter, L. C. (1986). Retinopathy in older type II diabetics: association with glucose control. *Diabetes*, 35, 797-801.
- National Health Medical Research Council (1997). *Management of diabetic retinopathy*. NHMRC, Canberra.
- North, R. V. (1985). The relationship between the extent of visual field and driving performance: a review. *Ophthalmic & Physiological Optics*, 5, 205-210.
- Owsley, C., Ball, K., McGwin, G. Jr., Sloane, M. E., Roenker, D. L., White, M. F. & Overley, E. T. (1998). Visual processing impairment and risk of motor vehicle crash among older adults. *JAMA*, 279(4), 1083-1088.
- Owsley, C., Ball, K., Sloane, M. E., Roenker, D. L. & Bruni, J. R. (1991). Visual/cognitive correlates of vehicle accidents in older drivers. *Psychology & Aging*, 6(3), 403-415.
- Owsley, C. & McGwin, G. Jr. (1999). Vision impairment and driving. *Survey of Ophthalmology*, 43(6), 535-550.
- Owsley, C., McGwin, G. Jr. & Ball, K. (1998). Vision impairment, eye disease, and injurious motor vehicle crashes in the elderly. *Ophthalmic Epidemiology*, 5(2), 101-113.
- Owsley, C., McGwin, G., Phillips, J. M., McNeal, S. F. & Stalvey, B. T. (2004) Impact of an educational program on the safety of high-risk, visually impaired, older drivers. *American Journal of Preventive Medicine*, 26, 222-229.
- Owsley, C., McGwin, G. Jr., Sloane, M. E., Wells, J., Stalvey, B. T. & Gauthreaux, S. (2002). Impact of cataract surgery on motor vehicle crash involvement of older adults. *JAMA*, 288, 841-849.
- Owsley, C., Stalvey, B. T. & Phillips, J. M. (2003). The efficacy of an educational intervention in promoting self-regulation among high-risk older drivers. *Accident Analysis & Prevention*, 35(3), 393-400.
- Owsley, C., Stalvey, B., Wells, J. & Sloane, M. E. (1999). Older drivers and cataract: driving habits and crash risk. *Journals of Gerontology Series A-Biological Sciences & Medical Sciences*, 54(4), 203-211.
- Owsley, C., Stalvey, B., Wells, J., Sloane, M. E. & McGwin, G. Jr. (2001). Visual risk factors for crash involvement in older drivers with cataract. *Archives of Ophthalmology*, 119(6), 881-887.

- Pambakian, A. L. M., Wooding, D. S., Patel, N., Morland, A. B., Kennard, C., Mannan, S. K. (2000). Scanning the visual world: a study of patients with homonymous hemianopia. *Journal of Neurology, Neurosurgery & Psychiatry*, 69(6), 751-759.
- Parrish, R. K., Gedde, S. J., Scott, I. U., Feuer, W. J., Schiffman, J. C., Mangione, C. M. & Montenegro-Piniella, A. (1997). Visual function and quality of life among participants with glaucoma. *Archives of Ophthalmology*, 115, 1447-1455.
- Pelli, D.G., Robson, J.G. & Wilkins, A.J. (1988). The design of a new letter chart for measuring contrast sensitivity. *Clinical Vision Science*, 2, 187-199.
- Phillipov, G., Alimat, A., Phillips, P. J. & Drew, A. C. (1995). Screening for diabetic retinopathy. *Medical Journal of Australia*, 162, 518-520.
- Quigley, H. A. (1996). Number of people with glaucoma worldwide. *British Journal of Ophthalmology*, 80(5), 389-393.
- Ramrattan, R.S., Wolfs, R.C., Panda-Jonas, J.B., Bakker, D., Pols, H.A., Hofman, A. & de Jong, P.T. (2001). *Archives of Ophthalmology*, 119(12), 1788-1794.
- Racette, L. & Casson, E. J. (2005) The impact of visual field loss on driving performance: Evidence from on-road driving assessments, *Optometry and Vision Science*, 82(8) 668-674.
- Regan, D. (1993). Detection and discrimination of spatial form in participants with eye or visual pathway disorders. In R. M. Shapely & D. M. Lam (Ed), Contrast sensitivity. *Proceedings of the Retina Research Foundation Symposia*, Vol. 5. (pp.309-337).
- Resnikoff, S., Pascolini, D., Mariotti S. P. & Pokharel, G. P. (2008) Global magnitude of visual impairment caused by uncorrected refractive errors in 2004, *Bulletin of the World Health Organisation*, 86(1), 1-80.
- Reuben, D. R., Silliman, R. A. & Traines, M. (1988). The aging driver: medicine, policy, and ethics. *Journal of the American Geriatrics Society*, 36, 1135-1142.
- Rochtchina, E. & Mitchell, P. (2000). Projected number of Australians with glaucoma in 2000 and 2030. *Clinical & Experimental Ophthalmology*, 28(3), 146-148.
- Royal College of Ophthalmologists (2003). *Understanding nystagmus*. Retrieved June 6, 2003, from <http://www.rcophth.ac.uk/publications/nystagmus.html>.
- Rubin, B. E. K., Adamsons, I. A. & Stark, W. J. (1993). Comparison of acuity, contrast sensitivity, and disability glare before and after cataract surgery. *Archives of Ophthalmology*, 111, 56-61.
- Rubin, G. S., Roche, K. B., Prasada-Rao, P. & Fried, L. P. (1994). Visual impairment and disability in older adults. *Clinical Vision Sciences*, 71, 750-756.
- Rubin, G. S., Ng, E. S. W., Bandeen-Roche, K., Keyl, P. M., Freeman, E. E., West, S. K. & the, S. E. E. P. T. (2007) A Prospective, Population-Based Study of the Role of Visual Impairment in Motor Vehicle Crashes among Older Drivers: The SEE Study. *Investigative Ophthalmology and Visual Science*, 48, 1483-1491.

- Salzberg, P. & Moffat, J. (1998). *The Washington State Department of Licensing Special Exam Program: An Evaluation*. Washington Traffic Safety Commission. Olympia, WA.
- Satariano, W. A., MacLeod, K. E., Cohn, T. E. & Ragland, D. R. (2004) Problems with vision associated with limitations or avoidance of driving in older populations. *The journals of gerontology. Series B, Psychological sciences and social sciences*, 59, S281-6.
- Schiff, W. & Arnone, W. (1995). Perceiving and driving: Where parallel roads meet. In P. Hancock, J. Flach, J. Caird, & K. Vicente (Eds.), *Local applications of the ecological approach to human-machine systems* (pp. 1-35). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Scilley, K., Jackson, G. R., Cideciyan, A. V., Maguire, M. G., Jacobson, S. G. & Owsley, C. (2002). Early age-related maculopathy and self-reported visual difficulty in daily life. *Ophthalmology*, 109(7), 1235-1242.
- Sewell, J.H. (1983). Color counts too! *Academic Therapy*, 81, 329-37.
- Shinar, D. & Scheiber, F. (1991). Visual requirements for safety and mobility of older drivers. *Human Factors*, 33, 507-519.
- Silveira, S., Jolly, R., Heard, R., Clunas N. & Kay, L. (2007) Current licensing authority standards for peripheral visual field and safe on-road senior aged automobile driving performance. *Clinical and Experimental Ophthalmology*, 35, 612-620.
- Sims, R. V., McGwin, G. Jr., Allman, R. M., Ball, K. & Owsley, C. (2000). Exploratory study of incident vehicle crashes among older drivers. *Journals of Gerontology Series A - Biological Sciences & Medical Sciences*, 55(1), M22-27.
- Sivak, M., (1996). The information that drivers use: is it indeed 90 % visual? *Perception*, 25, 1081-1089.
- Sokol, S., Moskowitz, A., Skarf, B., Evans, R., Molitch, M. & Senior, B. (1985). Contrast sensitivity in diabetics with and without background retinopathy. *Archives of Ophthalmology*, 103, 51-54.
- Stalvey, B. T. & Owsley, C. (2000). Self-perceptions and current practices of high-risk older drivers: Implications for driver safety. *Journal of Health Psychology*, 5(4), 441-456.
- Steward, J. M. & Cole, B. L. (1989). What do colour defectives say about everyday tasks? *Optometry & Vision Science*, 66, 288-295.
- Stewart, R. B., Moore, M. T., Marks, R. G., May, F. E. & Hale, W. E. (1993). Driving cessation and accidents in the elderly; an analysis of symptoms, diseases, cognitive function and medications. *AAA Foundation for Traffic Safety*, Washington, DC.
- Stutts, J. C. (1998). Do older drivers with visual and cognitive impairments drive less? *Journal of the American Geriatrics Society*, 46, 854-861.

- Swedish Road Administration. (1998). *Swedish Road Administration Statute Book*. Sweden.
- Szlyk, J. P., Alexander, K. R., Severing, K. & Fishman, G. A. (1992). Assessment of driving performance in participants with retinitis pigmentosa. *Archives of Ophthalmology*, *110*(12), 1709-1713.
- Szlyk, J. P., Fishman, G. A., Severing, K., Alexander, K. R. & Viana, M. (1993). Evaluation of driving performance in participants with juvenile macular dystrophies. *Archives of Ophthalmology*, *111*(2), 207-212.
- Szlyk, J., Mahler, C., Seiple, W., Edward, D. & Wilensky, J. (2005a) Driving performance of glaucoma patients correlates with peripheral visual field loss. *Journal of Glaucoma*, *14*, 145-150.
- Szlyk, J. P., Mahler, C. L., Seiple, W., Vajaranant, T. S., Blair, N. P. & Shahidi, M. (2004) Relationship of retinal structural and clinical vision parameters to driving performance of diabetic retinopathy patients. *Journal of Rehabilitation Research and Development*, *41*, 347-58.
- Szlyk, J. P., Pizzimenti, C. E., Fishman, G. A., Kelsch, R., Wetzel, L. C., Kagan, S. & Ho, K. (1995). A comparison of driving in older subjects with and without age-related macular degeneration. *Archives of Ophthalmology*, *113*(8), 207-212.
- Szlyk, J. P., Seiple, W., Stelmack, J. & McMahon, T. (2005b) Use of prisms for navigation and driving in hemianopic patients. *Ophthalmic & Physiological Optics*, *25*, 128-35.
- Szlyk, J. P., Taglia, D. P., Paliga, J., Edward, D. P. & Wilensky, J. T. (2002). Driving performance in participants with mild to moderate glaucomatous clinical vision changes. *Journal of Rehabilitation Research and Development*, *39*(4), 467-481.
- Tagarelli, A., Piro, A., Tagarelli, G., Lantieri, P. B., Risso, D. & Olivieri, R. L. (2004) Colour blindness in everyday life and car driving. *Acta Ophthalmologica Scandinavica*, *82*, 436-42.
- Talbot, E. M. & Perkins, A. (1998). The benefit of second eye cataract surgery. *Eye*, *12*, 983-989.
- Tant, M. L. M., Brouwer, W. H., Cornelissen, F. W., & Kooijman, A. C. (2002). Driving and visuospatial performance in people with hemianopia. *Neuropsychological Rehabilitation*, *12*(5), 419-437.
- Taylor, H. R. (2001) *Eye care for the community*, Report prepared for the Centre for Eye Research Australia, Melbourne, Australia.
- Taylor, H. R., Keeffe, J. & Mitchell, P. (2004). *Clear Insight. The Economic Impact and Cost of Vision Loss in Australia*. Access Economics Report for the Centre for Eye Research Australia and the Eye Research Australia Foundation.
- Taylor, H. R., Livingston, P., Stanislavsky, Y., & McCarty, C. (1997). Visual impairment in Australia: distance visual acuity, near visio, and visual field

- findings of the Melbourne Visual Impairment Project. *American Journal of Ophthalmology*, 123, 328-337.
- Tielsch, J. M., Sommer, A., Katz, J., Royall, R. M., Quigley, H. A. & Javitt, J. (1991). Racial variation in the prevalence of primary open angle glaucoma: the Baltimore Eye Survey. *JAMA*, 266(3), 369-374.
- Troutbeck, R. & Wood, J. M. (1994). Effect of restriction of vision on driving performance. *Journal of Transport Engineering*, 120(5), 737-752.
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.
- VanNewkirk, M. R., Nanjan, M. B., Wang, J. J., Mitchell, P., Taylor, H. R. & McCarty, C. A. (2000). The prevalence of age-related maculopathy. *Ophthalmology*, 107(8), 1593-1600.
- VanNewkirk, M. R., Weih, L., McCarty, C. A. & Taylor, H. R. (2001). Cause-specific prevalence of bilateral visual impairment in Victoria, Australia. *Ophthalmology*, 108(5), 960-967.
- Verriest, G., Naubauer, O., Marre, M. & Uvijls, A. (1980). New investigations concerning the relationships between congenital colour vision defects and road traffic security. *International Ophthalmology*, 2, 887-889.
- Vingrys, A. J. & Cole, B. L. (1988). Are colour vision standards justified in the transport industry? *Ophthalmic & Physiological Optics*, 8, 257-274.
- Vingrys, A. J. (2002). The case against protan drivers holding professional driving licenses. *Clinical & Experimental Optometry*, 85(1), 46-48.
- Vision Australia Foundation (2002a). *Statistics on eye health in Australia*. Retrieved May 27, 2003, from <http://www.visionaustralia.org.au/index>
- Vision Australia Foundation (2002b). *Diabetic Retinopathy*. Retrieved May 27, 2003, from <http://www.visionaustralia.org.au/index>
- Vision Channel (2003a). *Refractive Errors*. Retrieved May 28, 2003, from www.visionchannel.net/refractiveerrors/
- Vision Channel (2003b). *Retinitis Pigmentosa*. Retrieved May 29, 2003, from www.visionchannel.net/retinitis/
- Wang, J.J., Forans, S. & Mitchell, P. (2000). Age-specific prevalence and causes of bilateral and unilateral visual impairment in older Australians: The Blue Mountains Eye Study. *Clinical and Experimental Ophthalmology*, 28, 268-273.
- Wolfs, R. C. W., Klaver, C. C. W., Ramrattan, R. S., van Duijn, C. M., Hofman, A. & de Jong, P. T. V. M. (1998). Genetic risk of primary open angle glaucoma: Population-based familial aggregation study. *Archives of Ophthalmology*, 116(12), 1640-1645.

- Wolfe, R. J. B. (2002). Protans and driving safety. *Clinical & Experimental Optometry*, 85(6), 399-400.
- Wood, J. M. (1999). How do visual status and age impact on driving performance as measures on a closed circuit driving track? *Ophthalmic & Physiological Optics*, 19(1), 34-40.
- Wood, J. M. & Carberry, T. P. (2006) Bilateral cataract surgery and driving performance. *British Journal of Ophthalmology*, 90, 1277-1280.
- Wood, J. M., Dique, T. & Troutbeck, R. (1993). The effect of artificial visual impairment on functional visual fields and driving performance. *Clinical Vision Sciences*, 8, 563-575.
- Wood, J. M., & Mallon, K. (2001). Comparison of driving performance of young and old drivers (with and without visual impairment) measured during in-traffic conditions. *Optometry & Vision Science*, 78(5), 343-349.
- Wood, J. M., McGwin, G., Elgin, J., Vaphiades, M. S., Braswell, R. A., DeCarlo, D. K., Kline, L. B., Meek, C., Cearcey, K. & Owsley, C. (2009) On-road driving performance by persons with hemianopia and quadrantanopia, *Investigative Ophthalmology and Visual Science*, In press.
- Wood, J. M. & Owens, D. A. (2005) Standard measures of visual acuity do not predict drivers' recognition performance under day or night conditions, *Optometry and Vision Science*, 82(8) 698-705.
- Wood, J. M. & Troutbeck, R. (1994). Effect of visual impairment on driving. *Human Factors*, 36(3), 476-487.
- Wood, J. M. & Troutbeck, R. J. (1996). Visual impairment and driving. In A. G. Gale et al. (Eds), *Vision in Vehicles - V* (pp. 229-308). Amsterdam: North Holland.
- World Health Organisation (WHO) (2009) *Visual impairment and blindness: Factsheet*, Available online: www.who.int/mediacentre/factsheets/fs282/en/index.html, accessed on 16/06/09.
- Zihl, J. (1995). Visual scanning behaviour in patients with homonymous hemianopia. *Neuropsychologia*, 33(3), 287-303.

CHAPTER 4 SUMMARY AND RECOMMENDATIONS

4.1 SUMMARY OF RISK ASSOCIATED WITH MEDICAL CONDITIONS

Using the evidence from studies identified in the 1980 to May 2003 and post-May 2003 to June 2009 review periods presented in Chapter 3, a rating system was applied to estimate risk associated with all conditions of interest. The ratings were based on evidence for crash involvement only, since this was deemed to be of more direct relevance in assessing crash risk than both citations and driving performance. The rating provided a means of identifying those conditions that presented the greatest risk. Three authors rated the risk for each medical condition independently and in the few cases where there were discrepancies, a consensus was reached.

Three main levels of ratings were applied:

- Higher (H):
 - Slightly high (*): RR: 1.1-2.0
 - Moderately high (**): RR: 2.1-5.0
 - Considerably high (***) RR: 5.0+;
- No difference (N) (nominally RRs \approx 1);
- Inconclusive (I) (evidence highly equivocal or no evidence).

