

**Driving Performance of Older Adults with Early Dementia with Lewy Bodies or Early
Alzheimer's Disease**

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ABSTRACT

Little is known about the specific cognitive impairments that may be the cause of the reported increased crash rate in individuals with early dementia. Though, it is widely accepted that attention, visuospatial and perceptual abilities are central in being able to operate a vehicle safely. This study had three objectives. The first was to clarify the neuropsychological profile, with an emphasis on attention, visuospatial and perceptual abilities, of individuals with early dementia with Lewy bodies (DLB), the next was to examine the driving performances of two groups of individuals with early dementia (i.e., early Alzheimer's disease, AD, and early DLB) and the last was to examine the degree of association between neuropsychological impairments and driving impairments in hopes of predicting poor driving outcomes. Fifty-six participants were recruited from three groups; 20 individuals diagnosed with early AD, 15 individuals diagnosed with early DLB and 21 healthy age-matched controls. All participants were administered the following neuropsychological tests: the Mini-Mental Status Exam (MMSE), the Dementia Rating Scale (DRS-2), the Boston Naming Test (BNT), the Test of Everyday Attention (TEA), the Visual Object and Space Perception Test (VOSP) and the Useful Field of View (UFOV). Additionally, a simulated driving task was completed, with data being collected through primary measures recorded by the simulator as well as an experimenter based driving assessment using a demerit-point test. Results indicated that individuals with early DLB were found to be most impaired in their visuospatial abilities, selective and divided attention abilities, and were found to have significant cognitive fluctuations. Driving performances confirmed that drivers with early dementia were at greater risk for motor vehicle collisions (MVC) and they were found to commit a significant number of driving errors during the driving simulation. Finally, this study was able to demonstrate that in drivers with early AD, attentional impairments

were the strongest predictors of driving impairment, whereas in drivers with early DLB, visuospatial impairments were indicative of driving impairment.

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INTRODUCTION

Driving in our society.

Transportation is an essential need in our society that is tied to acquiring one's essential needs (i.e., shelter and food), maintaining successful employment, taking care of dependents and much more. Transportation is also necessary for maintaining social interactions as well as having a high quality of life (Steg & Gifford, 2005). In Europe, public transportation, walking and bicycling are common traveling means. However, in Canada, Australia and the United States, most transportation is completed by automobile, either as a driver or passenger (OECD, 2001). In Canada, people spend approximately 60 to 80 minutes a day traveling and often make up to three to four trips daily, and this time can increase substantially depending on the distance of a home to the city centre (Environment Canada, 1996). Historically, our reliance on automobiles has changed substantially with a sharp increase following WWII, due to lower fuel prices, higher household incomes, smaller sized households, and women entering the workforce (Environment Canada, 1996). This change is well illustrated in vehicle ownership records in Canada, which indicates that in 1951 there were five individuals for every vehicle registered, whereas recent estimates demonstrate that there are less than two individuals per registered vehicle (Statistics Canada, 2006).

In Canada, as in most industrialized countries, there is a growing tendency for the population to congregate in large city centres, where there is better access to public transportation (OECD, 2001). Regardless of this trend, dependence on automobiles increased between 1992 and 2005. In fact, the General Social Survey (2005) demonstrated that people aged 18 and over who used their automobiles exclusively for all of their transportation needs rose

from 68% in 1992 to 74% in 2005. This trend may be explained by the fact that many residents of metropolitan areas live at a significant distance from the city centre in areas called the suburbs. “Suburbanisation” is a growing trend in most large North American cities and this urban trend is also increasing, though at a slower rate, in European and Asian countries (OECD, 2001). In moving away from urban and rural areas and into suburban dwellings, families had larger layouts than urban apartments, had large backyards, and a garage for an automobile. A private home in the suburbs became a standard of successful living for North American families and resulted in an increased reliance on automobiles (Coughlin & D’Ambrosio, 2012).

In addition to geographic differences in reliance on automobiles, there are also sex and age differences. The General Social Survey (2005) demonstrated that on any given day in 2005, 81% of Canadian men aged 18 years and over made at least one trip behind the wheel of an automobile, in comparison to only 66% of women. This sex difference remained constant even after controlling for all other factors. In addition, women were found to be much more likely to use public transportation in comparison to men. Age differences were also found, where middle aged and older adults were more likely to use an automobile in comparison to younger adults. In fact, baby boomers (i.e., those born between 1946 and 1964) were particularly likely to exclusively rely on their automobiles as their mode of transportation (General Social Survey, 2005).

The baby boomer generation aging into older adulthood will represent a shift in the way mobility is viewed in this population. In 2011, the oldest members of this generation turned 65, which is typically considered the age of retirement. Yet given current societal trends, researchers anticipate that many will not retire at this age. They will continue to work and play vigorously, and they will continue to drive as they have done their entire lives. In 2021, the oldest members

of this generation will turn 75, typically the age where age associated changes start to impact mobility (Coughlin et al., 2012). This generation will most likely continue to demand a high level of mobility, including the use of their vehicles, and this will be a significant change from previous generations (Coughlin et al., 2012). This trend is demonstrated by the fact that in 1940 less than 6% of drivers in the United States were 60 years of age and older, whereas today older drivers represent more than 25% of drivers. The number of older drivers is expected to continue to increase and it is estimated that in 2050, older drivers will represent 40% of all drivers (National Center for Statistics and Analysis, 1995).

Certainly there are many benefits associated with driving an automobile, including better control of transportation timing, widespread accessibility of locations, better access to employment and essential needs, increased social contact, and a sense of autonomy and independence. In fact, Carp (1988) draws an important connection between mobility and quality of life, stating that “well-being depends on success in meeting life-maintenance and higher-order needs. Satisfaction of any need depends on congruence between the need and the resources for meeting it. Mobility is a key factor in determining congruence, because community services and facilities are irrelevant if they are inaccessible.” Thus, even though mobility is often measured in the number of trips an individual undertakes, the concept may be more related to the ability to access services and social interaction. Though there are many modes of transportation, for reasons mentioned above, the automobile has become the preferred mode (Coughlin et al., 2012).

Collision risk.

An increase reliance on automobiles does not come without a cost. Costs associated with driving an automobile include risk of collision, injury, disability and fatality. A Transport

Canada (2001) report indicated that from 1988 to 1997 fatal crashes accounted for 0.4% of all collisions, while injury inducing crashes accounted for 24.1% of all collisions, and the majority of fatalities were drivers of motor vehicles accounting for 50.4% of all fatalities. Interestingly, recent reports have demonstrated that the number of fatalities and serious injuries on Canada's roads are decreasing despite the increasing number of vehicles on the road (Transport Canada, 2010). This report found that the rate of fatalities per 100 000 population was 6.5 in 2010. Despite a reduction in vehicle-related fatalities in recent years, reliance on automobiles carries a risk of potential injury and fatality.

Increased reliance on automobiles for transportation also carries a risk for property damage which is associated with financial and social costs. Transport Canada (2001) demonstrated that collisions resulting in only property damage consisted of 75.5% of all MVCs. In Canada, this property damage resulted in \$25.1 billion in financial costs and \$17.9 billion in social costs. Personal injury and fatality represents the largest component of social costs at about \$15 billion, with \$11 billion accounted by the costs of fatalities. Other major contributors to social costs involved in collisions include property damage and other losses normally paid out through insurance, traffic delay costs, tow truck services, fire department response, police services, and many more (Vodden, Smith, Eaton, & Mayhew, 2007). Our reliance on automobiles holds both benefits (e.g., freedom, mobility, access to services, etc) and costs (e.g., mortality, morbidity, social, financial, etc). However, when we examine collision risk it is evident that not all subpopulations are equivalent.

Research studies examining the risk of motor vehicle collisions (MVC) have demonstrated multiple driver characteristics as being causal, including male gender, decreased cognitive abilities, and driving inexperience, though the most researched driver characteristic is

the driver's age (Begg & Langley, 2001; Vaez & Laflamme, 2005; Valcour, Masaki & Blanchette, 2002). In fact, the age group most frequently associated with a higher risk of MVC's are young adults (Williams, 2003), whereas the age group least likely to be cited are older adults (i.e., over the age of 65 years). Doherty, Audrey, and MacGregor (1998) examined police records in 1998 for Ontario, and they concluded that drivers 16-19 years of age had a higher risk of MVC than drivers over the age of 20 years. Similarly, a National study examined self-reported MVC injuries from the National Population Health Survey (NPHS) and police records from Transport Canada's Traffic Accident Information Database (TRAID) and concluded that adolescents were at greatest risk of injury as a result of a MVC (Roberts, Vingilis, Wilk & Seeley, 2008). Several researchers have attributed the over-representation of young adults in MVC's to the tendency of young drivers to engage in risky driving behaviours (Clarke, Ward & Truman, 2005; Hatfield & Fernandes, 2009; Ulleberg, 2001).

The MVC rates for older drivers are the lowest than for any other age group. Though, an important consideration in this rate is that there are fewer older adults who are licensed to drive and as a group they also tend to drive fewer miles (Lyman, McGwin & Sims, 2001). In fact, MVC statistics by The Insurance Institute of Highway Safety (2003) demonstrated that after the age of 65 years, older adults experience an increase in their MVC risk per mile driven. This analysis also showed that by the age of 75 years, older adults are involved in as many MVC per kilometer driven as 16 year old drivers. In contrast, other researchers have demonstrated that older adults are implicated in significantly more MVC's than younger adults (Stamatiadis, 1996; Stamatiadis & Deacon, 1997). In addition, when older adults experience a MVC, they are much more likely to be injured or killed in comparison to all other age groups, when comparing crashes of similar intensity (Evans, 1991). For example, the National Highway Traffic Safety

Administration (1997) described that in 1996 the fatality rate per million miles driven for older adults was 17 times greater than for those between the ages of 25 and 65 years. Certainly the increase in fatality rate associated with MVC in older adults can be attributed to their greater fragility (Augenstein, 2001). Other researchers have found that older adults increase in MVC and associated fatalities per mile driven may also be partially accounted by the finding that many older adults compensate for their perceived increase in driving difficulties by driving less and avoiding highways (De Raedt & Ponjaert-Kristoffersen, 2000; Festa, Ott, Manning, Davis, & Heindel, 2013; Staplin & Lyles, 1992). Researchers have determined that MVC rate per mile driven is higher for low-mileage older drivers than for high-mileage drivers (Langford, Methorst, Hakamies-Blomqvist, 2006). In addition, research has also demonstrated that highways are in fact the safest type of road, and when older adults avoid them they tend to drive on streets with intersections, which are less safe (Janke, 1991).

Older drivers also experience different types of MVC when compared to younger drivers. First, older drivers are more often involved in MVCs that occur in good weather and during daylight hours (Coughlin et al., 2012). Older drivers also account for a large share of MVC involving at least one other vehicle, and they have a smaller share of single-vehicle and speed-related MVC typically associated with young adults. A great proportion of older adults' MVC occur at intersections where the older driver is turning against oncoming traffic with right of way on the main road (Langford & Koppel, 2006; McGwin & Brown, 1999). Research has also demonstrated that older adults were usually involved in multi-vehicle crashes, especially at intersections, characterized by errors related to failure to yield right of way, difficulties merging into traffic, changing lanes, leaving a parking position, and reversing (Hakamies-Blomqvist,

1993; Langford et al., 2006). Finally, researchers also demonstrated that older drivers have a tendency to be at-fault in their MVC (Larsen & Kines, 2002; McGwin et al., 1999).

Unlike young drivers whose MVC risk may be due to inexperience and risk-taking behaviours that seems to decrease with age, older drivers' difficulties stem from perceptual errors where critical stimuli are neglected in the driving environment, resulting in a MVC (Caird, Edwards, Creaser & Hottry, 2005; Skyving, Berg & Laflamme, 2009). Older drivers' primary strategy to ease their perceived driving difficulties is to drive slower, which is a particularly ineffective strategy when driving through intersections as this is not a self-paced task. Therefore, in intersections, older drivers may be forced to perform under a time pressure that actually exceeds their capabilities (Hakamies-Blonqvist, 1993). In addition, negotiating an intersection is a complex task requiring perceptual and motor functions that are well known to decline with age. For example, while driving through an intersection, older adults have to divide their attention between scanning the environment outside of the vehicle while handling the vehicle. They must also select, sustain and focus their attention between task-relevant stimuli (Kausler, 1991). Thus, negotiating intersections likely represents a "testing of limits" for many older adults, who may have experienced some age-related cognitive decline.

Driving research implicating older adults is crucial at this point in Canadian history, because older adults constitute the fastest growing segment of the Canadian population. In 2011, it was estimated that there were 5 million Canadians over the age of 65 years, more than two thirds the population that was present in 1981 (Human Resources and Skills Development Canada, 2011). By comparison, during the same time period, the remaining age groups within the population experienced an overall growth of merely 25%. As the baby boomers age, the Canadian senior population is expected to reach 6.7 million in 2021 and a staggering 9.2 million

in 2041. In fact, the fastest growth in the senior population is occurring amongst the oldest Canadians. In 2041, individuals over the age of 85 years are expected to reach 4% of the overall Canadian population. This population trend is due to several factors, including reduced fertility rates, aging baby boomers, as well as increased longevity. In 1997, life expectancy at birth for Canadians reached 75.8 years for men and 81.4 years for women. Life expectancy is expected to continue to grow, reaching 81 years for men and 86 years for women in 2041 (Health Canada: Division of Aging and Seniors, 2002). As the Canadian senior population increases, so too will the number of older individuals driving.

Reasons for increased collision risk among older adults.

Concerns about older adults' ability to drive emerged in the scientific literature and became a social issue in the late 1960s and early 1970s (Coughlin et al., 2012). At that time, the focus was on age-related safety problems and functional decline of older drivers. The main safety measure involved attempting to screen for high-risk older drivers so as to remove them from the roads. Research in the 1980s and 1990s focused on the epidemiology of MVCs among older adults in order to better understand their causes and prevent their occurrences. Concerns about older adults' ability to drive was reconsidered and shifted from safety to mobility (Evans, 1991). At this time, the driver's environment was also investigated to see if it could be improved to reduce overall driving risk. Research in the 1980s and early 1990s also attempted to explain the nature of older drivers' difficulties and most came to the conclusion that there was no straightforward answer. In fact, gerontological research demonstrated that individual variance on most performance measures increased significantly with age. Other research studies also demonstrated that certain groups of older adults posed a specific safety concern, as opposed to the entire population of older adults (OECD, 2001). In particular, research since the 1990s

focused on studying high-risk sub-groups of the older driver population (Hu, Trumble, Foley, Eberhard, & Wallace, 1998).

Certainly the 20th century has been a time of advancement in most domains, including the improvement of health across all age groups in all industrialized countries. These improvements in health have brought a significant increase in life expectancy, from just 47 years in 1900, to approximately 80 years in 2000 (He, Sengupta, & Velkoff, 2005). Though the current generation of older adults can expect to live longer and have a generally healthy life compared to previous generations, this will not be without chronic conditions and disability in later life. Recent estimates of the older adult population have shown that 80% have at least one chronic condition and 50% have at least two (Coughlin et al., 2012). The most common chronic conditions include hypertension, heart disease, stroke, diabetes, cancer, and arthritis (Coughlin et al., 2012). In addition, current estimates suggest that approximately one third of older adults have a disability of some kind (OECD, 2001). Chronic conditions in the older adult population invariably have an impact on quality of life, overall functioning and the ability to live independently.

The most common chronic conditions in the older adult population that were found to have an impact on driving abilities include visual conditions, cardiovascular disease, cerebrovascular conditions, insulin-dependent hypoglycaemia, reduced memory, cognitive skills and processing, dementia, mental illness, severe muscular and skeletal disorders, loss of upper body strength, loss of lower body strength, and neurological disorders (Fildes, Fitzharris, Charlton, & Pronk, 2001). Older adults who have one or more of these conditions are considered medically-at-risk drivers. Medically-at-risk drivers are defined as individuals, regardless of age, who have at least one medical condition that could have an impact on their ability to drive safely (Charlton et al., 2004; Diller, Cook & Leonard, 1998; Dobbs, 2005; Vernon & Diller, 2002). McGwin,

Sims, Pulley and Roseman (1999) reviewed records of older adults involved in at-fault MVC from the Alabama Department of Public Safety for the 1996 calendar year. They determined that the presence of a chronic medical condition in the older adult population increased the risk of being involved in an at-fault MVC when compared to their healthy counterparts. Thus, it is not the entire older adult population that is at higher risk of MVC but a specific subset of medically-at-risk older adults. Medically-at-risk older adults fall broadly into three main categories of ailments based on their cause, including physical, sensory and cognitive conditions.

Physical conditions can impair driving due to their impact on gross and fine motor skills. Physical chronic conditions are often called musculoskeletal disorders. Due to the nature of age-related changes that occur in bones, muscles, tissues and internal organs, older adults are particularly vulnerable to a variety of disorders and degenerative diseases that affect the musculoskeletal system. Musculoskeletal disorders that impact driving significantly in the older adult population include osteoarthritis and rheumatoid arthritis. A study of drivers with musculoskeletal disorders found that these individuals had difficulty with various manoeuvres whilst driving their vehicle. In particular, it was found that drivers with rheumatoid arthritis had the most difficulties steering and cornering, whereas drivers with osteoarthritis had the most difficulties with reversing their vehicle (Jones, McCann, & Lassere, 1991). Whether this increased difficulty with certain driving manoeuvres translates into an increased MVC risk is unclear. One study concluded that there were no significant differences in the overall risk for MVCs between drivers with musculoskeletal disorders and healthy controls (Koepsell, Wolf, McCloskey, 1994). However, other researchers have concluded that drivers with musculoskeletal disorders are in fact at higher risk for at-fault MVC than healthy controls (McGwin, Sims, Pulley & Rossman, 2000, Vernon et al., 2002). Thus, it seems that drivers with musculoskeletal

disorders are likely to be at higher risk for at-fault MVC, though their overall MVC rate may not be any different than the general population. In the past few decades, there have been significant improvements in the adaptation of vehicles to meet the needs of drivers with physical handicaps so that these drivers can continue to drive safely (Shaheen & Niemeier, 2001).

There are several sensory chronic disorders that can develop with age, including visual, auditory and tactile conditions, though hearing and tactile conditions do not necessarily have a significant impact on driving. In fact, in Canada, being legally deaf restricts commercial licenses but there is no restriction on a passenger vehicle license (i.e., G license in Ontario). However, visual disorders cause the most impact on driving in the older adult population. Specifically, it is estimated that vision may be responsible for 90 to 95 percent of the sensory input necessary for driving (Hills, 1980; Shinar & Scheiber, 1991). The most common vision disorders that have a negative impact on driving include refractive errors, cataracts, glaucoma, age-related macular degeneration, and diabetic retinopathy (Vision Australia Foundation, 2002). Effective surgical treatment is available for the treatment of refractive errors and cataracts when caught early, though glaucoma, macular degeneration and diabetic retinopathy can only be medically managed. Some studies have studied the risk of being involved in a MVC when suffering from a untreatable visual disorder. For example, Owsley, McGwin and Ball (1998) found that drivers diagnosed with glaucoma were 3.6 times more likely to be involved in an injurious MVC, whereas drivers diagnosed with macular degeneration were 3.3 times more likely than healthy drivers. It is clear that visual sensory disorders can have a significant negative impact on driving, though research has identified that cognitive disorders have the most severe impact on driving abilities (Charlton et al., 2004; Diller et al., 1998; Dobbs, 2005; McCracken, Caprio Triscott, & Dobbs, 2001; Vernon et al., 2002).

The risk of MVC among individuals with disorders that impair cognition has been shown to be comparative to the risk of driving a vehicle while impaired on alcohol. , Unlike intoxicated drivers, drivers with cognitive deficits are impaired 24 hours a day, 7 days a week (Diller et al., 1998; Dobbs, 2008; Krüger, Kazenwadel, & Vollrath, 2000). Dementia and cerebrovascular accidents are the most common causes of cognitive impairment in the older adult population. Drivers with cerebrovascular accidents are not a homogenous group and thus difficult to study in terms of their risk for MVC. In fact, they can range from debilitating impairment (i.e., cognitively ravaged and functionally paralyzed) to virtually no impact (i.e., no difference on neuropsychological testing). Dementia is the most common cause of cognitive impairment in the older adult population (Gogia & Rastogi, 2009). Once again, drivers with dementia are not a homogenous group as they start off with mild cognitive impairments and eventually develop more serious impairment (Gogia et al., 2009). Research has demonstrated that on average, drivers with dementia continue to drive for approximately 4 years after the onset of their symptoms (Hopkins, Kilik, Day, Rows, & Tseng, 2004). In addition, it has also been found that one in four Canadians with serious cognitive impairments continue to have a valid driver's license and continue to drive regularly (Bess, 1999). Thus, there seems to be some difficulty in identifying drivers who are medically-at-risk cognitively.

With the review of the previous literature it is clear that the older driver population is not uniform. In fact, the research points out that the medically-at-risk older adults pose the highest risk of MVC. Older drivers who are medically-at-risk typically suffer from physical, sensory or cognitive conditions. However, older drivers suffering from cognitive disorders, more specially, dementia, pose the highest risk. A Canadian researcher estimated that the number of drivers with dementia in Ontario will increase by 3 times between 2000 and 2026 (Bess, 1999). With such

population trends, it has become increasingly important to study older drivers with dementia in order to identify their specific challenges and see how these might translate to difficulties on the road.

Cognitive abilities involved in driving.

By reviewing the literature on medically-at-risk older adults, it becomes clear that one's cognitive abilities are critical in being able to drive safely. Driving is considered to be a complex and dynamic task involving primarily cognitive (e.g., attention), perceptual (e.g., visual perception) and psychomotor processes (e.g., reaction time). Various driving models have been proposed to help explain the behavior of drivers (e.g., Anstey, Wood, Lord & Walker, 2005, Bekiaris, Amditis & Panou, 2003; Fuller, 2005; Hancock & Berwey, 1997; Galski, Bruno & Ethle, 1992; Grandenigo, 2002; Linstrom-Forneri, Tuokko, Garrett & Molnar, 2010; Michon, 1985; Salvucci, 2006; Sheridan, 2004). In order to understand the complex demands involved in driving as an older adult it is first essential to review one of the most influential driving models as well as specific cognitive abilities associated with driving successfully.

Michon's Model (1985):

Michon (1985) created one of the most influential models in the driving literature on older drivers (Unsworth, Lovell, Terrington, & Thomas, 2005; van Zomeren, Brouwer, & Minderhoud, 1987). Michon's driving model involves three conceptual levels organized in a hierarchical fashion, including the strategic, tactical and operational level. The strategic level refers to behaviors that are spread over a long period of time (i.e., from minutes to days). For example, when planning a road trip a route is selected based on its difficulty, the cost of the trip and consideration of weather conditions. The second level is the tactical level, which includes

behaviors deployed in driving situations that have windows of 5 to 60 seconds. For example, maintaining and controlling speed, negotiating a curve, and overtaking another car. Finally, the operational level corresponds to behaviors generated to maneuver the vehicle in real time and is fairly automatic (i.e., time window of approximately .5 to 3 seconds). An example of this would be steering the wheel, applying gas or brake pressure, as well as being able to attend, process and respond to stimuli in the environment. These levels are organized in a hierarchical fashion meaning that difficulties with the operational level (i.e., lowest level) will have trickle down effects on both the tactical and strategic levels.

When Michon's driving model (1985), including the strategic, tactical and operational level, is applied to the cognitively medically-at-risk older drivers, it becomes obvious that the majority of these drivers experience their difficulties at the operational level, which then impacts the tactical and strategic levels. Using this logic, drivers who compensate for operational difficulties by engaging in strategical and tactical compensation should improve their driving performance. Unfortunately, this logic has never been tested with cognitively medically-at-risk older drivers, but a study examining unsafe older drivers found that when older drivers engaged in strategical and tactical compensation they could in fact improve their on-road safety (De Raedt et al., 2000). This particular study brings strength to the overall logic behind Michon's model.

Since the operational level of Michon's model (1985) seems to be associated with difficulties present in cognitively medically-at-risk older adults it should be examined in further detail. The operational level is comprised of basic cognitive abilities. Basic cognitive abilities include perception (i.e., recognition and interpretation of sensory stimuli), attention (i.e., ability to sustain concentration on an object, action, or thought and ability to manage competing demands), memory (i.e., short-term and long-term memory), motor (i.e., ability to mobilize

muscles and bodies and ability to manipulate objects), language (i.e., ability to generate verbal output and translate sounds into words), and visual and spatial processing (i.e., ability to process incoming visual stimuli and understand spatial relationships between objects). The tactical and strategical levels include higher order cognitive abilities. Higher order cognitive abilities include executive functions (i.e., theory of mind, anticipation, problem-solving, decision making, working memory, emotional self-regulation, sequencing, and inhibition). Executive functions need intact basic cognitive abilities in order to function effectively (Lezak, Howieson, & Loring, 2004). Thus, Michon's model (1985) is quite logical in that if the operational level is dysfunctional (i.e., basic cognitive abilities), then the tactical and strategical levels will also suffer as a result (i.e., higher order cognitive abilities).

Driving a vehicle requires a seamless interaction of numerous cognitive abilities, including attention, perceptual and motor skills, memory, and decision-making. The research on the cognitive abilities required to drive safely is based on studying populations with specific cognitive deficits (Anderson, Rizzo, Shi, Uc, & Dawson, 2005). Though the research in this field of research is limited, there is some data to suggest that certain cognitive abilities may be more crucial than others in the ability to operate a vehicle safely. In fact, deficits in attention and visual processing speed have been found to be associated with a higher MVC risk (Ball & Owsley, 1993; Ball et al., 1993; Duchek et al., 1998; Parasuraman & Nestor, 1991). Speed of processing has also been studied through simple (i.e., responding to a stimulus as fast as possible) and choice reaction time (i.e., responding to a choice of stimuli as fast as possible). Marottoli and colleagues (1998), for example, found that there were small associations between simple reaction time and self-report MVC, and moderate associations for choice reaction time.

Finally, memory, poor recall and recognition scores have also been moderately correlated with driving outcomes (Odenheimer et al., 1994).

Current licensing regulations in Canada.

Thus far, a general approach to the review of the literature has been taken. General driving trends and the MVC risks for various populations including the increased risk found in older adults were presented. Following this, the reasons for the increased MVC risk in the older adult population were reviewed, clarifying that in fact the cognitively medically-at-risk older adult population, which are typically individuals diagnosed with dementia, were the culprit for a great portion of that increased risk. Finally, the cognitive abilities implicated in driving safely were reviewed. As previously mentioned, with the current population trends, it has become increasingly important to study older drivers with dementia in order to identify their specific challenges and see how these might translate to difficulties on the road. More specifically, since the literature has demonstrated that the majority of older adults diagnosed with dementia continue to drive for an average of 4 years following diagnosis (Hopkins et al., 2004), it would be especially important to concentrate efforts on understanding the driving abilities of older adults with early stage dementia. Though, before this topic is examined in greater detail the current driving legislation that is applicable to older adults with dementia should be reviewed.

In Canada, licensing decisions fall under the provincial or territorial licensing authorities. Across the 13 provinces and territories, policies and practices vary considerably with differences in renewal periods and requirements for testing (Coughlin et al., 2012). According to the AAA Foundation for Traffic Safety Driver Licensing Policies and Practices, the standard renewal interval for a driver's license varies from 1 to 5 years, with the majority of provinces and

territories requiring a 5 year interval (e.g., including Ontario). With the exception of Alberta and Ontario, all provinces continue to use the 5 year license renewal interval with older adults. Alberta and Ontario have an accelerated renewal interval (i.e., two year renewal interval) starting at the age of 80 for older adults. In addition, Ontario has a special licensing program, the Senior Driver Renewal Program for drivers 80+, which requires an eye exam, a 90-minute interactive group education session, a multiple-choice test about the rules of the road and traffic signs, and at times an on-road test (i.e., if the older adult has demerit points on their driving record or if he/she has difficulty understanding the written test or group discussion). Finally, medical reports are required in order to maintain a driver's license into old age for Alberta, British Columbia, Newfoundland, Northwest Territories, Nunavut, Ontario, Quebec, and Yukon.

At-risk drivers can be reported to their respective ministry of transportation (i.e., provincial or territorial) by physicians, law enforcement personnel, or, in select areas, by family or friends of the driver. In fact, physicians in all provinces and territories are required to report drivers whom they deem at-risk, with the exception of Alberta, Nova Scotia, and Quebec. Once the at-risk driver has been reported, the driver's file and condition are reviewed and three outcomes can occur: 1) immediate revocation of the driver's license, 2) the driver's license may become restricted to certain conditions (only available in certain provinces/territories), or 3) the driver may be required to be evaluated further in order to maintain their driver's license. In most provinces, with the exception of Ontario, a restricted license may be issued. Depending on the province or territory the following restricted licenses may be available: (1) daytime/daylight only; (2) lower speed/no freeway or limited access; (3) within a specific distance from home; (4) for a specific length of time; (5) specified destinations or trip purposes; (6) passenger presence

required; (7) passenger presence prohibited; and (8) required vehicle equipment (AAA Foundation for Traffic Safety Driver Licensing Policies and Practices).

The majority of reports identifying at-risk drivers in Canada are made by physicians. Physicians are required to determine if a driver with a specific medical condition is considered at-risk. This determination is not always clear, especially when considering drivers with a cognitive condition, such as dementia. All physicians are aware that dementia eventually progresses to a point where a driver is no longer safe to drive, though the determination of the point where a driver becomes unsafe to drive can be tricky (Breen, Breen, Moore, Breen, O'Neil, 2007; Carmody, Granger, Lewis, Traynor, & Iverson, 2013; Wilson & Pinner, 2013). Added to this tricky determination is the knowledge that for many older adults, driving is highly related to their mobility, independence and quality of life (Carp, 1988; Curl, Stowe, Cooney & Proulx, 2013). Thus, more research is necessary in the field of cognition and driving, and more specifically, on the cognitive impairments that are associated with unsafe driving, in order to be able to effectively screen older adults who may be at-risk.

What is dementia and early stage dementia?

The literature reviewed has indicated that drivers with dementia were at increased risk for a MVC. Additionally, the majority of older adults diagnosed with dementia continue to drive for an average of four years following diagnosis while still in the early stages (Hopkins et al., 2004). The determination of whether or not a driver with early stage dementia is considered at-risk is typically completed by a physician. This decision can be a tricky one and can have a significant impact on the older driver's life (Carp, 1988). The following section will present research on dementia and early stage dementia.

Dementia originally meant “out of one’s mind”, from the Latin words *de* (i.e., out of) and *mens* (i.e., the mind), which of course today is known to be an inaccurate description of dementia. Dementia should be seen as a disorder of the brain. This an important point because dementia should not be seen as being under conscious control, nor due to ‘letting go’, which has been of speculation in the media and in the general public (Maj & Sartorius, 2000). With that in mind, dementia can be defined as a clinical syndrome characterized by progressive deteriorations in multiple cognitive domains that are severe enough to interfere with daily functioning (Maj et al., 2000). Dementia is often described as a disease in the literature but it is better served when conceptualized as a syndrome. The term disease infers that there is a recognized etiologic agent, an identifiable group of signs and symptoms, and consistent anatomical alterations, which is not the case in dementia. Whereas a syndrome infers that there is a collection of signs and symptoms known to frequently appear together and that there is an unknown etiology (Maj et al., 2000). In dementia, brain pathology has been identified, however there is no definitive causal link between the pathology and the symptoms commonly associated with dementia.

Thus far, diagnostic manuals have had little success at creating diagnostic criteria for dementia that are useful. The International Classification of Diseases (10th Revision; ICD-10) diagnostic guidelines for dementia are nearly identical to the criteria found in the Diagnostic and Statistical Manual (American Psychiatric Association, 2000) as they both state that the following should be present:

1. A decline in memory to an extent that it interferes with everyday activities, or makes independent living either difficult or impossible.
2. A decline in thinking, planning and organizing day-to-day things, again to the above extent.

3. Initially, preserved awareness of the environment, including orientation in space and time.
4. A decline in emotional control or motivation, or a change in social behaviour, as shown in one or more of the following: emotional lability, irritability, apathy or coarsening of social behaviour, as in eating, dressing and interacting with others.

These criteria are non-specific and do not help in the differential diagnosis of dementia. In fact, there are several disorders which present with clinical symptoms that are very similar to dementia and some even co-occur with dementia. Differential diagnoses for dementia include depressive disorders, delirium, mild to moderate mental retardation, states of below normal cognitive functioning attributable to impoverished social environments and education, and Iatrogenic mental disorders due to medications. However, diagnostic criteria specific to the type of dementia have been created and are currently thought to be the gold standard and these will be reviewed in greater detail in further sections of this introduction (Sunderland, Jeste, Baiyewu, Sirovatka, & Regier, 2007). Nonetheless, clinical diagnosis of dementia is only probable, as the only fully accurate diagnosis is through an autopsy, and in order to increase diagnostic accuracy it is best to use a multi-modal approach (refer to Appendix N).

Studies of incidence and prevalence are complicated by the fact that there is no clear-cut definition of dementia. Prevalence studies demonstrate a wide range, where in individuals over the age of 65 years there is a prevalence of 6-10%, whereas, in individuals over the age of 85 years there is a prevalence of 30-50% (van der Flier & Scheltens, 2005). However, studies have demonstrated that dementia is more common in industrialized countries, which is thought to be due to longer lifespans and possible exposure to more toxins (van der Flier et al., 2005). In addition, it is widely known that women have a higher prevalence of dementia, which can

partially be explained by the estrogen hypothesis. The estrogen hypothesis posits that post-menopausal women are at higher risk of dementia because women's brains benefited from the protective properties of estrogen and that once it is gone this creates an increased vulnerability in the brain (Zarit & Zarit, 2007). In addition, women do live longer and thus live longer with dementia (Johanson & Zarit, 1995). Thus, with the current prevalence rates and the increasingly aging population, dementia will possibly become one of the biggest public health crises of the current century.

There are several types of dementia. In the dementia literature there is a clear distinction between dementia diagnosis prior and after the age of 65 years (Zarit et al., 2007). In fact, diagnosis of dementia prior to the age of 65 is thought to be much more genetically based, though a genetic predisposition certainly plays a role even at older ages (Gogia et al., 2009). In addition, there are also various types of dementia, the most common types being Alzheimer's disease (AD), dementia with Lewy bodies (DLB), and vascular dementia (VaD). Several medical conditions can also lead to the development of dementia, including Huntington's disease, Multiple Sclerosis, HIV, Parkinson's disease, Pick's disease and Progressive Supranuclear Palsy (Gogia et al., 2009). Furthermore, some conditions can mimic the symptoms of dementia in what is called a delirium, such as a high fever, dehydration, vitamin deficiency and poor nutrition. The dementia symptoms caused by delirium are reversible but the degree of reversal depends on how quickly the underlying cause of delirium is treated (Gogia et al., 2009). Dementia can also be caused by brain damage. For example, dementia pugilistica is a type of dementia that affects professional boxers and other athletes who suffer repeated concussions (Gogia et al., 2009). Through this brief review of dementia it is clear that dementia is an umbrella term for a wide

variety of pathology and clinical symptoms. For the purpose of this thesis, DLB and AD will be the focus of the literature review.

While normal aging is associated with some mild cognitive decline, some forms of cognitive impairment may be early manifestations of dementia. Most researchers agree that there is a continuum between normal aging and dementia, including an intermediate stage called Mild Cognitive Impairment (MCI) (Bennett, Schneider, Bienias, Evans, & Wilson, 2005; Albert et al., 2011; Mayo Clinic, 2012; Petersen, 2004). MCI is a clinical condition in which individuals afflicted have some impairment in cognition, such as memory, language, attention, reasoning, judgment, reading, and writing (Caselli & Tariot, 2010). The Mayo Clinic has devised a set of criteria in order to differentiate MCI from normal aging, which are currently the gold standard in diagnosis (Petersen, Stevens, Ganguli, Tangalos, Cummings, et al., 2001), and these include: (1) Memory complaint, preferably corroborated by an informant; (2) Objective memory impairment (for age and education); (3) Preserved general cognitive function; (4) Intact activities of daily living; (5) Not demented. Though the chief complaint in MCI is memory there are nonetheless two main types of MCI, including amnesic MCI and nonamnesic MCI. Not surprisingly amnesic MCI is the most common type. In amnesic MCI, memory is the dominant problem and approximately 10-15% convert to a diagnosis of AD after just one year (Petersen, Stevens, et al., 2001). In nonamnesic MCI, memory is not the predominant problem, instead other cognitive impairments are predominant, such as language, visuospatial perception, and attention. Nonamnesic MCIs convert to atypical AD at a rate of 2% per year, but the majority actually convert to other types of dementia, such as DLB or VaD (University of California Davis, 2007). Along with their being a continuum from normal aging to dementia, including an intermediate

phase of MCI, there is also a continuum in dementia, including, early, moderate and severe stages (Caselli et al., 2010).

It is estimated that approximately 80% of individuals with MCI will progress to early dementia within a 10-year period (Petersen, Smith, Waring, Ivnik, Tangalos, & Kokmen, 1999). Early stage dementia can be defined as a mild impairment in cognition that has an impact on an individual's functioning (Caselli et al., 2010). Unfortunately, MCI and early stage dementia are often equated by the media and lay people. The main difference between MCI and early stage dementia is that in early stage dementia the cognitive impairment causes functional impairment (Caselli et al., 2010). Functional impairment is measured through the assessment of activities of daily living (ADL; i.e., eating, bathing, dressing, toileting, transferring and/or continence) and/or the instrumental activities of daily living (IADL; i.e., housework, shopping, managing finances, taking medications as prescribed, and/or driving a vehicle) (Hall, Vo, Johnson, Barber, O'Bryant, 2011). Once a person is diagnosed with dementia, they are usually staged with the Global Deterioration Scale (Reisberg, Ferris, deLeon, & Crook, 1982; Refer to Appendix B). Early stages are described as stages 3 and 4 on this scale due to the fact that they demonstrate a functional impact. Due to this functional impairment in individuals with early stage dementia, there is great concern about their ability to drive a vehicle safely. Specifically, since AD and DLB are the two most common types of dementia, accounting for over 60% of all dementia diagnoses, it would be particularly important to study driving ability in this population (Lovestone, 1998). Prior to such an investigation, however, a brief review of the literature on AD and DLB is necessary.

A brief overview of Alzheimer's disease.

AD is defined as a progressive, degenerative disease of the brain, which causes thinking and memory to become seriously impaired. The medical identification of AD was fairly recent as it was first recognized in 1906 by a German physician, Alois Alzheimer. Alzheimer had a patient under his care, a female in her fifties, who died after years of experiencing memory problems, confusion, and difficulty understanding questions. After her death, he completed an autopsy and found dense deposits, now called amyloid plaques, outside and around the neurons in her brain. He linked these deposits to the symptoms he had observed in his patient. The disease was later given his name (NIH National Institute on Aging, 2012).

AD is the most commonly diagnosed form of dementia. It is not a part of normal aging, as healthy brain tissue degenerates causing a steady decline in mental abilities. AD is considered to be progressive with an insidious onset. The prevalence rates change radically depending on the sample, for example about 5% of people between the ages of 65 and 74 have AD, whereas nearly half the people over the age of 85 have AD. The average duration is approximately 10 years but includes a range of anywhere from 3 to 20 years (Hebert, Scherr, Bienias, Bennett, & Evans, 2003). The American Alzheimer's Association (2012) estimated that one in eight older Americans (i.e., over the age of 65 years) has AD. One of the most startling statistics is that approximately 15% of American older adults diagnosed with AD live alone in the community (Hebert et al., 2003). When examining prevalence a little more closely, gender differences emerge. Specifically, more women than men have AD as well as other types of dementias. In fact, almost two-thirds of Americans with AD are women (Hebert et al., 2003). This increased prevalence in women is likely accounted by the fact that women live longer and not because they are at particular risk. In addition, epidemiological studies demonstrate that having fewer years of

education is associated with a higher risk of developing AD and other dementias (Evans et al., 1997; Evans et al., 2003; Fitzpatrick et al., 2004; Gurland et al., 1999; Kukull et al., 2002; Plassman et al., 2007; Stern, Gurland, Tatemichi, Tand, Wilder, & Mayeux, 1994). Researchers theorize that having higher levels of education serve as a “cognitive reserve” to compensate for changes in the brain that are a result of dementia or AD (Roe, Xiong, Miller, & Morris, 2007; Stern, 2006). In terms of prevalence among racial groups, the majority of older adults with AD are non-Hispanic whites, though this is likely because of their longer longevity (Dilworth-Anderson, Hendrie, Manly, Khachaturian, & Fazio, 2008). When mortality is controlled, it becomes clear that older African-Americans and Hispanics are proportionally more likely to be diagnosed with AD than white older individuals (Dilworth-Anderson et al., 2008; Manly & Mayeux, 2004). This racial difference may be accounted by socio-economic factors (i.e., lack of adequate nutrition and education).

According to the American Alzheimer’s Association Report (2012), AD has become the sixth leading cause of death in the United States. In fact, between five and 15 percent of all deaths among older adults can be attributed to AD (Aguero-Torres, Fratiglioni, Guo, Vitanen, & Winblad, 1999; Ganguli, Dodge, Shen, Panday, & DeKosky, 2005). Individuals with AD rarely succumb to the illness, rather they typically die from related causes such as immobility, swallowing disorders and malnutrition. Pneumonia is the most common cause of death as individuals with AD have difficulty with their activities of daily living (ADLs) and thus forget to chew their food and swallow it, causing an aspiration of food (Alzheimer’s Society of Canada, 2007).

Brain Pathology:

Prior to discussing the brain pathology associated with AD it is important to state that there are several different types of AD that can be easily divided into two categories, including typical and atypical presentation. Typical AD represents the vast majority of AD diagnoses and classically presents as an amnesic condition marked by pathology in the medial temporal lobe (Galton, Patterson, Xuereb, & Hodges, 2000). Atypical AD can include the following types: progressive visual dysfunction, progressive biparietal syndrome or a progressive aphasia (Galton et al., 2000). These atypical types of AD are caused by brain pathology being marked in other areas of the brain. For the purpose of this thesis only typical AD will be discussed.

There are three main types of brain pathology commonly associated with AD, these include β -amyloid plaques, neurofibrillary tangles, and neuronal degeneration (Caselli et al., 2010). β -amyloid plaques (i.e., senile plaques and neuritic plaques) are one of the hallmarks of AD. These plaques accumulate between the nerve cells of the brain (i.e., neurons) and cause a disruption in communication between neurons. Older plaques will also attach themselves to neurons causing the eventual death of the cell as they can be seen as being parasitic in nature. When enough neurons die this can cause brain atrophy, which is a hallmark of AD. Amyloid is a general term for protein fragments that the body produces normally. β -amyloid is a fragment of a protein that is snipped from another protein called Amyloid Precursor Protein (APP). In a healthy brain, these protein fragments would be broken down and eliminated but in AD the fragments accumulate to form hard and insoluble plaques. It is unclear as to why this occurs in AD, however, it is well known that the APP is coded by chromosome 19, which has three variants, APOE 2, 3, and 4. In a famous study conducted by David Snowdon, on a group of religious sisters, it was found that the APOE4 gene is a risk factor for AD, whereas the APOE2

gene is a protective factor (Maj et al., 2000; Snowdon, 2003). An individual with one allele of the APOE4 gene has 3 times the risk of developing AD, whereas an individual with two alleles of the APOE4 gene increases their risk 10 times.

Neurofibrillary tangles are another type of pathology that are found in the brains of individuals with AD. Tangles consist of insoluble twisted fibers that are found inside neurons. They primarily consist of a protein called Tau, which forms part of a structure called a microtubule. Microtubules are responsible for transporting nutrients and other important substances from one part of the neuron to another. In AD, the Tau protein is abnormal and the microtubule structures collapse. This collapse causes the neuron to eventually die and with enough neuronal loss this will cause brain atrophy (Zarit et al., 2007).

In AD, neuronal degeneration does occur and is the main culprit for this degeneration are the plaques and tangles which cause neuronal death (Caselli et al., 2010). When there is accumulation of neuronal death this results in brain atrophy. A common finding emerges when the brains of individuals with AD-related brain atrophy are examined using CT scans. Specifically, atrophy is more severe in the medial temporal lobe which is consistent with the severe memory impairment that is associated with this disease. Unfortunately, although there are specific pathological changes in the brains of individuals with AD there is no definitive causal relationship between the presence of pathology and AD. In fact, plaques are commonly found in the normal aging brain but not to the same degree (Zarit et al., 2007).

Clinical Presentation and Diagnostic Criteria:

AD presents insidiously and progresses in a gradual and relentless fashion. The diverse clinical features of cognitive decline associated with this syndrome reflect the dysfunction of

widespread areas of the neocortex (Caselli et al., 2010). Memory loss is the most common presenting complaint in AD (Caselli et al., 2010). In fact, it is widely accepted that amnesic symptoms progress very gradually for many years before impairment in any other cognitive domain, such as language, semantic memory and visuospatial function, becomes apparent (Grady et al., 1988; Hodges & Patterson, 1995; McKhann, Drachman, Folstein, Katzman, Price, & Stadlan, 1984; Welsh, Butters, Hughes, Mohs, Heyman, 1992). Initially, individuals with AD primarily have short-term memory loss, manifested by difficulty with recall of new information (e.g., difficulty recalling details of recent conversations and news items or remembering appointments) (Caselli et al., 2010). Though, from a clinically perspective, individuals with AD may appear to have difficulties with recall when observed, it is actually their difficulty to encode new material that is at fault. Additionally, individuals with AD may ask the same question repeatedly or repeat the same stories. Related to this memory loss is a chronic disorientation to time, place and person. In addition, caregivers will often describe that individuals with AD have difficulty keeping information in their mind for a short period of time, such as remembering a telephone number (Zarit et al., 2007). These difficulties are due to a breakdown of the working memory system, where information that is the immediate focus of attention is temporarily held in mind (Baddeley, 2003). Finally, remote memory, which is initially fairly preserved, declines as the disease progresses. In the later stages, previously well-consolidated and over-learned information, such as the name of a spouse or children, is also lost (Zarit et al., 2007).

Individuals with AD also demonstrate great difficulty performing ADLs and iADLs. In fact, caregivers to individuals with early AD will often describe them as being inattentive, having difficulty concentrating, and becoming easily confused. These observational difficulties with iADLs have been found to be associated with attentional deficits (Carlson, Fried, Xue, Bandeen-

Roche, Zeger, Brandt, 1999; Hall et al., 2011). In fact, several cross-sectional and longitudinal studies have demonstrated that attentional impairments are the first non-memory domain to be affected in AD (Perry & Hodges, 1999).

Language deficits are also a prominent feature of AD. The first language deficits to appear include reduced expressive output, word-finding difficulties, and vague speech. In addition, since their ability to process information is delayed, they may also take significantly longer to respond. As the disease progresses, an individual with AD becomes increasingly non-fluent and verbally disorganized, and this may lead to global aphasia (Williams et al., 2007).

Memory, attention, and language impairment are the most commonly observed deficits in AD, but there are other cognitive deficits that are commonly associated with AD. Visuospatial deficits may occur and manifest themselves through impaired driving, wandering, and difficulty with drawing figures (Caselli et al., 2010). Executive dysfunction due to pathology in the frontal lobe can result in deficits in problem solving, abstraction, reasoning, decision making, and judgment. An individual with AD may display difficulty with organizing complex tasks such as planning a day of errands (Ballard et al., 2001). Impaired judgment often renders AD patients susceptible to solicitors and telemarketers (Templeton & Kirkman, 2007). Executive dysfunction may also manifest itself through difficulty with calculations which impairs an individual's ability to handle money and finances (Ballard et al., 2001). Apraxia, or the inability to produce purposeful movements despite normal strength and coordination, leads to difficulties in operating appliances, using utensils, or dressing. Agnosia often presents later in the disease course and manifests as failure to recognize objects and people.

Finally, in addition to cognitive decline, behavioural and psychiatric symptoms are common in AD. Specifically, depressive symptoms can occur in up to 50% of individuals with AD. Delusions of the paranoid type are also very common (e.g., fears of attack from others, theft, and infidelity). Agitation and sleep difficulties can also be present and are the principle cause for the administration of anti-psychotic medications (Maj et al., 2000; Zarit et al., 2007).

The DSM-IV criteria (American Psychiatric Association, 2000) are not particularly useful in aiding the diagnosis of AD. Not only is this diagnosis non-specific but it also does not aid in the differential diagnosis in any way (i.e., AD vs. VaD or DLB). Therefore, this diagnosis is not used to diagnose AD, instead the NINCDS-ADRDA AD criteria are used as they are the current gold standard in diagnosis of probable AD (refer to Appendix M; McKhann et al., 1984). These criteria were proposed in 1984 by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association. In a multisite reliability and validity study in which clinicians from each site diagnosed 60 case summaries using the NINCDS-ADRDA AD criteria and where cases were then verified by autopsy, it was found that the criteria had good sensitivity (0.83) and good specificity (0.84; Blacker, Albert, Bassett, Go, Harrell, & Folstein, 1994).

Neuropsychological Profile of Early AD

There is a considerable amount of research on AD and early AD. The neuropsychological profile of patients with early AD has been well established. One of the earliest sites of brain pathology in AD include the medial temporal lobe (e.g., hippocampus and entorhinal cortex), which has a great impact on episodic memory, the earliest symptom of AD (Braak & Braak, 1991). As a result, individuals with early AD have difficulty learning and remembering new

information. In fact, studies have been done to characterize the type of episodic impairment in AD. They used word lists, such as the Consortium to Establish a Registry for Alzheimer Disease (CERAD) and the California Verbal Learning Test (CVLT), to demonstrate that patients with AD rapidly forget information over time, but are equally impaired on recognition and free recall when compared to their age-matched counterparts (Delis et al., 1991; Welsh, Butters, Hughes, Mohs, Heyman, 1991). Thus, this pattern indicates an impairment in consolidation rather than ineffective retrieval. This might explain why patients with early AD have difficulty recalling new information but seem to have no difficulty recalling remote memories. Episodic impairments are the first symptom to appear in early AD because these symptoms are typically present prior to a diagnosis of AD (i.e., present in amnesic MCI; Backman, Small, & Fratiglioni, 2001) and they continue to deteriorate until functional impairment is measured and AD is diagnosed.

Though language is not often thought to be greatly impacted in early AD, studies have demonstrated that individuals with early AD are often mildly to moderately impaired on tests of object naming (Bayles & Tomoeda, 1983; Bowles et al., 1987; Hodges et al., 1991; Martin & Fedio, 1983), verbal fluency (Butters et al., 1987; Martin et al., 1983; Monsch et al., 1992), and semantic categorization (Aronoff et al., 2006). Severity of impairment is related to neuropathology in the temporal, frontal and parietal association cortex (Hodges et al., 1995).

Deficits in attention are often prevalent in early AD and are related to functional deficits (Carlson et al., 1999; Hall et al., 2011). Attention can be better examined by separating the general construct into three separate attentional abilities, including selective, sustained, and divided attention, all of which are impaired in early AD. Selective attention has been found to be impaired in early AD in a number of different tasks, such as trail making (Grady et al., 1988), Stroop (Spieler, Balota, & Faust, 1996), dichotic listening (Grady, Grimes, Patronas, Sunderland,

Foster & Rapport, 1989) and visual search (Nebes & Brady, 1989). In addition, shifting attention can also be impacted in early AD. For example, research on the Stroop test was found to be particularly sensitive to minute deficits in attention (Bench et al., 1993). Perry and colleagues (1999) theorize that patients with early AD have difficulty performing on the Stroop test because they have difficulty performing in old tasks in a novel way. Thus, rather than read the word they are asked to inhibit that response and state the color of the writing. Patients with early AD have a breakdown of that inhibitory process. Similarly, patients with AD have particular difficulty dividing their attention (Corbetta et al., 1991). Finally, research on sustained attention has demonstrated that difficulties are present even in early stages of AD (Rosvold et al., 1956).

The majority of abilities that fall under executive functions are relatively preserved in early AD, with the exception of mental manipulation, which is particularly impaired in early states. In fact, patients with early AD were found to be moderately impaired on tests that require set shifting, self-monitoring, or sequencing, but not on tests that require cue-directed attention or verbal problem-solving (Lafleche & Albert, 1995). These impairments are likely due to a breakdown of the attentional system which occurs early on.

Thus, it can be seen that early AD is often characterized by marked memory deficits, particularly in episodic memory. In addition, patients with early AD have significant impairment in the attentional domain. These attentional difficulties are often the culprit of functional deficits. Patients with early AD can also have mild to moderate deficits in language and executive functions.

An overview of dementia with Lewy bodies.

Dementia with Lewy Bodies (DLB) is a neurodegenerative disease that may develop in old age, producing a combination of dementia, parkinsonism and mental disturbances in the form of hallucinations (Loy-English, 2004). The average age of onset of DLB is 67 years, with males being more affected than females (Ransmayr, 2000). The average duration of the illness is nine years, although estimates of speed and progression vary considerably from study to study (Barber, Panikkar, & McKeith, 2001). The pathological hallmark of DLB is the presence of Lewy bodies, which are spherical, cytoplasmic inclusions located within neurons. Lewy bodies occur throughout the brain in patients with DLB, however, the brainstem, subcortical nuclei, limbic cortex, and neocortex are more afflicted. This pathology can also be found in other diseases, such as Parkinson's disease (PD) and Parkinson's disease dementia (PDD), although the distribution throughout the brain is thought to be different in DLB.

DLB only accounts for 1.9% of the primary diagnoses of dementia, however, it is considered to be the second or third most common cause of dementia, depending on the diagnostic criteria used, in older adults after AD (Loy-English, 2004). Currently, the neuropathological diagnosis is made more frequently than the clinical diagnosis of DLB. According to autopsy reports, DLB actually accounts for 15-20% of all dementias (Lovestone, & Gautier, 1998). In fact, many researchers agree that further studies are required in order to enhance the clinical diagnosis of this condition (Loy-English, 2004).

The development of the concept of DLB mirrors the history of neuroscience over the last 200 years. The first contributions were based on clinical observations, which were later supplemented with pathological data of increasing sophistication. Progress accelerated very

quickly with the advent of immunohistochemical techniques, which allowed for the identification of the Lewy body protein. With the identification of the Lewy body clinicians could then go back and improve the descriptions of the clinical features and their epidemiology. More recently, an era of molecular genetics has contributed to the understanding of the condition (O'Brien, McKeith, Ames, & Chiu, 2006).

The first detailed description of a Lewy body disorder can be found in James Parkinson's essay on the Shaking Palsy in 1817. He observed and described the motor symptoms of six of his patients. The motor symptoms outlined belong to a disease that we now call PD. At the time he made no mention of the cognitive aspects of PD, this may be due to the fact that he did not have the opportunity to follow his six patients long-term. In fact, James Parkinson believed that the intellect remains uninjured. However, in the late 19th century the idea that dementia may complicate the course of PD was recognized. In 1861 Charcot and Vulpian stated that "psychic faculties" were definitely impaired in PD. It was later added that the mind becomes clouded and that memory is lost. This idea was amplified by Charcot's pupil Ball, who in 1882 followed and described the progression of PD in seven patients. Charcot and Ball's description of PD has had lasting influence on the current views of PD (O'Brien et al., 2006).

Pathological classification of PD began in 1912 with Friedrich Lewy's identification of a particular protein. Specifically, he observed intracytoplasmic inclusion bodies in the basal forebrain and dorsal motor nucleus of the vagus nerve in the brains of individuals with PD. The term Lewy body was coined by Tretiakoff in 1919, who observed Lewy bodies in the substantia nigra of patients with PD and stressed the importance of this structure being afflicted in terms of motor impairments. In 1923 Friedrich Lewy went on to describe Lewy bodies in the cerebral cortex of patients who had marked mental alterations. Since then, the presence of cortical Lewy

bodies has been widely confirmed. Specifically, it was found that significant cortical Lewy bodies was associated with DLB and Parkinson's disease dementia (PDD) but less so with PD (O'Brien et al., 2006).

Following the discovery that cortical Lewy bodies caused dementia symptoms, clinical descriptions of DLB or PDD became more precise and specific. The discovery that Alzheimer's pathology existed in 60-90% of all patients with dementia with Parkinsonism further complicated the diagnosis of DLB or PDD. In particular, researchers were unsure about which pathology should be given more importance, that is, the Lewy body or the Alzheimer-type changes. This uncertainty is reflected in the terms that were used to describe the emerging diagnosis. Some authors stressed the Alzheimer-type pathology using terms like 'Alzheimer disease with Parkinson's disease changes', 'Alzheimer's disease with incidental Lewy bodies', or 'Lewy body variant of Alzheimer's disease' (Ditter & Mirra, 1978; Hansen, Slamon, & Galasko, 1990; Joachim, Morris, & Selkoe, 1988). Others focused on the importance of cortical Lewy bodies using terms like 'diffuse Lewy body disease', 'dementia with cerebral Lewy bodies', or 'diffuse cortical Lewy body disease' (Eggerton & Sima, 1986; Gibb, Esiri, & Lees, 1987; Kosaka et al., 1984). Due to the fact that so many terms were being used to describe the same set of symptoms a certain amount of confusion was created among clinicians. Many clinicians felt that it was impossible to diagnose DLB as there was no set consensus on the data that was available.