Information on post-treatment risk was also considered. Evidence relating to treatment was relatively sparse and for the majority of conditions, no evidence could be found for post-treatment crash risk. In some studies that did report crash data during or after treatment, serious methodological issues generally precluded the separate identification of treatment effects from the effects attributable to the disorder itself. Understandably, the comparison of treatment groups with non-treatment groups is difficult for obvious ethical reasons. Post-treatment crash risk was rated as:

- Higher (H);
- Lower (L);
- Inconclusive (I) (evidence highly equivocal or no evidence).

Table 48 summarises the risk ratings for all conditions of interest.

Table 48 Summary of crash risk associated with specific medical conditions and post-treatment risk based on evidence from pre- and post-May 2003 reviews

Condition	Prevalence %	Overall Crash Risk	Post-Treatment Crash Risk
ALCOHOL ABUSE & DRUG DEPENDENCE	0.82% ¹	H**	I
CARDIOVASCULAR DISORDERS	17.9% ²	H* ₋ **	I
CVA (Stroke, heart and vascular diseases)	3.83% ³	I	I
COGNITIVE			

Condition	Prevalence %	Overall Crash Risk	Post-Treatment Crash Risk
IMPAIRMENT			
Dementia	1.0% ⁴	H**	I
TBI	.03 -.25% ⁵	I	I
DIABETES MELLITUS	3.55% ⁶	H*	I
Severe hypoglycaemia		I	I
Hypoglycaemic unawareness		I	I
EPILEPSY	0.7% ⁷	H*_*_*_*	I
MUSCULOSKELETAL DISORDERS		H*	I
Rheumatoid arthritis	2.4% ⁸	H* (females)	I
Osteoarthritis	7.8% ⁹	H*	I
Spinal cord injury		I	I
Amputation		I	I
NEUROLOGICAL DISORDERS (as a group)		H*	I
Parkinson's disease	0.1% ¹⁰	I	I
Multiple Sclerosis	0.03% ¹¹	H**	I
Cerebral Palsy	0.2% of live births ¹²	I	I
Spina Bifida	0.09% of live births ¹³	I	I
PSYCHIATRIC DISORDERS (as a group)	25% (at some time in life; includes substance abuse) ¹⁴	H*_*_*	H (Benzodiazepine) (method. problem distinguishing risk assoc. with drug vs. condition)
Schizophrenia	1% ¹⁵	H**	I
Depression	3-5% ¹⁶	I	H (Antidepressants-tricyclics) (method. problem distinguishing risk assoc. with drug vs. condition)
Anxiety disorders	4.91% ¹⁷	I	I
Personality disorders	1-10% ¹⁸	I	I
ADHD	3-7% ¹⁹ (school-aged children)	I	I
RESPIRATORY DISORDERS	7-16% ²⁰	H*	I

Condition	Prevalence %	Overall Crash Risk	Post-Treatment Crash Risk
Sleep apnoea	.3-7.5% ²¹	H**_***	L (CPAP lowers the crash risk to that of controls without the condition)
Narcolepsy	0.06% ²²	I	I
VESTIBULAR DISORDERS		I	I
VISION CONDITIONS (as a group)		N-H*	I
Cataracts	2-5% (40-49 yr olds) ²³	H**	L (Cataract surgery lowers crash risk compared with un-treated; inconclusive compared with no-cataract)
Glaucoma	0.43% (75+ yrs) ²⁴	H*_**	I
Age-Related Macular Degeneration	13%(90+yrs) ²⁵	I	I
Diabetic Retinopathy	1.8% ²⁶	I	I
Retinitis Pigmentosa	1% (40 yrs+) ²⁷	I	I
Defective Colour vision	7-8% (male) ²⁸	N	N/A
Monocular vision	1.8 – 5% (40+yrs) ²⁹	I	I
Corneal pathology	1.1 – 1.9% (40+ yrs) ³⁰	I	I
Nystagmus	.1% ³¹	I	I
Reduced Visual acuity	6/12 or worse: 0.6% - 3.6% (40-59 yrs) 1.1% - 8.2% (60-69yrs) 5.4%- 20.1% (70-79yrs) 26.3% - 52.2%. (80+ years) ³²	I	I
Dynamic visual acuity	Unknown	I	I
Visual field defects	3% (16-60yrs) ³³ 7% (60-65yrs) 13% (65+yrs)	I	I
Reduced Contrast sensitivity	Unknown	H*	I

1. ABS 2004-2005 survey

2. ABS 2004-2005 survey

3. ABS 2004-2005 survey

4. AIHW, 2006

5. Fortune & Wen, 1999 (world)

6. ABS 2004-2005 survey

7. ABS 2004-2005 survey

8. ABS 2004-2005 survey

9. ABS 2004-2005 survey

10. Mehta et al. (2007) Australia

11. WHO, 2008

12. Sanner, 1996; cited in Falkmer & Gregersen, 2000

18. Martin & Sugarman, 1997

19. Barkley, 1997

20. WHO, 2008

21. Sharma et al. (2008) world

22. Medical standards (2003)

23. ABS 2004-2005 survey

24. ABS 2004-2005 survey

25. Taylor et al. 2004

26. NHMRC, 1997

27. CERA, 2004

28. Montgomery, 2003

29. Cedrone et al., 2006

Condition	Prevalence %	Overall Crash Risk	Post-Treatment Crash Risk
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13. Spina Bifida Association of America, 2003
14. WHO, 2004
15. WHO, 2004
16. ABS 2004-2005 survey
17. ABS 2004-2005 survey

30. Khandekar et al., 2000
31. Dobbs, 2001
32. Wang, Forans & Mitchell, 2000
33. Johnson & Keltner, 1983

Risk ratings were based primarily on available Relative Risk data, with Odds Ratios and other statistical comparisons used for supportive evidence. It should be noted that comparisons across risk ratings are not strictly valid because each condition was compared with a different control group. For example, those studies examining the risk of crashes amongst drivers with cataracts generally recruited older participants (both cases and controls) because the condition is more prevalent in the older population. On the other hand, comparisons involving multiple sclerosis were more likely to include drivers older than 25 years and younger than 50 years, an age group whose risk of crashes is generally lower than older drivers. Hence, differences in control groups prevent a direct comparison of risk ratios. Nevertheless, what can be established is that the conditions that were rated high risk (moderately to considerably elevated) had substantially elevated crash risks compared with their relevant controls.

Based on the evidence from studies reviewed in Chapter 3, and taking into account new evidence from studies published post-May 2003, eight conditions were found to have an elevated risk of crash involvement compared with their relevant control group. Specifically, these were the same conditions as those high-risk conditions identified in the pre-May 2003 review: alcohol abuse and dependence, dementia, epilepsy, multiple sclerosis, psychiatric disorders (considered as a group), schizophrenia, sleep apnoea and cataracts.

The quality of evidence for elevated crash risk was modest. Table 49 summarises the quality of evidence, including additional supportive evidence identified in the post-May 2003 review .

Table 49 Summary of quality of evidence for high-risk medical conditions based on evidence from pre- and post-May 2003 review periods

Condition	Review period	Quality of Evidence
Alcohol Abuse and Dependence ⁷	Pre-May 2003	Main evidence from 3 studies: <ul style="list-style-type: none"> - 1 population-based case-control study with unrestricted drivers, minimal bias (no exposure measure) H*-.** - 1 study of adequate sample size, some bias (self-reported crashes), no exposure measure H** - 1 study of adequate sample size, some bias (self-reported crashes), adjusted for exposure H*-.**
	Post-May 2004	Not reviewed in the post-May 2003 report update
Dementia	Pre-May 2003	12 studies; main evidence from 1 large sample case-control study (minimal bias) (H**)

7

Condition	Review period	Quality of Evidence
		Supportive evidence: - 2 strong studies (RR not quantifiable)
	Post-May 2003	2 studies supporting previous conclusion
Epilepsy	Pre-May 2003	1 strong, study, minimal bias (H***) Supportive evidence: - 3 studies H*
	Post-May 2003	3 studies supporting previous conclusion
Multiple sclerosis	Pre-May 2003	1 study, adequate sample size, minimal bias (H**)
	Post-May 2003	No new evidence
Psychiatric disorders (as a group)	Pre-May 2003	1 large sample, population-based case-control, minimal bias (no exposure measure) (H*-.**); Supportive evidence: - 1 strong study for tricyclic antidepressant users, minimal bias, valid crash measure, not possible to distinguish role of treatment from disorder per se (H**) - 2 strong studies for benzodiazepine users, minimal bias, valid crash measure, not possible to distinguish role of treatment from disorder per se (H*-.**)
	Post-May 2003	1 study which does not add to previous conclusion
Schizophrenia	Pre-May 2003	1 study, adequate sample size, some bias (self-reported crashes), adjusted for exposure (H**)
	Post-May 2003	No new evidence
Sleep apnoea	Pre-May 2003	1 strong study, adequate sample size, minimal bias, valid crash measure, corrected for exposure (H**) Supportive evidence: - 2 studies, adequate sample size, some bias (self-report crash measure) (H***) <u>Treatment effect:</u> 3 studies: - 1 large sample study, valid crash measures, reversal of crash risk post treatment - 1 study, adequate sample size (self-report crash measure) 75% reduction pre-post treatment effect; - 1 small sample, valid crash measure, weak evidence for reversal of risk post-treatment compared with population;
	Post-May 2003	Conclusion unchanged: supportive evidence from 1 case control study of adequate size and 3 studies with weaker evidence.
Cataract	Pre-May 2003	Evidence from 1 strong study, adequate sample size, valid crash measure, corrected for exposure (H**) Supportive evidence : - one weak study (sampling bias towards more impaired participants), valid crash measures (H*) <u>Treatment effect:</u> - 1 study adequate sample size, case-control, 50% reduction in crash risk post-surgery compared with untreated.
	Post-May 2003	Conclusion unchanged: supportive evidence from 1 study

Only one study used a population-based, prospective design (see Skurveit et al., 2008, section 3.5). Generally, the best studies that were used to establish the risk ratings employed retrospective, case-control design, with adequate sample size, reliable diagnosis of condition and valid measures of crash involvement. However, most had some potential bias, such as recruitment of non-representative cases (including severity, type of disorder, time since onset), and lack of control of confounding variables such as comorbidity and driving exposure. A summary of quality of evidence for specific medical conditions.

4.2 HIGH-RISK MEDICAL CONDITIONS AND RISK FOR OTHER KNOWN HIGH-RISK GROUPS

It is instructive to examine the risk associated with medical conditions in the context of other road user high-risk groups. Table 50 summarises the data for high-risk medical conditions and other groups. Well-established risk estimates for drink driving show that a blood alcohol concentration (BAC) of 0.05 results in a relative risk of crash involvement of around 1.5 (Borkenstein et al., 1964). The relative risk increases as the severity of crash increases. Drivers with a BAC of 0.05 or more had at least five times the risk of being killed in a crash, relative to drivers with a nil BAC (Maycock, 1997). Another high-risk group of drivers is the under-20 year olds. Recent figures from Australian crash data showed that drivers younger than 20 years have around 9 times the relative risk of serious casualty crash involvement per distance travelled compared to drivers aged 40-54 years (safest age group).

An important factor not yet discussed is the prevalence of the condition amongst licensed drivers. This is informative because it enables us to estimate the size of the problem. However, for many conditions, specific prevalence data for the driving population are difficult to establish. Data from the large population-based study (State of Utah, U.S.A.) by Vernon et al. (2002) are available for psychiatric conditions and epilepsy. A substantial discrepancy can be seen between population prevalence and prevalence amongst licensed drivers. The lower prevalence figures reported for drivers in Utah may be due to under-reporting of medical conditions (because of fear of losing driving privileges) and a tendency for only those with more serious conditions to be reported to the authority. In a longitudinal study in Finland Tervo and colleagues (2008) found that out of 522 fatal motor vehicle accidents recorded between 1995-2005, 54 were due to a medical condition. The main cause of death was heart attack experienced by drivers who had a prior history of heart disease.

Table 50 Comparison of crash risk associated with medical conditions and other high-risk groups based on evidence from pre- and post-May 2003 review periods

Condition	Prevalence ¹ %	Overall Crash Risk
Alcohol Abuse and Dependence	0.82%	H**
Dementia	1.0%	H**
Epilepsy	0.7%	H*_***
Multiple Sclerosis	0.03%	H**
Psychiatric disorders	0.4% of licensed drivers, (Vernon et al., 2002) 25% (total population; at some time in life; includes substance abuse)	H*_**
Schizophrenia	1%	H**
Sleep apnoea	.3-7.5%	H**_***
Cataracts	2-5% (40-49 yr olds)	H**
Young drivers: <20 yrs (compared with drivers aged 40-55 years)	5-6% (NSW; USA)	H***
BAC: (0.05)	0.4% (Victoria)	H* (all crashes) H**(fatal crashes)

As summarised in Table 50, the risk across all groups is moderately to highly elevated (with the exception of BAC of 0.05 for all crashes). Thus, on the basis of prevalence data, if young drivers and older drivers are considered as a unit, the risk overwhelms all of the risks associated with medical conditions combined, to such an extent that the impact of any single medical condition might seem minor. Nevertheless, the high risk associated with some medical conditions cannot be discounted. Hansotia and Broste (1991) make the point that a ban on all young male drivers would have a significant impact in enhancing road safety. However, they conclude that this would represent an unacceptable restriction of individual freedom. Clearly, decision-making about driving restrictions for high-risk groups is complex and politically and legally sensitive. The decision-making process should incorporate a range of relevant issues and should weigh up individual needs for mobility, while maintaining an acceptable level of safety for all road users. In the case of drivers with medical conditions, important factors might also include the driver's capacity for rehabilitation, as well as their lifestyle and mobility needs (proximity to services; access to alternative transport, etc). These management factors are considered in more detail below.

¹ Prevalence for medical conditions is expressed in terms of population data (sources as cited in Chapter 3) and where available, per licensed drivers (Vernon et al., 2000). In the case of high-risk groups, prevalence is expressed as a proportion of licenced drivers (sources: for New South Wales (NSW): Roads and Traffic Authority, 2001; for USA: Federal Highway Administration, 2003; for Victoria: Victoria Police, Traffic Alcohol Section, 2002).

4.3 MANAGEMENT OF CRASH RISK AMONGST DRIVERS WITH MEDICAL CONDITIONS

In Chapter 1, a useful framework was presented for understanding the relationship between medical, functional impairment and crash risk (OECD, 2001, p. 25). This framework is summarized as follows:

- Determine which health and medical conditions have functional impairments that affect driving;
- If there are functional impairments, determine whether they necessarily lead to increased crash risk;
- If there is substantial injury risk, identify and implement countermeasures (treatment, rehabilitation or other compensatory strategies) to reduce the risk;
- If no effective countermeasures exist, decision needs to be made regarding continuation of driving.

In the following section the implications of this risk management approach are considered. In Figure 1 below, two high-risk medical conditions are discussed, highlighting different management outcomes.

In sleep apnoea, a number of functional impairments have been identified including excessive daytime sleepiness, depression, difficulty concentrating and impaired cognitive ability. These impairments are likely to impact on aspects of driving by causing inattention, drowsiness while driving and poor judgements. The review of evidence showed a moderately to considerably elevated crash risk. However, there is also strong evidence for a reduction of risk with Continuous Positive Airways Pressure (CPAP) treatment. Indeed the risk reduced to levels equal to drivers without sleep apnoea. Therefore, it would appear to be entirely appropriate to allow drivers with sleep apnoea undergoing CPAP treatment to continue to drive.

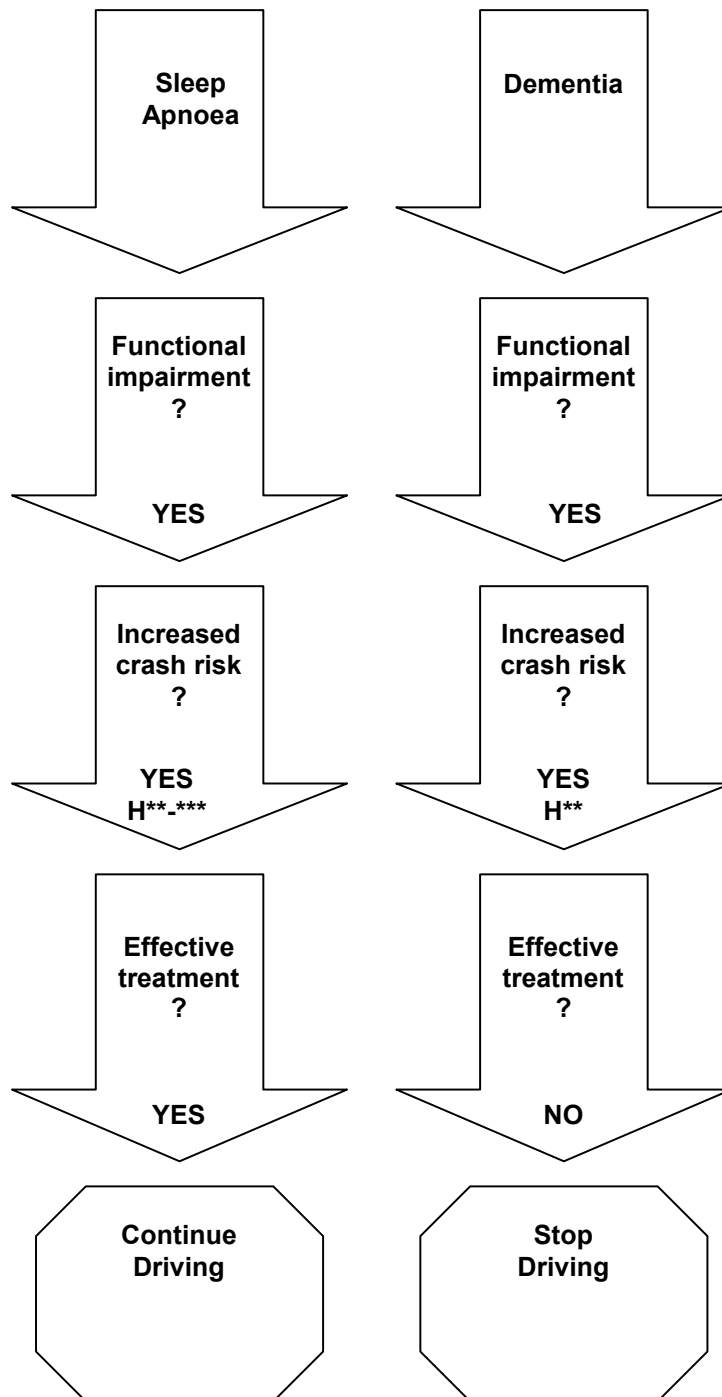


Figure 1 Management of risk for selected medical conditions

In the case of dementia, there are also a number of functional impairments across a wide range of cognitive areas that are required for safe driving; for example, difficulties with decision-making, planning, attention and memory. The review of evidence showed a moderately elevated crash risk for drivers with a clinical diagnosis of dementia. However, in contrast to sleep apnoea, there is no evidence for an effective treatment in lowering crash risk. Based on this evidence, a conservative decision would be to remove licensing privileges. Such an approach also seems prudent given the likelihood of lack of insight associated with this disorder. As discussed in Chapter 3, the position taken by Canada fits with this conservative approach. This, however, does not take into account the severity of impairment. The approach adopted by other jurisdictions (UK, Utah,

Australia, New Zealand) is to recommend the provision of a conditional licence including a regular review, which is sensible given the progressive nature of the disorder and wide individual differences in the nature and extent of cognitive decline. Sweden's position also takes into account the severity of impairment and in the case of mild impairment, driving may be permitted if skills are judged to be adequate. The problem with this, however, is that there are inadequate tools to make this assessment.

Cessation of driving has important implications for both the individual and society. For example, for an individual who is no longer able to drive, other transport options become increasingly important in order to maintain mobility and independence. Alternative transport options might include public transport, car passenger, walking, cycling and scooters. However, these options may not necessarily be available, accessible or safer than driving. The OECD (2001) report on ageing and transport showed that the crash injury risks associated with walking and cycling are not insignificant (see Figure 2). Moreover, these data do not include other accident risks that might occur while walking or using public transport, such as falls.

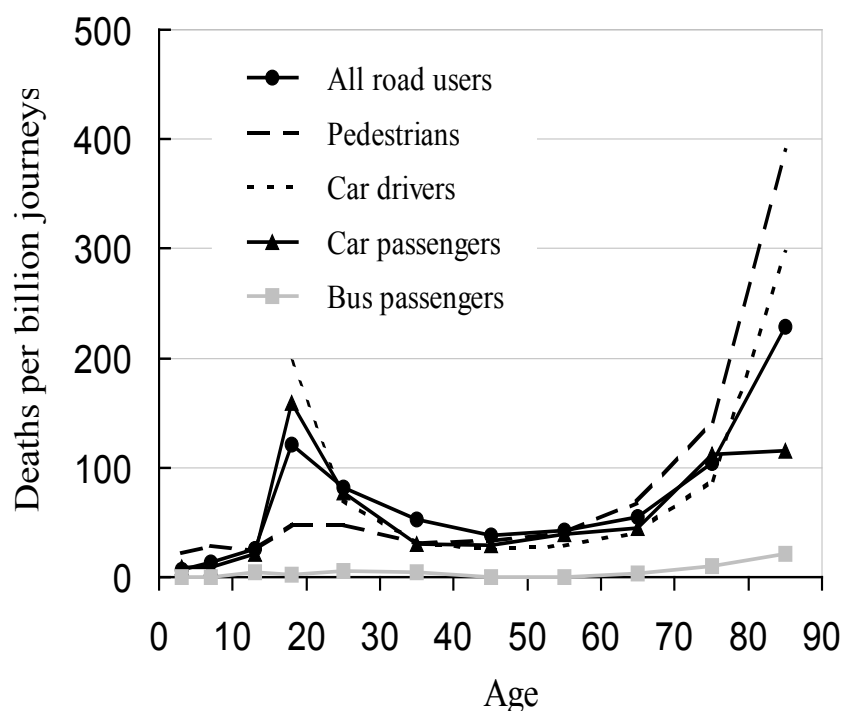


Figure 2 Fatality rate per journey, U.K., 1998 (from OECD, 2001, p. 46)

4.4 CONCLUSION AND RECOMMENDATIONS

This review presents evidence in relation to medical conditions and driver risk. One of the most striking observations that can be made is that the quality and quantity of evidence does not do justice to the serious consequences associated with motor vehicle crashes. Methodological limitations were evident in most studies, including a lack of standardisation of inclusion criteria for medical conditions and unreliable measures of crash involvement (i.e. self-report).

The review of evidence for crash risk was compared with guidelines regarding fitness to drive from selected jurisdictions. These comparisons revealed a number of

inconsistencies across the jurisdictions and in some cases the guidelines did not appear to reflect the available evidence for crash risk.

Information about management of medical conditions was also reviewed. Intuitively, it would be reasonable to expect that well-established treatments might reduce risk. Indeed, the treatment of sleep apnoea was shown to significantly reduce crash risk to the same level as those without the condition. However, for most conditions there was extremely limited evidence for this in the literature. In the case of treatments for psychiatric disorders, benzodiazepines and antidepressants (tricyclics) were found to increase risk. Other methods of management include special licensing conditions or restrictions. For example:

- A driver diagnosed with visual impairment may drive only when wearing corrective lenses;
- A driver with diabetes may be required to take insulin on a regular basis;
- A driver who has lost a limb may only drive whilst wearing a prosthesis;
- In addition, self-regulation is also a potentially useful management approach. For example drivers with epilepsy are often advised not to drive if they are tired and to avoid precipitating factors such as emotional or physical stress. However, self-regulation is only likely to be effective if the driver has insight into the factors that place them at risk. In the case of dementia and psychiatric illness, the capacity for insight is likely to be impaired. Moreover, there is little evidence that specifically addresses the benefit of self-regulation in reducing crash risk.

In the light of the available information presented in this review, a number of recommendations can be made:

- Develop reliable methods of identifying and referring those who are potentially at-risk as a result of medical conditions;
- Promote public awareness, particularly amongst the driving population, about the known crash risks and effective management for particular medical conditions or impairments - this is important particularly because most jurisdictions are reliant on self-referral or voluntary reporting of medical conditions and hence, the onus is on the driver to determine whether they have a condition that affects their driving;
- Improve knowledge within the health professions about the known crash risks and effective management for particular medical conditions or impairments;
- Develop and implement valid and standardised assessments to identify the functional impairments of drivers with specific medical conditions at an increased risk;
- Review licensing guidelines for fitness-to-drive in the light of all available evidence regarding crash risk;

- Investigate the capacity for the use of medical technologies for more effective monitoring of driver risk (e.g., in-vehicle blood glucose monitoring system);
- Investigate the capacity for the use of adaptive technologies and intelligent transport systems (ITS) to enhance driver safety (e.g., safe following distance devices and rear collision warning and avoidance systems);
- Review of chronic alcohol and drug abuse in a broader framework, including drugs and alcohol abuse and high level dose/usage;
- Advance high-quality scientific knowledge linking medical conditions and crash risk in order to improve the evidence base for formulating policy about licensing and fitness to drive;
- Educate drivers with non-insulin dependent diabetes about hypoglycaemic awareness.

Future research

It is recommended that a cooperative international approach to future research be adopted. This should take the form of a large scale, prospective study (or group of studies) using a population-based or case-control design to investigate the following:

- underlying impairments or mechanisms that contribute to crash risk for particular medical conditions;
- the effectiveness of treatments, rehabilitation and countermeasures, including ITS and other advanced technologies, in reducing crash risk;
- the effectiveness of mandatory and voluntary reporting and assessment of medical conditions;
- risk and risk reduction strategies for targeted high-risk sub-groups, particularly with multiple medical conditions prevalent in the ageing population;
- the social, health and economic consequences of licensing restrictions in at-risk populations.

References

- Austroroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia*. Sydney, Australia.
- Borkenstein, R.F. et al (1964). *The role of the drinking driver in traffic crashes*. Dept. of Police Administration, Indiana University, Bloomington, Indiana, USA.
- Canadian Medical Association. (2000). *Determining Medical Fitness to Drive: A Guide for Physicians*. 6th Ed. Ottawa, Canada.
- Drivers Medical Group DVLA Swansea. (2003). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive*. Swansea, UK.
- Federal Highway Administration (2003). Accessed on 14th of July, 2003 from <http://www.fhwa.dot.gov/ohim/>
- Fildes, B., Fitzharris, M, Charlton J. & Pronk, N. (2001). *Older driver safety – A challenge for Sweden's 'Vision Zero'*. Paper presented to the 24th Australasian Transport Research Forum Conference, Hobart, Australia.
- Hansotia, P. & Broste, S.K. (1991). The effect of epilepsy or diabetes mellitus on the risk of automobile accidents. *New England Journal of Medicine*, 324, 22-26.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive*. Wellington, New Zealand.
- Maycock G (1997). *Drinking and driving in Great Britain – A review*. Report 232, TRL Ltd., Crowthorne, UK.
- OECD (2001). *Ageing and transport. Mobility needs and safety issues*. Paris: OECD Publications.
- Roads and Traffic Authority (2001). Accessed on 14th of July, 2003 from http://www.rta.nsw.gov.au/publicationsstatisticsforms/downloads/stats_annual_2001.pdf
- Swedish Road Administration. (1998). *Swedish Road Administration Statute Book*. Sweden.
- Utah State Driver License Medical Advisory Board. (1992). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.

APPENDIX A DETAILS OF LITERATURE SEARCH

The ARRB literature search request:

Requested articles dealing with “chronic illness and road accident involvement” and “managing the risk of injury within the road system resulting from chronic illness (or impairment of cognitive, sensory and physical abilities.”

The Key words included in this literature search were:

chronic illness; medications; functional ability/disability/impairment; driving/driving performance/ assessment of driving; crash risk; injury risk; education tools/resources; driver training/rehabilitation; community awareness; medical assessment; licensing; licence restrictions.

Medline and PsychLit Searches

The Medline, PsychLit and other relevant databases were searched using combinations of the following key words and phrases for accident involvement and medical conditions:

Search topic	Key words and phrases
Accident Involvement:	accident risk, automobile accidents, crash risk, driving patterns, driving performance, driving restrictions, driving safety, driving tasks, driver training, rehabilitation, fitness to drive, motor vehicle accidents, motor-vehicle related injury, risk of injury, traffic accidents, traffic safety
Alcohol:	use disorders, problems, abuse and dependence, Korsakoff’s syndrome
Cardiovascular:	conditions, diseases, disorders, heart – disease, attack, implantable cardioverter defibrillators, severe angina, tachycardia, ventricular fibrillation, arrhythmia, syncope
Cerebrovascular:	accidents, disease, damage, stroke, transient ischemic attacks, cerebrovascular accident
Cognitive:	ability, impairment, mild cognitive impairment (also see Neurological)
Epilepsy:	Epilepsy, seizure disorders
Medical:	chronic illness, co-morbidity, conditions, impairment

Medications:	Anticonvulsants, antidepressants, antihistamines, antipsychotic, benzodiazepines, insulin, neuroleptics, polypharmacy, prescribed, psychotropic, sedatives, tranquillisers, side effects
Metabolic:	condition, disorders, diabetes, hypoglycaemia, hypothyroidism, low blood sugar, pituitary, parathyroid,
Musculoskeletal:	conditions, impairment, arthritis, osteoporosis, motor conditions, physical impairment, back pain, lower back pain, spinal injuries, rheumatoid arthritis, osteoarthritis
Neurological:	conditions, impairment, cerebral palsy, Huntington's disease, multiple sclerosis, Parkinson's disease, spina bifida Alzheimer's disease, brain-injury, brain impairment, dementia, vascular dementia, head injury, closed head injury, traumatic brain injury, acquired brain injury
Psychiatric:	disorders, anxiety disorders, attention deficit, ADHD, depression, mood disorders, personality disorders, schizophrenia
Respiratory:	conditions, disease, disorders, failure, asthma, bronchitis, chronic obstructive lung disease, COPD, emphysema
Sleep:	conditions, disorders, apnoea, narcolepsy; obstructive sleep apnoea
Vestibular:	conditions, disorders, vestibular, balance Ménière's disease, vertigo, benign paroxysmal positional vertigo
Vision:	acuity, cataracts, colour vision, contrast sensitivity, deficits, standards diabetic retinopathy, diplopia, eye disease, field of vision, visual field loss, glaucoma, macular dystrophies/degeneration, monocular vision, night myopia, nystagmus, ocular conditions, peripheral vision, retinitis pigmentosa, visual attention

APPENDIX B REVIEW CHECKLIST

Paper Title:		
Disease/Condition:		Check if 'Yes'
Road Safety Evidence Investigated:	<input type="checkbox"/> Crashes <input type="checkbox"/> Citations <input type="checkbox"/> Driving Performance	
Type Of study	<ul style="list-style-type: none"> • Case Control <input type="checkbox"/> • Cross-Sectional <input type="checkbox"/> • Cohort <input type="checkbox"/> • Review <input type="checkbox"/> • Other - describe <input type="checkbox"/> 	
Adequate Definition of Condition	Was there an adequate method of defining/detecting the condition/disease? Consider potential bias in defining/detecting the condition (e.g. if the condition is diabetes, was it detected from medical records, self-report, medical assessment. Accuracy of medical records or self-report are likely to be lower than assessment by medical practitioner)	<input type="checkbox"/>
Adequate definition of key outcome measures	Was there an adequate method of assessing the outcome? Consider any potential bias associated with this method (e.g. if driving infringements are the outcome, are these self-reported or from police records –as self-reported infringements may be less reliable.)	<input type="checkbox"/>
Study Design	Was the method of recruitment adequate to attract an unbiased sample? (e.g. if all vision impaired participants were recruited from newspaper ads, they may be a healthier group than if recruited from a low vision clinic; and each of these examples is likely to be less representative than a random sample of the population of all vision impaired 'medical review cases' in a jurisdiction)	<input type="checkbox"/>
	Were controls adequately recruited & matched? (see if the paper has a table to compare case & control characteristics, such as mean age, gender, etc)	<input type="checkbox"/>
	Were sample numbers large (n>30)?	<input type="checkbox"/>
	Are data sources adequately described & an indication of data quality provided?	<input type="checkbox"/>
	Was there adequate control of other potential confounds (e.g. exposure) ?	<input type="checkbox"/>
Results	Are the analyses/ statistical techniques explained & justified?	<input type="checkbox"/>
	Is there a precise statement of the association between illness & outcome (Odds ratio, risk ratio, hazard ratio)?	<input type="checkbox"/>
	If so, rate the risk (e.g. RR of controls) for the condition: <div style="text-align: right;"> Slightly Higher 1.1-2.0 <input type="checkbox"/> Mod Higher 2.1-5.0 <input type="checkbox"/> Considerably Higher 5.0+ <input type="checkbox"/> No difference <input type="checkbox"/> Lower <input type="checkbox"/> </div>	
	If no precise statement of the association, please briefly outline findings	
Discussion	Are interpretation of results & conclusions made by authors clear & justifiable?	<input type="checkbox"/>
	Are the limitations of the study addressed in the interpretation?	<input type="checkbox"/>
Rate the empirical strength of study:	Weak 1	Adequate 2
		Strong 3

Comments:

APPENDIX C FITNESS TO DRIVE GUIDELINES

The licensing requirements for six countries reviewed in this report were Canada, Australia, UK, USA (the state of Utah), New Zealand and Sweden. The specific criteria that must be met by drivers of private vehicles with particular medical conditions are set out in the Licensing Guidelines tables in the relevant sections of Chapter 3 together with commercial driving guidelines pertaining to cardiac disorders, diabetes, epilepsy and sleep apnoea. Guidelines for commercial drivers with all other conditions are presented in the tables below.

Private and Commercial Licences

The licensing guidelines of each of the countries surveyed for this literature review draw a distinction between the stringency of licensing criteria for private and commercial licences. Due to the higher danger potential to the public and the environment that driving commercial vehicles carries (eg transporting dangerous goods, larger freight loads and passengers for hire, and the longer periods spent driving as well as the size and weight of the vehicle), drivers of these vehicles are required to undergo a more rigorous assessment prior to licensing. In comparison, the daily driving habits of a private licence holder may only involve driving to the shops or work and, hence, a less rigorous approach is indicated.

In addition, some countries allow scope to apply differing degrees of latitude when licensing both commercial and private drivers, depending on the driving circumstances. For example, in Australia, a farmer may require a commercial licence to drive heavy vehicles on the farm, rather than on the open road. Such a scenario would not present a grave threat to public safety and less strict criteria could be applied (Austroads, 2006). In addition, “grandfather rights” (less stringent test standards) apply to those who have held commercial licences prior to certain dates in the UK, Sweden and Utah. Conversely, a more rigorous approach may be called for. For example, in the UK, the House of Commons Transport Select Committee has recommended that all people seeking a taxi licence should be required to pass a medical exam. Similarly, the relevant authorities for commercial, taxi, police, ambulance and health service vehicle drivers may impose licensing and medical requirements over and above that set out in the guidelines (DVLA, 2008).