In 1995, there was a consensus conference in Newcastle in order to reunite some of the most important researchers in the field of DLB or PDD. During this conference there was recognition of the critical importance of cortical Lewy bodies and as such the term dementia with Lewy bodies was coined (McKeith, Galasko, & Kosaka, 1996). The recognition of DLB as a specific entity, independent of AD, created a need for operational, clinically applicable

diagnostic criteria. AD pathology was recognized as a secondary co-occurring pathology in DLB as a small minority of patients were found to have pure DLB with no AD pathology. McKeith and colleagues (1996) created a set of diagnostic criteria and have since updated it twice (McKeith, Perry, & Perry, 1999; McKeith et al., 2005). The latest version is currently used to diagnose DLB (see Appendix A).

The debate on whether DLB is a separate entity from AD has been resolved, but a new debate has arisen. In PD it is well recognized that dementia can develop over time. Specifically, in a study they found that 78% of individuals with PD developed dementia in an eight year period after controlling for attrition due to death (Aarsland et al., 2003). In fact, many researchers are now postulating that as life prolonging treatments increase all patients with PD will eventually convert to dementia because there is an accumulation of cortical Lewy bodies over time (O'Brien et al., 2006). The debate that currently exists is whether PDD and DLB are separate entities. There is an arbitrary temporal cut-off between the two diseases. Specifically, DLB is diagnosed if dementia occurs within 12 months of the onset of Parkinsonism, whereas PDD is diagnosed if the onset of dementia occurs after 12 months have elapsed from the onset of Parkinsonism (O'Brien, 2006). Many researchers believe that both of these diseases occur on the same spectrum of dementia related to Lewy bodies as few studies have been able to identify clear differences between these two diagnostic categories (O'Brien et al., 2006).

Brain Pathology:

Currently, DLB is most often diagnosed post-mortem with the presence of specific neuropathological characteristics. These characteristics include Lewy bodies that have predilection for certain areas of the brain, and atrophy specific to certain regions of the brain. In

the majority of DLB patients (approximately 75% of cases) there is the presence of secondary pathology, which is primarily the presence of AD pathology (O'Brien, 2006).

Lewy bodies are spherical, eosinophilic intracellular inclusions within neurons and glial cells that are composed of neurofilaments, crystalline, ubiquitin, and α -synuclein (see Figure X). In fact, α -synuclein is a protein that accumulates and which is important in numerous neurodegenerative diseases, such as PD, AD, and Shy-Drager syndrome. α -synuclein is the targeted protein when staining for Lewy bodies through the injection of α -synuclein antibodies (Loy-English, 2003).

Lewy bodies were initially described in the brainstem nuclei in patients with PD. However, in DLB these proteins seem to occur throughout the brain. In fact, researchers have found that patients with DLB consistently had Lewy bodies in the cerebral cortex, brain stem, and peripheral nervous system. Lewy bodies do not occur diffusely, in fact, they have predilection for certain areas of the brain, including the neocortex (i.e., mostly the temporal, parietal and frontal lobe), the limbic cortex, subcortical nuclei, and the brainstem. In the neocortex, the temporal lobe is more affected than both the frontal lobe and the parietal lobe (Graeber, et al., 2003). The presence of Lewy bodies in the temporal lobe is correlated with the presence of visual hallucinations and is not thought to be associated with the cognitive impairments found in DLB (Harding, Broe, & Halliday, 2002). Additionally, the amygdala is the most vulnerable of the limbic structures (Hamilton, 2000). Therefore, it is clear that certain areas are more affected by Lewy bodies than others.

The question that is often posed is what these Lewy body pathologies cause in DLB. Studies have demonstrated that the total Lewy body density correlates weakly with disease

duration but does not correlate directly with specific cognitive symptoms (Graeber, et al., 2003). This may be because Lewy bodies are not markers of dying neurons. Therefore, the presence of Lewy bodies in a certain brain region does not indicate impairment of the cognitive functions associated with that brain area. In fact, current theories have concentrated on the fact that the presence of Lewy bodies may indicate impaired cellular functioning, disrupted synaptic connections, and critical neurochemical changes, as the cause of cognitive impairments found in DLB (Galvin, Lee, & Trojanowski, 2001). For example, a study found that patients with DLB had decreased regional glucose metabolism in the occipital association cortex and the primary visual area (Albin, Minoshima, D'Amato, Frey, Kuhl, & Sima, 1996).

Numerous studies have tried to link the cognitive impairments found in DLB to brain atrophy. Average whole-brain volume has been found to be lower in patients with DLB when compared to normal individuals, which would suggest the presence of moderate widespread atrophy (Graeber, et al., 2003). Bozzali and colleagues (2005) conducted a volumetric analysis of the atrophy found in DLB and demonstrated that there was a significant reduction of grey matter volume and no difference in white matter volume.

The focus of recent studies has been to try and identify the main areas of grey matter tissue damage as to try and link them to specific cognitive impairments found in DLB. Voxel-based morphometry reveals bilateral gray matter loss in the temporal and frontal lobes also involving the insular cortex (Burton et al., 2002). In a later study, using MRI they were able to find grey matter atrophy in the thalamus, the caudate, and the substantia nigra (Burton, McKeith, Burn, Williams, & O'Brien, 2004). However, studies on brain atrophy are inconclusive as many studies have demonstrated no significant brain atrophy associated with DLB (Cordery, Tyrrell, Lantos, & Rossor, 2001).

The presence of Alzheimer-type pathology is common in DLB, which is not surprising given the advanced age of most patients and the prevalence of Alzheimer pathology with increasing age. Senile plaques exist in individuals with DLB. Senile plaques are complex and heterogenous lesions that are defined by the presence of extracellular deposits of β -amyloid protein. Some neuritic plaques contain neuritis that have abnormal aggregates of tau protein that are similar to those found in neuronal cell bodies in neurofibrillary tangles. Tau-positive neuritic plaques are commonly found in AD, while tau-negative plaques are found in older adults and individuals with DLB. Most patients with AD have amyloid deposits not only in senile plaques, but also within the walls of blood vessels, which is called cerebral amyloid angiopathy (CAA). Given that Alzheimer-type pathology is common in DLB, it is not surprising that many individuals with DLB also have CAA. The severity of CAA is a function of both age and the presence of APOE4 genotype, which is a risk factor for both AD and DLB (Dickson et al., 1992; O'Brien, 2006).

Neurofibrillary tangles are neuronal lesions formed by progressive accumulation of abnormally phosphorylated tau protein within the perikarya and proximal dendrites. This pathology is very common in AD but fairly rare in DLB. This may be due to the fact that AD pathology is primarily due to a dysfunction of the tau protein, while in DLB tau protein function is relatively preserved (Dickson et al., 1992; O'Brien, 2006).

Clinical Presentation and Diagnostic Criteria:

DLB has three core features, including cognitive impairments, visual hallucinations, and Parkinsonism symptoms. In most dementias, such as AD and VaD, memory impairment is the presenting issue. However, in DLB the most common presenting feature, in 33-65% of all cases,

are visual hallucinations (O'Brien et al., 2006). These hallucinations often take the form of people, animals, or objects, and are rarely described as distressing. Additionally, delusions are common in patients with DLB. Delusions and hallucinations are often useful for the diagnosis of DLB and can help differentiate the dementia from other types early. However, if a diagnosis of DLB is missed and neuroleptics are prescribed to control a patient's psychotic behaviors this can have tragic consequences. Severe neuroleptic sensitivity has been reported in 81% of all patients with DLB, with symptoms of sedation, confusion, rigidity and immobility, and even death (McKeith, 1992).

As with most dementias, DLB is marked by a progressive cognitive decline that interferes with normal life. Cognitive impairments are usually marked by fluctuations that occur hour to hour. It is postulated, that the pattern of dementia is a mixed cortico-subcortical dementia since patients have significant frontal-subcortical dysfunction (i.e., impairment of attention, visuospatial function, executive functions, thought, regulatory changes in praxis and gnosis). Although memory impairments are not usually found in the beginning stages of DLB they become marked once the disease has fully developed.

One of the most impaired cognitive abilities present in DLB is visuoperceptual and spatial functions. Within this domain they have particular difficulty with visual perception, making it difficult to identify objects and pictures. It is theorized that this impairment may cause their visual hallucinations, which is a hallmark of the disease (Oda, Yamamoto, & Maeda, 2009). In addition, patients with DLB have difficulty with visual construction, which can be seen through difficulty putting things together (i.e., blocks). These particular impairments are not associated with overall disease severity as multiple studies have shown that visuoperceptual and spatial functions are not correlated with overall Mini-Mental State Examination (MMSE) score

(Oda et al., 2009). Thus, visuoperceptual and spatial dysfunction in DLB are not due to overwhelming pathology across the brain caused by disease severity, but more likely due to a specific location of pathology.

In addition to visuoperceptual and spatial impairments, patients with DLB also suffer from attentional deficits. Ballard and colleagues (2001) demonstrated that patients with DLB had slowed processing speed, attentional impairments, and fluctuations in attentional impairments. In terms of attentional abilities, sustained attention is more severely impacted in DLB. Thus, patients with DLB can often have difficulty maintaining their attention on a task.

Memory impairment is not marked in DLB but as the disease progresses it can become significant. In particular, their verbal memory is relatively preserved until later stages of DLB. Visual memory is impacted significantly but researchers theorize that this is likely due to their visuoperceptual and spatial impairments rather than due to true memory impairment (Oda et al., 2009).

Finally, patients with DLB also have spontaneous motor features of Parkinsonism (i.e., tremors, rigidity, bradykinesia of bilateral onset, and postural instability). In fact, they occur in approximately 80% of all patients with DLB. However, in patients with DLB these symptoms are not typically present at the onset of the disease. Parkinsonism symptoms usually have their onset towards the middle of the disease process. Additionally, frequent falls, orthostatic hypotension, syncope, and a transient loss of consciousness occur in approximately 30-40% of patients with DLB (Graeber, et al., 2003). REM sleep behavior disorder often develops in individuals with DLB. During the REM stage (i.e., stage 5 where rapid eye movements occur) individuals experience muscle paralysis and as such they cannot act out their dreams. However,

this paralysis does not occur in REM sleep behavior disorder and individuals can experience violent movements during this stage of sleep. Therefore, it is evident that this disease is multifaceted as there is a wide range of clinical features presented.

The clinical diagnosis is based on the consortium conferences on DLB (McKeith et al., 1996; McKeith et al., 1999; McKeith et al., 2005). The clinical diagnosis has good sensitivity (0.83) and excellent specificity (0.95) when it is used accurately. The clinical diagnosis allows for a diagnosis of probable DLB including the central feature (i.e., progressive dementia) and two core features (i.e., Parkinsonism, visual hallucinations, or cognitive fluctuations) and possible DLB including the central feature and one core feature. However, it is important to remember that diagnosis can only be confirmed on autopsy as it is important to identify the presence of Lewy bodies.

Neuropsychological Profile of Early DLB

Currently, there are no studies looking at the neuropsychological profile of patients with early DLB to date. The majority of research in the last decade has concentrated on trying to differentiate DLB from AD in terms of neuropsychological profile due to the finding that patients with DLB were often being erroneously diagnosed as AD (Loy-English, 2004; Oda et al., 2009). In fact, this research has clearly demonstrated that patients with DLB have more severe impairments in visual perception and construction than patients with AD (Ala, Hughes, Kyrrouac, Ghobrial, & Elble, 2001; Cormack, Aarsland, Ballard, & Tovee, 2004; Ferman et al., 2006). In addition, a recent review article stated that patients with DLB may have more severe impairments in attention, especially sustained attention, when compared to AD patients (Oda et al., 2009). Finally, research has demonstrated that patients with AD have more severe memory

impairments, especially verbal memory, when compared to patients with DLB (Lambon, Powell, Howard, Whitworth, & Hodges, 2001; Shimomura et al., 1998).

Based on the research one could theorize that patients with early DLB would likely have significant visuoperceptual and spatial impairment. This is based on the research that the majority of patients with DLB present initially to psychiatric clinics with visual hallucinations (O'Brien et al., 2006) and the fact that the presence of visuoperceptual and spatial impairments are associated with visual hallucinations (Oda et al., 2009). In addition, it is likely that patients with early DLB have at least mild impairments in attentional abilities, since they become marked as the disease progresses (Oda et al., 2009). Based on the literature, it is unlikely that patients with early DLB show significant memory impairment, since this is typically associated with disease severity (Oda et al., 2009). Clearly, significant research is needed to better ascertain the cognitive impairments associated with early DLB. This research would not only help clinicians with early diagnosis but would also provide some insight into possible associated functional deficits in early stages.

Dementia and driving.

The need for research on dementia and driving is well established. Of particular importance would be the study of driving abilities in older adults with early AD and DLB, the two most common types of dementia (Lovestone, 1998). Research on dementia and driving is still in its infancy, though its roots can be traced back as early as the 1960s. For example, Walker (1967) estimated the MVC risk of older “senile” drivers with cardiovascular disease to be approximately four times higher when compared to healthy controls. Since then, the literature on dementia and driving has grown but there continues to be a serious lack of research studies in

this field. The majority of research in the field has concentrated on examining drivers with early stage dementia who still maintain a driver's license and drive (Carr, Jackson & Alquire, 1990; Hopkins et al., 2004; Odenheimer, 1993; Waller, 1967). Three themes are commonly explored with drivers diagnosed with dementia in the literature, including: 1) the associated MVC risk; 2) the typical driving errors committed; and 3) the predictive value of neuropsychological tests to determine fitness to drive. In addition, driving is measured through examination of driving records, caregiver report, on-road examination, or through the use of a simulated drive. The most pertinent research will be reviewed in this section.

Since the publication of Walker et al. (1967), a number of studies have examined the occurrence of MVCs in drivers with dementia (Cooper, Tallman, Tuokko, Beattie, 1993; Dubinsky, Stein, & Lyons, 2000; Dubinsky, Williamson, Gray, Glatt, 1992; Friedland et al., 1988; Gilley, Wilson, Bennett, Bernard, & Fox, 1991; Lucas-Blaustein, Flipp, Dungan, Tune, 1988; Tuokko, Tallman, Beattie, Cooper, & Weir, 1995). Several of these authors have estimated that the risk of MVC among drivers with dementia was at least twice as high as their healthy counterparts (Cooper et al., 1993; Tuokko et al., 1995), whereas other researchers estimated much higher risks (Dubinsky et al., 2000; Dubinsky et al., 1992; Friedland et al., 1988). Interestingly, drivers with dementia were typically found to be at-fault in these MVC (Cooper et al., 1993; Lucas-Blaustein et al., 1988). Drachman and Swearer (1993) examined the risk of MVC in the years following the diagnosis of dementia and concluded that although drivers with dementia had an increased overall risk, their risk during the first three years post-diagnosis was comparable to their healthy counterparts. This finding indicates that not all drivers with early dementia are impaired but there becomes a point where a driver with a diagnosis of dementia is no longer safe to operate a vehicle.

Several studies have demonstrated that dementia patients lack insight into their decline and their competency to drive safely, and thus most continue to drive until they have experienced one or more MVC (Friedland et al., 1988; Kapust & Weintraub, 1992; Kaszniak, Keyl, & Albert, 1991; Meng, Siren, & Teasdale, 2013). In fact, a study by Cooper and colleagues (1993) demonstrated that over 80% of drivers diagnosed with dementia who were involved in a MVC continued to drive afterwards. They also indicated that 40% of those went on to have at least one more MVC. This research clearly points to the fact that self-initiated driving cessation in drivers with dementia is unlikely and this responsibility should be taken diligently by physicians who can assess for driving risk. Based on this research, other researchers have studied the impact of an innovative approach whereby older adults were asked to sign an Advance Driving Directive (ADD), in much the same way that they would for end-of-life care (Betz, Lowenstein, & Schwartz, 2012). These researchers found that signing an ADD was beneficial because it allowed the older adult to express their wishes to their family and physician. In addition, they found that older adults who had signed an ADD were more likely to comply with the directives and were more accepting of their need to cease driving when it became necessary.

Several research studies have looked at predicting the risk of future MVC in patients with either early dementia or mild cognitive impairment. These studies demonstrate that a history of crash in the previous one to five years or a traffic citation in the previous two to three years was predictive of future risk of MVC (De Raedt et al., 2000; Diagneault, Joly, & Frigon, 2002; McGwin et al., 2000). In another study that examined drivers with mild AD over the age of 55, it was found that drivers who reduced their weekly mileage were more likely to experience on-road difficulties (Cushman, 1996). Another study found that drivers who restricted their driving due to feeling unsafe on the road had a fivefold increased risk of MVC (Lesikar, Gallo, Rebok, & Keyl,

2002). Similarly, another study found a moderate correlation between cognitively impaired drivers who self-reported that they avoided driving at night with failing an on-road driving examination (Baldock, Mathias, McLean & Berndt, 2006). Patients with early dementia lack insight into their condition and their perception of their own ability to drive safely is often overestimated. To illustrate this point, Hunt, Morris, Edwards and Wilson (1993) reported that 100% of patients with early AD who failed an on-road driving test considered themselves to be safe drivers. Thus, self-reported driving ability is not an accurate predictor of MVC in early dementia. Some researchers studied the report of caregivers instead. Brown, Ott, Papandonatos, Sui, and Ready (2005) found that caregivers for patients with mild AD had a sensitivity of 47.8% and a specificity of 81.8% vs neurologists who had a sensitivity of 60.7% and a specificity of 90.9% in identifying unsafe drivers. They also indicated that caregivers had a tendency to overestimate driving abilities in the individual they were caring for.

Other researchers have approached the study of driving and early dementia by examining the typical driving errors committed by this group. Though this literature is limited, interesting findings have been listed. In particular, compared to healthy older adults, drivers with dementia are at an increased risk of unsafe operation of a vehicle (Man-Son-Hing, Marshall, Molnar, & Wilson, 2007). Research has demonstrated that several unsafe driving behaviors are more likely to occur in drivers with early dementia, including becoming lost in familiar areas (Silverstein, Flaherty, & Tobin, 2002; Uc et al., 2004), incorrect turning (Uc et al., 2005), impaired signaling (Duchek et al., 2003), decreased comprehension of traffic signs (Carr et al., 1998), and greater lane deviations (Dawson, Anderson, Uc, Dastrup, & Rizzo, 2009; Uc et al., 2005). Other researchers have found that drivers with dementia are more likely to drive the wrong way in

roundabouts and drive too slowly (Logsdon et al., 1992; Lucas-Blaustein et al., 1988; Odenheimer et al., 1994; O'Neil et al., 1992).

An interesting study was conducted wherein 100 older adults with cognitive decline, most of whom were diagnosed with early dementia, were evaluated on the road and compared to age-matched healthy controls as well as younger drivers (Dobbs, 1997). These three groups were asked to drive a specific on-road route with a driving instructor who evaluated their performance. The results of this study indicated that there was a triad of driving errors committed by older adults with cognitive impairments. One set of errors was committed almost exclusively by the group of older adults with cognitive impairment and these included errors termed as hazardous or potentially catastrophic because they could have resulted in a MVC if the driving instructor had not taken control of the vehicle. A second set of errors was committed by all three groups, though differentially. These errors included poor positioning on turns and errors of observation. These errors were committed rarely by younger adults, more so by older adults, and most often by older drivers with cognitive impairment. The last set of errors were errors that could result in an automatic fail on a licensing road test (e.g., rolled stops, speed errors). These errors were found to be committed equally by all three groups. Thus, taken together, this study indicates that older drivers with cognitive impairment not only commit more driving errors but also commit driving errors that are different and riskier in comparison to other driving populations.

Interesting studies have been conducted using simulators to test driving abilities of older drivers with early AD. In particular, one study found that patients with AD were less likely to comprehend and operate the simulator when compared to healthy older adults (Cox, Quillain & Thorndike, 1998). They also found that drivers with AD were more likely to drive off the road, spend more time driving considerably slower than the posted speed limit, and less time driving

faster than the speed limit. Drivers with AD typically applied less break pressure in stop zones, spent more time negotiating left turns, and drove poorly overall. Interestingly, they found a correlation between the AD drivers who were unable to operate the simulator and severity of MMSE score as well as fewer miles driven annually. A recent study examined drivers with mild AD, drivers with MCI and healthy older adults using a STISIM standardized simulated road driving session (Fritelli et al., 2009). They found impaired driving performance in the AD driver group by examining the length of the run, the mean time to first collision, and the number of off-road events in comparison to the MCI drivers and healthy older drivers. They found no statistical difference between the groups when examining the number of infractions and traffic light stops. They also found that, when examining the time to first collision, the MCI drivers were also impaired compared to their healthy counterparts but not as impaired as the AD drivers.

Other studies have examined the predictive value of neuropsychological tests to determine fitness to drive in older drivers with dementia. These studies have focused on establishing associations between driving outcomes and findings on neuropsychological testing. Findings on neuropsychological tests may be complementary to that of a bedside examination and an informant interview. Research in this field is deficient as the few studies that exist are unclear about their findings, they are not replicable, and their recommendations for assessments are lacking (Lundberg et al., 1997). In fact, psychologists and neuropsychologists were surveyed in the United Kingdom and 70% of them reported that they used neuropsychological testing to make recommendations about the fitness to drive (Christie, Savill, Buttress, Newby, & Tyerman, 2001). However, 51% of responders reported not being confident about their recommendations and felt as though there was little knowledge about the relationship between cognitive testing and driving ability.

In September 1994, the Swedish National Road Administration invited researchers from North America and Europe to prepare a consensus statement on dementia and driving (Johansson & Lundberg, 1997). Several recommendations were made and findings were discussed in relation to driving and dementia, including the cognitive impairments in dementia that are likely to cause driving impairment. Specifically, they reported that dementia causes impairments of visuospatial skills, attention, memory, and judgment, all of which are important functions for operating a vehicle safely. In particular, they stated that visuospatial skills were essential for appropriate positioning of the vehicle, estimating distances, and interpreting a current traffic situation and predicting its outcome. Attentional abilities, including selective, divided, and sustained attention, are necessary to predict potential hazards, deal with competing stimuli at intersections and to preserve optimal alertness while driving. Short-term memory and working memory are also important in driving safely. In particular, working memory allows the driver to retain incoming information (e.g., when looking in the rear-view mirror). Impairments in short-term memory combined with visuospatial impairments can contribute to drivers becoming lost, driving errors and violations. In addition, language deficits may impact the route the driver chooses or the planned maneuvers regarding traffic signs. Finally, they stated that impaired judgment not only impacted the ability to drive safely but also the insight into one's ability to drive safely and the related compensatory behavior. The consensus group concluded that drivers with moderate to severe dementia should likely not drive and that more research was needed on drivers with mild dementia.

Researchers have taken two main approaches at examining the relation between neuropsychological tests and driving performance. In particular, researchers have either studied global measures of functioning (e.g., CDR, MMSE) or they have used measures that target

specific abilities (e.g., UFOV for visual attention, trail making for attention). One particular study used the Clinical Dementia Rating scale (CDR) to examine driving risk in drivers with early AD (Dubinsky, Stein & Lyons, 2000). To evaluate driving risk they examined crash statistics, evaluation of driving performance, and analysis of driving task components. They found that drivers who had a CDR score of 0.5 (i.e., mild impairment) had mild impairments in driving comparable to youths (i.e., drivers between the ages of 16 and 21) and those driving under the influence at a blood alcohol level below 0.08%. However, drivers with AD with a CDR score of 1 (i.e., moderate impairment) posed a significant traffic safety problem. Though, another study found that the CDR was not an accurate measure of driving ability since 85% of the CDR 0.5 group and 76% of the CDR 1 group went on to pass an on-road examination test (Brown et al., 2005).

Research on the MMSE is also unclear as a predictor of ability to drive safely in the dementia population. For example, O'Neil (1992) demonstrated that MMSE scores did not discriminate individuals experiencing diminished driving ability from those with preserved driving abilities. However, MMSE have been found to be significantly related to driving in few other studies (Fitten et al., 1995; Logsdon et al., 1992; Marottoli et al., 1994; Rebok et al., 1994). A review article indicated that an MMSE score equal or less than 24 was possibly useful in identifying patients at an increased risk for unsafe driving (Iverson, Gronseth, Reger, Classen, Dubinsky & Rizzo, 2010). Though, depending on the range of variability of global MMSE score in the sample, these scores may not relate well to driving ability. Thus, it seems that global measures of neuropsychological functioning may not relate well to driving ability. However, deficits in specific cognitive domains such as attention or visuospatial skills may predict driving skills better.

One recent study sought to examine the association between driving errors and global functioning as well as cognitive functioning in specific domains in patients with early AD (Dawson, Anderson, Uc, Dastrup, & Rizzo, 2009). They found that drivers with AD committed significantly more driving errors in comparison to healthy controls, where the most common type of errors were lane violations. After adjustment for age and gender, they also found that their global measure of neuropsychological functioning, COGSTAT, was a significant predictor of safety errors in subjects with AD. When examining specific measures of cognitive functioning, they found significant increases in safety errors were also associated with AD patients who had poorer scores on the Benton Visual Retention Test, Complex Figure Test-Copy, Trail Making Subtest-A, and the Functional Reach Test. They concluded that visual memory, visuospatial skills, attention, and motor abilities may be most related to the ability to drive a vehicle safely.

Reger and colleagues (2004) conducted a meta-analysis to determine the relationship between neuropsychological functioning and driving ability in dementia. A total of 27 studies were included that examined on-road tests, simulated drives and caregiver reports on driving ability. They found that caregiver reports on driving ability correlated modestly with mental status and tests of visuospatial ability. For studies including an on-road test, they found strong correlations between driving performance and tests of attention and concentration and tests of visuospatial skills. Finally, in studies using a simulated drive, they found strong correlations between driving performance and mental status and tests of visuospatial abilities and modest correlations with tests of memory and tests of executive functions. They concluded that studies using caregiver report were a less reliable measure of a patient's driving ability in comparison to objective driving tests, but that it was likely that caregiver reports were the most used in physician assessment of MVC risk.

Some researchers have tried to examine cognitive domains in more detail by examining the specific constructs involved. For example, Duchek and colleagues (1997) argued that selective attention was more associated to driving impairment in patients with dementia than any other component of attention, including divided and sustained attention. They stated that identifying important information in the driving environment while ignoring irrelevant information may be a skill that is impaired in drivers with dementia. It is likely that as the body of literature continues to grow in this field, specific aspects of attention and visuospatial processing will be found to relate to driving ability better than most cognitive domains as a whole.

A comprehensive study examined the relationship between performance on neuropsychological tests, simulated driving performance, and state crash records (Anderson, Rizzo, Slu, Uc, & Dawson, 2005). They examined drivers with early AD and healthy older adults. The simulated composite score was found to be significantly correlated with overall cognitive ability as well as with individual cognitive tests of attention, memory, visuospatial and visuomotor abilities. Drivers who crashed during their intersection incursion scenario (i.e., participant had to avoid a vehicle that illegally incurred from the right side into an intersection) performed significantly worse on overall cognitive ability than those who successfully steered around the incurring vehicle. Crashers had specific cognitive deficits on measures of visuomotor abilities and attention. Memory test performance for both verbal information and visual material were associated with an increased risk of subsequent MVC as measured by state crash records. They concluded that their findings provide support for the validity of driving simulation as a safe means for evaluating driving ability and that performance on certain cognitive tests may be predictive of driving ability.

A thorough review of the research on the driving abilities of individuals with early dementia has been conducted. In particular, studies looking at driving records, caregiver reports, on-road examinations and simulated drives were presented. The research findings indicate that drivers with early dementia have a significant increase in the risk of MVC. They also indicate that driving errors are often committed by drivers with early dementia, though studies have found varying data on the types of errors most commonly committed. Finally, the predictive value of neuropsychological tests were reviewed and it was found that although the research on global tests of cognitions are unclear, specific tests examining attention and visuospatial abilities can be associated with driving outcome. At this point, more research needs to be conducted on the types of errors committed by drivers with early dementia to further clarify the results. In addition, research needs to be conducted on the predictive value of neuropsychological tests. Specifically, it would be of particular importance to study the specific components of each cognitive domain (i.e., examine selective, sustained and divided attention).

Research Design

The literature review has indicated that there is a dearth of research on the neuropsychological profile of individuals with early DLB. As a first step, this thesis aimed to qualify the nature of impairments in these early stages. As previously stated, based on the research one could theorize that participants with early DLB would likely have significant visuoperceptual and spatial impairment and at least mild impairments in attentional abilities. Though global cognitive functioning was assessed, this study focused on ascertaining the magnitude and quality of attentional and visuoperceptual and spatial impairments in individuals with early DLB.