Classification of Private and Commercial Vehicles

Australia

Private vehicle licences are issued for:

- Cars that are 4.5 tonnes or less and in which there are no more than 11 adult passengers;
- Vehicles classified as “light rigid” and whose gross vehicular mass (GVM) is over 4.5 tonnes and up to 8 tonnes, or that seats more than 11 adult passengers, or has a trailer that is no more than 9 tonnes; and
- Motorbikes or motor trikes.

Commercial licences are required for the following classes of vehicles:

- Any of the above types of vehicles listed under private licences where drivers apply to transport public passengers for hire or reward, or carry bulk dangerous goods;
- Medium rigid (2 axle) or heavy rigid (3 or more axles) vehicles that have a gross vehicular mass that exceeds 8 tonnes;
- Heavy combination vehicle – “prime mover & single semi-trailer or a rigid vehicle plus trailer greater than 9 tonnes GVM and any unladen converter dolly trailer” (Austroads, 2006, p12); and
- Multi-combination vehicle i.e. “a heavy combination vehicle with more than 1 trailer” (Austroads, 2006, p12).

Sweden

Licences are classified into three different groups: Group 1, 2 and 3. Group 1 is the equivalent of the private vehicular class and Group 2 and 3 relate to commercial vehicles.

Group 1 comprises:

- Private motorcars, light lorries, light trailers, cross-country vehicles, or “class I power-driven equipment in tow” (SRA, 1998, p4). Included in this group are trailers attached to any of the aforementioned vehicles;
- Tractors; and
- Motorcycles – light (max 125cc) or heavy.

Group 2 consists of:

- Heavy lorries. May tow any light trailer; and
- Trailers – no weight or number restrictions.

Group 3 covers licences for:

- Buses or buses with trailers (irrespective of number and weight). and
- Taxis.

New Zealand

Vehicles are classified into 6 different categories, again with a distinction being made between private (Classes 1 and 4) and commercial licences (Classes 2, 3, 5 and 6):

Private or lower licence classes are:

- All private cars including tractors and combination vehicles with a gross laden weight of up to 4,500 kg, and forklifts that weigh up to 1,500kg (Class 1); and
- Motorcycles, mopeds, and all-terrain vehicles (Class 6).

Commercial licences are required for the following vehicle types:

- Any rigid vehicle or tractor that has a gross laden weight that exceeds 4,500kg; and
- All combination vehicles ranging with a gross combined weight over 4,500kgs and over 25,000kgs towing a light trailer.

Canada

Licences are divided into 6 different classes in Canada.

Private licences are covered by Classes 5 and 6:

- Any motor vehicle or small truck. If a vehicle is being towed, it must not weigh more than 4,600 kg and must not drive an ambulance, a taxicab or a bus or to pull a semi-trailer (Class 5).
- Motorcycle, motor scooter, or minibike (class 6).

Commercial licences comprise Classes 1 to 4:

- Classes 1- 3 allow a vehicle of any type or size to be driven. Classes 1, 2 and 4 allow passengers to be aboard. Classes 2 and 3 prohibit a semi-trailer to be towed; and
- Class 4- taxis, buses that carry 24 or fewer passengers, and all emergency vehicles such as ambulances, fire-trucks and police cars.

In some instances a Class 5 licence may also be included in the commercial licence grouping, based on the amount driven (see CMA, 2006, p 5).

UK

Private licence holders are classified as Group 1, which includes:

- Motor cars (Category B); and
- Motorcycles.

Group 2 refers to commercial licence holders:

- Large lorries (Category C);
- Medium size lorry with a weight ranging from 3.5 to 7.5 tonnes (Category C1);
- Buses (Category D); and
- Minibus with between 9 to 16 seats, but not for hire or reward (Category D1).

Volunteer drivers may drive a minibus of up to 16 seats without having to obtain category D1 entitlement.

Private licences are classified into 4 separate categories:

- Vehicle or combination of vehicles that have a gross vehicle weight of 26,001 or more. The vehicle being towed must weigh over 10,000 pounds (Class A).
- Single vehicle that has a gross vehicle weight of less than 26,001 pounds. May tow a vehicle that has a gross vehicle weight of 10,000 pounds or less, or a farm trailer that weighs 20,000 pounds or less. May drive a bus that seats up to 24 (Class B).
- Single vehicle that has a gross vehicle weight of less than 26,001 pounds. May tow a farm trailer that weighs 20,000 pounds or less (Class C).
- Motorcycle or moped (Class M).

Commercial licences are categorised into 3 groups with 5 different codes:

- Any combination of vehicles that have a combined gross vehicle weight of 26,001 or more. The vehicle being towed must weigh over 10,000 pounds (Class A).
- Any single vehicle with a gross vehicle weight of 26,001 or more. May tow a vehicle that has a gross vehicle weight of 10,000 pounds or less. Any vehicle that carries up to 24 people (Class B).
- Vehicle or combination of vehicles that carries 16 to 23 people or that transports hazardous materials (Class C).
- The 5 different codes authorise the driver to cover hazardous material, passengers, double and triple trailers, and tanks.

Types of licences (Conditional and Unconditional)

Within each of the two broad licence classes (private and commercial), drivers may qualify for either an unconditional or conditional licence. An unconditional licence places no restrictions on the driver except those required by the specific licence class. However, some drivers may not meet the criteria required to obtain an unconditional licence and must apply for a conditional licence that places certain restrictions or conditions on their driving. These types of licences are often sought due to medical disorders or disabilities that impair driving and may require the person to undergo medical assessment, driver assessment and/or notification to the relevant driver licensing authorities prior to being licensed. Conditional licences commonly require that the medical conditions be successfully managed by treatment, or modifications be made to either the drivers' car or person, which will allow licence holders to drive without incurring unacceptable risk levels either to themselves or to others. For example, a driver diagnosed with visual impairment may drive only when wearing corrective lenses or, in the case of night blindness, may drive during daylight hours only; or diabetics may be required to take insulin on a regular basis; or a person who has lost a limb may only drive whilst wearing a prosthesis. A restricted licence is granted subject to the driver abiding by its conditions. Thus, a conditional licence is issued on the basis that

the extra road safety risk that the person may pose due to a medical condition is of an acceptable size (Austroads, 2006).

Appeal rights

In New Zealand, Australia and the USA (Utah), drivers have the right to appeal any decision to refuse, revoke or restrict their licence. In New Zealand drivers may take their case to the District Court. In Canada, appeals are only possible in certain jurisdictions and drivers must make their case to the licensing authority. In Utah, USA drivers may make an appeal to the Medical Advisory Board panel within 10 days of being advised of a licensing decision, if this decision was reached without the convening of the panel. In Australia, provision is also made for drivers to have their licence status reconsidered if their medical conditions have cleared or improved. In such cases, the medical practitioner must notify the DLA in writing and the DLA will reconsider reinstating the licence.

Medico-legal issues

It is not the patient's GP or medical specialist who makes the decision whether a licence is to be refused, revoked or restricted. In Australia, New Zealand, Canada and the UK it is the Driver Licensing Authority (DLA) that makes the decision as to who may or may not drive. In the UK, it is the Secretary of State for Transport who makes this decision although, in practice, this responsibility falls to the DLA. In Utah, USA the Medical Advisory Board panel makes licensing decisions. However, in order to make these decisions the licensing authorities require medical reports and other driver assessment reports.

In Australia, New Zealand, the UK, some Canadian provinces and Utah, USA, it is the individual driver's legal responsibility to report his/her medical condition to the DLA. Should the individual refuse to take the necessary action and thus put lives at risk, then the responsibility may fall onto his/her doctor. These countries recognize that doctors may have a duty-of-care obligation to report these instances to the relevant licensing authorities, and may need to breach patient confidentiality to do so. Different provinces in Canada have either mandatory (nine provinces) or discretionary (three provinces) reporting of patients by their GPs. In those States with mandatory reporting responsibilities, GPs may be liable in a court of law for any subsequent crash involvement by the patient, should they renege on their duty.

Most of the countries surveyed have indemnity legislation in place should a medical practitioner need to report a patient who cannot or will not comply with the self-notification requirements. Specifically, all states in Australia (except Tasmania), all provinces in Canada (except British Columbia), New Zealand and the UK provide legal protection for GPs if they report patients who are medically unfit to drive. The law in several of these countries also places certain restrictions or requirements on the medical practitioners who do report their patients. For example, in New Zealand and the USA (Utah) there is also the stipulation that the medical practitioner must make the report in good faith. In the UK, the doctor is required to apprise the patient of his/her intention to notify DLA and must also advise the patient in writing after this has been done.

Other general factors to be considered by physicians when assessing fitness to drive

The exact medical criteria that drivers must meet to obtain licences are stated explicitly by each of the countries surveyed and are set out in the Licensing Guideline Tables that

follow. In addition to these, many of the countries also provide extra factors/requirements to be considered either in all cases or as a blanket requirement for individuals with any particular condition. These are described below.

The New Zealand licensing guidelines recognise the diverse nature of the symptoms of medical conditions and individuals' varying response to treatment. Therefore, it is possible for some of the assessment requirements to be modified to suit individual cases, usually with a supportive medical report. The guidelines also list a number of additional, general factors over and above those required for each specific medical condition that the GP is to consider when assessing fitness to drive. These are:

- The person's ability to drive safely;
- The MVC risk that might arise should the person experience a sudden onset of symptoms;
- The class of licence – private or commercial;
- Medication side effects and the likelihood of patient compliance with treatment;
- The driver's MVC history with particular emphasis on previous medically related crashes;
- The presence of other medical conditions; and
- The presence of other risk factors, for example, alcohol, smoking and family history.

The Australian guidelines emphasise that during assessment, the physician is to take into consideration the following:

- Licensing responsibility resides with the DLA, although the doctor provides medical advice to the DLA;

Where conditional licences are recommended, the GP is required to outline the unconditional licence inclusion criteria that the patient does not meet and any monitoring that may be necessary;

- The presence of multiple disabilities and their combined impact on driving;
- GPs are to consider the demands of the driving task as well as the medical condition when making recommendations to the DLA. For instance, will the driver merely be making excursions to the local shops or hauling freight long-distance?; and
- GPs must advise patients of the impact that the medical disorder has on driving ability and the patient's legal responsibility to inform DLA if it is a notifiable condition.

The Canadian guidelines, unlike those of the other five jurisdictions surveyed, state that driving a commercial vehicle requires greater physical stamina than that needed for driving a private vehicle. Physicians are, therefore, advised that "this group should be

expected to meet higher medical standards than private drivers” (CMA, 2006, p.5) when determining fitness to drive and the medical assessment standards must be adhered to.

The Swedish licensing guidelines state that all medical conditions are to be assessed from a “traffic safety point of view” (SNRA, 1998, p.5). However, when assessing risk, not only are the symptoms of the actual medical condition to be considered, but also the person’s individual circumstances.

Blanket requirements for specific medical conditions

Some countries have blanket licensing requirements or amendments for specific conditions or faculties that apply to all drivers, or to drivers with a particular medical disability or disorder. These are in addition to those set down in the tables of licensing guidelines that follow.

Vision

UK law requires a vehicle licence holder to have the ability to read a licence plate at a distance of 20 to 20.5 metres, with or without corrective lenses. If this requirement is not met, then licence revocation or refusal will result. People who have only partial sight are generally also considered unfit to drive, however, the criteria set out in the actual guidelines are to be used in determining driver fitness.

The Canadian guidelines recognise that some people, although not meeting the required standards for specific visual defects, may have learned to compensate for their disability to such an extent that they are able to drive safely. In these exceptional cases, a licence may be granted if it is supported by a report by an optometrist or ophthalmologist, the visual problem is stable and the person has a good driving record. Conversely, a driver might meet the visual requirements to obtain a licence but may not drive safely, in which case, it may be reasonable to issue a restricted licence only.

Epilepsy

According to Section 92 of the Road Traffic Act in the UK, epilepsy is described as a “prescribed disability” and, as such, represents a legal bar to driving. Therefore, for a person with epilepsy to obtain a licence, relevant conditions set down in the statutory regulations may need to be met first.

Diabetes

The Road Traffic Act in the UK lists insulin-treated diabetes as a “prospective disability”. This means that the medical condition is progressive and may eventually develop into a “prescribed disability” and, thus, become a legal bar to driving. Drivers with a “prospective disability” are required to undergo periodic medical reviews.

Head Injuries/Learning/Behavioural Disabilities

Guidelines in some US jurisdictions recommend that GPs take a very conservative approach when assessing fitness to drive for people who have sustained head injuries or have cognitive problems arising from behavioural or learning disabilities. Such an approach will allow for the times when the person’s ability may fluctuate.

Psychiatric Conditions

Guidelines in some US jurisdictions state that due to the nature of these illnesses, a person's driving history of crashes or traffic violations is a more valid indicator of future road safety risk than the current status of the illness. To assist with this assessment, the GP is referred to a phone number to obtain further information on individual driving records.

Hearing

In Sweden, and some jurisdictions in the USA, all commercial licence holders are required to pass a hearing test. In the USA, the driver must be able to hear a forced whisper made from a distance of 5 feet in the ear with the most acute hearing, with or without a hearing aid. A hearing loss of more than 65 decibels (dB) will result in licence refusal or revocation. In Sweden, commercial drivers must be able to hear a normal speaking voice at a distance of 5 metres, with or without a hearing aid. This criterion has been set so that drivers can communicate with both passengers and other road users. The Canadian guidelines stipulate that the only classes of licence that require a specific level of hearing are Classes 2 and 4 (passenger-carrying vehicles and emergency response vehicles). These drivers must have "a corrected hearing loss of no more than 40dB averaged at 500, 1000 and 2000Hz and a correct word recognition score of at least 50-60%" (CMA, 2006, p.51). This requirement has been set so that these drivers can hear what their passengers are saying without taking their eyes off the road.

Cardiovascular Conditions

The Canadian guidelines specify that when determining fitness to drive for those with cardiovascular disease the risk of "sudden incapacitation" resulting from loss of consciousness, death etc (CMA, 2006, p.56) must be carefully considered. For commercial drivers the acceptable risk level is placed at 1% per annum.

References

- Austrroads. (2006). *Assessing Fitness to Drive for Commercial and Private Vehicle Drivers. Medical Standards for Licensing and Clinical Management Guidelines. A Resource for Health Professionals in Australia..*Sydney, Australia.
- Canadian Medical Association. (2006). *Determining Medical Fitness to Drive: A Guide for Physicians.* 7th Ed. Ottawa, Canada.
- Drivers Medical Group DVLA Swansea. (2008). *For Medical Practitioners. At a Glance Guide to the Current Medical Standards of Fitness to Drive.* Swansea, UK.
- Land Transport Safety Authority. (2002). *Medical Aspects of Fitness to Drive.* Wellington, New Zealand.
- Pakola, S.J., Dinges, D.F., & Pack, A.I. (1995). Review of regulations and guidelines for commercial and noncommercial drivers with sleep apnea and narcolepsy. *Sleep*, 18(9), 787-796.

Swedish National Road Administration. (1998). *Swedish National Road Administration Statute Book*. Sweden.

Texas Department of Public Safety. (2000). *Texas Drivers Handbook*. Texas, USA.

Utah State Driver License Medical Advisory Board. (2006). *State of Utah Functional Ability in Driving: Guidelines and Standards for Health Care Professionals*. Utah, USA.

APPENDIX D FITNESS TO DRIVE GUIDELINES FOR COMMERCIAL DRIVERS

Table C.1 Commerical licensing guidelines for drivers with alcohol dependency and alcohol abuse

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Alcoholism/ Alcohol Dependency	<p><i>Diagnosis of Dependency:</i> Desist from driving all vehicles.</p> <p>Driving may resume if following conditions are met: 1. Must complete recognised treatment program. 2. Must be monitored by a specialist 3. Must remain alcohol free for 12 months.</p>	<p>Person may not hold an unconditional licence.</p> <p>A conditional licence may be issued if person: 1. Has abstained from drinking for a “substantial period”. 2. Has insight into the condition. 3. Complies with treatment. 4. Has no end organ damage that may impair driving.</p> <p>Periodic review required.</p>	<p>Licence denial if alcoholism has been present in the previous 3 years.</p> <p>Restoration of licence may occur if “satisfactory” medical reports obtained from the person’s GP.</p> <p>May also require independent verification via medical + blood tests organised by DVLA + support/ referral to appropriate consultants.</p>	<p><i>Chronic Alcohol Use:</i> No driving if there is impairment of motor +/- or intellectual functions.</p> <p><i>Alcohol use causing intermittent functional impairment outside of work + driving hours:</i> May not drive.</p>	<p>In general, no restrictions on driving.</p> <p><i>Exceptions:</i> Dependency has affected the person’s cognitive, perceptual + motor skills so that the ability to drive safely is impaired.</p> <p>Therefore, person to desist from driving until “effective treatment has been established” (p141).</p> <p>In addition, care needs to be taken as alcohol may exacerbate other existing medical conditions eg epilepsy.</p>	<p><i>Diagnosed Dependency:</i> Licence denied or revoked.</p> <p>Licence may be reinstated after a sober lifestyle has been demonstrated for a period of 6 – 24 months + continued sobriety is likely. For institutionalised people, the sobriety period commences after release.</p> <p>Sobriety to be confirmed via regular medical assessment + laboratory tests.</p> <p><i>Exceptions:</i> Person may retain their licence if there is evidence of other favourable circumstances eg very good progress in a rehabilitation program.</p> <p>In all cases above, 3 reviews are required – the first at 6 months, then 1 year + finally 2 years.</p>
Misuse of Alcohol	<i>Drink-driving:</i> Desist from driving	<i>History of alcohol abuse:</i>	<i>Persistent alcohol misuse:</i>	<i>Alcohol use without adverse personal or</i>	Not specifically addressed.	<i>Gross Drunk Driving Conviction:</i>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
	<p>all vehicles.</p> <p>Driving may resume if following conditions are met:</p> <ol style="list-style-type: none"> 1. Must complete recognised treatment program. 2. Must be monitored by a specialist 3. Must remain alcohol free for 12 months. 	<p>Confirmed by biochemical results.</p> <p>Person may not hold an unconditional licence.</p> <p>A conditional licence may be issued if person:</p> <ol style="list-style-type: none"> 1. Has abstained from drinking for a “substantial period”. 2. Has insight into the condition. 3. Complies with treatment. 4. Has no end organ damage that may impair driving. <p>Periodic review required.</p>	<p>Licence refused or revoked upon medical diagnosis or confirmation via blood markers.</p> <p>May resume driving after person has abstained or controlled his/her drinking for a period of at least 12 months.</p> <p>It is recommended that the person obtain advice/ counselling during the non-driving period.</p>	<p><i>social outcomes in the past 1 to 3 months:</i> May not drive.</p> <p><i>Alcohol use without adverse personal or social outcomes in the past 6 months:</i> May hold a restricted commercial licence. Restricted to intrastate driving + subject to review by the Medical Advisory Board.</p>		<ol style="list-style-type: none"> 1. A statement that complies with the Driving Licences Ordinance is to be obtained two months prior to applying for a licence. 2. A medical certificate shall be obtained from a medical specialist + contain pertinent information on person’s alcohol habits, laboratory test results + if necessary, psychological test results. 3. The person is subject to a monitoring period of 3 – 6 months, during which time 2 laboratory tests are to be conducted. <p>A review is to undertaken at 6 months and then 12 months. Further reviews may be required on a case-by-case basis.</p>
Alcohol-Related Disorders	<p><i>Alcohol-induced seizures:</i> Desist from driving all vehicles.</p> <p>Driving may resume if following conditions are met:</p> <ol style="list-style-type: none"> 1. Must complete recognised treatment program. 	<p><i>Epilepsy:</i> Epileptics who are frequently intoxicated are considered unfit to drive.</p> <p><i>Diabetes:</i> Insulin-dependent diabetics may forget to take medication + maintain food</p>	<p><i>Seizures:</i> Single seizure: Licence denial or revocation for 5 years following the seizure.</p> <p>Licence may be restored if person:</p> <ol style="list-style-type: none"> 1. Has not taken anti-convulsant drugs for 5 years 	<p><i>Impairment of motor +/or intellectual functions.</i> No driving.</p>	<p><i>Seizures:</i> Care is recommended about the possibility of alcohol exacerbating other existing medical conditions eg epilepsy.</p>	Not specifically addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
	2. Must be monitored by a specialist 3. Must remain alcohol free for 12 months.	balance whilst intoxicated. It is recommended that they desist from driving. <i>End Organ Effects:</i> End organ effects that impair driving must not be present. If they are present, the person does not meet the requirements for a conditional licence.	2. Has abstained from alcohol if history of alcoholism. 3. Has no “underlying cerebral structural abnormality” (p28). 4. Has been assessed by a neurologist + addiction specialist. Multiple seizures: person must comply with the epilepsy licensing requirements. <i>Impairment from Alcohol-Induced Cirrhosis/Psychosis</i> Recommendation that licence be revoked or denied.			

** No distinction is made in this manual between alcohol use/misuse/abuse. Distinction is made in terms of functional ability only.

Table C.2 Commerical licensing guidelines for drivers with cardiovascular disease

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Acute Myocardial Infarct (AMI) (Exercise tolerance measured on Bruce Treadmill Test (BTT) or similar & exercise ECG).	Desist from driving for 3 months after hospital discharge for AMI.	<i>Uncomplicated:</i> Desist from driving for minimum of 3 months after AMI. A conditional licence may be issued if: 1. Person has history of minimal symptoms. 2. Exhibits exercise tolerance on BTT (or similar) of more than 9 minutes for males & more than 6 minutes for females. 3. Does not have severe ischaemia 4. Has an ejection fraction of 40% or more. Periodic review required.	Disqualified from driving for minimum of 6 weeks. Re-licence if person can pass exercise test requirements & no other disqualifying condition is present.	Desist from driving for 6 weeks or until the condition has stabilised. May hold an unrestricted licence if the person: 1. Has symptoms only with strenuous exercise 1 year following surgery. Yearly review required. A treadmill stress test should be repeated at 6 months.	<i>Uncomplicated:</i> Desist from driving for minimum of 4 weeks. Resume driving only on specialist's advice.	Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving. Assessments are to take account of the causes, development & treatment of the disease. Licence denial in cases of ischaemic heart disease if any of the following are present: 1. Tested work capacity is well below expected normal limits. 2. The left heart ventricle is operating at reduced capacity, with cardiac failure symptoms. 3. Serious paroxysmal arrhythmia occurs. 4. Angina occurs whilst at rest or with emotional arousal. 5. Angiography shows "haemodynamically significant stenosis of the coronary blood vessels" (p9). A licence may still be issued if a favourable

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
						medical report is obtained & the person poses a negligible safety risk to traffic.
Angina Pectoris	<p><i>Stable angina:</i> No restrictions and no waiting period.</p> <p><i>Unstable angina:</i> 48 hours after percutaneous coronary intervention if performed during hospital stay and 7 days after discharge if PCI not performed during initial stay.</p>	<p>Licence restriction if person has Angina.</p> <p>A conditional licence may be issued if: 1. Exhibits exercise tolerance on BTT (or similar) of more than 9 minutes for males & more than 6 minutes for females & there is no evidence of myocardial ischaemia. 2. If myocardial ischaemia is detected, person must exhibit “lumen diameter reduction of <70% in a major coronary branch or <50% in left main coronary artery”.</p> <p>Periodic review required.</p>	<p>Licence revoked if symptoms continue when driver is at rest or with emotion.</p> <p>May re-licence if symptom-free for 6 weeks & person can pass exercise test requirements & no other disqualifying condition is present.</p>	<p><i>For any diagnosis of heart disease:</i></p> <p>No licence restrictions if: 1. Complete recovery. 2. Symptom-free or no undue symptoms with normal activity. 3. Slight physical limitations with mild exertion.</p> <p>Periodic review required.</p>	<p>Desist from driving if symptoms occur at rest or with minimal exertion despite medical treatment.</p>	<p>Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Licence denial if angina occurs whilst at rest or with emotional arousal.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p> <p>A licence may still be issued if a favourable medical report is obtained & the person poses a negligible safety risk to traffic.</p>
Heart Failure	Disqualified from driving if mild to moderate functional limitation. (NYHA	May not hold an unconditional licence.	Licence disqualification if the person has symptoms.	<p><i>For any diagnosis of heart disease:</i></p> <p>No licence</p>	Person generally considered unfit to drive.	Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>Class III or Class IV)</p> <p>Person may drive if no functional limitations and an ejection fraction \geq 35% .</p>	<p>A conditional licence may be issued if the person:</p> <ol style="list-style-type: none"> 1. Exhibits exercise tolerance on BTT (or similar) of more than 9 minutes for males & more than 6 minutes for females 2. "Has an ejection fraction of 40% or over" (p44). 3. The underlying reason for heart failure is "considered". <p>Annual review required.</p>	<p>May be re-licensed if:</p> <ol style="list-style-type: none"> 1. LVEF is good i.e. greater than 0.4. 2. No other conditions are present that would make the person unfit to drive. <p>Exercise or functional testing may be required depending on the cause of heart failure.</p>	<p>restrictions if:</p> <ol style="list-style-type: none"> 1. Complete recovery. 2. Symptom-free or no undue symptoms with normal activity. 3. Slight physical limitations with mild exertion. <p>Periodic review required.</p>	<p>A conditional licence may be issued if supported by a specialist's report.</p>	<p>in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p> <p>Licence denial in cases of ischaemic heart disease if any of the following are present:</p> <ol style="list-style-type: none"> 1. Tested work capacity is well below expected normal limits. 2. The left heart ventricle is operating at reduced capacity, with cardiac failure symptoms. 3. Serious paroxysmal arrhythmia occurs. 4. Angina occurs whilst at rest or with emotional arousal. 5. Angiography shows "haemodynamically significant stenosis of the coronary blood vessels" (p9). <p>A licence may still be issued if a favourable medical report is obtained & the person poses a negligible safety risk to traffic.</p>
Heart Transplant	Desist from driving for 6 months after		Disqualified from driving if the driver has			

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>hospital discharge.</p> <p><i>Conditions:</i></p> <ol style="list-style-type: none"> 1. Must only involve mild functional limitation (NYHA Class I) 2. Ejection fraction greater than 35%. 3. Requires annual review of ischemic burden 		<p>symptoms.</p> <p>May be re-licensed if:</p> <ol style="list-style-type: none"> 1. LVEF is good i.e. greater than 0.4. 2. Person passes exercise test. 3. No other conditions are present that would make the person unfit to drive. 			
Pacemaker	<p>Desist from driving for 1 week after implant.</p> <p><i>Conditions:</i></p> <ol style="list-style-type: none"> 1. No impaired level of consciousness may be present. 2. ECG to display “normal sensing & capture” . 3. Pacemaker must perform according to specifications. 	<p>Desist from driving for minimum of 1 month.</p> <p>Not eligible to hold an unconditional licence.</p> <p>Conditional licence may be issued after taking into account the risks of the pacemaker malfunctioning & the opinion of a cardiologist.</p> <p>Periodic review required.</p>	<p>Disqualified from driving for 6 weeks.</p> <p>May resume driving if there are no other conditions present that would make the person unfit to drive.</p>	Not specifically addressed.	<p>Desist from driving for minimum of 1 month after successful pacemaker insertion.</p> <p>May resume driving on specialist advice if:</p> <ol style="list-style-type: none"> 1. During moderate exercise haemodynamic response are normal. 2. No other conditions are present that would make the person unfit to drive. <p>May be required to undergo periodic medical review.</p>	Not specifically addressed.
CABG	Desist from driving for 3 months after hospital discharge.		Disqualified from driving for at least 3 weeks.			

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
			May be re-licensed if: 1. LVEF is good i.e. greater than 0.4. 2. Person passes exercise test. 3. No other conditions are present that would make the person unfit to drive.			
Hypertension	<p>No driving restrictions on people with hypertension that is less than 170/110.</p> <p>No driving is recommended if sustained hypertension is over 170/110.</p> <p>Driver must undergo comprehensive cardiovascular examination (electrocardiogram, chest radiography, fundoscopic examination and measurement of blood urea nitrogen) and referred to an internist if necessary.</p>	<p>No driving restrictions on people with hypertension that is less than 200/110, whether treated or untreated.</p> <p>No notification to DLA is required.</p> <p>Periodic medical review required to monitor the condition.</p> <p>An unconditional licence may NOT be held by those with: 1. Hypertension that is continually above 200/110. 2. End organ damage that interferes with driving. 3. Medication</p>	<p>Licence disqualification if resting blood pressure is consistently \geq 180mm Hg systolic and/or $>$100mm Hg diastolic.</p> <p>Re-licensing may occur if: 1. Condition is controlled. 2. Side effects of medication do not interfere with driving.</p>	<p>Drivers taking antihypertensive medications should be questioned about side effects such as orthostatic hypotension, syncope, drowsiness/sedation, or dizziness.</p> <p>No driving restrictions if 1. Hypertension is controlled by medication & diastolic blood pressure is less than 120 mm/Hg.</p> <p>A restricted licence may apply if diastolic persistently above 120mm.Hg and/or systolic over 200mm.Hg;</p> <p>Periodic reviews</p>	<p><i>Severe hypertension:</i> Person is unfit to drive if: 1. Resting blood pressure is consistently \geq 200mm Hg systolic or $>$110mm Hg diastolic. 2. Medication impairs alertness or results in significant postural hypotension. 3. End organ damage interferes with driving.</p>	<p>Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p> <p>A licence may still be issued if a favourable medical report is obtained & the person poses a negligible safety risk to traffic.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		<p>impairs alertness or results in significant postural hypotension.</p> <p>A conditional licence may be issued if blood pressure is controlled and medication does not have any significant side-effects.</p> <p>Periodic review required.</p>		required.		
Dysrhythmia/ Arrhythmia	<p><i>Ventricular fibrillation or sustained ventricular tachycardia:</i> Disqualified from driving.</p> <p><i>Chronic atrial fibrillation:</i> No restrictions if cerebral ischaemia is not present & no underlying heart disease. Anti-coagulation is required, if indicated.</p> <p><i>Paroxysmal atrial fibrillation, or non-sustained</i></p>	<p>Person may not hold an unconditional licence if arrhythmia is recurrent & may result in syncope or other disabling symptoms.</p> <p>A conditional licence may be issued if:</p> <ol style="list-style-type: none"> 1. Surgical cure has been effected. 2. Anti-coagulant therapy has been satisfactory. 3. Arrhythmia has been treated successfully for 3 	<p>Disqualified from driving if any incapacity results or may result from the condition.</p> <p>Re-licensing may occur when the arrhythmia has been controlled for a minimum of 3 weeks, & the LV is good (i.e. LVEF is >0.4) & there is no other underlying condition that may impair driving.</p> <p>Transient dysrhythmias and arrhythmias associated with acute coronary syndromes do not apply under this</p>	<p>No licence restrictions for arrhythmias that occurred</p> <ol style="list-style-type: none"> 1. In childhood. 2. Over 5 years ago. 3. Arrhythmias that have been controlled or stable for 3 months minimum. <p>Two-yearly review required for 1 & 2. Three -monthly review required for 3.</p>	<p>Persons with recurrent arrhythmias or arrhythmias that may lead to syncope or death are unfit to hold a licence.</p> <p>No licence restrictions for arrhythmias without complications. A minimum symptom-free period of 6 months is required.</p> <p>Annual cardiac assessment may be required.</p>	<p>Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving.</p> <p>Licence denial if there serious paroxysmal arrhythmia occurs.</p> <p>Assessments are to take account of the causes, development & treatment of the disease.</p> <p>A licence may still be issued if a favourable medical report is obtained & the person poses a negligible safety risk to traffic.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<i>paroxysmal ventricular fibrillation, or paroxysmal supraventricular tachycardia:</i> No restrictions if cerebral ischaemia or underlying heart disease is not present or, if present, both are satisfactorily controlled.	months minimum. Periodic review required.	section.			
Angioplasty	Desist from driving for 7 days following percutaneous coronary intervention (PCI) if procedure performed during hospital stay or 30 days following discharge if PCI performed after initial hospital stay.	Desist from driving for 4 weeks. Person may not hold an unconditional licence. A conditional licence may be issued if: 1. Person's medical history typified by minimal symptoms. 2. Has an exercise tolerance of >9 minutes (males) or > 6 minutes (females) on the Bruce Treadmill Test or similar test. 3. Has no severe ischaemia. 4. Has an "ejection	Disqualified from driving for 6 weeks minimum. Re-licensing may occur if the person can meet the exercise test criteria & there is no other underlying condition that may impair driving.	<i>For any Cardiac Surgery:</i> May hold an unrestricted licence if the person: 1. Has symptoms only with strenuous exercise 1 year following surgery. 2. Is symptom-free whilst resting 3 months post-surgery.	Desist from driving for 4 weeks minimum. Persons with complications should not drive. Driving may resume if: 1. No AMI occurred before, after or during surgery. 2. Absence of myocardial ischaemia with adequate stress testing. 3. Minimal myocardial ischaemia at moderate or high stress levels but complete revascularisation at angiography. Annual medical reviews may be required.	Licence denial for any CVA disease that results in acute impairment of the cerebral functions involved in safe driving. Licence denial if the person's tested work capacity is well below expected normal limits. Assessments are to take account of the causes, development & treatment of the disease. A licence may still be issued if a favourable medical report is obtained & the person poses a negligible safety risk to traffic.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Heart Disease	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		fraction of 40% or over" (p41). Periodic review required.				