The majority of research on dementia and driving has focused on examining driving performance in a mixed group of dementia drivers (i.e., AD, VaD, DLB). The few studies that have targeted a particular dementia group have examined drivers with AD, though their research findings are confounding. The main goal of this thesis aimed to examine the driving performance of drivers with early AD so as to provide more support to the currently lacking literature. In addition, associations between findings on neuropsychological testing and driving performance were evaluated. Specifically, this study examined neuropsychological tests of global performance, attention and visuoperceptual and spatial abilities. This study also examined specific components of attention (e.g., selective, sustained and divided attention) and visuoperceptual and spatial abilities (e.g., object perception, spatial orientation).

There are currently no studies on early DLB and driving performance to date. Another main goal of this thesis was to conduct an exploratory study of the driving performance of drivers with early DLB as well as the associated findings on neuropsychological testing. The same objectives mentioned above in the study of early AD were sought in this population. It is anticipated that this investigation will lay the foundation in the evaluation of driving abilities of drivers with early DLB.

These three research goals were examined through one large comprehensive study. In particular, fifty-six participants were recruited from three groups; 20 individuals diagnosed with early AD, 15 individuals diagnosed with early DLB and 21 healthy age-matched controls. All participants were administered two global tests of cognitive functioning, including the Mini-Mental Status Exam (MMSE) and the Dementia Rating Scale (DRS-2). Participants were administered a common test of word finding ability called the Boston Naming Test (BNT). Participants were also administered a neuropsychological battery that assessed different facets of

attention, called the Test of Everyday Attention (TEA). A second neuropsychological battery that assessed different facets of visuoperceptual and spatial abilities was administered, termed the Visual Object and Space Perception Test (VOSP). Finally, a test of visual field and visual attention was also administered, termed the Useful Field of View (UFOV). A simulated driving task was completed, with driving performance being assessed by the simulator as well as an experimenter based assessment using a demerit-point assessment.

METHODS

Participants

There were three participant groups ($N = 56$) including a group of healthy older adult controls and two groups of individuals diagnosed with early stage dementia (DLB and AD). All participants were over the age of 65 years and English speaking. In addition, all participants had valid driver's licenses, although by self-report about a third of participants had given up driving by their own accord. The mean age of the control group was 77.00 years ($SD = 5.86$) with a range of 68 to 86 years, the mean years of education was 13.14 ($SD = 3.18$) and the group was comprised of 52.4% women and 47.6% men. The mean age of the early AD group was 78.50 years ($SD = 7.22$) with a range of 66 to 90 years, the mean years of education was 13.05 ($SD = 3.94$) and the group was comprised of 45% women and 55% men. Finally, the early DLB group had a mean age of 76.40 ($SD = 6.59$) with a range of 68 to 88 years, the mean years of education was 14.20 ($SD = 4.55$) and the group was comprised of 40% women and 60% men. Participants were matched for age, gender, and years of education.

A convenience sample of healthy older adult controls ($N = 21$) was obtained through brief community newspaper ads (Appendix I). These participants underwent a 20-minute screening call in order to determine if they qualified to participate in this study (Appendix C & D). The exclusion criteria included any serious visual or hearing impairments left uncorrected (e.g., cataracts, and color blindness), serious health problems (e.g., mental illnesses, history of head injury, epilepsy, apoplexy, heart attacks, hypertension, and sleep apnea), any medications that could alter cognitive abilities, any history of substance abuse, and any history of learning disabilities. These exclusion criteria were based on research that indicated that certain conditions

could have an effect on cognitive abilities (Ahto, Isoaho, Puolijoki, Laippala, Sulkava, Kivela, 1999; Sheahan, Coons, Robbins, Martin, Hendricks, & Latimer, 2005). Control participants with abnormal Mini-Mental State Exam (MMSE) scores (< 25) were also excluded from this study (Folstein, Folstein, & McHugh, 1975).

Participants diagnosed with early stage dementia were a convenience sample recruited from the Memory Clinic at the Bruyère Continuing Care Center (a tertiary care facility; EBRI) in Ottawa through the collaboration of two supervising neurologists. Previous studies involving clinical groups, such as participants with AD and DLB, have identified that recruiting participants can be challenging and time consuming. Due to this limitation, this study focused on recruiting approximately 15-25 participants per group. Such sample sizes were feasible for the present study according to the intake rate of participants with such dementias at the Memory Clinic. Additionally, a literature review of studies examining the cognitive impairments associated with different types of dementia revealed small to moderate effect sizes ($d = .3-.5$) with such a sample size (Bäckman, Jones, Berger, & Laukka, 2005). Unfortunately, effect size estimates could not be found in the driving and dementia population.

Participants who had a diagnosis of probable early DLB or AD at the memory clinic were contacted in order to determine their willingness to participate in the study (Appendix F). Participants were assessed for severity, using the Global Deterioration Rating Scale and only participants in the early stages of dementia were included in this study (Stages 3 and 4). Once again, the same exclusion criteria were used with the exception of medications, as most participants from the EBRI were taking psychoactive medications, such as acetylcholinesterase inhibitors (Appendix C). Additionally, participants were also excluded if they had participated in more than three previous studies in order to reduce over-recruitment. Participants in the early

stages of dementia were grouped in one of the two dementia groups depending on the diagnosis determined at the memory clinic. Following group assignment participants underwent neuropsychological testing over two sessions and the simulated drive during the second session.

Procedures

Potential control participants were contacted by a research assistant or by the investigator and were asked if they had time to respond to a short questionnaire on their demographic information (Appendix C & D). If they met the inclusion criteria (i.e., no serious visual or hearing impairment left uncorrected, no serious health problem, no medications that could alter cognitive abilities, no history of substance abuse, and no history of learning disabilities) they were asked to participate in the study and were informed that all testing would occur at the EBRI. Newspaper ads continued until the number of participants had reached sufficient numbers for the study.

All patients at the EBRI are asked if they would like to participate in future research studies when they seek services at the Memory Clinic. If interested, they completed a consent form that is added to their file. Individuals with a signed consent form and who had a diagnosis of early DLB or AD in their file were telephoned by the investigator or research assistant in order to verify that they were willing to participate and that they did not meet the exclusion criteria (Appendix O). Potential participants were informed that testing would take place at the EBRI. When a participant agreed to participate they were asked if they had time to answer a short questionnaire to collect their demographic information (Appendix C & F). In the event that they were unable to answer the demographic information on their own, a date was scheduled to have a phone interview with the participant and their primary caregiver. The recruitment of each

participant was followed by a brief conversation with the supervising neurologists about the potential participant's ability to provide consent, to review the diagnosis and dementia stage. This recruitment process continued until sufficient participants had been reached and when groups were considered to be equivalent.

In this study, all participants with dementia were diagnosed by supervising neurologists at a tertiary care memory clinic. All diagnoses of dementia were accomplished using the general guidelines for the assessment of dementia in hospitals (Appendix N). These guidelines offer a multi-modal approach to diagnosis of dementia which greatly reduces diagnostic error (Cummings, 2002). Additionally, diagnosis of specific dementia group (AD and DLB) was accomplished using the current gold standards in diagnosis of dementia. DLB was diagnosed using the diagnostic criteria outlined by the first symposium on DLB, which has good predictive validity (Appendix A; McKeith et al., 1996). AD was diagnosed using the diagnostic criteria outlined by the National Institute of Neurological and Communicative Diseases and Stroke/Alzheimer's Disease and Related Disorders Association, which has excellent predictive validity (NINCDS-ADRDA) (Appendix M; McKhann, Drachman, Folstein, Katzman, Price, & Stadlan, 1984). Using this method of diagnosis ensured that diagnostic groups had a good level of reliability.

All participants who met the inclusion criteria at the end of the demographic information questionnaire were asked for their availability. At this point the first of two testing sessions was scheduled with the participant. Participants were expected to travel to the EBRI, all expenses paid (i.e., taxi fare, bus fare, parking fare), alone or with their primary caregiver for the first testing session. Both testing sessions took place in a testing room at the EBRI within the memory disorders clinic. Specifically, testing occurred in a room that was equipped with all the testing

materials, a table, and two chairs. The room was sealed off from any distractions that could impact performance. All participants began the first testing session by completing the consent form as approved by the ethics committee at the EBRI and at the University of Ottawa (Appendix H & P).

During the first testing session, participants with AD or DLB were asked questions pertaining to the Global Deterioration Rating Scale (GDS) in order to verify if they were in fact in the early stages of dementia (stages 3 & 4); participants whose disease was at other stages were excluded. Additionally, they completed the Mini-Mental State Exam (MMSE). The control participants were also asked to complete the MMSE. Scores of 25 and above indicated normal cognitive functioning therefore all potential control participants who scored below a score of 25 on this test were excluded. Following this screening, testing commenced immediately.

Participants that met all the criteria participated in a first session of testing immediately following the screening. This session lasted approximately two and a half hours and participants were offered as many breaks as needed during the testing. All participants underwent a neuropsychological and computerized assessment including a test of general cognitive functioning (DRS-2), visuospatial/perceptual abilities (VOSP), word finding (BNT), attention (TEA), and processing speed (UFOV). Participants also underwent a simulated drive of approximately 20 minutes. All neuropsychological and computerized testing was completed according to the protocol specified by each test. The neuropsychological and computerized testing was administered in the presence of the participant and the investigator only. Once the first session of testing was completed (i.e., consent signed, GDS completed for participants with AD and DLB, GDS depression, MMSE, DRS-2, the BNT and the VOSP) another testing session was booked. During this second session, the DRS-2 was administered a second time, the TEA

was completed, the UFOV and the simulated driving task were administered. The DRS-2 and its alternate form were used and the order from session 1 to 2 was counterbalanced between participants. Both sessions lasted approximately 2 and a half hours for a total of 5 hours of testing (Appendix L).

Primary Outcome Measures

1) Diagnostic Measures. These measures were administered during the first session of testing by the experimenter and they were used to determine the participant eligibility.

Global Deterioration Scale (GDS) (Reisberg, Ferris, deLeon, & Crook, 1982). The GDS is an instrument that was developed in order to stage the magnitude of cognitive impairments in dementia (Appendix B). The theory is based on the idea that dementia has identifiable ordinal stages of regression. There are seven identifiable stages; 1) no cognitive decline, 2) very mild cognitive decline based on subjective complaints, 3) mild cognitive decline based on objective testing, 4) early dementia, 5) moderate dementia, 6) moderately severe dementia, 7) severe dementia. Descriptions are given for each stage of decline. In this study, mild dementia was described as stages 3 and 4. Stages 1 and 2 were excluded, as these stages are often described as no dementia present.

Overall, the psychometric properties of this test are strong. Reisberg, Ferris, Steinberg, Shulman, and deLeon (1982) reported good rates of test-retest reliability ($r = .92$) for intervals of testing of 7 days to 4 months. In terms of inter-rater reliability, Foster, Sclan, Welkowitz, Boksay, and Seeland, (1988) found excellent reliability amongst psychiatrists ($r = .97$) and research assistants ($r = .92$) when the GDS was administered at a psychiatric hospital. Several studies have examined the construct validity of this scale including the original author (Reisberg,

et al., 1982). Specifically, the GDS was found to be strongly correlated ($r = .97$) with the Functional Capacity Scale (FCS), a 7-point scale that is an ordinal staging instrument used in dementia research. Each point on the FCS represents progressively greater disability (Foster, et al., 1988). Therefore, the psychometric data on this test seems to indicate that the GDS is both a valid and reliable measure.

Geriatric Depression Scale (GDS Depression) (Brink, Yesavage, Lum, Heersema, Adey & Rose, 1982; Yesavage et al., 1983). The GDS is currently one of the most used self-report measures of depression in old age. The administration is fairly quick, taking approximately 5 minutes to complete. All questions are answered with a yes or no response. There are a total of 30-items. A score of 0 to 9 indicates normal functioning, 10 to 19 indicates a mild depression, and 20 to 30 indicates a severe depression.

The GDS is recommended for use with community dwelling older adults. The GDS was validated against scores from two other assessment instruments of depression, including the Zung Self-Rating Scale for Depression and the Hamilton Depression Rating Scale. Correlations between the GDS and both of these measures for no depression were good ($r = .82$), adequate for mild depression ($r = .69$) and good for severe depression ($r = .83$) (Yesavage et al., 1983). In addition, they also found that sensitivity for the measure was 84% and specificity was 95%. Though the Cornell Scale for Depression has a higher sensitivity and specificity for the dementia population the GDS has still been found to have a sensitivity ranging from 82-90% and a specificity ranging from 75-94% in the dementia population (Korner et al., 2006).

Mini Mental State Exam (MMSE) (Folstein, et al., 1975). The MMSE is probably one of the most widely used brief screening instruments in dementia. The administration is very quick,

at only five to ten minutes per participant. Scores below 24 usually indicate abnormality or probable dementia. A cut-off score of 27 is usually used to identify normal healthy adults. In the aging population, scores above 25 are used to identify normal healthy older adults, as such, only healthy controls with scores above and including 25 participated in this study.

The MMSE has often been recommended as a screening tool for cognitive impairments in community dwelling older adults, as it has good sensitivity (80%) and high specificity (98%). The MMSE correlates ($r = .79$) with the cognitive and self-contained part of the Cambridge Examination for Mental Disorders of the Elderly (CAMCOG), the gold standard screening tool (Blake, McKinney, Treece, Lee, & Lincoln, 2002). Although the MMSE is not the gold standard, it is still the most widely used because it can be rapidly administered. Additionally, high 24-hour test-retest reliability was found in the original standardization study whether the same examiner was used ($r = .89$) or a different one ($r = .83$) (Folstein, et al., 1975). In a study that examined specific items on the MMSE, it was found that the three-word recall was most sensitive to dementia participants. The second most sensitive item was the drawing task. In fact, only 3% of demented individuals passed both items successfully. This study indicates that the MMSE should accurately discriminate between the control group and early dementia group (Galasko, Klauber, & Hofsetter, 1990). Overall, the MMSE has strong psychometric properties as a screening tool for cognitive impairments.

2) Neuropsychological Tests. The following measures were administered during the first and second session of testing by the investigator. They included tests of cognitive fluctuations, visuospatial/perceptual abilities, semantic memory and attention functions. The neuropsychological tests were selected because of their psychometric properties. Additionally, all these tests have been widely used in studies examining the cognitive impairments found in

dementia (Jendroska, 1997; Lovestone et al., 1998; Mori et al., 2000; Preobrazhenskaya, et al., 2006; Walker, et al., 2000).

Mattis Dementia Rating Scale (DRS-2 and DRS-2-Alternate) (Coblentz, Mattis, Zingesser, Kasoff, Wisniewski, & Katzman, 1973 ; Jurica, Leitten, & Mattis, 2001; Mattis, 1976). The DRS-2 assesses attention, memory, visuospatial construction, conceptualization, and initiation/perseveration. It was originally designed to assess adults with suspected dementia. It is organized so that the most difficult items in each domain are given first. The purpose of this administration scheme is to save time when testing less impaired participants. As such, high functioning older adults can complete the battery in less than 10 minutes, but other participants may take as long as 45 minutes. The DRS-2 consists of 36 tasks and 32 stimuli, yielding five subscale scores and an assessment of the participant's overall level of cognitive functioning. The entire test was administered according to discontinue rules.

The DRS-2 shows high sensitivity and specificity, even in the early stages of dementia (Green, Woodard, & Green, 1995). The reliability and validity properties of the DRS-2 are excellent. A test-retest reliability correlation coefficient was .97 with subscale correlation coefficients ranging from .61 to .94. The DRS was administered twice with a 1-week interval between administrations to a group of 30 participants diagnosed with dementia of the Alzheimer's type. A split-half reliability coefficient was .90, utilizing a sample of 25 participants aged 65 to 94 years who received diagnoses of either organic brain syndrome or senile dementia. A t-test indicated no significant differences between scores on the two halves. The alpha coefficients were calculated for four DRS subscales using a combined dementia sample. The alpha coefficients were Attention (.95), Initiation-Perseveration (.87), Conceptualization (.95), and Memory (.75). The DRS-2 was compared with the Mini-Mental State Examination (MMSE),

which displayed a significant correlation ($r = .82$) with the DRS-2 showing a greater sensitivity to change than the MMSE. In addition, correlations with the Wechsler Adult Intelligence Scale indicated a correlation of .75 between the WAIS full scale and the DRS-2 total score (Johnson-Greene, 2003).

The DRS-2 and its alternate version were administered to all participants, with one version being administered in the first session and the second version in the second session. This was done in order to measure cognitive fluctuations between testing sessions, which are well documented in individuals with DLB.

Visual Object and Space Perception Test (VOSP) (Warrington & James, 1991). This is a measure of visuoperceptual and spatial abilities. Specifically, this test assesses object and space perception. This battery was originally standardized on two cognitively intact medical samples in England. However, recently several studies have tested the psychometric properties of this test in different countries (Bird, Papadopoulou, Ricciardelli, Rossor, & Cipolotti, 2004; Bonello, Rapport, & Millis, 1997; Graham et al., 2004). All eight subtests of this battery were administered. It should be noted that there was a semantic component to this test, as accurate performance relied on naming on certain subtests. When this was the case the experimenter assisted participants who clearly knew the answer but were unable to state the appropriate word. Assistance was given to the participants by asking them to describe the object/animal or to state its use. Scoring for this test was completed for each individual subtest. In addition, a VOSP object perception composite score was calculated by adding the first four subtests and a VOSP space perception composite score was calculated by adding the last four subtests, these calculations were extracted from the user manual. In addition, a VOSP total score was also computed by adding all subtests.

I. Incomplete letters: Participants were asked to name 20 capital letters in which the clarity of the letter has been degraded making it difficult to identify the letters. According to the normative data, the cut-off score for this task is 19 correct answers. The specificity of this task was found to be excellent (98.2%). Internal consistency reliability was evaluated using the alpha coefficient and it was found to be low ($\alpha = .54$). Other psychometric properties have not been assessed. In this study, the total number of letters identified was the participant's score (max score = 20).

II. Silhouette naming: Participants were asked to name silhouette drawings of 30 common animals and objects which had been rotated to varying degrees. This rotation increases the demand on perceptual skills. It should be noted that there is a semantic component to this test, as accurate performance relies on naming. According to the normative data, the cut-off score for this task is 16 correct answers. The specificity of this task was found to range from an adequate to good level (68.5-82.4%). The sensitivity of this task was found to be at a good level (77%). Internal consistency reliability was evaluated using the alpha coefficient and it was found to be adequate ($\alpha = .78$). In an analysis of test-retest reliability it was found that this test had a good level of reliability ($r = .88$). Other psychometric properties have not been assessed. In this study, the total number of animals or objects identified was the participant's score (max score = 30).

III. Object decision: Participants were shown a selection of four silhouette drawings, including a rotated real object, and three object-like distracters, and they were asked to point to the real object. There was a total of 20 items on this test. According to the normative data, the cut-off score for this task is 16 correct answers. The specificity of this task was found to be good (83.8%). Internal consistency reliability was evaluated using

the alpha coefficient and it was found to be low ($\alpha = .58$). Other psychometric properties have not been assessed. In this study, the total number of objects identified was the participant's score (max score = 20).

IV. Progressive Silhouettes: Similarly to the silhouette naming subtest, participants were asked to name silhouette drawings of 10 common animals and objects which were rotated to varying degrees, however these images have the defining features of the object primarily hidden. Cards were presented in a graded fashion. That is, the first card that was presented had a very incomplete picture and with each additional card that was presented the picture became increasingly complete (i.e., for a total of 10 cards of increasing completeness). The participants were asked to guess the picture as soon as they could. The score was the total number of cards presented, where lower scores meant a better performance. The specificity of this task was found to be excellent ($> 90\%$). Other psychometric properties have not been assessed. In this study, the total number of cards presented was the participant's score (max score = 100, best score = 10).

V. Dot counting: Participants were shown a selection of between five and nine black dots, and they were asked to count them. There was a total of ten items in this test. According to the normative data, the cut-off score for this task was nine correct answers. The specificity of this task was found to be excellent (92.8%). Internal consistency reliability was evaluated using the alpha coefficient and it was found to be low ($\alpha = .41$). Other psychometric properties have not been assessed. In this study, the total number of correctly counted dots per item was the participant's score (max score = 10).

VI. Position Discrimination: Participants were presented with cards on which two squares appeared. Within each square there was a dot and the participant was asked to identify which square had a centered dot in it. There were a total of 20 stimulus cards. The specificity of this task was found to be good ($> 80\%$). Internal consistency reliability was evaluated using the alpha coefficient and it was found to be adequate ($\alpha = .58$). Other psychometric properties have not been assessed. In this study, the total number of correctly identified squares with a centered dot was the participant's score (max score = 20).

VII. Number location: Participants were shown two squares, one square had randomly placed numbers and the other had one dot in a position that corresponded to one of the numbers in the other square. The task was to name the number that was in the same position as the dot. There were ten items on this test. According to the normative data, the cut-off score for this task is seven correct answers. The specificity of this task was found to be adequate (71.2%). The sensitivity of this task was also found to be adequate (73.4%). Internal consistency reliability was evaluated using the alpha coefficient and it was found to be good ($\alpha = .84$). Other psychometric properties have not been assessed. In this study, the total number of correctly identified dot positions was the participant's score (max score = 10).

VIII. Cube analysis: Participants were asked to count the number of cubes represented in two dimensional drawings of arrangements of between five and 12 cubes. There was a total of ten items. According to the normative data, the cut-off score for this task was nine correct answers. The specificity of this task was found to be excellent (92.8%). The sensitivity of this task was found to be good (87.4%). Internal consistency reliability was

evaluated using the alpha coefficient and it was found to be adequate ($\alpha = .77$). Other psychometric properties have not been assessed. In this study the total number of correctly counted cubes was the participant's score (max score = 10).

Test of Everyday Attention (TEA) (Robertson, Ward, Ridgeway, & Nimmo-Smith, 1994).

This is a measure of attention. Specifically, it is aimed at assessing various types of attention in an ecologically valid manner as the tests are related to everyday tasks. This test has been normed on individuals from 18 to 80 years of age. There are eight subtests and individual psychometric properties were not found. However, overall psychometric properties for this battery are available. Coefficients for test-retest reliability were excellent over a one-year period ($r = .86$) (Robertson et al., 1994). The sensitivity of this task was found to be adequate (77.2%). The specificity of this task was also found to be good (87.2%) (Robertson et al., 1994). Scores on this test were computed for each individual subtest as a composite score could not be calculated.

I. Map search task: This is a timed task of selective attention. Participants were given two minutes to locate target symbols on a map which contained numerous irrelevant distracter symbols. There were 80 items that could be identified (max score = 80). This subtest was found to correlate significantly with another test of selective attention, the Ruff selective attention test ($r = .621$; Bate et al., 2001). Two separate scores were derived from this subtest, the first is the number of identified targets in 1 minute and the second is the number of identified targets in 2 minutes.

II. Elevator counting: This is a test that measures sustained attention. Participants were asked to listen to a recording of seven strings of tones of various lengths and were then asked to count the number of tones. There were seven items (max score = 7). This

subtest was found to correlate significantly with another test of sustained and selective attention, the Stroop modified color-word test ($r = .664$; Bate, Mathias, Crawford, 2001).

III. Elevator counting with distraction: This is a test of auditory selective attention. Participants were asked to listen to a recording of ten series of tones, and then were asked to count the high tones while ignoring the low tones. There were ten items on this test (max score = 10). This subtest was found to correlate significantly with another test of selective attention, the Stroop modified color-word test ($r = .243$; Bate et al., 2001).

IV. Visual elevator: This is a measure of attentional switching and cognitive flexibility. Participants were asked to count a series of drawings of elevator doors that were presented in rows on the pages of a presentation booklet. The task was self-paced. The drawings of the elevator doors are interspersed with large up- or down-pointing arrows, indicating that the direction of counting should change in line with the arrow (i.e., up or down). Two separate scores were derived from this subtest: the first score represents the number of visual strings counted correctly (max score = 10), while the second score was the timing score calculated by dividing the total time taken for the correct items by the total number of switches for the correct items. Lower values represented a superior performance to higher values on this timing score.

V. Auditory elevator with reversal: This is a measure of timed attentional switching and cognitive flexibility. This subtest was the same as the visual elevator subtest except that it was presented at a fixed speed on the audiotape. The score represented the number of strings of tones counted correctly (max score = 10).

VI. Telephone search: This is a measure of selective attention. Participants were asked to look for key symbols and ignore other symbols, while searching for plumbers in the simulated telephone directory. The score was calculated by dividing the total time taken by the number of symbols detected (max score = 20). Lower values represented higher scores on this test.

VII. Telephone search dual task: This is a measure of divided attention. Participants were asked to again search in the directory while simultaneously counting strings of tones presented through a sound system. The participant's score on this subtest was calculated by subtracting the time per target score on the previous subtest from the time per target score on the current subtest, which had been weighted for accuracy of tone counting. Lower and negative values represented a superior performance to higher values.

VIII. Lottery: This is a measure of sustained attention. Participants were asked to listen to their winning number in a lottery list, which they knew ended in "55". They had to listen to a 10-min series of tape-presented numbers of the form "BC143", "LD967", and so forth. Participants were specifically asked to write down the two letters preceding all numbers that ended in 55. There were 10 winning numbers within the 10 minute presentation. The participants score was the number of correctly recorded numbers (max score = 10).

Boston Naming Test (BNT) (Kaplan, Goodglass, & Weintraub, 1983). This is a test of word finding ability. Specifically, this measure consists of 60 large ink drawings that are presented in order of increasing difficulty. The participant was asked to identify the picture correctly. In dementia research, it is common to provide cues to individuals who cannot name the

picture (Mack et al., 1992). At first a semantic cue is given (e.g., for a pelican, a semantic cue would be “it’s a bird”), if the individual still does not respond a phonetic cue is provided (e.g., for a pelican, “pe...”). This test was normed on a population of 25 to 85 years of age. Normative data indicates that any score below 45 is abnormal. In this study, the total number of correctly identified drawings, whether the participant was cued or not, was used as the participant’s score on this test (maximum score = 60).

This test has been found to be reliable and valid in the assessment of word finding deficits in dementia. This test was found to be correlated with other measures of mental status, including the MMSE ($r = .65$). In terms of correctly identifying a semantic deficit in individuals with dementia, the BNT was found to have good sensitivity (76%) and excellent specificity (100%) (Calero, Arnedo, Navarro, Ruiz-Pedrosa, & Carnero, 2002). Test-retest reliability has been found to be high ($r = .94$).

3) Computerized Tasks. The following tests were administered using a computer and a touch screen monitor. One of these tasks was used as a measure of attention, whereas the other was used as a measure of driving behavior.

Useful Field of View (UFOV; Ball, Beard, Roenker, Miller, & Griggs, 1988). The UFOV© task is a measure of attention that examines three test variables and is composed of three subtests. In terms of test variables, an individual’s score on the UFOV© is a function of the duration of target presentation, the level of complexity of a secondary central task, and the salience of a peripheral target. The three subtests are entitled processing speed, divided attention, and selective attention. All of the subtests follow the same basic procedure with some subtle manipulations. In the processing speed subtest a silhouette of either a car or a truck was

presented in the center of the screen for a fraction of a second. Participants were then required to indicate whether they saw either a car or a truck. Participants completed successive trials in which the time of stimuli presentation was manipulated. In the divided attention subtest, participants were presented with either a car or a truck and required to indicate which icon they were presented with. Unlike the processing speed subtest, however, in this case participants were also presented with an additional icon at one of three eccentricities (10°, 20°, or 30°) and were required to only indicate the location of the icon. Finally, the selective attention subtest contains the same two components as the divided attention subtest, with the addition of the peripheral target being embedded in distractor visual stimuli which were triangles.

Performance on the UFOV is often cited as being related to performance on functional activities requiring sustained attention, such as driving (e.g., Myers, Ball, Kalina, Roth, & Goode, 2000). Among individuals with dementia, UFOV scores were more predictive of driving performance than dementia severity (Duchek et al., 1998). Test-retest reliability for the UFOV is high ($R=.88$; Edwards, Vance, Wadley, Cissell, Roenker, & Ball, 2005).

Simulated Driving Task. Participants were asked to complete a simulated driving scenario mimicking an on-road examination. The STISIM drive software was implemented on a 15 inch laptop screen with an associated steering and brake/throttle console (refer to Figure 1). This software was created (Systems Tech, California) as a tool to generate laboratory tasks relevant to psychomotor and cognitive demands of real-world driving. This software allows for the design of interactive vehicles on all lanes, buildings, traffic control devices, and pedestrians. The simulated drive was accompanied by realistic audio effects that provide acceleration and deceleration cues. Instructions to the drivers (e.g., turn left/right, lane changes and speed maintenance, etc.) were given through laptop speakers.

Figure 1.
Apparatus Setup for the Simulated Driving Task.

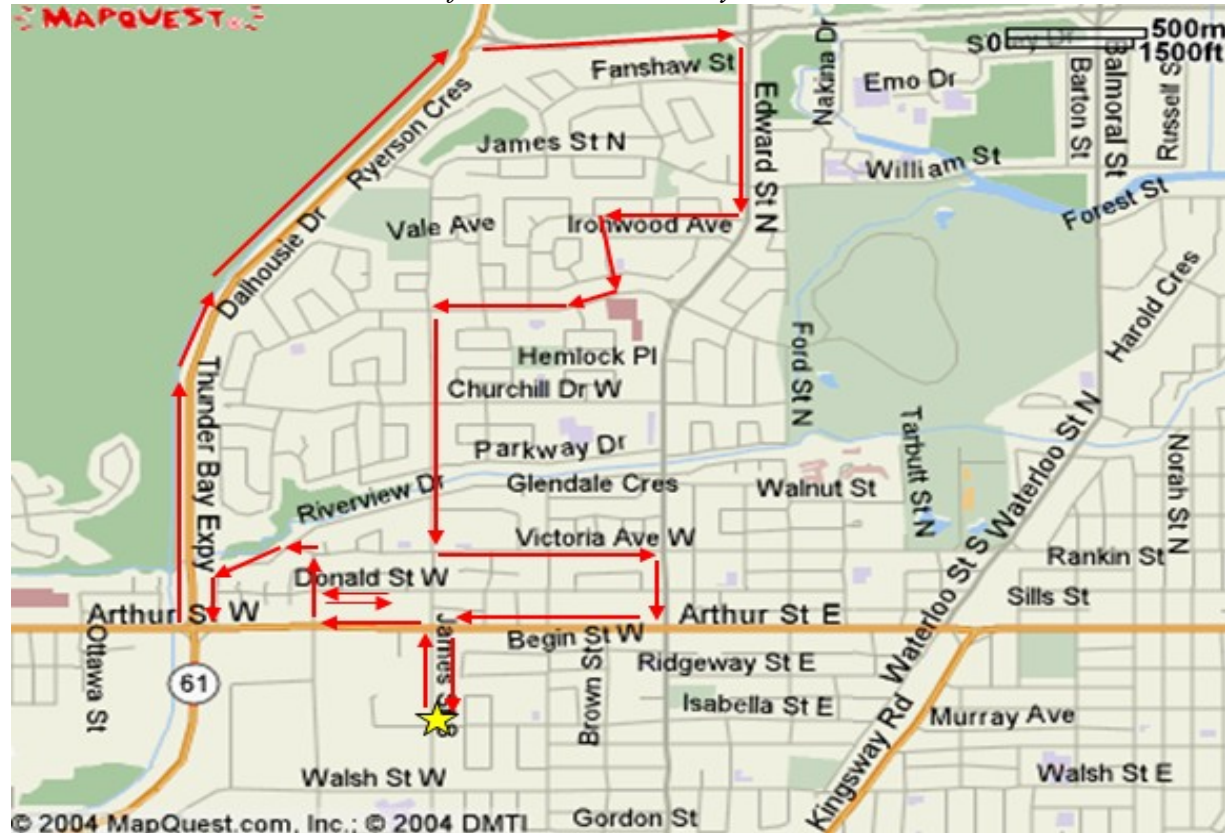


Before beginning, participants completed a comprehensive training session including a thorough explanation of the task, practice operating the pedals and steering wheel, followed by a training course that took approximately 20 minutes to complete. The training course was designed to ensure that participants practiced maintaining their speed, braking, stopping, making left and right turns and negotiating traffic. The experimenter ensured that all participants understood how the simulator operated before moving on to the test course. The test course presented in this study was 12 kilometers long, based on a real segment of road found in Thunder Bay, Ontario, and included driving in residential, highway and urban environments (refer to Figure 2). Measurements of the simulated driving task were generated both from driving performance as recorded by the computer as well as through a demerit-point assessment

(Appendix Q). Driving errors and parameters were recorded (e.g., speed exceedances, stop sign violations, traffic light violations, etc.) by the simulator.

Figure 2.

Scenario based on 12 kilometers of road in Thunder Bay, Ontario.



The demerit-point assessment is a written checklist used in licensing drivers in Manitoba and includes measures such as signal violations, passing and turning. All simulated drives were recorded and scored by two independent raters. Inter-rater reliability was found to be extremely high ($r = .99, p < .001$) and as such an average of both scores was taken as the participants score. For the purpose of this experiment, sections one through nine were scored and summed as the participants score.

RESULTS

This results section is partitioned into three separate sections. The first section focuses on qualifying the nature of cognitive impairments in the early stages of DLB. The second section aims to examine the driving performance of drivers with early AD and DLB. Finally, the last section presents the correlations between driving performance and findings on neuropsychological testing. As can be seen in Tables 1 and 2 all groups were appropriately matched for age, gender and years of education, as evidenced by the lack of statistically significant differences when comparing the clinical groups to the control group.

Table 1.
One-way ANOVA Demonstrating that Groups are Matched for Age and Years of Education.

Comparisons	Variables	<i>df</i>	<i>F</i>	<i>p</i>
Controls vs. AD	Age	1, 39	.54	.47
	Education	1, 39	.01	.93
Controls vs. DLB	Age	1, 34	.08	.78
	Education	1, 34	.67	.42
AD vs. DLB	Age	1, 33	.78	.38
	Education	1, 33	.64	.43

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$.

Table 2.
Chi-Square Demonstrating that Groups are Matched for Gender.

Comparisons	Group	Gender Female	Gender Male	χ^2	Φ
Controls vs. AD	Control	11	10	.22	.64
	AD	9	11		
Controls vs. DLB	Control	11	10	.54	.46
	DLB	6	9		
AD vs. DLB	AD	9	11	.09	.77
	DLB	6	9		

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$.

Section 1: Cognitive profile of older adults with DLB

The purpose of this first section of the thesis was to qualify the nature of cognitive impairments associated with early DLB since few studies have examined the associated profile. In order to accomplish this, a series of one-way ANOVAs were conducted between individuals with early DLB and healthy controls on several neuropsychological tests, including measures of global functioning, word finding abilities, attention and visuospatial abilities. These types of analyses were selected in lieu of a MANOVA because the assumptions for a MANOVA could not be met (i.e., insufficient cases per dependent variable).

As previously mentioned, groups were matched for age, gender and years of education. In addition, several other descriptive factors, thought to be associated with cognition, were examined (Lezak et al., 2004). When participants were asked about their handedness, 81% of the control group and 87% of the early DLB group reported being right handed, 14% of the control group and 13% of the of the early DLB group stated being left handed, while only 5% of the control group reported being ambidextrous. The number of comorbid medical illnesses, number of total medications taken regularly, number of Acetylcholinesterase inhibitors (AChEI) taken regularly, score on the geriatric depression scale, score on the global deterioration scale and presence of insomnia were also examined (refer to Tables 3 and 4). Several differences emerged between the control group and the early DLB group. In particular, the early DLB group was found to take significantly more medications on a daily basis than the control group, $F(1, 34) = 13.77, p < .001$. Not surprisingly, the early DLB group was found to take significantly more AChEIs than the control group, though what was surprising is that some participants were on more than one AChEI concurrently, $F(1, 34) = 128.92, p < .001$. Finally, dementia staging was determined using the Global Deterioration Scale and findings suggested that the early DLB

group had a mean of 3 ($SD = .00$), demonstrating that the group was in the early stages of dementia as defined by this study protocol, and the control group had a mean of 1.19 ($SD = .40$), meaning no presence of dementia. No difference was found between the groups in the number of comorbid illnesses reported, scores on the geriatric depression scale, and the presence of insomnia.

Table 3.

One-way ANOVA between Controls and Early DLB Demonstrating Group Composition.

Variables	Mean DLB	SD DLB	Mean Controls	SD Controls	<i>df</i>	<i>F</i>	<i>p</i>
Medical Illnesses	3.33	1.80	2.48	1.75	1, 34	2.05	.16
Number of Medications	5.20	3.19	2.10	1.81	1, 34	13.77**	.00
Number of AChEIs	.87	.35	.00	.00	1, 34	128.92**	.00
Geriatric Depression Scale	6.47	4.22	5.95	4.98	1, 34	.11	.75
Global Deterioration Scale	3.00	.00	1.19	.40	1, 34	300.83**	.00

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$.

Table 4.

Chi-Square between Controls and Early DLB Demonstrating Group Composition.

Variable	Group	YES	NO	χ^2	Φ
Presence of Insomnia	Control	13	8	.01	.91
	DLB	9	6		

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$.

In examining the results of the neuropsychological testing, it is evident that the early DLB participants performed significantly worse than healthy controls on most measures (refer to Table 5). This is not a surprising result given that a certain amount of global deterioration is expected in early dementia. In addition, scores significantly different from healthy controls do not necessarily indicate impairment. In examining test scores, neuropsychologists must often

decipher when a result is within the normal range, borderline range or if it represents a meaningful impairment. The vast majority of neuropsychologists use at least two standard deviations from the mean to diagnose impairment, which would indicate that at least 98% of the population obtained higher scores. It is widely accepted that 1-1.5 standard deviations from the mean is indicative of a MCI (i.e., borderline score), whereas at least two standard deviations from the mean is indicative of early dementia (Giovanetti, Schmidt, Gallo, Sestito, & Libon, 2005; Lezak et al., 2004; Lonie et al., 2010; Schinka et al., 2010; Schoenber & Scott, 2011; Schultz, Davis, & King, 2004). Thus, examining all significant results that are at least two standard deviations from the mean would be helpful in determining the profile of individuals with early DLB.

Table 5.

One-way ANOVA between Controls (Ctrls) and Early DLB on neuropsychological tests.

Neuropsych. Tests	Mean DLB	SD DLB	Mean Ctrls	SD Ctrls	df	F	p
MMSE	22.40	2.80	29.00	1.30	1, 34	90.24**	.00
DRS	108.60	17.57	136.38	4.43	1, 34	48.72**	.00
Attention	33.00	4.31	36.29	.72	1, 34	11.88**	.00
Initiat./ Persever.	27.47	5.59	34.91	3.25	1, 34	25.34**	.00
Construction	4.00	1.25	6.00	.00	1, 34	54.10**	.00
Conceptualization	30.40	6.87	35.52	1.81	1, 34	10.75**	.00
Memory	13.73	3.24	23.67	0.80	1, 34	183.93**	.00
BNT	46.6	9.74	53.62	3.26	1, 34	9.51**	.00
VOSP	96.40	11.85	113.43	7.67	1, 34	27.450**	.00
VOSP1	17.27	2.71	19.24	1.30	1, 34	8.46**	.01
VOSP2	14.13	3.11	18.62	4.56	1, 34	10.87**	.00
VOSP3	14.60	2.38	17.29	1.77	1, 34	15.12**	.00
VOSP4	15.20	2.98	12.81	3.25	1, 34	5.07*	.03
VOSP5	8.27	1.28	9.67	0.48	1, 34	21.13**	.00
VOSP6	16.93	4.43	19.14	1.11	1, 34	4.85*	.04
VOSP7	6.13	3.38	8.62	1.91	1, 34	7.90**	.01
VOSP8	3.87	3.66	8.05	2.73	1, 34	15.45**	.00
VOSP Object (1-4)	61.20	4.35	67.95	5.82	1, 34	14.41**	.00
VOSP Space (5-8)	35.20	10.58	45.48	4.62	1, 34	15.76**	.00
TEA1: 1 minute	9.47	4.44	28.62	10.26	1, 34	45.84**	.00
TEA1: 2 minutes	14.93	9.16	52.19	11.87	1, 34	103.39**	.00
TEA2	5.87	1.69	7.05	0.74	1, 34	8.19**	.01
TEA3	4.87	2.59	7.29	2.90	1, 34	6.64*	.01
TEA4: Raw score	2.73	3.15	6.91	1.95	1, 34	24.11**	.00
TEA4: Time score	9.34	10.96	5.98	2.01	1, 34	1.91	.18
TEA5	1.67	1.40	4.81	2.11	1, 34	25.21**	.00
TEA6	11.66	8.98	3.93	1.01	1, 34	15.51**	.00
TEA7	9.45	6.56	2.23	1.46	1, 34	24.08**	.00
TEA8	4.00	3.00	7.95	1.69	1, 34	25.40**	.00
UFOV1	272.07	120.32	24.91	16.74	1, 34	87.25**	.00
UFOV2	432.20	135.17	152.52	116.45	1, 34	44.16**	.00
UFOV3	490.20	37.96	304.76	115.13	1, 34	35.86**	.00
DRS Differences	5.73	4.94	4.00	3.38	1, 34	1.57	.22
Attention	3.53	3.54	0.81	0.75	1, 34	11.81**	.00
Initiat./ Persever.	2.93	3.63	2.38	3.35	1, 34	.22	.64
Construction	1.40	1.24	0.00	0.00	1, 34	26.99**	.00
Conceptualization	2.80	2.27	2.10	1.84	1, 34	1.05	.21
Memory	2.47	1.99	1.10	0.70	1, 34	8.54*	.01

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$. **Bold** values indicate significant findings that are > 2 SDs away from the mean of healthy controls.

Global measures of cognitive functioning were found to be at least two standard deviations from the mean in comparison to healthy controls. Specifically, participants with early DLB performed significantly worse on the MMSE than healthy controls, $F(1, 34) = 90.24, p < .001$. Participants with early DLB also performed significantly worse on the DRS than healthy controls, $F(1, 34) = 48.72, p < .001$, and they were at least two standard deviations from the mean on all subtests including attention, initiation/perseveration, construction, conceptualization, and memory. Similarly, participants with early DLB performed significantly worse on a measure of word finding, the BNT, and their performance was at least two standard deviations from the mean of healthy controls, $F(1, 34) = 9.51, p < .001$.

Though participants with early DLB were found to have significantly lower scores on all subtests measuring visuospatial/perceptual abilities, only a few scores were at least 2 standard deviations from the mean of healthy controls. Specifically, participants were found to have a significantly lower performance on the VOSP5 than healthy controls, indicating that they had difficulty counting the number of dots that were spatially dispersed, $F(1, 34) = 21.13, p < .001$. In addition, scores on the VOSP6 were found to be significantly lower than healthy controls, indicating that they had difficulty discriminating the position of targets, $F(1, 34) = 4.85, p < .001$. Both of these scores load into the conglomerate VOSP Space score, which is why participants with early DLB were found to have performed significantly worse on this conglomerate measure of spatial perception, $F(1, 34) = 15.76, p < .001$.

Similarly, all subtests of attentional functioning on the TEA and UFOV were found to be significantly different in participants with early DLB than healthy controls, though only some were found to be at least two standard deviations away from the mean of healthy controls. Specifically, scores on the TEA1 (i.e., selective attention) at 2 minutes were found to be

significantly lower in participants with early DLB than healthy controls, indicating that they identified fewer targets on the map, $F(1, 34) = 103.39, p < .001$. In addition, scores on the TEA6 (i.e., selective attention) were significantly higher for participants with early DLB than healthy controls, indicating that it took them longer to find the target phone numbers, $F(1, 34) = 15.51, p < .001$. On the TEA7 (i.e., divided attention) participants with early DLB had significantly higher time per target scores than healthy controls, $F(1, 34) = 24.08, p < .001$. Finally, participants with early DLB identified significantly fewer lottery number on the TEA8 (i.e., prolonged sustained attention) in comparison to healthy controls, $F(1, 34) = 25.40, p < .001$. In terms of the UFOV, participants with early DLB had significantly longer processing speed times on the UFOV1 in comparison to healthy controls, $F(1, 34) = 87.25, p < .001$. In addition, participants with early DLB had significantly longer divided attention scores on the UFOV2 in comparison to healthy controls, $F(1, 34) = 44.16, p < .001$.

The DRS was administered during the first session and the second session in order to capture cognitive fluctuations that are thought to be present in DLB. Findings on all subtests were compared using the absolute difference and these differences were analyzed. Three subtests were found to have absolute differences that were at least two standard deviations away from the absolute difference of healthy controls. Specifically, attention scores were found to fluctuate significantly more in the early DLB group than in healthy controls, $F(1, 34) = 11.81, p < .001$. Similarly, participants with early DLB were found to fluctuate significantly more on measures of visual spatial construction than healthy controls, $F(1, 34) = 26.99, p < .001$. Finally, participants with early DLB fluctuated significantly more on memory measures than healthy controls, $F(1, 34) = 8.54, p < .05$.

Section 2: Driving performance associated with early AD and early DLB

The purpose of this second section of the thesis was to examine the driving performance of drivers with early AD and DLB. This was accomplished through a simulated drive with outcomes measured by the simulator and through a rater scored measure of driving performance based on a demerit-point assessment. The simulator produced two types of outcomes, including driving errors (e.g., stop signs missed, off road accidents, etc.) and driving parameters (e.g., total run length, percent of time out of lane, etc.). Variables related to driving performance were subsequently analyzed through a series of one-way ANOVAs that were conducted between early AD or DLB and healthy controls. Once again ANOVAs were selected over conducting a MANOVA due to not meeting the assumptions for MANOVA.

As previously mentioned, groups were matched for age, gender and years of education. In addition, several other descriptive factors, thought to be associated with cognition, were examined (Lezak et al., 2004). When participants were asked about their handedness, 81% of the control group and 95% of the early AD group reported being right handed, 14% of the control group and 5% of the of the early AD group stated being left handed, while only 5% of the control group reported being ambidextrous. The number of comorbid medical illnesses, number of total medications taken regularly, number of Acetylcholinesterase inhibitors (AChEI) taken regularly, score on the geriatric depression scale, score on the global deterioration scale and presence of insomnia were also examined (refer to Tables 6 and 7). Several differences emerged between the control group and the early AD group. In particular, the early AD group was found to take significantly more medications on a daily basis than the control group, $F(1, 34) = 11.11, p < .001$. Not surprisingly, the early AD group was found to take significantly more AChEIs than the control group, though what was surprising is that some participants were on more than one

AChEI concurrently, just as the DLB group presented in section one, $F(1, 34) = 108.64$, $p < .001$. Finally, dementia staging was determined using the Global Deterioration Scale and findings suggested that the early AD group had a mean of 3.10 ($SD = .31$), demonstrating that the group was in the early stages of dementia as defined by this study protocol, and the control group had a mean of 1.19 ($SD = .40$), meaning no presence of dementia. Interestingly, group differences were found in the presence of insomnia, in that participants with AD reported significantly less insomnia than healthy controls, $\chi^2(1, N = 41) = 11.90$, $p < .001$. No differences were found between the groups in the number of comorbid illnesses reported and scores on the geriatric depression scale.

Table 6.

One-way ANOVA between Controls and Early AD Demonstrating Group Composition.

Variables	Mean AD	SD AD	Mean Controls	SD Controls	<i>df</i>	<i>F</i>	<i>p</i>
Medical Illnesses	2.45	1.43	2.48	1.75	1, 39	.01	.96
Number of Medications	5.11	3.66	2.10	1.81	1, 39	11.11**	.00
Number of AChEIs	0.94	0.42	0.00	0.00	1, 39	108.64**	.00
Geriatric Depression Scale	3.70	2.87	5.95	4.98	1, 39	3.11	.09
Global Deterioration Scale	3.10	0.31	1.19	0.40	1, 39	289.14**	.00

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$.

Table 7.

Chi-Square between Controls and Early AD Demonstrating Group Composition.

Variable	Group	YES	NO	χ^2	Φ
Presence of Insomnia	Control	13	8	11.90**	.00
	AD	2	18		

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$.

In examining the driving performance results, it is evident that early AD drivers had significantly worse outcomes in comparison to healthy older drivers (refer to Table 8). The experimenter rated driving performance as measured by a demerit-point assessment as well as all driving errors, with the exception of the number of pedestrians hit ($p = .31$) and the total number of stop signs missed ($p = .95$), were all significantly worse in drivers with early AD than healthy control drivers. When examining driving parameters, participants with early AD spent significantly more time over the speed limit of 50 km/h in comparison to health control drivers, $F(1, 39) = 18.11, p < .001$. In addition, drivers with early AD spent significantly more time out of their lane than healthy control drivers, $F(1, 34) = 5.53, p < .05$.

Table 8.

One-way ANOVA between Controls (Ctrls) and Early AD on measures of driving performance.

	Mean AD	SD AD	Mean Ctrls	SD Ctrls	df	F	p
Demerit-point Average	261.00	96.49	107.74	59.64	1, 39	37.84**	.00
Total Number of Off Road Accidents (ORA)	2.05	2.69	0.14	0.36	1, 39	10.42**	.00
Total Number of Collisions (C)	3.65	3.27	1.05	1.24	1, 39	11.59**	.00
Total Number of Pedestrians Hit (PH)	0.05	0.22	0.00	0.00	1, 39	1.05	.31
Total Number of Speed Exceedances (SE)	16.15	4.97	12.24	4.74	1, 39	6.66*	.01
Total Number of Traffic Light Tickets (TLT)	2.15	0.93	0.43	0.60	1, 39	49.97**	.00
Total Number of Stop Signs Missed (SSM)	1.50	1.28	1.52	0.98	1, 39	.01	.95
Total Number of Centerline Crossings (CC)	7.70	6.11	2.62	3.34	1, 39	11.08**	.00
Total Number of Road Edge Excursions (REE)	14.35	13.45	7.52	9.76	1, 39	3.49	.07
Total Number of Stops at Traffic Lights (STL)	0.80	0.83	1.91	1.00	1, 39	14.77**	.00
Total Errors	50.55	24.04	27.86	16.18	1, 39	12.69**	.00
Total Run Length-Time (RLT)	1054.27	257.68	1151.50	122.18	1, 39	2.42	.13
Over the Speed Limit Percent of Time (SLT)	28.89	15.07	12.95	8.04	1, 39	18.11**	.00
Out of Lane Percent of Time (OLT)	9.00	9.28	3.46	5.40	1, 39	5.53*	.02

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$. **Bold** values indicate significant findings that are > 2 SDs away from the mean of healthy controls.

Once again, it was of interest to examine truly impaired performance as it is more meaningful than simply examining difference between groups. Scores that are 1-1.5 standard deviations from the mean are considered borderline, whereas scores at least two standard deviations from the mean of healthy controls are considered truly impaired as these scores are in the bottom 2% (Giovanetti et al., 2005; Lezak et al., 2004; Lonie et al., 2010; Schinka et al., 2010; Schoenber et al., 2011; Schultz et al., 2004). Scores on the experimenter rated demerit-

point assessment indicated that drivers with early AD made significantly more errors on the road than healthy control drivers, $F(1, 39) = 37.84, p < .001$. In addition, the total number of off-road accidents as measured by the simulator were found to be significantly higher in drivers with early AD than healthy drivers, $F(1, 39) = 10.42, p < .001$. The total number of collisions as measured by the simulator were also found to be significantly higher in early AD drivers, $F(1, 39) = 10.59, p < .001$. Finally, the total number of traffic light tickets as measured by the simulator was found to be significantly higher in early AD drivers, meaning that they had a tendency to drive through red lights, $F(1, 39) = 49.97, p < .001$.

The driving performances of drivers with early DLB in comparison to healthy controls were also examined. These two groups have been thoroughly described in the previous section of this thesis. Similarly to the early AD drivers, it is evident that the early DLB drivers had significantly poorer outcomes in comparison to healthy older drivers (refer to Table 9). The experimenter rated driving performance as measured by the demerit-point assessment as well as all driving errors, with the exception of the number of pedestrians hit ($p = .24$) and the total number of off-road accidents ($p = .13$), were all significantly worse in drivers with early DLB than healthy control drivers. When examining driving parameters, participants with early DLB spent significantly more time over the speed limit of 50 km/h in comparison to healthy control drivers, $F(1, 34) = 6.82, p < .05$. In addition, drivers with early DLB spent significantly more time out of their lane than healthy control drivers, $F(1, 34) = 9.01, p < .05$.

Table 9.

One-way ANOVA between Controls (Ctrls) and Early DLB on measures of driving performance.

	Mean DLB	SD DLB	Mean Ctrls	SD Ctrls	<i>df</i>	<i>F</i>	<i>p</i>
Demerit-Point Average	284.17	80.00	107.74	59.64	1, 34	57.61**	.00
Total Number of Off Road Accidents (ORA)	1.27	3.28	0.14	0.36	1, 34	2.45	.13
Total Number of Collisions (C)	3.27	1.75	1.05	1.24	1, 34	19.81**	.00
Total Number of Pedestrians Hit (PH)	0.07	0.26	0.00	0.00	1, 34	1.42	.24
Total Number of Speed Exceedances (SE)	17.00	5.64	12.24	4.74	1, 34	7.53**	.01
Total Number of Traffic Light Tickets (TLT)	2.47	0.52	0.43	0.60	1, 34	113.62**	.00
Total Number of Stop Signs Missed (SSM)	2.47	0.83	1.52	0.98	1, 34	9.13**	.01
Total Number of Centerline Crossings (CC)	9.67	4.34	2.62	3.34	1, 34	30.39**	.00
Total Number of Road Edge Excursions (REE)	17.80	10.43	7.52	9.76	1, 34	9.17**	.01
Total Number of Stops at Traffic Lights (STL)	0.60	0.51	1.91	1.00	1, 34	21.64**	.00
Total Errors	57.07	18.03	27.86	16.18	1, 34	25.95**	.00
Total Run Length-Time (RLT)	1129.14	255.25	1151.50	122.18	1, 34	0.12	.73
Over the Speed Limit Percent of Time (SLT)	23.78	16.53	12.95	8.04	1, 34	6.82*	.01
Out of Lane Percent of Time (OLT)	12.65	12.56	3.46	5.40	1, 34	9.01**	.01

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$. **Bold** values indicate significant findings that are > 2 SDs away from the mean of healthy controls.

The same protocol to determine meaningful impairment (i.e., two standard deviations away from the mean) as used for early AD drivers was used for early DLB drivers. Scores on the experimenter rated demerit-point assessment indicated that drivers with early DLB made significantly more errors on the road than healthy control drivers, $F(1, 34) = 57.61$, $p < .001$. In addition, the total number of traffic light tickets as measured by the simulator was found to be

significantly higher in early DLB drivers, meaning that they had a tendency to drive through red lights, $F(1, 34) = 113.62, p < .001$.

Section 3: Prediction of impaired driving in early AD and DLB

The purpose of this section was to attempt to accurately predict impaired driving (i.e., performance at least two standard deviations away from the mean of healthy control drivers) with the use of neuropsychological test performance. In order to predict impaired driving in early AD neuropsychological test performance had to be analysed (refer to Table 10). The same procedure to determine impairment, as outlined in section one, was used here. The first finding was that global measures of cognitive functioning were found to be at least two standard deviations from the mean in comparison to healthy controls. Specifically, participants with early AD performed significantly worse on the MMSE than healthy controls, $F(1, 39) = 20.72, p < .001$. Participants with early AD also performed significantly worse on the DRS than healthy controls, $F(1, 39) = 50.95, p < .001$, and they were at least two standard deviations from the mean on the initiation/perseveration and memory subtests. Similarly, participants with early AD performed significantly worse on a measure of word finding difficulties, the BNT, and their performance was at least two standard deviations from the mean of healthy controls, $F(1, 39) = 14.59, p < .001$.

Table 10.

One-way ANOVA between Controls (Ctrls) and Early AD on neuropsychological tests.

Neuropsych. Tests	Mean AD	SD AD	Mean Ctrls	SD Ctrls	df	F	p
MMSE	24.00	4.86	29.00	1.30	1, 39	20.72**	.00
DRS	115.00	12.96	136.38	4.43	1, 39	50.95**	.00
Attention	35.15	2.06	36.29	0.72	1, 39	5.67*	.02
Initiation/ Perseveration	27.20	7.43	34.91	3.25	1, 39	18.81**	.00
Construction	5.80	0.52	6.00	0.00	1, 39	3.07	.09
Conceptualization	32.80	3.49	35.52	1.81	1, 39	9.99**	.00
Memory	14.05	3.79	23.67	0.80	1, 39	129.36**	.00
BNT	43.00	12.30	53.62	3.26	1, 39	14.59**	.00
VOSP	107.50	13.26	113.43	7.67	1, 39	3.11	.09
VOSP1	18.50	3.10	19.24	1.30	1, 39	1.00	.32
VOSP2	15.30	5.01	18.62	4.56	1, 39	4.94*	.03
VOSP3	16.35	2.50	17.29	1.77	1, 39	1.93	.17
VOSP4	13.35	4.04	12.81	3.25	1, 39	.22	.64
VOSP5	9.55	1.96	9.67	0.48	1, 39	.07	.79
VOSP6	17.85	3.35	19.14	1.11	1, 39	2.82	.10
VOSP7	9.45	3.30	8.62	1.91	1, 39	.99	.33
VOSP8	7.15	2.78	8.05	2.73	1, 39	1.09	.30
VOSP Object (1-4)	63.50	7.13	67.95	5.82	1, 39	4.82*	.03
VOSP Space (5-8)	44.00	7.41	45.48	4.62	1, 39	.59	.45
TEA1: 1 minute	19.85	14.68	28.62	10.26	1, 39	4.96*	.03
TEA1: 2 minutes	30.35	19.71	52.19	11.87	1, 39	18.68**	.00
TEA2	6.75	0.72	7.05	0.74	1, 39	1.71	.20
TEA3	4.30	2.62	7.29	2.90	1, 39	11.93**	.00
TEA4: Raw score	3.05	3.25	6.91	1.95	1, 39	21.45**	.00
TEA4: Time score	9.47	9.05	5.98	2.01	1, 39	2.97	.09
TEA5	1.60	1.70	4.81	2.11	1, 39	28.57**	.00
TEA6	8.66	8.35	3.93	1.01	1, 39	6.65*	.01
TEA7	64.21	135.01	2.23	1.46	1, 39	4.43*	.04
TEA8	3.75	3.04	7.95	1.69	1, 39	30.32**	.00
UFOV1	143.15	142.79	24.91	16.74	1, 39	14.21**	.00
UFOV2	406.10	130.90	152.52	116.45	1, 39	43.05**	.00
UFOV3	452.40	90.86	304.76	115.13	1, 39	20.64**	.00

Note: * indicates significance at the $p < .05$ and ** indicates significance at the $p < .001$. **Bold** values indicate significant findings that are > 2 SDs away from the mean of healthy controls.