Table C3. Commerical licensing guidelines for drivers with cognitive impairment (dementia)

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Dementia	<p>Recommended that patients diagnosed with mild dementia are given a comprehensive on and off-road driving test at a specialized centre, as approved by provincial or territorial transportation ministries.</p> <p>Re-evaluation should occur approximately every 6–12 months.</p> <p>Moderate or severe dementia patients are ineligible for any licence.</p>	<p>May not hold an unconditional licence if dementia is present. A conditional licence may be issued on specialist's advice & taking into account treatment response & results of neuropsychological & practical driving tests. Subject to periodic review.</p>	<p>Licence refusal or revocation.</p>	<p>Frequent review of driving abilities may be required.</p> <p>Special restrictions apply as recommended by medical staff.</p> <p>DLD must be notified.</p> <p><i>Moderate, severe or profound cognitive impairment:</i> No driving.</p>	<p>May not drive.</p>	<p>Licence denied or revoked.</p>

Table C4. Commerical licensing guidelines for drivers with cognitive impairment (TBI)

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Minor Head Injuries	Minor head injury is not expected to impair driving for longer than a few hours – symptoms should be monitored, cognitive and motor functions should be determined.	Desist from driving immediately following the injury. If loss of consciousness does not last more than 24 hours & there are no complications, the person is not viewed as posing a road safety risk. An unconditional licence may not be held if the person sustains chronic functional impairments. A conditional licence may be issued subject to medical & neuropsychological assessments & practical driver assessment, and if there are no other disabilities that may interfere with driving ability. Subject to periodic review.	Not specifically addressed.	Special restrictions apply for cognitive & communication impairment resulting from closed head injury as recommended by medical staff. Regular reviews required. DLD must be notified.	If no loss of consciousness, or other complications, desist from driving for a minimum of 3 hours. If loss of consciousness occurs, desist from driving for 24 hours & obtain medical assessment. Longer stand-down periods may be required if the person displays any of the following: 1. Impaired judgment, vision or intellectual capacity. 2. Loss of motor skills. 3. Seizures. Person must obtain GP clearance before driving is resumed.	Not specifically addressed.
Serious Head Injuries	If concussion, post-traumatic amnesia or any residual brain damage	An unconditional licence may not be held if the person sustains chronic	Recommended that licence be revoked or refused.	Evaluation by a State driver licence examiner required.	Desist from driving for a minimum of 12 months.	Licence denial or revocation if serious cognitive disturbances result from injury.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>results, a full medical evaluation is required prior to resumption of driving. Patients with moderate to severe TBI (Glasgow coma scale <13 or requiring hospital admission) will need comprehensive assessment</p> <p><i>Single post-traumatic seizure:</i> No driving for 12 months & a full neurological exam and ECG to be conducted.</p> <p><i>Post-traumatic epilepsy:</i> The guidelines for “Diagnosis of Epilepsy” apply (see Epilepsy Table).</p>	<p>functional impairments.</p> <p>A conditional licence may be issued subject to medical & neuropsychological assessments & practical driver assessment, and if there are no other disabilities that may interfere with driving ability.</p> <p>Subject to periodic review.</p>	<p>May resume driving subject to the risk of seizure falling to no more than 2% per annum.</p>	<p>No driving If there is moderate, severe or profound cognitive impairment.</p>	<p>If post-traumatic seizures occur (except those that occur in the first 24 hours after the event), the same guidelines required for tonic clonic epilepsy apply.</p> <p>For most severe head injuries, the person is generally considered unfit to drive.</p> <p>In some cases driving may resume subject to a full neurological assessment and if the person has recovered sufficiently to drive safely.</p> <p>Assessment by an occupational therapist is recommended.</p>	<p>Medical assessment will take into account disturbances in judgement, memory, vision, psychomotor & emotional functioning.</p> <p>The extra safety risk that exists with driving commercial vehicles will also be taken into account during assessment.</p>

Table C5 Commercial licensing guidelines for drivers with diabetes mellitus

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Diabetes	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Diabetes controlled by diet alone	May drive if: There are no other disqualifying complications.	No licence restriction. Periodic review by GP recommended.	Licence granted provided complications do not develop eg visual acuity & visual field problems.	<i>Condition is Mild & Stable:</i> No licence restrictions. Yearly review required. Appropriate snacks must be readily available for consumption whilst the driver is on duty.	Generally considered fit to drive	Licence denial for diabetes that is not sufficiently controlled. Applications considered in light of road safety risk from diabetic complications eg vision & CVA conditions. Reappraisals carried out on a case-by-case basis or discontinued if unnecessary.
Non-insulin treated diabetes	May drive if: There are no other disqualifying complications and are not subject to hypoglycaemia. However class I, II, III, & IV require an annual medical review.	Person may not hold an unconditional licence. A conditional licence may be issued if: 1. Diabetes is controlled & person complies with treatment. 2. No hypoglycaemia episodes & person has hypoglycaemic awareness. 3. No end organ effects which may impair driving. Annual review required.	Licence granted provided complications do not develop eg visual acuity & visual field problems or if insulin treatment begins. Licence may be refused, revoked or a short period licence granted if disabilities do develop or if insulin treatment becomes necessary.	<i>Condition is Mild & Stable:</i> No licence restrictions. Yearly review required. Appropriate snacks &/or anti-diabetic drugs must be readily available for consumption whilst the driver is on duty.	Conditional licence may be issued if: 1. There is no history of hypoglycaemia. 2. Person has hypoglycaemic awareness. 3. Person complies with treatment. 4. There are no complications associated with diabetes. In addition, regular meal breaks & shift work must be adhered to. Annual medical review & two-yearly specialist	Licence denial for diabetes that is not sufficiently controlled. Applications considered in light of road safety risk from diabetic complications eg vision & CVA conditions. Reappraisals carried out on a case-by-case basis or discontinued if unnecessary.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Diabetes	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
					review required.	
Insulin-treated diabetes	<p>Disqualified from driving if:</p> <ol style="list-style-type: none"> 1. Hypoglycaemic episode occurred in last 6 months & required outside intervention or had no warning symptoms. 2. Insulin treatment has changed in last month. Monthly assessments required until stability reached. 3. Visual impairment or progressive retinopathy are present. 4. Peripheral neuropathy with functional loss is present. 5. Cardiovascular disease with arrhythmia, angina, or myocardial infarction occurred in last year. 6. Poor self-monitoring of blood glucose. <p>When driving the person must:</p> <ol style="list-style-type: none"> 1. Always carry self- 	<p>Person may not hold an unconditional licence.</p> <p>A conditional licence may be issued if:</p> <ol style="list-style-type: none"> 1. Diabetes is controlled & person complies with treatment. 2. No hypoglycaemia episodes & person has hypoglycaemic awareness. 3. No end organ effects which may impair driving. <p>Annual review required.</p>	<p><i>Licence applications made after 1/4/91</i></p> <p>Licence denial for drivers of HGV or PCV vehicles.</p> <p>Exceptions may be made for class CI vehicles, conditional on yearly medical assessments.</p> <p><i>Existing licence applications made before 1/4/91:</i></p> <p>Assessed on a case-by-case basis & subject to annual medical assessment.</p> <p>Drivers can also reapply for a commercial license if insulin treatment is discontinued.</p>	<p>According to federal guidelines the person is not fit to drive.</p> <p>However, a licence may be issued if there has been:</p> <ol style="list-style-type: none"> 1. No seizures, comas, loss of consciousness, or diabetic ketoacidosis resulting from hypoglycaemia for 5 years. 2. A complete medical & driving history & medical report submitted to DLA. <p><i>In the State of Utah</i></p> <p>A conditional licence may be issued for intrastate travel if:</p> <ol style="list-style-type: none"> 1. The above federal conditions are met & there have been no episodes of ketosis or altered states of consciousness in the previous year 	<p>A conditional licence may be issued if:</p> <ol style="list-style-type: none"> 1. Person has hypoglycaemic awareness. 2. Person complies with treatment. 3. There are no significant diabetic complications. 4. GP has evidence that the person self-tests blood glucose levels & these are satisfactory. <p>In addition, regular meal breaks & shift work must be adhered to.</p> <p>Six-monthly medical & annual specialist review required.</p>	<p>Licence denied or revoked.</p> <p><i>Exceptions:</i></p> <ol style="list-style-type: none"> 1. If the condition is controlled, licence for Group 3 may be issued provided that the person does not drive in traffic designated as commercial. 2. Persons with an existing licence who subsequently develops diabetes requiring insulin treatment may retain their licence if the condition is under control & requires the licence for their livelihood. Other compelling & persuasive arguments will also be considered.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Diabetes	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>monitoring equipment & a glucose source that can be quickly absorbed & syringes, pump, or injector.</p> <p>2. Must test blood glucose 1 hour prior to driving & at 4-hourly intervals whilst driving.</p> <p>3. Must stop driving if > 10% of blood glucose is below 4 mmol/L. Resume driving after eating & glucose level has risen.</p> <p>Must attend annual reviews including an eye examination.</p>					

Table C6 Commerical licensing guidelines for drivers with epilepsy

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Auras & minor epilepsy (absences)	<p>Patients with auras with somatosensory, special sensory symptoms or nondisabling focal motor seizures may be eligible to drive commercial vehicles (Classes 1 – 4) if:</p> <ul style="list-style-type: none"> • seizures are benign for at least 3 years • no generalised seizures • neurologist gives approval • no impairment of cognition or consciousness • no head or eye deviation with seizures 	Not addressed.	Must be seizure-free for 10 years & not taking anti-epileptic drugs & not a source of danger whilst driving.	<p>Disqualified from holding an unrestricted licence.</p> <p>A restricted licence may be issued if:</p> <p>1. Seizure or episode- free for 5 years & no medication for 3 years.</p> <p>OR</p> <p>2. Seizure or episode- free for 1 year without medication or with medication but no side effects.</p> <p>Restricted to intrastate travel & medical approval required.</p> <p>For 2. above person is also restricted to driving light vehicles only.</p>	<p>Usually regarded as permanently unfit to drive.</p> <p>May be considered fit to drive after a 5-year seizure-free period without medication with a neurologist-supported claim</p>	Not specifically addressed.
First, isolated epileptic seizure	All passenger-carrying drivers to stop driving	A conditional licence may be issued if ALL of the	Must be seizure-free for 10 years & not taking anti-epileptic	Disqualified from holding an unrestricted licence.	Usually regarded as permanently unfit to drive.	Licence denied due to any of the following: 1. Seizure in the last 5

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
(prior to epilepsy diagnosis)	<p>immediately.</p> <p>Desist from driving for 3 months. Complete neurological exam required including EEG & CT.</p> <p>May resume driving if free of seizures for 1 year.</p>	<p>following apply:</p> <ol style="list-style-type: none"> 1. Is a single, provoked seizure. 2. Person can avoid provoking factors. 3. No seizures in past year. 4. Not on anti-epileptic drugs. 5. No evidence of epileptiform activity on EEG. <p>Other factors to be considered are working hours & tasks, vehicle size & condition. Specialist may advise restrictions.</p>	<p>drugs & not a source of danger whilst driving.</p> <p>After a single seizure provoked by drugs and/or alcohol, driver must be seizure free for at least 5 years without medication.</p>	<p>A restricted licence may be issued if:</p> <ol style="list-style-type: none"> 1. Seizure or episode- free for 5 years & no medication for 3 years. <p>OR</p> <ol style="list-style-type: none"> 2. Seizure or episode- free for 1 year without medication or with medication but no side effects. <p>Restricted to intrastate travel & medical approval required.</p> <p>For 2. above person is also restricted to driving light vehicles only.</p>	<p>May be considered fit to drive after a 5-year seizure-free period without medication with a neurologist-supported claim.</p> <p>Special circumstances may apply if seizure provoked by medication taken for another condition & the medication has been discontinued. Written report from neurologist required.</p>	<p>years.</p> <ol style="list-style-type: none"> 2. EEG test & medical history show high risk of loss of consciousness. 3. No evidence of epileptiform activity on EEG.
Epilepsy diagnosis	<p>Drivers with a past history of seizures should not hold any licence other than a class V or VI, and only then they must conform to the following criteria: Physician believes they are truthful about their seizures Physician believes the</p>	<p>A conditional licence may be issued if any of the following apply:</p> <ol style="list-style-type: none"> 1. History of benign childhood epilepsy or febrile seizures & not on anti-epileptic drugs & no evidence of epileptiform activity on EEG. 2. History of single 	<p>Must be seizure-free for 10 years & not taking anti-epileptic drugs & not a source of danger whilst driving.</p> <p>Exceptions can be made for seizures occurring at the time of an acute head trauma or intracranial</p>	<p>Disqualified from holding an unrestricted licence.</p> <p>A restricted licence may be issued if:</p> <ol style="list-style-type: none"> 1. Seizure or episode- free for 5 years & no medication for 3 years. <p>OR</p>	<p>Usually regarded as permanently unfit to drive.</p> <p>May be considered fit to drive after a 5-year seizure-free period without medication with a neurologist-supported claim</p>	<p>Licence denied due to any of the following:</p> <ol style="list-style-type: none"> 1. Seizure in the last 5 years. 2. EEG test & medical history show high risk of loss of consciousness. 3. No evidence of epileptiform activity on EEG.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>patient will adhere to medication. Driver must be under adequate supervision. The seizures must be preventable by medication.</p> <p>However if driver has been seizure free on or off medication for 5 yrs and have received a favourable report from their physican they may hold any licence.</p>	<p>seizures or can avoid provocative factors that lead to seizures & no seizures in past 5 years & not on anti-epileptic drugs & no evidence of epileptiform activity on EEG.</p> <p>3. Epilepsy treated by surgery & no seizures in past 5 years & undergoes annual review & no evidence of epileptiform activity on EEG.</p> <p>4. Epilepsy treated by drugs & no seizures in last 5 years & under regular review & no evidence of epileptiform activity on EEG.</p> <p>5. Had single provoked seizure & can avoid provocative factors & no seizures in past 1 year & not on anti-epileptic drugs & no evidence of epileptiform activity on EEG.</p>	<p>surgery with no recurrence thereafter. Seizre risk msut have fallen to 2% or less before driving can resume.</p>	<p>2. Seizure or episode- free for 1 year without medication or with medication but no side effects.</p> <p>Restricted to intrastate travel & medical approval required. For 2. above person is also restricted to driving light vehicles only.</p>		

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		The size & condition of vehicle & work hours are to be taken into consideration & restrictions may apply.				
Epilepsy occurring while asleep	May resume driving after 5 years free of seizures & off medication. May differ between patients.	Not addressed.	Must be seizure-free for 10 years & not taking anti-epileptic drugs & not a source of danger whilst driving.	Disqualified from holding an unrestricted licence. A restricted licence may be issued if: 1. Seizure or episode- free for 5 years & no medication for 3 years. OR 2. Seizure or episode- free for 1 year without medication or with medication but no side effects. Restricted to intrastate travel & medical approval required. For 2. above person is also restricted to driving light vehicles only.	Usually regarded as permanently unfit to drive. May be considered fit to drive if seizure pattern during sleep & upon waking has been stable for 5 years & no other seizures have occurred & a neurologist-supports the claim.	Licence denied due to any of the following: 1. Seizure in the last 5 years. 2. EEG test & medical history show high risk of loss of consciousness. 3. No evidence of epileptiform activity on EEG.
Medication Withdrawal	Desist from driving for 6 months after withdrawal or	Cannot drive if medication withdrawn.	Desist from driving during withdrawal period & for 6 months	Disqualified from holding an unrestricted licence.	Not addressed for commercial licences.	Licence denied due to any of the following: 1. Seizure in the last 5

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	<p>change of medication.</p> <p><i>If seizures recur:</i> Can resume driving on resumption of previously effective medications. Resume driving after 6 months if seizure free.</p> <p><i>Long-term withdrawal</i> Patients can drive any class of vehicle after being seizure free for 5 years and if no epileptiform activity is recorded during a waking and sleep EEG obtained in the 6 months prior to driving</p>		<p>after this. Exceptions can be made depending on the physician's advice.</p>	<p>A restricted licence may be issued if:</p> <ol style="list-style-type: none"> 1. Seizure or episode- free for 5 years & no medication for 3 years. <p>OR</p> <ol style="list-style-type: none"> 2. Seizure or episode- free for 1 year without medication or with medication but no side effects. <p>Restricted to intrastate travel & medical approval required. For 2. above person is also restricted to driving light vehicles only.</p>		<p>years.</p> <ol style="list-style-type: none"> 2. EEG test & medical history show high risk of loss of consciousness. 3. No evidence of epileptiform activity on EEG.
Epilepsy Treated by Surgery	May resume driving after 5-years seizure-free period after surgery.	A conditional licence may be issued if the person has been seizure-free for the past 5 years & there is no epileptiform activity on the EEG & a yearly review is undertaken.	Not addressed.	Not addressed.	Not addressed.	Not addressed.

Table C.7 Commercial licensing guidelines for drivers with musculoskeletal disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2003)</i>	<i>Utah Driver License Division (1992)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
Limb Amputation	<p>May continue to drive subject to satisfactory driving assessment with prosthesis.</p> <p><i>Upper limb amputation:</i> private licence only.</p> <p><i>Limb amputation below knee in 1 or 2 legs:</i> Must have prosthesis & “full strength & movement in back, hips & knee joints” & subject to satisfactory driving assessment to be eligible to operate a commercial licence.</p>	<p><i>Complete or partial limb amputation:</i> May not hold an unconditional licence.</p> <p>A conditional licence may be issued following a practical driving assessment & if prosthesis is worn & suitable car modifications are made.</p> <p>Periodic review required.</p> <p><i>Both thumbs missing:</i> May not hold an unconditional licence.</p> <p>A conditional licence may be issued following a practical driving assessment & vehicle is modified.</p> <p>Periodic review required.</p>	<p>May be licensed if driving ability is unimpaired.</p> <p>Vehicle modifications may be required.</p> <p>Annual review required.</p>	<p><i>Limb amputation:</i> If person has no “driving limitations” & subject to further driving assessment with prosthesis &/or car modifications, an unrestricted licence will be issued with a waiver.</p> <p>Medical Advisory Board approval required.</p> <p>Annual review required.</p>	<p>Licence denied if it is impossible to compensate with modifications.</p> <p><i>Both arms or both legs amputated:</i> Licence denied</p> <p><i>Both thumbs missing:</i> May continue to drive if s/he can meet driving performance requirements.</p> <p>A complete on & off road assessment by a trained occupational therapist may be required.</p>	<p>Licence denied if ability to drive safely is impaired.</p> <p>May continue to drive if prosthesis &/or vehicle modifications can compensate for disability.</p> <p>If person has a bus or taxi licence, s/he must be able to help passengers to enter & alight from the vehicle & buckle their seat belts.</p>
Arthritis & Joint Problems	Not addressed.	May not hold an unconditional licence if chronic pain present which interferes with concentration or	Licence denial or revocation if the person is disabled. May be licensed if	<i>With mild or moderate “residual loss of function”:</i> An unrestricted licence will be issued with a	Driving assessment is required if locomotor functioning is impaired. If condition interferes	Licence denied if ability to drive safely is impaired. May continue to drive

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2003)</i>	<i>Utah Driver License Division (1992)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
		restriction/ loss of joint movement that impairs driving performance. A conditional licence may be issued following a practical assessment of ability to operate vehicle get in and out of it.	driving ability is unimpaired. Vehicle modifications may be required. Annual review required.	waiver. Medical Advisory Board approval required. A restricted licence may be issued if the person has impaired psychomotor function but can drive the vehicle, with or without modifications. Restricted to intrastate with a waiver. One or two-yearly review required.	with ability to drive safely, then driving restrictions may apply.	vehicle modifications can compensate for disability. If person has a bus or taxi licence, s/he must be able to help passengers to enter & alight from the vehicle & buckle their seat belts.
Spinal Conditions	<i>Cervical vertebrae:</i> Some reduction in head & neck movement is permitted providing vehicle is fitted with outside mirrors on both the right & left hand sides. Must be able to move shoulders sufficiently and pass a road test. <i>Lumbar Spine:</i> Must be free of pain that restricts movement or judgement ability or is distracting.	May not hold an unconditional licence if cervical spine movement is restricted to less than 45 degrees in either direction.	Not addressed.	<i>With mild or moderate "residual loss of function":</i> An unrestricted licence will be issued with a waiver. Medical Advisory Board approval required. A restricted licence may be issued if the person has impaired psychomotor function but can drive the vehicle, with or without modifications. Restricted to intrastate	Driving assessment is required if locomotor functioning is impaired. Desist from driving if severe back, neck, shoulder or pelvic pain. Persons with cervical spine movement that is restricted to less than 45 degrees in either direction may continue to drive if assessment demonstrates that they can safely drive.	Licence denied if ability to drive safely is impaired. May continue to drive vehicle modifications can compensate for disability. If person has a bus or taxi licence, s/he must be able to help passengers to enter & alight from the vehicle & buckle their seat belts.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2003)</i>	<i>Utah Driver License Division (1992)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish Road Administration (1999)</i>
				with a waiver. One or two-yearly review required.		

Table C.8 Commercial licensing guidelines for drivers with a neurological condition (excluding epilepsy)

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Parkinson's	<p><i>Early stages of disease:</i> No restrictions. Must be closely monitored.</p> <p><i>Mild loss of muscle strength or control:</i> Car modifications may be necessary to ensure safe driving. Driving assessment required.</p> <p><i>When safe driving compromised:</i> No driving.</p>	<p>An unconditional licence may not be held if the disease impairs driving.</p> <p>A conditional licence may be issued subject to the results of a driving assessment & treatment response & with appropriate vehicle modifications.</p> <p>Subject to yearly reviews (minimum).</p>	<p><i>Condition stable & driving unimpaired:</i> Licence may be issued subject to yearly assessment and consideration given on an individual basis.</p> <p><i>Condition progressive or disabling:</i> Recommendation that licence be refused or revoked.</p>	<p>An unrestricted licence may be issued if the person is able to control equipment & has no or minimal neurological impairment.</p> <p>Periodic review required.</p> <p>A restricted licence may be issued if the person is able to control equipment despite slight neurological impairment.</p> <p>Restricted to intrastate driving.</p> <p>Annual review required.</p>	<p>Licence revocation or denial.</p> <p><i>Exceptions:</i> 1. Subject to the results of on & off-road assessment indicating safe driving ability, persons with minor muscular weakness may continue to drive. Periodic review may be required. 2. Persons with drug-induced Parkinson's disease who are expected to fully recover when drugs are withdrawn & the disease being so treated does not preclude them from driving.</p>	<p>Licence denial or revocation if disease impairs driving ability & so renders the person a traffic safety risk.</p> <p>Risk assessment to include an appraisal of the stage of the disease & treatment response as well as the extra dangers posed by holding this class of licence.</p> <p>Periodic review required on a case-by-case basis.</p>
Multiple Sclerosis	<p><i>Early stages of disease:</i> No restrictions. Must be closely monitored.</p> <p><i>Mild loss of muscle strength or control:</i> Car modifications may be necessary to ensure safe driving.</p>	<p>An unconditional licence may not be held if the disease impairs driving.</p> <p>A conditional licence may be issued subject to the results of a driving assessment & treatment response &</p>	<p><i>Condition stable & driving unimpaired:</i> Licence may be issued subject to yearly assessment and consideration given on an individual basis.</p> <p><i>Condition</i></p>	<p>An unrestricted licence may be issued if the person is able to control equipment & has no or minimal neurological impairment.</p> <p>Periodic review required.</p>	<p>Licence revocation or denial.</p> <p><i>Exceptions:</i> Subject to the results of on & off-road assessment indicating safe driving ability, persons with minor muscular weakness may</p>	<p>Licence denial or revocation if disease impairs driving ability & so renders the person a traffic safety risk.</p> <p>Risk assessment to include an appraisal of the stage of the disease & treatment response as well as the</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	Driving assessment required. <i>When safe driving compromised:</i> No driving.	with appropriate vehicle modifications. Subject to yearly reviews (minimum).	<i>progressive or disabling:</i> Recommendation that licence be refused or revoked.	A restricted licence may be issued if the person is able to control equipment despite slight neurological impairment. Restricted to intrastate driving. Annual review required.	continue to drive. Periodic review may be required.	extra dangers posed by holding this class of licence. Periodic review required on a case-by-case basis.
Motor Neurone Disease and Peripheral Neuropathy	<i>Early stages of disease:</i> No restrictions. Must be closely monitored. <i>Mild loss of muscle strength or control:</i> Car modifications may be necessary to ensure safe driving. Driving assessment required. <i>When safe driving compromised:</i> No driving.	An unconditional licence may not be held if the disease impairs driving. A conditional licence may be issued subject to the results of a driving assessment & treatment response & with appropriate vehicle modifications. Subject to yearly reviews (minimum).	<i>Condition stable & driving unimpaired:</i> Licence may be issued subject to yearly assessment and consideration given on an individual basis. <i>Condition progressive or disabling:</i> Recommendation that licence be refused or revoked.	An unrestricted licence may be issued if the person is able to control equipment & has no or minimal neurological impairment. Periodic review required. A restricted licence may be issued if the person is able to control equipment despite slight neurological impairment. Restricted to intrastate driving.	Licence revocation or denial. <i>Exceptions:</i> Subject to the results of on & off-road assessment indicating safe driving ability, persons with minor muscular weakness may continue to drive. Periodic review may be required.	Licence denial or revocation if disease impairs driving ability & so renders the person a traffic safety risk. Risk assessment to include an appraisal of the stage of the disease & treatment response as well as the extra dangers posed by holding this class of licence. Periodic review required on a case-by-case basis.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				Annual review required.		

Table C.9 Commercial Licensing guidelines for drivers with psychiatric illness

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006**)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Anxiety or Depression	<p>If the physician believes that the person is impaired to drive any type of vehicle due to impairment in judgement or psychomotor activity then the patient should be advised not to drive any type of vehicle until recovered.</p>	<p>An unconditional licence may not be held if the mental disorder impairs the person's cognitive, perceptual or psychomotor functioning OR Taking medication that impairs driving performance.</p> <p>A conditional licence may be issued if the condition is under control & person complies with treatment & the side effects of medication minimally interfere with driving.</p> <p>Subject to periodic review.</p>	<p><i>Without Significant Symptoms:</i> May continue to drive if illness is brief.</p> <p>If medication is taken which adversely affects driving ability, driving is to cease.</p> <p>No need to notify DVLA.</p> <p><i>Severe anxiety or depression (including significant memory or concentration problems, agitation or behavioural disturbances)::</i> Driving may resume if: 1. Condition is stable for 6 months & person is well. 2. Side-effects of medication do not impair driving ability. 3. Symptoms of enduring illness are absent with medication & with no</p>	<p>Unrestricted licence may be issued if the condition has been stable for 1 to 2 years without medication, or with medication that does not impair alertness or psychomotor functioning.</p> <p>A restricted licence may be issued if the condition has been stable for 3 months without medication, or with medication that does not impair alertness or psychomotor functioning. Licence restricted to intrastate travel.</p> <p>Medical recommendation required.</p> <p>Reviews conducted six monthly or as required.</p>	<p><i>Mental Disorder that May Impair Driving:</i> Assessment is to be based on the impact that the disorder has on behaviour, mood & psychomotor functioning. Other factors to consider are the insight the person has into the illness & medication (effectiveness & side effects).</p> <p>The extra stresses of driving commercial vehicles are to be considered (eg deadlines, long hours, contact with the public).</p> <p>In addition, it is recommended that the person refrains from driving during periods of suicide ideation.</p> <p><i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive.</p> <p>Driving may resume after an observation period of 12 months if: 1. Treatment has been</p>	<p><i>Condition stable & minimal risk of symptom manifestation:</i> Licence may be retained.</p> <p><i>Serious disorder:</i> Licence denial if the disorder results in serious disturbances of behaviour, judgement or adaptability.</p> <p>Particular attention is to be paid to the increased traffic safety risk associated with commercial licences.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006**)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
			<p>side effects that impair driving. Psychiatric reports may be required.</p> <p>If the illness involves substance misuse, continuing misuse is not acceptable for driving.</p>		<p>satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving.</p> <p>The waiting period can be reduced in exceptional circumstances eg condition is stable & person is symptom& free for a “satisfactory” period, low risk of recurrence, no residual impairment & favourable psychiatric assessment. The extra stresses of driving commercial vehicles are to be considered (eg deadlines, long hours, contact with the public).</p>	
Manic-Depression (bipolar disorder)	If the physician believes that the person is impaired to drive any type of vehicle due to impairment in judgement or psychomotor activity then the patient should be advised not to drive any type of vehicle until recovered.	<p><i>Acute phase of illness:</i> Desist from driving.</p> <p>May not hold an unconditional licence if:</p> <ol style="list-style-type: none"> 1. Condition is acute or chronic. <p>OR</p> <ol style="list-style-type: none"> 2. On medication that impairs driving in the long-term. <p>OR</p> <ol style="list-style-type: none"> 3. There is a 	<p>Driving to cease until medical evaluation is undertaken confirming the following points:</p> <ol style="list-style-type: none"> 1. Illness is stable for 3 years & person is well, treatment compliant and with insight into their illness. At this point, a psychiatric evaluation is to be conducted. 	<p><i>Acute phase of illness:</i> No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.</p>	<p><i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive.</p> <p>Driving may resume after an observation period of 12 months if:</p> <ol style="list-style-type: none"> 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. <p>The waiting period can be reduced in exceptional</p>	<p>Licence denial or revocation in cases of serious disturbance.</p> <p>May continue to drive if the condition is stable & the risk of symptoms assessed as minimal.</p> <p>Desist from driving for 1 year following a relapse of the illness. This period may be reduced if the relapse was into a depressive phase.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006**)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		<p>significant likelihood of relapse, according to medical opinion.</p> <p>A conditional licence may be issued if the condition is under control & person complies with treatment & the side effects of medication minimally interfere with driving.</p> <p>Subject to periodic review.</p>	<p>3. Side-effects of medication do not impair driving ability.</p> <p>4. Low likelihood of recurrence of illness.</p>		<p>circumstances eg condition is stable & person is symptom& free for a “satisfactory” period, low risk of recurrence, no residual impairment & favourable psychiatric assessment. The extra stresses of driving commercial vehicles are to be considered (eg deadlines, long hours, contact with the public).</p>	<p>The extra safety risks associated with this type of licence are also to be considered.</p>
Chronic Schizophrenia	Not specifically addressed.	<p><i>Acute phase of illness:</i> Desist from driving.</p> <p>May not hold an unconditional licence if:</p> <p>1. Condition is acute or chronic. OR 2. On medication that impairs driving in the long-term. OR 3. There is a significant likelihood of relapse, according to medical opinion.</p> <p>A conditional licence</p>	<p>Driving to cease until medical evaluation is undertaken confirming the following:</p> <p>1. Illness is stable for minimum of 3 years & person is well & has insight into their illness - after which time a psychiatric evaluation is required. 2. Medication should not impair driving ability & must be of minimum effective dose. 3. Low likelihood of</p>	<p><i>Acute phase of illness:</i> No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.</p>	<p><i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive.</p> <p>Driving may resume after an observation period of 12 months if:</p> <p>1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving.</p> <p>The waiting period can be reduced in exceptional circumstances eg condition is stable & person is symptom& free for a “satisfactory” period, low risk of</p>	<p>Licence denial or revocation in cases of serious disturbance.</p> <p>May continue to drive if the condition is stable & the risk of symptoms assessed as minimal.</p> <p>Particular attention is to be given to the existence of delusions, hallucinations, disorganised behaviour, anger & rage outbursts, alcohol/substance abuse & any residual problems after an active phase of the illness.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006**)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		may be issued if the condition is under control & person complies with treatment & the side effects of medication minimally interfere with driving. Subject to periodic review.	symptom recurrence		recurrence, no residual impairment & favourable psychiatric assessment. The extra stresses of driving commercial vehicles are to be considered (eg deadlines, long hours, contact with the public).	Desist from driving for 1 year following an active phase of the illness. The extra safety risks associated with this type of licence are also to be considered.
Psychotic Disorders	<i>After a single psychotic episode:</i> the driver may qualify for a licence after a period of satisfactory emotional and mental stability as evidenced by a psychiatrists report. <i>After recurrent episodes:</i> Driver is eligible for a class I, II, III or IV licence after six months free from psychiatric support. Evidence must be provided from a psychiatrists report.	<i>Acute phase of illness:</i> Desist from driving. May not hold an unconditional licence if: 1. Condition is acute or chronic. OR 2. On medication that impairs driving in the long-term. OR 3. There is a significant likelihood of relapse, according to medical opinion. A conditional licence may be issued if the condition is under control & person complies with treatment & the side	Driving to cease until medical evaluation is undertaken. Driving may resume if: 1. Illness is stable for minimum of 3 years & person is well & has insight into their illness - after which time a psychiatric evaluation is required. 2. Medication should not impair driving ability & must be of minimum effective dose. 3. Low likelihood of symptom recurrence	<i>Acute phase of illness:</i> No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.	<i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive. Driving may resume after an observation period of 12 months if: 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. The waiting period can be reduced in exceptional circumstances eg condition is stable & person is symptom & free for a "satisfactory" period, low risk of recurrence, no residual impairment & favourable psychiatric assessment. The extra stresses of driving commercial	Licence denial or revocation in cases of serious disturbance. May continue to drive if the condition is stable & the risk of symptoms assessed as minimal. Particular attention is to be given to the existence of delusions, hallucinations, disorganised behaviour, anger & rage outbursts, alcohol/substance & any residual problems after an active phase of the illness. Desist from driving for 1 year following an active phase of the illness. The extra safety risks

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006**)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		effects of medication minimally interfere with driving. Subject to periodic review.			vehicles are to be considered (eg deadlines, long hours, contact with the public).	associated with this type of licence are also to be considered.
Personality Disorders	Drivers with personality disorders should not be allowed to drive until adequately assessed by a psychiatrist.	People with personality disorders frequently exhibit a disregard for social values & the law & may have a history of aggressive & erratic behaviour. Psychiatric, legal & administrative assistance may be required with driver licensing. A conditional licence may be issued if: 1. The illness is controlled. 2. Person complies with treatment over a prolonged period. 3. Medication that minimises cognitive & other symptoms that impair driving. Subject to periodic review.	Licence will be refused or revoked if behavioural disturbances may cause dangerous driving. If medical advice confirms that driver is not a danger, then licence may be retored.	<i>Acute phase of illness:</i> No driving if person poses a risk to others or to self, or medication impairs alertness or psychomotor functioning or if person requires commitment.	<i>Mental Disorder that May Impair Driving:</i> Assessment is to be based on the impact that the disorder has on behaviour, mood & psychomotor functioning. Other factors to consider are the insight the person has into the illness & medication (effectiveness & side effects). The extra stresses of driving commercial vehicles are to be considered (eg deadlines, long hours, contact with the public). In addition, it is recommended that the person refrains from driving during periods of suicide ideation. <i>Severe & Chronic Mental Disorder:</i> Person is unfit to drive.	Licence denial or revocation in cases of serious disturbance. May continue to drive if the condition is stable & the risk of symptoms assessed as minimal. Particular attention is to be given to anti-social & borderline personality disorders. The extra safety risks associated with this type of licence are also to be considered.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006**)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
					<p>Driving may resume after an observation period of 12 months if:</p> <ol style="list-style-type: none"> 1. Treatment has been satisfactory. 2. Symptoms are absent or at a level that does not impair safe driving. <p>The waiting period can be reduced in exceptional circumstances eg condition is stable & person is symptom& free for a “satisfactory” period, low risk of recurrence, no residual impairment & favourable psychiatric assessment. The extra stresses of driving commercial vehicles are to be considered (eg deadlines, long hours, contact with the public).</p>	
ADHD	The physician should determine whether or not the driver can respond appropriately to traffic signs and signal situations. Higher standards are expected of those wishing to drive commercial vehicles.	<p>May not hold an unconditional licence.</p> <p>May be issued with a conditional licence if:</p> <ol style="list-style-type: none"> 1. Condition is under control & person complies with treatment over a long period of time. 	Drivers with minor symptoms can be considered for a licence. Individual assessment is required. Factors such as impulsivity and limited awareness of the impact of their behaviours need to be	Not specifically listed.	Not specifically listed.	Not specifically listed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006**)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		AND 2. Medication is being taken that minimises risk of symptoms that impair driving. Subject to periodic review & specialist advice.	considered.			