Most subtests of attentional functioning on the TEA and UFOV were found to be significantly different in participants with early AD than healthy controls, though only some were found to be at least two standard deviations away from the mean of healthy controls.

Specifically, the TEA4 raw score (i.e., attentional switching and cognitive flexibility) scores were found to be significantly lower in participants with early AD than healthy controls, $F(1, 39) = 21.45, p < .001$. Scores on the TEA6 (i.e., selective attention) were significantly higher for participants with early AD than healthy controls, indicating that it took them longer to find the target phone numbers, $F(1, 39) = 6.65, p < .05$. On the TEA7 (i.e., divided attention) participants with early AD had significantly higher time per target scores than healthy controls, $F(1, 39) = 4.43, p < .05$. Finally, participants with early AD identified significantly fewer lottery number on the TEA8 (i.e., prolonged sustained attention) in comparison to healthy controls, $F(1, 39) = 30.32, p < .001$. In terms of the UFOV, participants with early AD had significantly longer processing speed times on the UFOV1 in comparison to healthy controls, $F(1, 39) = 14.21, p < .001$. In addition, participants with early AD had significantly longer divided attention scores on the UFOV2 in comparison to healthy controls, $F(1, 39) = 43.05, p < .001$.

Now that neuropsychological test performance has been reviewed for participants with early AD, the focus can be shifted to attempting to accurately predict impaired driving with the use of neuropsychological test performance. First, correlations were computed between measures of impaired driving (i.e., at least two standard deviations from the mean of the control group) and neuropsychological test performance in both early AD and DLB (refer to Tables 11 and 12). As can be seen several correlations emerged between impaired driving and neuropsychological functioning. Namely, in the AD group, a total of 12 significant correlations emerged, while in the DLB group, 11 significant correlations emerged. Rather than examining these correlations blindly it would be more useful to specifically look at the correlations between impaired driving and impaired neuropsychological functioning. The rationale for concentrating efforts on the significant correlations with impaired performance on neuropsychological tests is that these

deficits are what define the population from a cognitive standpoint. Thus, it is more useful to look at the cognitive deficits that are characteristic of participants of early AD and early DLB, especially if the goal is to inform practitioners in identifying individuals with early dementia who may no longer be able to drive safely. Scatterplots of the significant correlations between impaired driving performance and impaired neuropsychological performance were then produced. Finally, sensitivity (i.e., $\text{true positives} / (\text{true positives} + \text{false negatives})$) and specificity (i.e., $\text{true negatives} / (\text{true negatives} + \text{false positives})$) for each neuropsychological measure were calculated.

Table 11.

Correlation coefficients for neuropsychological test performance and impaired driving performance for AD group.

Variables	Demerit-Point	ORA	C	TLT
MMSE	-.40	.13	.19	.08
DRS	-.38	.02	.38	.14
DRS Attention	-.26	.04	.42	.26
DRS Initia./Pers.	-.34	.05	.44	.16
DRS Construct.	-.31	.23	.39	.17
DRS Concept.	-.31	-.29	.23	.01
DRS Memory	-.17	.18	-.05	.00
BNT	-.53*	.01	.21	.06
VOSP	-.29	-.00	.49*	.21
VOSP1	-.35	.06	.29	.30
VOSP2	-.24	.08	.35	.08
VOSP3	-.24	-.26	.40	.02
VOSP4	.54*	.19	.25	.07
VOSP5	.14	.12	.31	.30
VOSP6	-.50	.04	.11	.11
VOSP7	-.13	.03	.15	.08
VOSP8	-.48*	-.43	.15	-.03
VOSP Object	-.10	.10	.65**	.23
VOSP Space	-.43	-.10	.25	.15
TEA1: 1 min.	-.19	-.04	.28	.01
TEA1: 2 min.	-.28	-.07	.37	-.12
TEA2	-.47*	-.46*	-.29	-.18
TEA3	-.37	.10	.00	-.06
TEA4 Raw	-.43	-.15	.07	-.33
TEA4 Time	-.23	-.05	.02	.41
TEA5	-.63**	-.27	-.38	-.62**
TEA6	.32	.45*	-.11	.01
TEA7	.32	-.02	-.25	-.22
TEA8	-.62**	-.27	-.08	-.10
UFOV1	.17	-.18	-.26	-.29
UFOV2	.56**	.30	.16	.12
UFOV3	.40	.24	.20	.22

Note: *p<.05; **p<.001

Table 12.

Correlation coefficients for neuropsychological test performance and impaired driving performance for DLB group. (11 correlations)

Variables	Demerit-Point	TLT
MMSE	-.62*	-.48
DRS	-.45	-.31
DRS Attention	-.34	-.13
DRS Initia./Pers.	-.27	-.30
DRS Construct.	-.30	-.11
DRS Concept.	-.41	-.26
DRS Memory	-.53*	-.39
BNT	-.30	-.27
VOSP	-.58*	-.33
VOSP1	-.17	.01
VOSP2	-.33	-.31
VOSP3	-.28	-.13
VOSP4	.65**	.31
VOSP5	-.71**	-.53*
VOSP6	-.49	-.36
VOSP7	-.54*	-.33
VOSP8	-.46	-.04
VOSP Object	-.05	-.08
VOSP Space	-.62*	-.33
TEA1: 1 min.	-.51	-.20
TEA1: 2 min.	-.65**	-.28
TEA2	-.40	-.17
TEA3	-.56*	-.32
TEA4 Raw	-.26	-.05
TEA4 Time	-.33	-.07
TEA5	.01	.33
TEA6	.40	.17
TEA7	-.29	.03
TEA8	-.24	.00
UFOV1	.55*	.28
UFOV2	.51	.30
UFOV3	.48	.25

*Note: *p<.05; **p<.001*

For the early AD drivers, driving performance was found to be impaired on the demerit-point assessment, number of off-road accidents, number of collisions, and number of traffic light tickets (refer to Table 8). In addition, participants with early AD were found to have impaired performance on several neuropsychological tests including the MMSE, DRS, DRS

initiation/perseveration subtest, DRS memory subtest, BNT, TEA4, TEA6, TEA7, TEA8, UFOV1, and UFOV2 (refer to Table 10). Measures of impaired driving performance and measures of impaired neuropsychological functioning performance were correlated for drivers with early AD (refer to Table 13).

Table 13.

Correlation coefficients between impaired neuropsychological test performance and impaired driving performance for AD group.

Variables	Demerit-Point	ORA	C	TLT
MMSE	-.40	.13	.19	.08
DRS	-.38	.02	.38	.14
DRS Initia./Pers.	-.34	.05	.44	.16
DRS Memory	-.17	.18	-.05	.00
BNT	-.53*	.01	.21	.06
TEA4 Raw	-.43	-.15	.07	-.33
TEA6	.32	.45*	-.11	.01
TEA7	.32	-.02	-.25	-.22
TEA8	-.62**	-.27	-.08	-.10
UFOV1	.17	-.18	-.26	-.29
UFOV2	.56**	.30	.16	.12

Note: * $p < .05$; ** $p < .001$

Correlations revealed that the demerit-point assessment correlated with several neuropsychological tests. Specifically, impaired performance on the demerit-point assessment was found to correlate significantly with impaired performance on the BNT in early AD participants, $r(39) = -.53$, $p < .05$. The scatterplot revealed that the BNT had a sensitivity of 75% at predicting impaired early AD drivers and a specificity of 62.5% at predicting non-impaired AD drivers (refer to Figure 3). In addition, impaired performance on the demerit-point assessment was found to correlate significantly with impaired performance on the TEA8 subtest (i.e., prolonged sustained attention) in early AD drivers, $r(39) = -.62$, $p < .001$. The scatterplot revealed that the TEA8 had a sensitivity of 50% at predicting impaired early AD drivers and a specificity of 62.5% at predicting non-impaired AD drivers (refer to Figure 4). Finally, impaired performance on the demerit-point assessment was found to also correlate significantly with

impaired performance on the UFOV2 (i.e., divided attention) in early AD drivers, $r(39) = .56$, $p < .001$. The scatterplot revealed that the UFOV2 had a sensitivity of 83.3% at predicting impaired early AD drivers and a specificity of 62.5% at predicting non-impaired AD drivers (refer to Figure 5). Impaired levels of off-road accidents was found to correlate significantly with impaired performance on the TEA6 (i.e., selective attention) in early AD drivers, $r(39) = .45$, $p < .05$. In addition, the scatterplot revealed that the TEA6 had a sensitivity of 38.5% at predicting impaired early AD drivers and a specificity of 71.4% at predicting non-impaired AD drivers (refer to Figure 6).

Figure 3.

Scatterplot of performance on BNT and the demerit-point assessment for drivers with early AD.

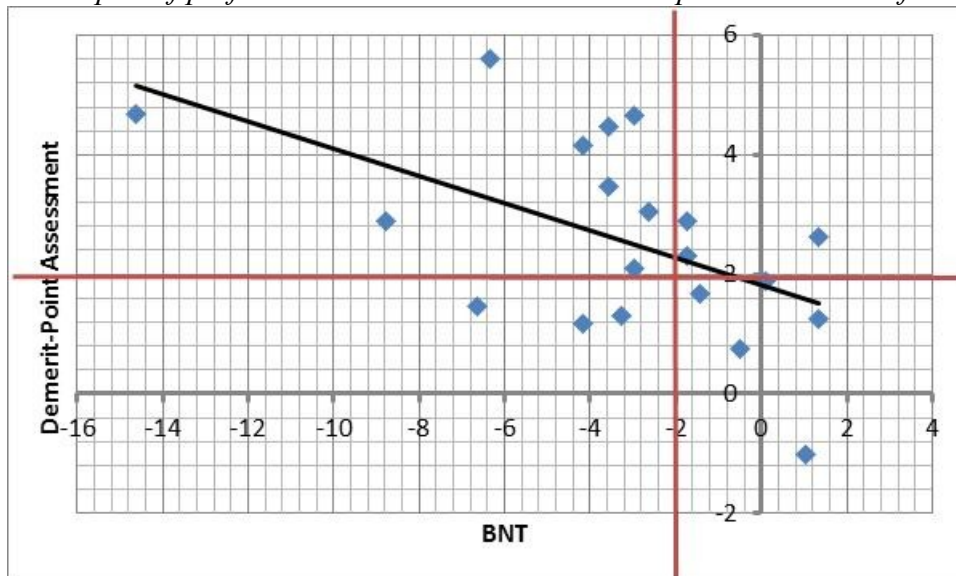


Figure 4.

Scatterplot of performance on TEA8 and the demerit-point assessment for drivers with early AD.

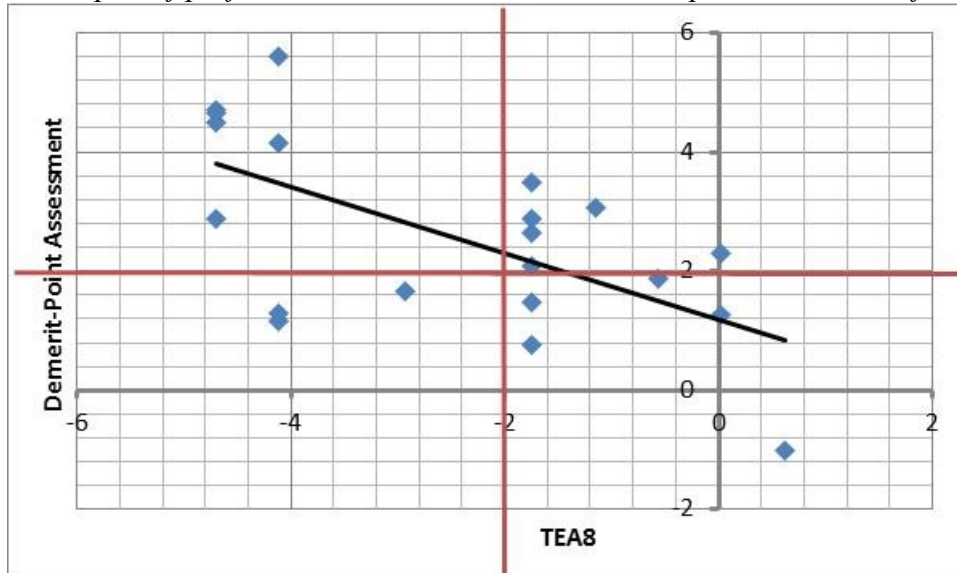


Figure 5.

Scatterplot of performance on UFOV2 and the demerit-point assessment for drivers with early AD.

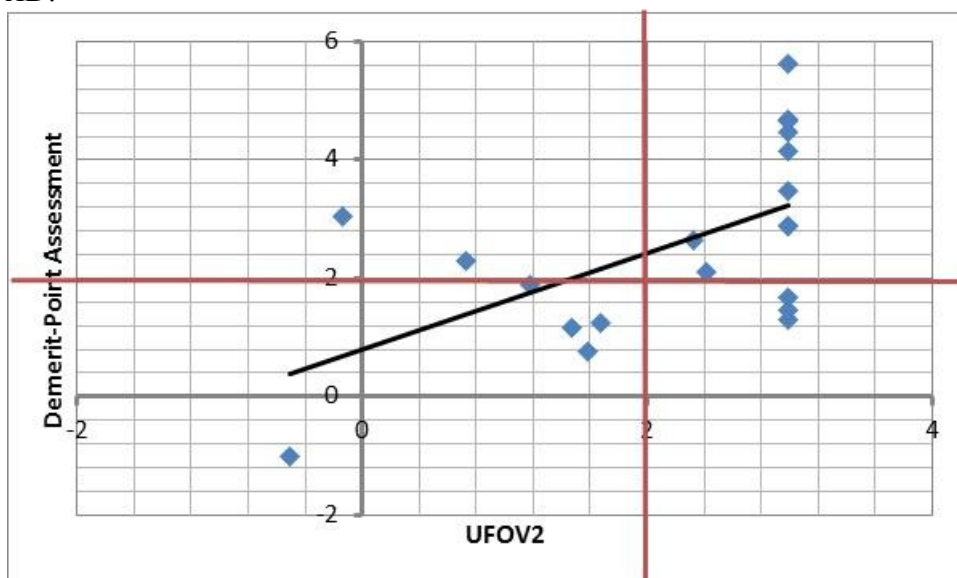


Table 14.

Correlation coefficients between impaired neuropsychological test performance and impaired driving performance for DLB group.

Variables	Demerit-Point	TLT
MMSE	-.62*	-.48
DRS	-.45	-.31
DRS Attention	-.34	-.13
DRS1 Initiation/Perseveration	-.27	-.30
DRS1 Construction	-.30	-.11
DRS1 Conceptualization	-.41	-.26
DRS1 Memory	-.53*	-.39
BNT	-.30	-.27
VOSP5	-.71**	-.53*
VOSP6	-.49	-.36
VOSP Spatial Perception	-.62*	-.33
TEA1 at 2 minutes	-.65**	-.28
TEA4 Raw	-.26	-.05
TEA6	.40	.17
TEA7	-.29	.03
TEA8	-.24	.00
UFOV1	.55*	.28
UFOV2	.51	.30

Note: * $p < .05$; ** $p < .001$

Correlations revealed that the demerit-point assessment correlated with several neuropsychological tests. Specifically, impaired performance on the demerit-point assessment was found to correlate significantly with impaired performance on the MMSE in early DLB participants, $r(34) = -.62$, $p < .05$. The scatterplot revealed that the MMSE had a sensitivity of 100% at predicting impaired early DLB drivers and a specificity of 33% at predicting non-impaired DLB drivers (refer to Figure 7). Impaired performance on the demerit-point assessment was also found to correlate significantly with impaired performance on the DRS memory subtest in early DLB, $r(34) = -.53$, $p < .05$. The scatterplot revealed that the DRS memory subtest had a sensitivity of 100% at predicting impaired early DLB drivers but a specificity of 0% at predicting non-impaired DLB drivers (refer to Figure 8). Impaired performance on the demerit-point assessment was also found to correlate significantly with impaired performance on the VOSP5 in

early DLB, $r(34) = -.71, p < .001$. The scatterplot revealed that the VOSP5 had a sensitivity of 58.3% at predicting impaired early DLB drivers and a specificity of 100% at predicting non-impaired DLB drivers (refer to Figure 9). Impaired performance on the demerit-point assessment was also found to correlate significantly with impaired performance on the VOSP Spatial Perception congrate score (VOSP5-8) in early DLB, $r(34) = -.62, p < .05$. The scatterplot revealed that the VOSP Spatial Perception congrate score had a sensitivity of 58.3% at predicting impaired early DLB drivers and a specificity of 100% at predicting non-impaired DLB drivers (refer to Figure 10). Impaired performance on the demerit-point assessment was also found to correlate significantly with impaired performance on the TEA1 at 2 minutes in early DLB, $r(34) = -.65, p < .001$. The scatterplot revealed that the TEA1 at 2 minutes had a sensitivity of 100% at predicting impaired early DLB drivers and a specificity of 33.3% at predicting non-impaired DLB drivers (refer to Figure 11). Finally, impaired performance on the demerit-point assessment was also found to correlate significantly with impaired performance on the UFOV1 in early DLB, $r(34) = .55, p < .05$. The scatterplot revealed that the UFOV1 had a sensitivity of 100% at predicting impaired early DLB drivers and a specificity of 33.3% at predicting non-impaired DLB drivers (refer to Figure 12).

Figure 7.

Scatterplot of performance on MMSE and the demerit-point assessment for drivers with early DLB.

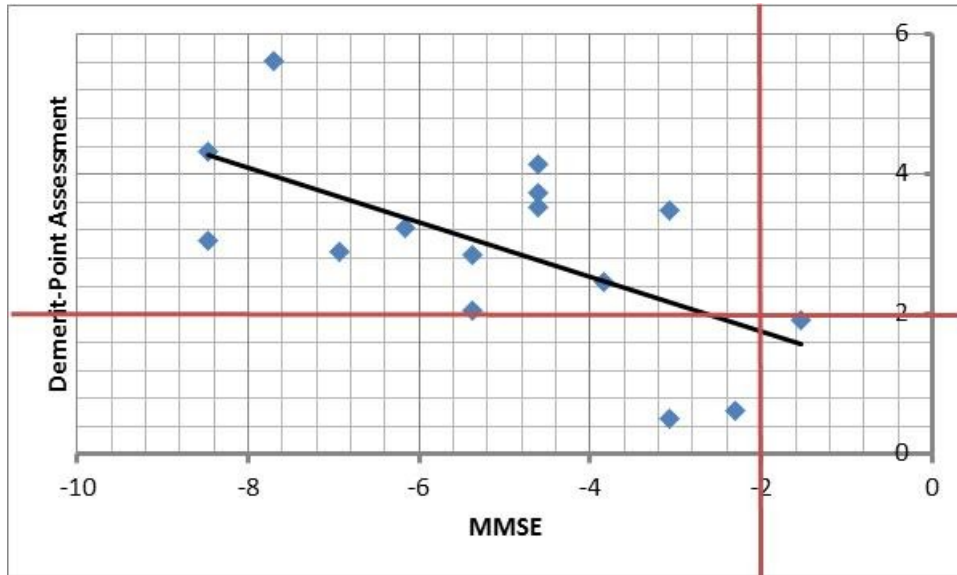


Figure 8.

Scatterplot of performance on the DRS memory subtest and the demerit-point assessment for drivers with early DLB.

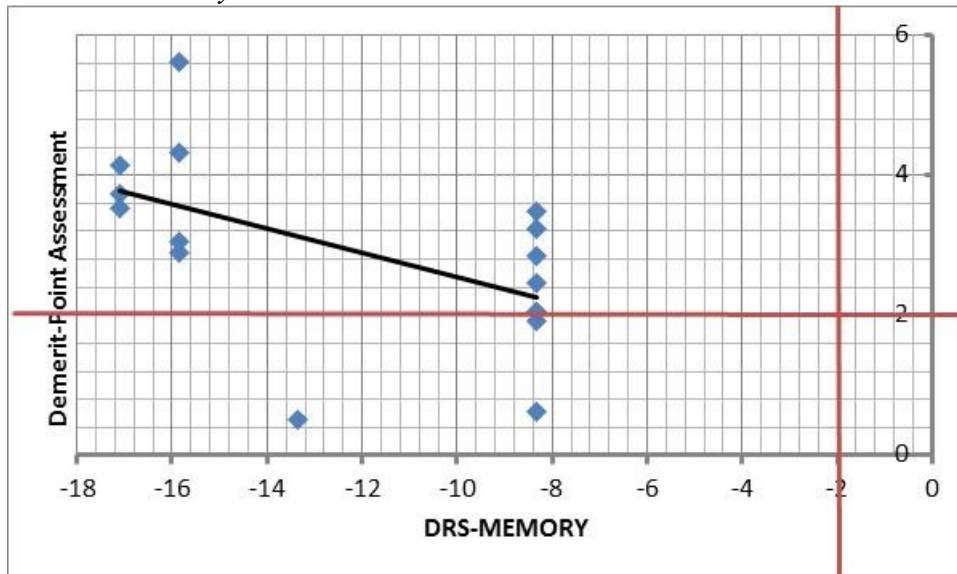


Figure 9.

Scatterplot of performance on the VOSP5 and the demerit-point assessment for drivers with early DLB.

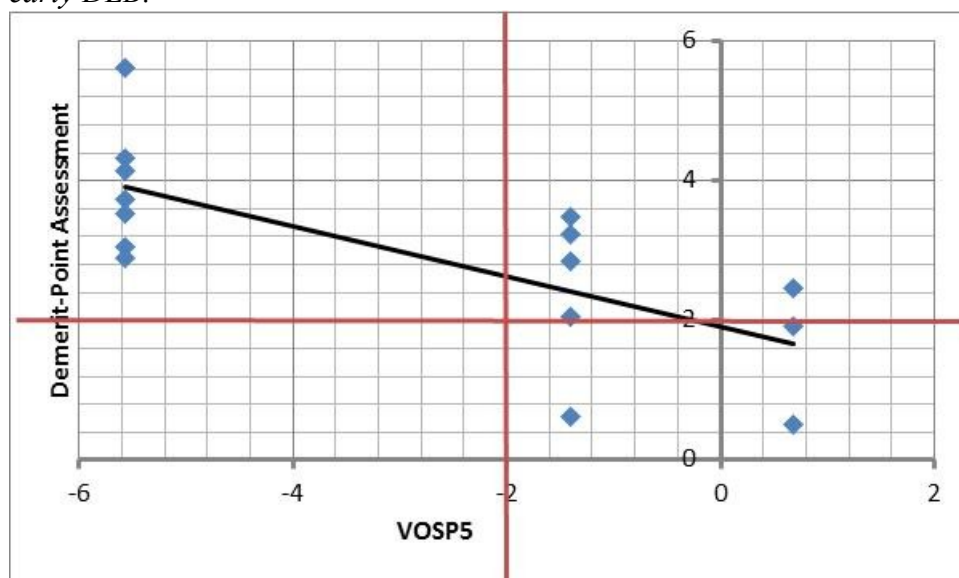


Figure 10.

Scatterplot of performance on the VOSP Spatial Perception congragate score (VOSP5-8) and the demerit-point assessment for drivers with early DLB.

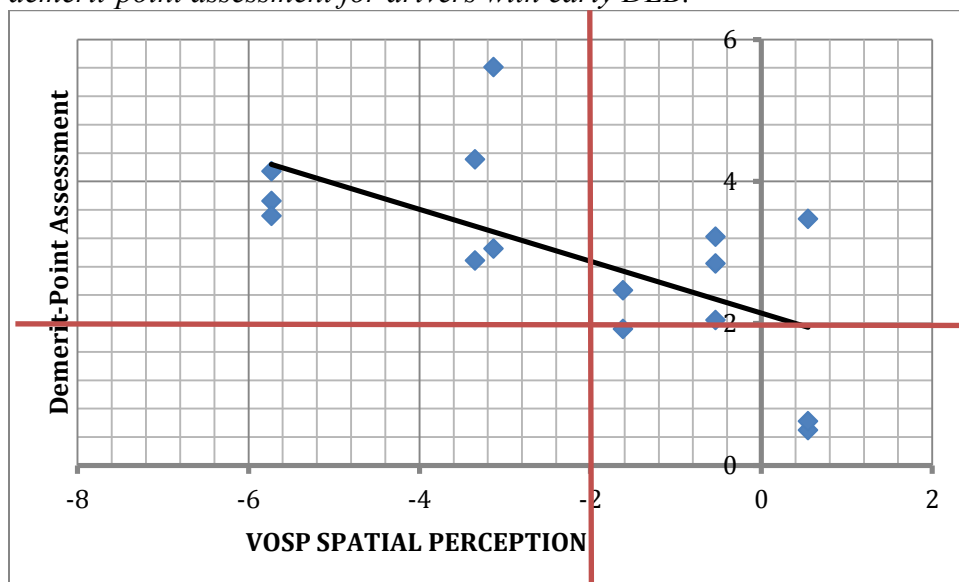


Figure 11.

Scatterplot of performance on the TEA1 subtest at 2 minutes and the demerit-point assessment for drivers with early DLB.

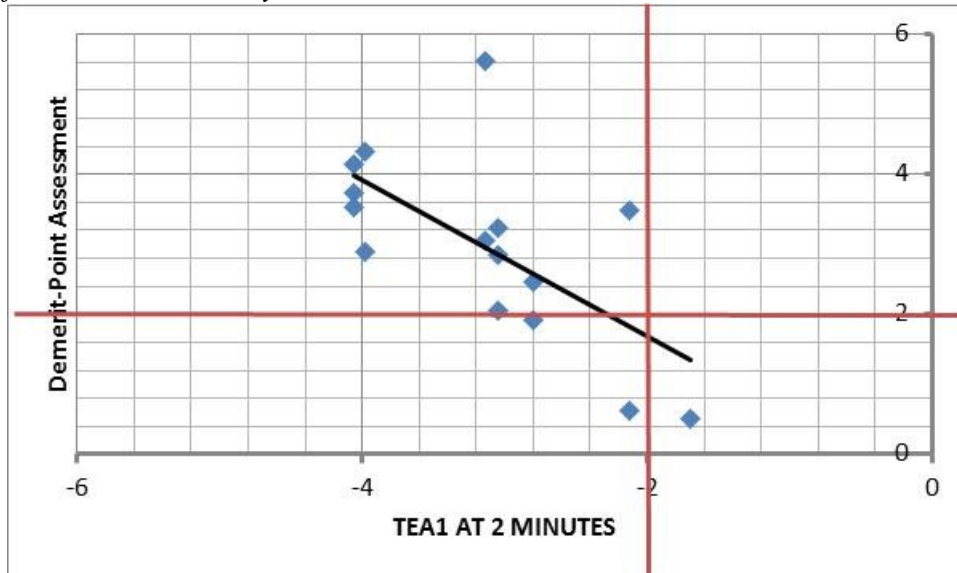
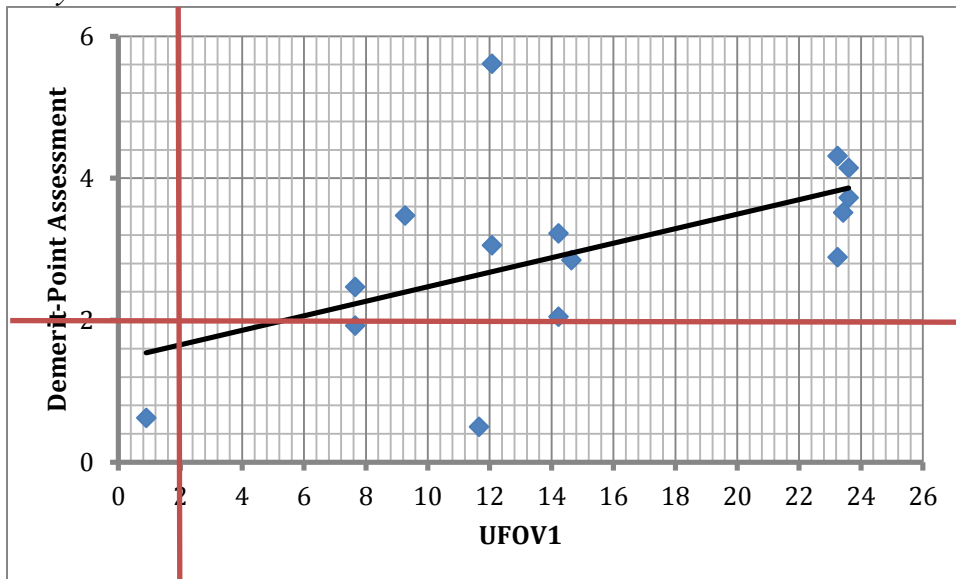


Figure 12.

Scatterplot of performance on the UFOV1 and the demerit-point assessment for drivers with early DLB.

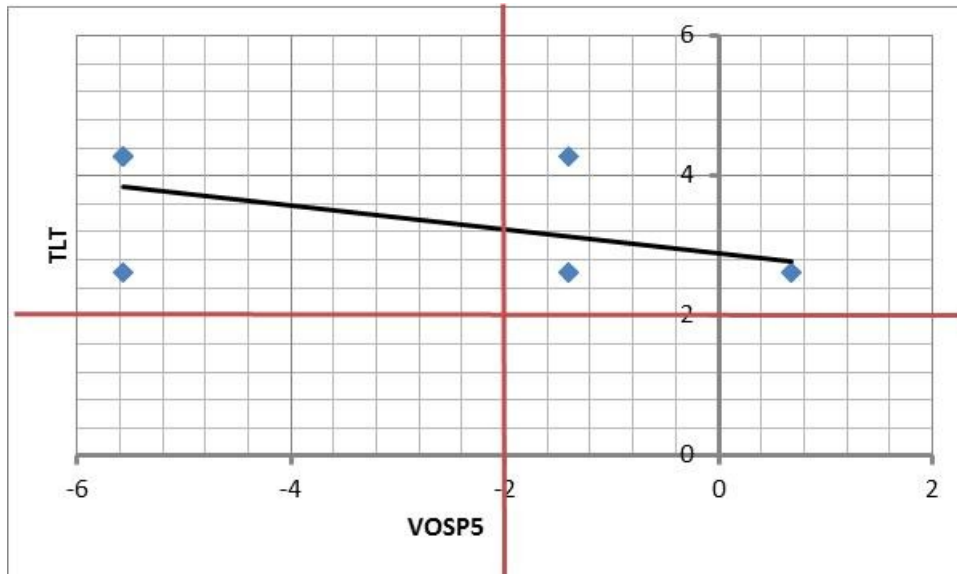


Finally, an impaired number of Traffic Light Tickets was found to correlate significantly with impaired performance on the VOSP5 in early DLB, $r(34) = -.53$, $p < .05$. The scatterplot

revealed that the VOSP5 had a sensitivity of 46.6% at predicting impaired early DLB drivers and sensitivity could not be computed since the number of Traffic Light Tickets did not identify any non-impaired drivers (refer to Figure 13).

Figure 13.

Scatterplot of performance on the VOSP5 and the Traffic Light Tickets for drivers with early DLB.



DISCUSSION

Transportation is an essential need in our society and in Canada most transportation is completed by automobile (OECD, 2001). Mobility trends have been changing significantly and are expected to continue to change, especially as the baby boomer generation moves into the older adult population. As a result, it is expected that there will be significantly more older drivers using their vehicles on a regular basis in the upcoming decades (Coughlin et al., 2012). Certainly, there are a lot of benefits for older adults driving an automobile, including better control of transportation timing, widespread accessibility of locations, better access to employment and essential needs, increased social contact, an increased sense of autonomy and independence, and ultimately a better quality of life. Along with benefits for older adults there are also costs to operating a motor vehicle, including a risk of collision, injury and fatality as well as risk of property damage, financial and social costs. These costs are significant, especially when considering that older adults are over-represented in MVC statistics (Stamatiadis, 1996; Stamatiadis et al., 1997).

When examining the population of older adults at risk of MVC's a little closer it becomes clear that the risk is not uniform across this age group. In fact, medically-at-risk drivers have a significantly higher MVC risk than the rest of the older adult population, especially those with cognitive impairments (Charlton et al., 2004; Diller et al., 1998; Dobbs, 2005; McCracken et al., 2001; McGwin et al., 1998; McGwin et al., 1999; McGwin et al., 2000, Vernon et al., 2002). The most common cause of cognitive impairment is dementia and research has demonstrated that on average, drivers with dementia continue to drive for approximately 4 years after the onset of their symptoms (Hopton et al., 2004). In many jurisdictions, physicians are often left with the sole determination of whether or not a driver with dementia is capable of operating a vehicle safely.

This determination is often difficult to ascertain in the early stages of dementia when cognitive deficits are often subtle and it is unclear whether these deficits will translate into driving impairment.

Two of the most common types of dementia include AD and DLB. AD has been extensively researched and it is well accepted that this disease includes deficits in language, attention, memory and visuospatial function, which translate into functional impairments (Grady et al., 1988; Hodges et al., 1995; McKhann et al., 1984; Welsh et al., 1992). In early stages of the disease, patients with AD present with deficits in encoding of new information, attentional deficits, working memory deficits, and mild to moderate language deficits. The clinical profile of individuals with DLB is not as clear as research in this field is still lacking. Aside from delusions, hallucination and motor impairments, patients with DLB have cognitive fluctuations and deficits in attention, visuoperceptual and spatial functioning, executive functions, and memory (Ballard et al., 2001; O'Brien et al., 2006; Oda et al., 2009; McKeith et al., 2005). Unfortunately, there is no research examining the cognitive profile of patients with early DLB, though one could speculate that there would likely be an early impairment in visuoperceptual and spatial abilities as well as attentional deficits. This lack of research inspired the first objective of this thesis, which was to qualify the nature of impairments in the early stages of DLB, with particular interest in visuoperceptual, spatial and attentional abilities since these are the hallmark of cognitive deficits in moderate to severe DLB.

The need for research on dementia and driving is well established, especially in the early stages of the illness when many diagnosed with dementia continue to drive for several years. The majority of research on dementia and driving has focused on examining driving performance in a mixed group of dementia drivers (i.e., AD, VaD, DLB). The few studies that have targeted a

particular dementia group have examined drivers with AD, though their research findings are confounding. In addition, there are currently no studies on early DLB and driving performance to date. These unclear research findings as well as lack of research inspired the main goal of this thesis, which was to examine the driving performance of drivers with early AD or DLB. Finally, this thesis aimed to explore the relationship between cognitive performance on neuropsychological tests and driving performance. This latter goal was completed with the hopes of identifying the specific neuropsychological impairments that may lead to driving impairment in early stages of dementia. Ultimately, the purpose of this investigation was to contribute to the body of research that seeks to identify a subset of individuals with early dementia who may no longer be able to drive an automobile safely, while allowing those with preserved functions to maintain their mobility.

Summary of Results

Three main research goals were examined through one comprehensive study, including (1) qualifying the nature of cognitive impairments in the early stages of DLB; (2) examining the driving performance of drivers with early AD and DLB; and (3) examining the degree of association between driving performance and findings on neuropsychological tests.

In the first part of the results section, results pertaining to the cognitive profile of individuals with early DLB were described. This was accomplished by examining scores that were significantly different from controls by at least two standard deviations, which would indicate meaningful impairment (Giovanetti et al., 2005; Lezak et al., 2004; Lonie et al., 2010; Schinka et al., 2010; Schoenber et al., 2011; Schultz et al., 2004). It was important to describe the impairments associated with early DLB rather than just examining significant differences from healthy controls, since differences do not necessarily indicate a clinically significant deficit.

Individuals with early DLB were found to be impaired on both global measures of cognitive functioning (i.e., the MMSE and the DRS). Interestingly, on the DRS, individuals with early DLB were impaired on all subtests, indicating that even in early stages there is a certain amount of global deterioration, though certain cognitive abilities may still be more impaired than others. Similarly, though language impairments are not typically considered to be a hallmark of DLB, individuals with early DLB were found to be impaired on a test of word finding abilities (i.e., BNT), which once again may be an indication of global cognitive impairment that exists in early DLB. On the one hand, one might hypothesize that this level of global impairment may be due to the group not actually being in the early stages of dementia. However, this was verified using the global deterioration scale and participants were confirmed to be in the early stages of dementia. Thus, these results reflect a global deterioration in cognitive abilities that occurs even in the early stages of DLB.

In terms of visuoperceptual and spatial abilities, which are considered to be a hallmark of the disease (Oda et al., 2009), participants were tested on a comprehensive battery measuring several facets of these abilities (i.e., VOSP). Individuals with early DLB were found to be impaired on two measures of visuospatial abilities as well as the congregate score for visuospatial functioning. Interestingly, though individuals with early DLB were found to have significantly different scores than healthy control participants on tests of visuoperceptual abilities, these scores were not considered to be impaired. Thus, the ability to correctly identify the spatial relationships amongst objects, the environment and oneself was considered to be impaired in early DLB but not the ability to recognize objects based on their form, pattern and color. To put it differently, the ‘What’ system is relatively spared, while the ‘Where’ system is impaired even in the early stages of DLB. This result is interesting because another study

concluded that the ‘What’ system was more impaired than the ‘Where’ system in a mixed group of individuals with DLB (i.e., not in the early stages) and that this may account for the visual hallucinations that are experienced in DLB (Creem & Proffitt, 2000; Oda et al., 2009). Thus, it can be concluded based on these results that the ‘Where’ system deteriorates first in early DLB.

Attentional abilities, which are also considered to be impaired in DLB patients (Ballard et al., 2001), were assessed using a comprehensive battery measuring multiple facets of attention (i.e., TEA) as well as a computerized test of visual attention (i.e., UFOV). Participants with early DLB were found to be impaired on measures of visual selective attention (i.e., TEA1 and TEA6). They were also found to be impaired on measures of simple divided attention between simultaneous auditory and visual stimuli (i.e., TEA7) but not on auditory complex selective attention with distraction (TEA3), attentional switching and cognitive flexibility (TEA4) and complex timed attentional switching (i.e., TEA5). One would expect to see impairments in auditory complex selective attention with distraction and attentional switching and cognitive flexibility, though through closer examination of the data it was seen that there was too much variability in the data to generate this conclusion. Specifically, a large amount of variability was measured for both controls and participants with early DLB on the measure of auditory complex selective attention with distraction, whereas a lot of variability was seen only in the early DLB group for the attentional switching and cognitive flexibility subtest. Similarly, one would expect to see impairments in complex timed attentional switching, though when examining the data there is a clear floor effect that emerged. Qualitatively, all participants were noted to struggle significantly on this subtest. Finally, participants with early DLB were found to have impairments in prolonged auditory sustained attention (i.e., TEA8), but not simple auditory

sustained attention (i.e., TEA2). Certainly, there was a clear ceiling effect on the simple auditory sustained attention subtest and qualitatively, participants reported finding this subtest quite easy.

In terms of visual attention, participants with early DLB were found to have impaired processing speed (i.e., UFOV1) and divided attention (i.e., UFOV2), but were not impaired on the selective attention with distraction (i.e., UFOV3). Though, at closer inspection it is clear that the selective attention with distraction subtest produced a floor effect as many participants achieved the maximum time of 500 ms, which skewed the results. Thus, when reviewing the attention domain we can conclude that individuals with early DLB are impaired on many facets of the attentional, especially on measures of selective attention and divided attention. They seem to be able to sustain their attention for short periods of time but when the demands are increased their performance deteriorates.

Cognitive fluctuations have been found to be present in DLB and this has been included in the diagnostic criteria for DLB (McKeith et al., 1996; McKeith et al., 1999; McKeith et al., 2005). Upon repeated testing (i.e., using the DRS administered at session1 and 2), individuals with early DLB were found to have significant fluctuations in memory, attention and visuospatial construction. Thus, the data confirms previous research that cognitive fluctuations are present in DLB even in early stages of the disease.

Several cognitive processes were found to be impaired in early stages of DLB, which help clarify the profile in early stages of the disease. Individuals with early DLB certainly suffer from a global deterioration of their cognitive abilities. However, when examining specific domains, participants with early DLB were more impaired in certain areas than in others. They were found to be impaired on measures of visuospatial abilities, selective and divided attention,

and were found to have cognitive fluctuations in memory, attention and visuospatial construction.

The purpose of the second section of the thesis was to examine the driving performance of drivers with early AD and DLB. This was accomplished through a simulated drive with outcomes measured by the simulator and through a rater scored measure of driving performance based on a demerit-point assessment. Once again, performances at least 2 standard deviations from the mean of healthy controls were considered truly impaired (Giovanetti et al., 2005; Lezak et al., 2004; Lonie et al., 2010; Schinka et al., 2010; Schoenber et al., 2011; Schultz et al., 2004).

Participants with early AD had significantly worse performance on all driving measures in comparison to healthy controls, with the exception of pedestrians hit and the total number of stop signs missed. In terms of pedestrians hit there was only one pedestrian present in the driving course and qualitatively one could remark that it would be quite difficult to hit that pedestrian, unless the driver swerved accidentally or intentionally in their direction, which was off-road. The total number of stop signs missed was not found to be significant. A surprising result is that healthy controls missed on average half of all stop signs (i.e., 1.52 missed stop signs out of 3). A missed stop sign is calculated when the driver fails to stop in front of the stop line and this result may be more indicative of difficulty adjusting to the simulator, even though there was extensive training provided to all groups. Drivers with AD were found to have impaired driving on several measures. In particular, their performance was found to be impaired as measured by the experimenter rated demerit-point assessment. This assessment is a written checklist that is used in licensing drivers in a province of Canada (i.e., Manitoba). What is particularly startling is that 12 out of 20 licensed AD drivers had impaired performances when compared to healthy controls. Based on these performances, one might question whether these participants would pass an on-

road examination. Their performance was also found to be impaired through driving errors calculated by the simulator, including the total number of off-road accidents, the total number of collisions, and the total number of traffic light tickets.

Similarly to AD drivers, drivers with DLB performed significantly worse on all driving measures in comparison to healthy controls with the exception of the number of pedestrians hit and the total number of off-road accidents. For reasons mentioned earlier, the lack of difference in the number of pedestrians hit is not surprising. Though, surprisingly, the total number of off-road accidents was not found to be different between the two groups even though the total out of lane percent of time for DLB drivers was 12.65% in comparison to 3.46% for healthy controls, which would increase their chance of off-road accidents. This lack of difference may be partially accounted by the large amount of variability in the off-road accidents found in the DLB group (i.e., $M = 1.27$; $SD = 3.28$). Individuals with early DLB also had impaired driving performances on many measures. Specifically, their performance was found to be impaired on the experimenter rated demerit-point assessment and once again a surprising 12 out of 15 drivers were found to be two standard deviations below the average of the healthy older drivers. In addition, their performance was found to be impaired on one driving error measured by the simulator, which was the total number of traffic light tickets.

The driving results of both the AD and DLB drivers are in line with the little amount of driving research in the dementia population that exists. Many researchers have estimated that drivers with dementia have a significantly higher risk of MVC, most stating at least twice as high as healthy counterparts (Cooper et al., 1993; Dubinsky et al., 1992; Dubinsky et al., 2000; Friedland et al., 1988; Tuokko et al., 1995). In this study, both early dementia groups had an elevated MVC risk of almost three times as high as their healthy counterparts. Drachman and

colleagues (1993) found that the risk of MVC for drivers with dementia was similar to healthy drivers in the first three years post-diagnosis. Though the dementia groups were considered to be in the early stages of dementia, a limitation of this study is that the number of years post-diagnosis is unknown. Certainly, in future research, it would be interesting to try to replicate this finding using years post-diagnosis as a dementia criterion variable.

In this study, both dementia groups were found to make significantly more errors as measured by the demerit-point assessment and driving errors calculated by the simulator than their healthy counterparts, which is in line with the literature that states that drivers with dementia are at an increased risk of unsafe operation of a vehicle (Man-Son-Hing et al., 2007). Both early dementia groups were also found to be involved in significantly more collisions than their healthy counterparts, findings that are in line with previous research by Dobbs (1997). In particular, her study findings of on-road performances demonstrated that older adults with cognitive impairment were more likely to engage in hazardous or potentially catastrophic errors that would have resulted in a MVC had a driving instructor not taken control of the vehicle. The increased number of driving errors committed by drivers with early dementia, including an increase in the total number centerline crossings and road edge excursions has been found in previous research of drivers with early dementia (Dawson et al., 2009; Uc et al., 2005). Interestingly, previous research has indicated that drivers with early dementia were more likely to drive more slowly on on-road examinations, however in this study both early dementia groups were found to have significantly more speed exceedances than their healthy counterparts (Lucas-Blaustein et al., 1988; Logsdon et al., 1992; O'Neil et al., 1992; Odenheimer et al., 1994). This result may be partially accounted by the fact that this study was done using a simulator and previous research has found that individuals with dementia were less likely to comprehend and

operate the simulator compared to healthy older adults (Cox et al. 1998). This study incorporated a lengthy training session but nonetheless it is possible that the simulator offers an additional level of complexity to which drivers with early dementia have difficulty adapting.

The driving performance of drivers with early dementia was considered to be impaired on the demerit-point assessment and the total number of traffic light tickets. In addition, drivers with early AD were also found to have impaired performance in the total number of off-road accidents and the total number of collisions. A recent study by Fritelli and colleagues (2009) found similar results with early AD drivers using a similar simulator as was used in this study. In particular, they found that drivers with early AD were more likely to have an increased number of off-road accidents, a result which has been replicated in this study.

The purpose of the last section of the thesis was to attempt to accurately predict impaired driving (i.e., performance at least two standard deviations away from the mean of healthy control drivers) with the use of neuropsychological test performance. In order to predict impaired driving in early AD, neuropsychological test performance was measured. The same procedure to determine impairment was used in this section. Both global measures of cognitive functioning were found to be impaired in individuals with early AD (i.e., MMSE and DRS). In addition, individuals with early AD were also found to be impaired on a subtest that measures frontal abilities (i.e., DRS initiation/perseveration) and on a subtest that measures memory abilities (i.e., DRS memory), which are both known to be predictive of IADLs (Greenaway, Duncan, Hanna & Smith, 2012). These results are not surprising since it is well established that the most common presenting problem in early AD are memory impairment as well as dysfunction of IADLs (Backman et al., 2001; Braak et al., 1991; Delis et al., 1991; Welsh et al., 1991). In addition, the memory subtest also includes orientation questions, which are also thought to become impaired

in early stages of AD (Zarit et al., 2007). Individuals with early AD were also found to be impaired in word finding abilities (i.e., BNT). This is another finding that is mirrored in the literature as individuals with early AD are often mildly to moderately impaired on tests of object naming (Bayles et al., 1983; Bowles et al., 1987; Hodges et al., 1991; Martin et al., 1983).

The visuospatial/perceptual domain was not found to be impacted in early AD. When examining differences between groups, a significant difference was detected on a perceptual naming subtest where subjects were asked to identify the name of a silhouette. This difference is credited for creating a significant difference between groups in the object perception composite score. This difference is likely accounted by word finding difficulties that are prevalent in the early AD population and do not reflect a meaningful impairment in visuoperceptual abilities. In addition, none of the subtests demonstrated impaired performance in the early AD group.

In terms of the attention domain, all attentional abilities were found to be significantly impacted in participants with early AD in comparison to their healthy counterparts, with the exception of simple auditory sustained attention (i.e., TEA2), which had a clear ceiling effect. Several attentional abilities were found to be impaired as per the criteria of two standard deviations from the mean of healthy older adults, which is not surprising since attentional deficits have been found to be present even in early stages of AD (Carlson et al., 1999; Hall et al., 2011). In particular, attentional switching and cognitive flexibility (i.e., TEA4 Raw) and simple divided attention between simultaneous auditory and visual stimuli (i.e., TEA7) were found to be impaired in individuals with early AD, but not auditory complex selective attention with distraction (TEA3) and complex timed attentional switching (i.e., TEA5). One would expect to see impairments in auditory complex selective attention with distraction, though by examining the data a little closer it can be seen that there was too much variability in the data. Similarly, one

would expect to see impairments in complex timed attentional switching, though a clear floor effect emerges on examination. Qualitatively, all participants were noted to struggle significantly on this subtest. In addition, both of these subtests include an auditory component that relies on the accurate perception of high pitched sounds. One cannot neglect the potential impact of presbycusis, a condition commonly associated with older age, which impacts the perception of higher frequency sounds. In addition, previous research has demonstrated that participants with early AD have impairments in shifting attention, cognitive flexibility, and dividing their attention (Bench et al., 1993; Corbetta et al., 1991; Perry et al., 1999). Participants with early AD were also found to be impaired on a measure of visual selective attention (i.e., TEA6), but not on another measure of visual selective attention (i.e., TEA1). The main difference between these two measures of simple visual selective attention is that one is a timed score per item (i.e., TEA6), whereas the other is the total number of correctly identified targets (i.e., TEA1). Certainly, one can observe that individuals with early AD are significantly slower than their healthy counterparts, though they may still be accurate in detecting targets. Though, other researchers have found that individuals with early AD are impaired on other tests of selective attention (Grady et al., 1988; Grady et al., 1989; Nebes et al., 1989; Spieler et al., 1996). Finally, individuals with early AD were found to be impaired on a prolonged measure of sustained attention (i.e., TEA8), but not on a simple measure of sustained attention (i.e., TEA2), which is a finding that has been documented in the literature (Rosvold et al., 1956).

In terms of visual attention, participants with early AD were found to have impaired processing speeds (i.e., UFOV1) and divided attention (i.e., UFOV2), but were not impaired on the selective attention with distraction (i.e., UFOV3). Though, it is clear that the selective attention with distraction subtest produced a floor effect as many participants achieved the

maximum time of 500 ms, which skewed the results. Thus, when reviewing the attention domain we can conclude that individuals with early AD are impaired on most facets of attention, whether the test is simple or complex.

Once the neuropsychological test performance was reviewed for participants with early AD, the focus shifted to attempting to accurately predict impaired driving with the use of neuropsychological test performance. This was completed by calculating correlations between impaired neuropsychological performance and impaired driving performance for both drivers with early AD and early DLB. The purpose in doing this was to be able to predict potential driving impairments in drivers with early AD and DLB by looking at associated neuropsychological performance impairments. For each association that was examined, sensitivity and specificity was computed so as to be able to inform the reader of the potential usefulness of using a particular neuropsychological impairment to predict unsafe driving. Typically for a test to be considered useful it must surpass current gold standards or at least be equivalent to them. Since, gold standards do not exist in identifying unsafe drivers within the population of older drivers with early dementia it is impossible to make this determination. However, when examining the medical and diagnostic literature on gold standards for tests or tools it is not uncommon to see specificity and sensitive levels as low as 70% accountability (Jaeschke, Guyatt & Sackett, 1994).

Within the early AD sample a correlation was found between impairments on the demerit-point assessment and a test of word finding difficulties (i.e., BNT). Sensitivity was found to be adequate at 75%, but specificity was lower at 62.5%. Impairments on the demerit-point assessment were also found to correlate with a test of prolonged sustained attention (i.e., TEA8), but sensitivity was found to be low at 50% and specificity was found to be modestly

better at 62.5%. Impairments on the demerit-point assessment were also found to correlate with impairments on a test of divided visual attention (i.e., UFOV2) with a good sensitivity of 83.3% and a lower specificity of 62.5%. Finally, impaired levels of off-road accidents were found to be correlated with impaired performance on a measure of simple visual selective attention (i.e., TEA6), but this measure had a low sensitivity of 38.5% and an adequate specificity of 71.4%.

Thus, within the context of this research, impaired divided visual attention is the cognitive ability best predicts driving behaviour within this clinical population and could be used to identify unsafe early AD drivers. This impairment would identify a substantial proportion of truly impaired drivers but unfortunately it would also incorrectly determine that 37.5% of good drivers were unsafe drivers (i.e., false positives), which is unacceptably high. Interestingly, several attentional measures are highly correlated with driving performance. Perfect sensitivity and specificity is an unrealistic goal in clinical determinations. Instead, the goal is in fact to be able to use clinical tools with clinical judgment in order to make a decision. In terms of being able to make a determination of impaired driving in early AD, a clinician would be best served by thoroughly assessing their client's attentional abilities, asking them and trusted informants (i.e., a caregiver) about recent driving behavior, and combining that with clinical judgment. A thorough assessment of an early AD driver's attentional abilities is warranted given the findings of this study as well as previous research. In particular, previous research has demonstrated that deficits in attentional abilities are prevalent in early AD (Bench et al., 1993, Corbetta et al., 1991, Grady et al., 1988, Grady et al., 1989, Perry et al., 1999, Rosvold et al., 1956, Spieler et al., 1996). In addition, deficits in attention have been found to be related to functional deficits seen in early AD, which include driving abilities (Carlson et al., 1999; Hall et al., 2011). Finally two studies have demonstrated a strong link between attentional impairments and driving impairment

in drivers with early AD as was concluded in this study (Anderson et al., 2005; Dawson et al., 2009).

In early DLB drivers, several correlations were found between impaired performance on the demerit-point assessment and impaired neuropsychological functioning. A correlation was found with an impaired performance on a global measure of cognition (i.e., MMSE), with a perfect sensitivity of 100% but a low specificity of 33%. A correlation was also found with impaired performance on a memory subtest (i.e., DRS Memory), again with a perfect sensitivity of 100% but a deplorable 0% specificity. Another correlation with impaired performance on a subtest of visuospatial functioning (i.e., VOSP5) was found revealing a poor sensitivity at 58.3% and an excellent specificity of 100%. Similarly, a correlation was found with impaired performance on a comprehensive visuospatial functioning assessment (i.e., VOSP Spatial), revealing a poor sensitivity at 58.3% and an excellent specificity of 100%. A correlation was also found with impaired performance on a measure of visual simple selective attention (i.e., TEA1), with an excellent sensitivity of 100% but a poor specificity of 33.3%. Finally, a correlation was also found with impaired performance on a test of processing speed (i.e., UFOV1), with an excellent sensitivity of 100% and a poor specificity of 33.3%. An impaired number of traffic light tickets as measured by the simulator was also correlated with an impaired visuospatial performance (i.e., VOSP5), with a poor sensitivity of 46.6%. Sensitivity could not be computed for this correlation since all drivers were found to be impaired on this measure.

Thus, impaired visuospatial performance is the cognitive ability most predictive of unsafe driving within the sample of early DLB drivers. This impairment would identify a lower number of truly impaired drivers but on the positive side it would not incorrectly identify any good drivers as bad drivers. Thus, similarly to drivers with early AD, in order to be able to make

a determination of impaired driving in early DLB, a clinician would be best served by thoroughly assessing their client's visuospatial abilities, asking them and trusted informants (i.e., a caregiver) about recent driving behavior, and combining that with clinical judgment. A thorough assessment of an early DLB driver's visuospatial abilities is warranted given the findings of this study as well as previous research. In particular, previous research has demonstrated that individuals with DLB are particularly impaired in visuoperceptual and spatial abilities and that these impairments are significantly worse than in individuals with AD (Ala et al., 2001; Cormack et al., 2004; Ferman et al., 2006; Oda et al., 2009). Though no specific studies have been conducted on the involvement of visuoperceptual and spatial abilities in the ability to drive safely in early DLB drivers, a meta-analysis did suggest that visuospatial abilities were modestly correlated with caregiver report on driving ability in individuals with dementia. In addition, they also found a strong correlation between on-road and simulator performance and performance on tests of visuospatial skills (Reger et al., 2004).