** No distinction is made in this manual between types of psychiatric disorders. Distinction is made in terms of functional ability.

Table C.10 Commercial licensing guidelines for drivers with respiratory disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Asthma	Not specifically addressed.	<p><i>Severe chronic asthma:</i></p> <p>Desist from driving for 2 weeks following an attack that required admission to an ICU or from which loss of consciousness ensued.</p> <p><i>Exception:</i> Specialist clearance is given.</p>	<p>Notification to DVLA not required.</p> <p><i>Exceptions:</i> Asthma causes debilitating dizziness, fainting or loss of consciousness.</p> <p>Regular (annual) review required.</p>	<p><i>Minimal Symptoms:</i> No licence restrictions if medication is infrequently required & FVC & FEV >70% of predicted normal.</p> <p><i>Other cases:</i> A restricted licence may be issued if:</p> <ol style="list-style-type: none"> 1. Respiratory symptoms occur when activity levels are greater than normal. FVC & FEV >50% of predicted normal. Restricted to intrastate driving. 2. Driver requires any supplemental oxygen, then licence is restricted to intrastate & light vehicles only. May not transport dangerous cargo. If passengers are carried, a "No Smoking" sign is to be displayed. <p>Annual review required.</p> <p><i>Severe Breathing Difficulties:</i> No driving if severe</p>	<p><i>Severe asthma attacks:</i> Person warned to desist from driving especially if severe emphysema or loss of consciousness may occur.</p>	Not addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				symptoms occur with any activity or PO2 < 50 &/or PCO2 > 50.		
COPD (Chronic Obstructive Pulmonary Disease)	<p><i>Mild impairment:</i> May drive.</p> <p><i>Moderate impairment</i> May be allowed to drive a class I to IV vehicle licence subject to individual assessment.</p> <p><i>Severe impairment:</i> No driving is permitted. May be able to hold a class V vehicle licence subject to individual assessment.</p>	<p>This disease has a variable effect on driving depending on its "type & phase" (p82).</p> <p><i>Severe:</i> Person may not hold an unconditional licence.</p> <p>A conditional licence may be issued depending on the level of severity & treatment response.</p> <p>Periodic review required.</p>	<p>Notification to DVLA not required.</p> <p><i>Exceptions:</i> COPD causes debilitating dizziness, fainting or loss of consciousness.</p>	<p><i>Minimal Symptoms:</i> No licence restrictions if medication is infrequently required & FVC & FEV >70% of predicted normal.</p> <p><i>Other cases:</i> A restricted licence may be issued if:</p> <ol style="list-style-type: none"> 1. Respiratory symptoms occur when activity levels are greater than normal. FVC & FEV >50% of predicted normal. Restricted to intrastate driving. 2. Driver requires any supplemental oxygen, then licence is restricted to intrastate & light vehicles only. May not transport dangerous cargo. If passengers are carried, a "No Smoking" sign is to be displayed. <p>Annual review required.</p> <p><i>Severe Breathing Difficulties:</i></p>	<p><i>Severe COPD Episodes:</i> Person warned to desist from driving especially if severe emphysema or loss of consciousness may occur.</p>	Not addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				No driving if severe symptoms occur with any activity or PO ₂ < 50 &/or PCO ₂ > 50.		
Respiratory Failure	Not specifically addressed.	<p><i>Severe:</i> Person may not hold an unconditional licence.</p> <p>A conditional licence may be issued depending on the level of severity & treatment response.</p> <p>Periodic review required.</p>	Not specifically addressed.	<p><i>Minimal Symptoms:</i> No licence restrictions if medication is infrequently required & FVC & FEV >70% of predicted normal.</p> <p><i>Other cases:</i> A restricted licence may be issued if:</p> <ol style="list-style-type: none"> 1. Respiratory symptoms occur when activity levels are greater than normal. FVC & FEV >50% of predicted normal. Restricted to intrastate driving. 2. Driver requires any supplemental oxygen, then licence is restricted to intrastate & light vehicles only. May not transport dangerous cargo. If passengers are carried, a "No Smoking" sign is to be displayed. <p>Annual review required.</p> <p><i>Severe Breathing</i></p>	<i>Severe & Chronic:</i> No driving.	Not addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				<i>Difficulties: No driving if severe symptoms occur with any activity or PO₂ < 50 &/or PCO₂ > 50.</i>		

Table C.11 Private licensing guidelines for drivers with sleep disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Sleep Apnoea (obstructive sleep apnoea & apnoea syndrome)	Driver may operate any class of vehicle once the condition has been adequately treated and controlled subject to medical surveillance.	<p>May not hold an unconditional licence if:</p> <ol style="list-style-type: none"> 1. Diagnosed with OSA via sleep study & have moderate or severe sleepiness & in GP's opinion pose significant driving risk. 2. Frequently feels sleepy or drowsy whilst driving or has MVCs caused by sleepiness or inattention. 3. High-risk OSA that is untreatable or person not compliant with treatment or unwilling to restrict driving whilst waiting for treatment. <p>A conditional licence may be issued if person is compliant with treatment and symptoms are responsive to treatment.</p> <p>Periodic review required.</p>	Desist from driving until symptoms are satisfactorily controlled. Medical confirmation of this is required.	<p>Only 3 States in the USA specifically mention sleep apnoea in their licensing guidelines (Pakola et al., 1995).</p> <p>May drive unrestricted if mild to moderate problems of alertness and excessive sleepiness (Epworth Sleep Scale [ESS] score 10-12).</p> <p>Review required annually or every 2nd year.</p> <p>Restricted licence may apply with moderate symptoms of hypersomnolence and alertness (ESS score 13 – 15).</p> <p>Six monthly review required.</p> <p>Severe symptoms of inattentiveness or hypersomnolence (ESS score > 15) - Restricted from</p>	<p>Desist or restrict driving for the following high- risk patients</p> <ol style="list-style-type: none"> 1. Suspect person has OSA with excessive daytime sleepiness whilst driving & awaiting confirmation of diagnosis. 2. Severe daytime sleepiness & history of sleep-related accidents 3. Severe OSA that is untreatable or person not compliant with treatment <p>May resume driving if symptoms satisfactorily controlled under specialist supervision. Periodic medical assessment may be required.</p>	<p>Licence issued if condition successfully treated.</p> <p>Licence denied if alertness is affected to a degree that person poses a road safety risk.</p> <p>Subject to periodic review on a case-by-case basis.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				driving.		
Narcolepsy	Drivers who suffer from narcolepsy are not allowed to hold a class I, II, III or IV licence.	A conditional licence may be granted if person responds to treatment, according to expert opinion. Periodic review required.	Desist from driving upon diagnosis. Driving may be permitted on a 1, 2 or 3 year licence if control of symptoms achieved with regular medical review. Licence up to age 70 may be restored if illness controlled for 7 years.	Only 6 States in the USA specifically mention narcolepsy in their licensing guidelines (Pakola et al., 1995). <i>Utah</i> Narcolepsy falls under the same guidelines set down for epilepsy. An unrestricted licence may be issued if seizure or episode-free for 5 years, without medication. OR seizure-free for 12 months without medication or with medication but no side effects. One or two-yearly review required. A restricted licence may be issued if seizure or episode-free for 3 to 6 months, without medication or with medication but no	Desist from driving if person is suspected of having narcolepsy that impairs safe driving ability (in medical opinion) & is awaiting confirmation of diagnosis. May resume driving after satisfactory response to treatment or the person does not exhibit cataplexy or other symptoms that pose significant road safety risk. Regular medical assessment may be required.	Licence issued if condition successfully treated. Licence denied if alertness is affected to a degree that person poses a road safety risk. Subject to periodic review on a case-by-case basis.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
				<p>side effects. Speed, area & time of day restriction apply, depending on the length of time without seizures. Six-monthly review required.</p> <p>Restricted from driving when episodes are uncontrolled and/or medications affect alertness and coordination.</p>		

Table C.12 Private licensing guidelines for drivers with vestibular disorders

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Meniere's disease	Individuals with true vertigo should not drive any vehicle until they have responded to treatment or until the condition has subsided.	<p>May not hold an unconditional licence</p> <p>A conditional licence may be issued subject to treatment response & person's functional ability to drive safely.</p> <p>Periodic review required.</p>	<p><i>Upon diagnosis:</i> Desist from driving.</p> <p>Driving may resume after satisfactory treatment of symptoms.</p> <p>Unrestricted licence will be reinstated if person remains free of symptoms.</p>	<p>An unrestricted licence may be issued if balance problems or episodes are rare, or never incapacitating for driving.</p> <p>Reviews required every 2 – 5 years.</p> <p>Those experiencing recurring or incapacitating episodes, but not in past 1 – 3 months may drive with medical practitioner approval. Reviews required every 6 to 12 months.</p> <p>Restricted from driving if balance problems are chronic and incapacitating.</p>	<p>Desist from driving if vertigo impairs driving ability & occurs suddenly.</p> <p>May resume driving when treated successfully.</p>	Licence denial if vertigo attacks are unexpected & impair safe driving.
Benign Paroxysmal Positional Vertigo	Not specifically addressed.	<p>No licence restrictions if no symptoms are experienced when upright.</p> <p>Desist from driving if symptoms are present in the upright</p>	Not specifically addressed.	<p>An unrestricted licence may be issued if balance problems or episodes are rare, or never incapacitating for driving.</p> <p>Reviews required</p>	<p>Desist from driving if vertigo impairs driving ability & occurs suddenly.</p> <p>May resume driving when treated successfully.</p>	Not specifically addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
		position.		<p>every 2 – 5 years.</p> <p>Those experiencing recurring or incapacitating episodes, but not in past 1 – 3 months may drive with medical practitioner approval. Reviews required every 6 to 12 months.</p> <p>Restricted from driving if balance problems are chronic and incapacitating.</p>	Some people may only be temporarily affected by vertigo & may only need to pull over to the side of the road until sufficiently recovered.	

Table C.13 Commercial licensing guidelines for drivers with visual conditions

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Visual Problems	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Visual Acuity (assessed using Snellen chart or similar)	<p>Minimum visual acuity of 6/9 with both eyes open for class I, II, III & IV.</p> <p>Minimum of 6/30 for the weaker eye.</p>	<p>Minimum visual acuity of 6/9 in better eye or minimum visual acuity of 6/18 in either eye is required.</p> <p>Conditional licence may be issued if:</p> <ol style="list-style-type: none"> 1. Meets the standard with use of corrective lenses. 2. Underlying conditions are considered. 3. If visual acuity is less than 6/18 in worst eye BUT is at least 6/9 in the better eye. <p>Periodic review required.</p>	<p>Minimum visual acuity of 6/9 in better eye & 6/12 in weaker eye is required (with or without corrective lenses) AND minimum visual acuity in each eye of 3/60 without corrective lenses.</p>	<p>Unrestricted licence issued if person has 20/40 in better eye.</p> <p>May hold a restricted licence if medical recommendation obtained and 20/40 in the stronger eye. Medical Advisory Board approval is also required. Review required every 2 years.</p>	<p>Minimum visual acuity in both eyes together of 6/9, with or without corrective lenses.</p>	<p>Minimum visual acuity of 0.8 in better eye & 0.5 in the weaker eye required.</p> <p>If corrective lenses are required to meet visual acuity standards, the lenses must not exceed a strength of “8 dioptres in the meridian with the highest refraction” (p6). If contact lens can be conveniently used, this requirement is not applicable.</p>
Visual Field Defect	<p>Visual field defects must be fully assessed by an optometrist or ophthalmologist.</p> <p>“150 continuous degrees along the horizontal meridian & 20 continuous degrees above & below fixation with</p>	<p>A conditional licence may be issued if:</p> <ol style="list-style-type: none"> 1. No significant visual field loss that may impair driving ability. 2. Meets minimum requirements for binocular visual field. 3. Any other underlying conditions are considered. 	<p>Must possess normal binocular field of vision (i.e. any defects in the field of one eye is compensated for by the other eye).</p>	<p>Unrestricted licence issued if the person has:</p> <ol style="list-style-type: none"> 1. “Monocular visual fields 120 degrees in each eye”. (p52) 2. “Binocular visual fields 70 degrees to the right & left in the horizontal meridian”. (p52) 	<p>Minimum visual field requirement must be met – i.e. “a binocular horizontal field of 140 degrees” with “no significant pathological defect encroaching within 20 degrees of the point of fixation”.</p>	<p>The field of vision must be normal.</p> <p>Exceptions: If one eye has a visual field defect that is limited in depth & extent AND it is completely compensated for by the better eye.</p> <p>SNRA to be consulted where doubt exists.</p>

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Visual Problems	<i>CCMTA (2009)</i>	<i>Austroroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
	both eyes open”.	Periodic review required.		A conditional licence for intrastate travel may be issued if the person has “at least 120 degrees in each eye” (p52). OR A conditional licence may be renewed only if the person has “at least 120 degrees total for both eyes” (p52). Approval by Medical Advisory Board required. Review required every 2 years.		
Monocular Vision (loss of vision in one eye)	Recent loss of sight in one eye may require a few months for adaptation to occur in order to adequately judge distance.	Requirements are the same as for visual acuity (above).	May not be licensed if has complete loss of vision in one eye or visual acuity is less than 3/60 in that eye.	A conditional licence for intrastate travel may be issued by the Medical Advisory Board in some cases.	Generally considered unfit to drive. Exceptions may be considered. All requests must be supported by optometrist or ophthalmologist. Must demonstrate that vision in the good eye meets combined visual acuity & visual field test criteria. Good eye must be free of disease which impairs driving ability. Probable licence condition requiring external rear vision	Licence denial or disqualification.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Visual Problems	<i>CCMTA (2009)</i>	<i>Austroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
					mirrors on both sides of vehicle. May be required to undergo a practical driving test.	
Diplopia (Double vision)	Referral to optometrist or ophthalmologist required if diplopia occurs within the central 40 degrees of gaze. May resume driving if condition is rectified with patch or prism. Must meet visual acuity & visual fields criteria. A 3-month adjustment period is required prior to driving.	Persons with diplopia (except physiological diplopia) when gazing at objects that are within 20 degrees of the primary direction of gaze do NOT meet the standards required for an unconditional licence.	Permanent licence revocation or refusal if diplopia cannot be overcome. Patches are not acceptable.	May only be licensed if medical recommendation obtained.	Refrain from driving if diagnosed with diplopia. A licence may be issued if diplopia is “resolved” or an optometrist or ophthalmologist issues a favourable report.	No double vision, in any direction of the gaze, is acceptable.
Night Blindness	Driving may need to be restricted to the daytime. No standardised tests are available at present.	No specific standard.	Must meet visual acuity and visual field requirements (as above). Cases will be considered individually.	No specific standard. However, some cases may be recommended to drive during daylight only.	Person is generally considered as being unfit to drive. A conditional licence may be issued if an optometrist or ophthalmologist issues a favourable report. Probable restriction of driving during daylight hours.	Licence disqualification or denial if person has total night blindness or night vision is seriously limited.
Colour Vision	No required standard.	No restrictions.	No restrictions.	Must be normal i.e.	No restrictions.	Not addressed.

Disorder	Canada	Australia	U K	USA	NZ	Sweden
Visual Problems	<i>CCMTA (2009)</i>	<i>Austrroads (2006)</i>	<i>Drivers Medical Group, Swansea (2008)</i>	<i>Utah Driver License Division (2006)</i>	<i>Land Transport Safety Authority (2002)</i>	<i>Swedish National Road Administration (1999)</i>
Defects	Driver must be able to discriminate among traffic lights.	Doctors should counsel drivers of difficulties in detecting red lights eg brake & traffic lights.	DVLA notification not required.	must be able to recognise red, green & amber lights to drive commercial interstate licence.		
Cataracts	Assessment by an ophthalmologist or optometrist recommended, if cataracts are suspected.	Regular monitoring of vision required. Must meet visual acuity & visual field standards.	Must satisfy visual acuity standards (above). Must be able to read car number plates in the presence of of glare.	Must meet visual acuity & visual fields standards.	Restrictions may be necessary due to glare or vision difficulties eg driving restricted to daylight hours only.	Not specifically addressed.
Glaucoma	Assessment by an ophthalmologist or optometrist recommended, if glaucoma is suspected.	Regular monitoring of vision required. Must meet visual acuity & visual field standards.	Normal field of vision is required i.e. any areas of defect are compensated for by the field of the other eye.	Must meet visual acuity & visual fields standards.	Must meet visual field requirements.	Not specifically addressed.