Limitations

There are several limitations in this study that should be taken into account when interpreting the results. The first has to do with sampling of populations. All three participant groups were found to be appropriately matched for age, gender and years of education. Ensuring that groups are appropriately matched is imperative because it allows the researcher to be able to make conclusions based on the data without worrying about already existing difference between the samples that are extraneous to the research question. Though, an important consideration is the fact that these samples may not be representative of the overall population of older adults, adults with early AD and early DLB. In fact, these samples are likely not a perfect match to the populations. The average age of the three samples is interesting for several reasons. First, most

individuals with AD are diagnosed after the age of 75 years and the average duration is of about 10 years, whereas most individuals with DLB are diagnosed around the age of 67 with an average duration of 9 years (American Alzheimer's Association, 2012; Ransmayr, 2000). Thus, one would expect the AD group to be older than the DLB group because of the general differences between the disease progression and certainly the AD sample in this study is a little older than the DLB sample but not significantly so. In addition, all of the groups are about equivalent in terms of sex distribution. This might be perceived as unusual because women are often overrepresented in the dementia population for several reasons, one being that have a longer longevity compared to men (Austad, 2006). However, there are more male older drivers and for that reasons the groups are likely a good representation of the population. Finally, all of the groups have a high level of education with the average for all groups being above high school, which is quite unusual for this cohort. However, this high level of education is not unusual for the region of Ottawa. Qualitatively, a high level of bilingualism was also noted in the samples, though this could not be analyzed as this information was not available for all participants. Bilingualism is also not uncommon for the Ottawa region. All of the noted particularities with the samples should be considered when drawing conclusions based on this data.

Another important limitation arises from the use of a simulator to study driving behavior. When considering the use of simulators to measure driving behavior the question that arises is if it is a valid measure of true driving abilities. There are two main considerations in making this determination. The first is the simulator's physical fidelity, which includes the correspondence of components, layout and dynamics with those experienced in a real world setting (Godley, Triggs, & Fildes, 2002; Liu, Macchiarella, & Vincenzi, 2009). In this study, a lower fidelity simulator

was used since it was based on a personal computer with an add-on steering wheel and pedal components that were originally designed for video game use, it provided no kinaesthetic feedback to the driver due to its fixed base, and it had limited visual graphic capabilities. Certainly, having used a low fidelity simulator is a limitation to this study. Another consideration is the behavioral validity of the simulator, which is the comparison of the operator's performance in the simulator versus that of the real world. This is in fact the most important consideration in determining if a simulator study is valid. Many presume that behavioral validity is highly correlated with a simulator's physical fidelity, though this has not been found to be the case (Godley et al., 2002). Though there is no sure way to determine a simulator's behavioral validity except comparing simulator behavior to on-road behavior, there are ways of increasing behavioral validity. Providing adequate instructions, trial runs and training, as well as allowing for questions ensures that a participant becomes more comfortable with the operation of the simulator, thus increasing behavioral validity. This study provided a lengthy training session that was participant focused. The experimenter ensured that the participant understood how to operate the simulator accurately before proceeding to testing. Therefore, though the use of a low fidelity simulator is certainly a limitation the comprehensive training session likely increased behavioral validity.

In this study, the use of a driving simulator was advantageous for several reasons. First, the simulator allowed for a safe collection of data about driving behavior with individuals who are considered at higher risk for MVC. Second, the use of simulators allowed for the potential of replicability, which is imperative to scientific rigor. Third, by controlling the driving conditions each participant can be exposed to the exact same conditions. Fourth, simulator studies are cost-effective and efficient ways to study targeted behaviors in a short period of time as the scenarios

can be manipulated. Thus, certainly for this study the use of the simulator was not only warranted but advantageous considering the few limitations it presented. Nevertheless, conclusions should be interpreted in light of the use of a simulator and should be interpreted with some caution when extrapolating to on-road behavior.

Another limitation of this study is based on the determination of impairment. Certainly, there were numerous ways that this data could have been examined. For example, the data could have been examined using a pass/fail approach to the driving task. This was considered as a possible analysis methodology, though the determination of a pass/fail on the driving task would have to have been either based on previous literature or validated through a pilot study. Unfortunately, there were no studies that could determine a pass/fail to the simulated drive and due to time constraints a validation study of such a pass/fail approach was not conducted. Without either of these to help support a pass/fail approach, this determination would have been fairly subjective. Thus, there was a decision to use two standard deviations to determine impairment, which was well founded in the literature (Giovanetti et al., 2005; Lezak et al., 2004; Lonie et al., 2010; Schinka et al., 2010; Schoenber et al., 2011; Schultz et al., 2004). Certainly, had 1 or 1.5 standard deviations been used to determine impairment this would have likely altered the calculations for sensitivity and specificity. Though, again, meaningful impairment is characterized as two standard deviations from the mean and using a more conservative approach is well founded.

A final limitation of this study is a pragmatic one in that time and costs limited the extent of the study. In terms of time, testing was lengthy at approximately 5 hours per participant, spread over two testing sessions. Though, the attention and visuoperceptual and spatial domain were tested extensively because they were both thought to be the most implicated in driving, it

would have been interesting to also examine other domains such as memory, executive functions, language, and so on, in greater detail. In addition, larger sample sizes would have been helpful and some results may have in fact been slightly different since some appeared to be approaching significance. However, the samples that were included in this study took approximately three years to recruit and test which is significant considering the timeframe of doctoral studies. Finally, a limitation of this study is that diagnosis was only probable and not confirmed by autopsy. However, a triangulated approach was used in hopes of greatly reducing diagnostic error. Nonetheless, autopsy confirmation would have been the gold standard in confirming diagnostic groups. Once again, due to financial and time efficiency reasons this was not possible within the context of this thesis.

General Conclusion

This thesis had three main objectives. The first was to clarify the neuropsychological profile of individuals with early DLB, the next was to examine the driving performances of two groups of individuals with early dementia (i.e., early AD and early DLB) and the last was to examine the degree of association between neuropsychological impairments and driving impairments in hopes of predicting poor driving outcomes. These three objectives were successfully met to varying degrees considering the limitations presented.

The neuropsychological profile of individuals with early DLB was successfully clarified. In particular, individuals with early DLB were found to be most impaired in their visuospatial abilities, selective and divided attention abilities, and were found to have significant cognitive fluctuations. Certainly, this research adds to the existing body of literature on DLB and their associated profile but more importantly it helps clarify the earliest impairments present in the disease. Researching early stages of dementia is particularly important since as dementia

progresses a global deterioration occurs due to the increasing pathology in the brain, and it becomes difficult to differentiate between different types of dementia. Thus, having research on the accurate profile in early DLB could lead to accurate differentiation of dementia types in early stages, when patients are most likely to present to a medical professional for diagnosis of their cognitive difficulties. Accurate early diagnosis is important for several reasons. First, DLB patients can have a severe reaction to neuroleptics which are routinely prescribed in patients who present with hallucinations and early diagnosis could prevent this potentially fatal mistake. Second, each dementia type progresses in different ways due to their pathological mechanism and providing information to the patient and caregivers early could be beneficial. Third, each type of dementia involves different hereditary components, information which could be useful to the family of the patient. These are just some of the potential benefits of having an accurate diagnosis early in the course of DLB.

A very limited body of literature exists on the driving performances of individuals with dementia. This study was successful at examining the general driving abilities of individuals with early dementia, as well as their particular risks and errors encountered. Driving performances confirmed that drivers with early dementia are at higher risks for MVC, which has been found repeatedly in the literature (Cooper et al., 1993; Dubinsky et al., 1992; Dubinsky et al., 2000; Friedland et al., 1988; Tuokko et al., 1995). In addition, drivers with early dementia committed a lot of driving errors during the driving simulation, which may lead one to question whether these individuals would pass an on-road test and if these individuals are at significant risk of committing catastrophic driving errors on the road. Finally, this study replicated previous results on the increased number of off-road accidents committed by drivers with early AD (Fritelli et al.,

2009). Certainly, the study of dementia and driving is in its infancy and this study contributes significantly to the limited research that currently exists.

There is a need for more simulator studies examining the specific driving errors committed by drivers with early dementia of various types. Given that participants committed a significant number of driving errors, it would be important to elucidate where these errors were committed and under what conditions. It would be interesting to create scenarios of various complexities and examine how individuals with dementia fare under different conditions (e.g., different weather conditions, unexpected events, and dividing their attention between driving and another task such as listening to the radio). On-road examinations would also be useful in advancing the field further. This could be completed using cars with passenger controls, such as those used by the majority of driving schools. On-road performance could then be compared to performance on a simulated drive. On-road examination could also be studied using Geographic Positioning System (GPS) and video recordings in order to examine if drivers with early dementia compensate for their deficits and if this compensation leads to a change in the MVC risk. Finally, provincial driving data could also be analyzed to look for trends in the types of MVC encountered by drivers with dementia. But as previously stated, each one of these presents its own set of unique limitations.

The last goal of this thesis was to attempt to identify certain cognitive impairments that could be used to predict impaired driving in individuals with early AD or early DLB. Though neuropsychological test performance has been associated with driving performance in a few studies, the idea of predicting impaired driving has never been studied. This goal was partially accomplished. This study was able to demonstrate that in drivers with early AD, attentional impairments would be the best predictor of driving impairment, whereas in drivers with early

DLB, visuospatial impairments would be the best predictor of driving impairment. It was the hope of the investigator to be able to specifically pinpoint a facet of a cognitive domain (e.g., selective attention) in being able to predict impaired driving, but none proved to be an excellent predictor of impaired driving on their own. Nonetheless, this study provides useful information as to the cognitive domains that lead to increased driving risk in individuals with early AD and with early DLB. The fact that visuospatial impairments and attention were found to be predictive of impaired driving is not surprising when considering the cognitive profiles of both AD and DLB as well as Michon's driving model (1985), that clearly implicates the need for intact processing of visuospatial and attentional information.

There is a continued need for significant research in this field as the potential public safety issue will continue to increase as the population ages and as the number of drivers with dementia increases as a result. The public safety issue surrounding drivers with dementia is not likely to be solved by any individual intervention, but it may be alleviated by a combination of evidenced-based efforts. Such efforts may include, valid assessment, license restrictions, cognitive training, promotion of compensatory behaviors, as well as vehicle and environmental adaptations that take attentional and visuospatial limitations into consideration. Ultimately, driving cessation is an inevitable outcome as individuals with dementia progress into moderate and severe stages of their illness but an effort to prolong driving so long as one can do so safely is necessary to maintain social and mobility needs.

REFERENCES

- Aarsland, D., Andersen, K., Larsen, J. P., Lolk, A., & Kragh-Sorensen, P. (2003). Prevalence and characteristics of dementia in Parkinson disease: an 8-year prospective study. *Archives in Neurology*, 60, 387-392.
- Agüero-Torres, H., Fratiglioni, L., Guo, Z., Viitanen, M., von Strauss, E., Winblad, B. (1998). Dementia is the major cause of functional dependence in the elderly. Three-year follow-up data from a population-based study. *American Journal of Public Health*, 88, 1452–1456.
- Ahto, M., Isoaho, R., Puolijoki, H., Laippala, P., Sulkava, R., & Kivela, S. L. (1999). Cognitive impairment among elderly coronary heart disease. *Gerontology*, 45, 87-95.
- Ala, T. A., Hughes, L. F., Kyrouac, G. A., Ghobrial, M. W., Elie, R. J. (2001). Pentagon copying is more impaired in dementia with Lewy bodies than in Alzheimer's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, 70, 483–488.
- Albert, M. S., DeKosky, S. T., Dickson, D., Dubois, B., Feldman, H. H., Fox, N. C., et al. (2011). The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia*, 7, 270-279.
- Albin, R. L., Minoshima, S., D'Amato, C. J., Frey, K. A., Kuhl, D. A., Sima, A. A. F. (1996). Fluorodeoxyglucose positron emission tomography in diffuse Lewy body disease. *Neurology*, 47, 462–466.

American Alzheimer's Association (2012). 2012 Alzheimer's Disease Facts and Figures.

Retrieved on October 3rd, 2012 from http://www.alz.org/downloads/facts_figures_2012.pdf.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC.

Anderson, S. W., Rizzo, M., Shi, Q., Uc, E. Y., & Dawson, J. D. (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*, pp 286-292. Rockport, ME, June 27 - 30, 2005. Iowa City, Iowa: The University of Iowa.

Anstey, K. J., Wood, J., Lord, S., & Walker, J. (2005). Cognitive, sensory and physical factors enabling driving safety in older adults. *Clinical Psychology Review*, 25, 45-65.

Aronoff, J. M., Gonnerman, L. M., Almor, A., Arunachalam, S., Kempler, D., & Andersen, E. S. (2006). Information content versus relational knowledge: Semantic deficits in patients with Alzheimer's disease. *Neuropsychologia* 44, 21-35.

Augenstein, J. (2001). Differences in clinical response between the young and the elderly. *Paper presented at the Aging and Driving Symposium, Association for the Advancement of Automotive Medicine*.

Austad, S. N. (2006). Why women live longer than men: Sex differences in longevity. *Gender Medicine*, 3, 79-92.

Backman, L., Small, B. J., & Fratiglioni, L. (2001). Stability of the preclinical episodic memory deficit in Alzheimer's disease. *Brain*, 124, 96-102.

- Backman, L., Jones, S., Berger, A.K., Laukka, E.J. (2005). Cognitive Impairment in preclinical Alzheimer's disease: A meta-analysis. *Neuropsychology*, 19, 520-531.
- Baldock MRJ, Mathias JL, McLean AJ, Kloeden CN, Berndt A (2006). Self-regulation of driving and older drivers' functional abilities, *Clinical Gerontologist*, 30(1), 53-70.
- Ball, K., Beard, B., Roenker, D. L., Miller, R., & Griggs, D. (1988). Age and visual search: expanding the useful field of view. *Journal of the Optical Society of America*, 5, 2210-2219.
- Ball, K. and Owsley, C. (1993). The Useful Field of View Test: A new technique for evaluating age-related declines in visual function. *Journal of the American Optometric Association*, 64, 71-79.
- 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(11), 3110-3123.
- Ballard, C., O'Brien, J., Gray, A., Cormack, F., Ayre, G., Rowan, E., et al. (2001). Attention and fluctuating attention in patients with dementia with Lewy bodies and Alzheimer disease. *Archives of Neurology*, 58, 977-982.
- Bate, A. J., Mathias, J. L., & Crawford, J. R. (2001). Courting the clinician : Performance on the test of everyday attention and standard tests of attention following severe traumatic brain injury. *The Clinical Neuropsychologist*, 15, 405-422.
- Barber, R., Panikkar, A., & McKeith, I. G. (2001). Dementia with Lewy Bodies: diagnosis and management. *International Journal of Geriatric Psychiatry*, 16, S12-S18.

- Bayles, K. A., & Tomoeda, C. K. (1983). Confrontation naming impairment in dementia. *Brain and Language*, 19, 98–114.
- Begg, D., & Langley, J. (2001). Changes in risky driving behavior from age 21 to 26 years. *Journal of Safety Research*, 32 (4), 491-499.
- Bekiaris, E., Amditis, A., & Panou, M. (2003). Drivability: A new concept for modelling driving performance. *Cognition Technology and Work*, 5(2), 152-161.
- Bench, C. J., Frith, C. D., Grasby, P. M., Friston, K. J., Paulesu, E., Frackowiak, R. S. J., Dolan, R. J. (1993) Investigations of the functional anatomy of attention using the Stroop test. *Neuropsychologia*, 31, 907–922.
- Bennett, D. A., Schneider, J. A., Bienias, J. L., Evans, D. A., & Wilson, R. S. (2005). Mild cognitive impairment is related to Alzheimer disease pathology and cerebral infarctions. *Neurology*, 64, 834-841.
- Bess, I. (1999). *Seniors behind the wheel*. Canadian social trends. Retrieved on October 3rd, 2011, from <http://www.statcan.gc.ca/daily-quotidien/990909/dq990909a-eng.htm>.
- Betz, M. E., Lowenstein, S. R. & Schwartz, R. (2012). Older Adult Opinions of “Advance Driving Directives”. *Journal of Primary Care and Community Health*, 4, 14-27.
- Bird, C. M., Papadopoulou, K., Ricciardelli, P., Rossor, M. N., & Cipolotti, L. (2004). Monitoring cognitive changes: Psychometric properties of six cognitive tests. *British Journal of Clinical Psychology*, 43, 197-210.
- Blacker, D., Albert, M. S., Bessett, S. S., Go, R. C., Harrell, L. E., Folstein, M. F. (1994). Reliability and validity of NINCDS-ADRDA criteria for Alzheimer's disease. The

- National Institute of Mental Health Genetics Initiative. *Archives of Neurology*, 51, 1198-1204.
- Blake, H., McKinney, M., Treece, K., Lee, E., & Lincoln, N. B. (2002). An evaluation of screening measures for cognitive impairment after stroke. *Age and Ageing*, 31, 451-456.
- Bonello, P. J., Rapport, L. J., & Millis, S. R. (1997). Psychometric properties of the visual object and space perception battery in normal older adults. *The Clinical Neuropsychologist*, 11, 436-442.
- Bowles, N. L., Obler, L. K., Albert, M. L. (1987). Naming errors in healthy aging and dementia of the Alzheimer type. *Cortex*, 23, 519-524.
- Bozzali, M., Falini, A., Cercignani, M., Baglio, F., Farina, E., Alberoni, M., et al. (2005). Brain tissue damage in dementia with Lewy bodies: an in vivo diffusion tensor MRI study. *Brain*, 128, 1595-1604.
- Braak, H., & Braak, E. (1991). Neuropathological stageing of Alzheimer-related changes. *Acta Neuropathologica*, 82, 239-259.
- Breen, D. A., Breen, D. P., Moore, J. W., Breen, P. A., & O'Neil, D. (2007). Driving and Dementia. *British Medical Journal*, 334, 1365-1369.
- Brink, T. L., Yesavage, J. A., Lum, O., Heersema, P., Adey, M., & Rose, T. L. (1982). Screening tests for geriatric depression. *Clinical Gerontologist*, 1, 37-44.

- Brion, J. P., & Couck, A. M. (1995). Cortical and brainstem-type Lewy bodies are immunoreactive for the cyclin-dependent kinase 5. *American Journal of Pathology*, 147, 1465-1476.
- Brown, L. B., Ott, B. R., Papandonatos, G. D., Sui, Y., Ready, R. E., Morris, J. C. (2005). Prediction of on-road driving performance in patients with early Alzheimer's disease. *Journal of American Geriatric Society*, 53, 94-98.
- Burton, E. J., Karas, G., Paling, S. M., Barber, R., Williams, E. D., Ballard, C. G., et al. (2002). Pattern of cerebral atrophy in dementia with Lewy bodies using voxel-based morphometry. *Neuroimaging*, 17, 618-630.
- Burton, E. J. McKeith, I. G. Burn, D. J. William, E. D., & O'Brien, J. T. (2004). Cerebral atrophy in Parkinson's disease with or without dementia: a comparison with Alzheimer's disease, dementia with Lewy bodies, and controls. *Brain*, 4, 791-800.
- Butters, N., Granholm, E., Salmon, D. P., Grant, I., Wolfe, J. (1987). Episodic and semantic memory: A comparison of amnesic and demented patients. *Journal of Clinical and Experimental Neuropsychology*, 9, 479-497.
- Caird, J. K., Edwards, C. J., Creaser, J. I. & Horrey, W. J. (2005). Older Driver Failures of Attention at Intersections: Using Change Blindness Methods to Assess Turn Decision Accuracy. *Human Factors: The Journal of the Human Factors and Ergonomics Society*, 47(2), 235-249.
- Calero, M. D., Arnedo, M. L., Navarro, E., Ruiz-Pedrosa, M., & Carnero, C. (2002). Usefulness of a 15-item version of the Boston naming test in neuropsychological

- assessment of low-educational elders with dementia. *Journal of Gerontology: Psychological*, 57, P187-P191.
- Carlson, M. C., Fried, L. P., Xue, Q. L., Bandeen-Roche, K., Zeger, S. L., Brandt, J. (1999). *The Journals of Gerontology, Series B, Psychological Sciences and Social Sciences*, 54, S262-270.
- Carmody, J., Granger, J., Lewis, K., Traynor, V., & Iverson, D. (2013). What factors delay driving retirement by individuals with dementia? (The doctor's perspective). *Journal of the Australasian College of Road Safety*, 24, 10-16.
- Carp, F. (1998). Significance of mobility for the well-being of the elderly. *Transportation in an aging society: improving mobility and safety for older persons*, 2, 1-20.
- Carr, D., Jackson, T., Alquire, P. (1990). Characteristics of an elderly driving population referred to a geriatric assessment centre. *Journal of American Geriatric Society*, 38, 1145-1150.
- 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. *Journal of Gerontology: Medical Sciences*, 53A, M135-M139.
- Caselli, R. J. & Tariot, P. N. (2010). Alzheimer's disease and its variants. Oxford American Neurology Library. New York: Oxford University Press.
- Charlton, J., Koppel, S., O'Hare, M., Andrea, D., Smith, G., Khodr, B., et al. (2004). Influence of chronic illness on crash involvement of motor vehicle drivers. Monash University Accident Research Centre.

- 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, 45–55.
- Clarke, D. D., Ward, P., & Truman, W. (2005). Voluntary risk taking and skill deficits in young driver accidents in the UK. *Accident Analysis & Prevention*, 37(3), 523-529.
- Cooper, P. J., Tallman, K., Tuokko, H., Beattie, B. L. (1993). Vehicle crash involvement and cognitive deficit in older drivers. *Journal of Safety Research*, 24, 9-17.
- Corbetta, M., Miezin, F. M., Dobmeyer, S., Shulman, G., & Petersen, S. E. (1991) Selective and divided attention during visual discrimination of shape, color and speed. *Journal of Neuroscience*, 11, 2383–2402.
- Cordery, R. J., Tyrrell, P. J., Lantos, P. L., & Rossor, M. N. (2001). Dementia with Lewy bodies studied with positron emission tomography. *Archives in Neurology*, 58 (3), 505-508.
- Cormack, F., Aarsland, D., Ballard, C., Tovee, M. J. (2004). Pentagon drawing and neuropsychological performance in dementia with Lewy Bodies, Alzheimer's disease, Parkinson's disease and Parkinson's disease with dementia. *International Journal of Geriatric Psychiatry*, 19, 371–377.
- Coughlin, J. E., & D'Ambrosio (2012). Aging America and transportation: personal choices and public policy. New York: Springer Publishing Company.
- Cox, D. J., Quillian, W. C., Thorndike, F. P., Kovatchev, B. P., & Hanna, G. (1998). Evaluating driving performance of outpatients with Alzheimer disease. *Journal of the American Board of Family Practice*, 11, 264-271.

- Creem, S. H., & Proffitt, D. R. (2000). Defining the cortical visual systems: “What”, “Where”, and “How”. *Acta Psychologica, 107*, 43-68.
- Cushman, L. A. (1992). The impact of cognitive decline and dementia in older adults. *AAA Foundation for Traffic Safety Technical Report*. Washington, DC: AAA Foundation for Traffic Safety.
- Cummings, J., Cherry, D., Kohatsu, N. D., Kemp, B., Hewett, L., & Mittman, B. (2002). Guidelines for managing Alzheimer’s disease: Part I. Assessment. *American Family Physician, 65*, 2263-2272.
- Curl, A. L., Stowe, J. D., Cooney, T. M., & Proulx, C. M. (2013). Giving up the keys: How driving cessation affects engagement in later life. *The Gerontologist*, advanced online access.
- Daigneault, G., Joly, P., & Frigon, J. (2002). Previous convictions or accidents and the risk of subsequent accidents of older drivers. *Accident Analysis and Prevention, 34*, 257-261.
- 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.
- Delis, D. C., Massman, P. J., Butters, N., Salmon, D. P., Cermak, L. S., Kramer, J. H. (1991). Profiles of demented and amnesic patients on the California verbal learning test: Implications for the assessment of memory disorders. *Psychological Assessment 3*, 19–26.
- De Raedt, R., & Ponjaert-Kristoffersen, I. (2000). Can strategic and tactical compensation reduce crash risk in older drivers? *Age and Ageing, 29*, 517-521.

- Dickson, D. W., Wu, E., Crystal, H. A., Mattiace, L.A., Yen, S-H., & Davies, P. (1992).
Alzheimer and age-related pathology in diffuse Lewy body disease. In: Boller, F.,
Forcetter, F., Khachaturian, Z., et al., eds. *Heterogeneity of Alzheimer's Disease*.
Springer-Verlag : Berlin, p. 169-186.
- Diller, D., L. Cook, & J. Leonard. (1998). Evaluating drivers licensed with medical conditions in
Utah 1992-96. NHTSA Technical Report: Washington.
- Dilworth-Anderson, P., Hendrie, H. C., Manly, J. J., Khachaturian, A. S., Fazio, S. (2008).
Diagnosis and assessment of Alzheimer's disease in diverse populations. *Alzheimer's &
Dementia*, 4(4):305–309.
- Ditter, S. M., & Mirra, S. S. (1987). Neuropathologic and clinical features of Parkinson's
disease in Alzheimer's patients. *Neurology*, 37, 754-760.
- Dobbs, A. R. (1997). Evaluating the Driving Competence of Dementia Patients. *Alzheimer
Disease and Associated Disorders*, 11, 8-12.
- Dobbs, B. (2005). Medical Conditions and Driving: A Review of the Literature (1960-2000).
National Highway Traffic Safety Administration: Washington, DC.
- Dobbs, B. M. (2008). Aging baby boomers: A blessing or challenge for driver licensing
authorities. *Traffic Injury Prevention*, 9, 379–386.
- Doherty, S. T., Andrey, J. C., & MacGregor, C. (1998). The situational risks of young drivers:
The influence of passengers, time of day and day of week on accident rates. *Accident
Analysis & Prevention*, 30(1), 45-52.

- Drachman, D. A., & Swearer, J. (1993). Driving and Alzheimer's disease: the risk of crashes. *Neurology*, 43, 2448-2456.
- Dubinsky, R. M., Williamson, A., Gray, C., Glatt, S. L. (1992). Driving in Alzheimer's disease. *Journal of American Geriatric Society*, 40, 1112-1116.
- Dubinsky, R. M., Stein, A. C., Lyons, K. (2000). The American Academy of Neurology. Report of the Quality Standards Subcommittee. Practice parameter, risk of driving and Alzheimer's disease (an evidence-based review). *Neurology*, 54, 2205-2211.
- Duchek, J. et al. (2003). Longitudinal driving performance in early-stage dementia of the Alzheimer type. *Journal of the American Geriatrics Society*, 51, 1342-1347.
- Duckek, J. M., Hunt, L., Ball, K., Buckles, V., and Morris, J. C. (1998). Attention and driving performance in Alzheimer's disease. *Journal of Gerontology: Psychological Sciences*, 53B, P130-P141.
- Duchek, J. M., Hunt, L., Ball, K., Buckles, V., & Morris, J. C. (1997). The role of selective attention in driving and dementia of the Alzheimer type. *Alzheimer Disease and Associated Disorders*, 11, 48-56.
- Edwards, J. D., Vance, D. E., Wadley, V. G., Cissell, G. M., Roenker, D. L., & Ball, K. K. (2005). Reliability and validity of useful field of view test scores as administered by personal computer. *Journal of Clinical and Experimental Neuropsychology*, 27, 529-543.
- Eggerton, D. E., & Sima, A. A. F. (1986). Dementia with cerebral Lewy bodies. *Archives in Neurology*, 43, 534-527.

- Environment Canada. (1996). The state of Canada's Environment- 1996. Accessed from <http://www.ec.gc.ca/soer-ree/English/SOER/1996report> on June 4th, 2011.
- Evans, L. (1991). *Traffic Safety and the Driver*. New York: Van Nostrand Reinhold.
- Evans, D. A., Hebert, L. E., Beckett, L. A., Scherr, P. A., Albert, M. S., Chown, M. J., et al. (1997). Education and other measures of socioeconomic status and risk of incident Alzheimer disease in a defined population of older persons. *Archives of Neurology*, 54(11):1399–405.
- Evans, D. A., Bennett, D. A., Wilson, R. S., Bienias, J. L., Morris, M. C., Scherr, P. A., et al. (2003). Incidence of Alzheimer disease in a biracial urban community: Relation to apolipoprotein E allele status. *Archives of Neurology*, 60(2):185–189.
- Festa, E. K., Ott, B. R., Manning, K. J., Davis, J. D., & Heindel, W. C. (2013). Effect of cognitive status on self-regulatory driving behavior in older adults: An assessment of naturalistic driving using in-car video recordings. *Journal of Geriatric Psychiatry and Neurology*, 28, 10-18.
- Ferman, T. J., Smith, G. E., Boeve, B. F., Graff-Radford, N. R., Lucas, J. A., & Knopman, D. S. et al. (2006). Neuropsychological differentiation of dementia with Lewy bodies from normal aging and Alzheimer's disease. *Clinical Neuropsychology*, 20, 623–636.
- 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.

- Fitten, L. J., Perryman, K. M., Wilkinson, C. J., Little, R. J., Burns, M. M., Pachana, N., et al. (1995). Alzheimer and vascular dementias and driving. A prospective road and laboratory study. *JAMA*, 273, 1360-1365.
- Fitzpatrick, A. L., Kuller, L. H., Ives, D. G., Lopez, O. L., Jagust, W., Breitner, J. C., et al. (2004). Incidence and prevalence of dementia in the Cardiovascular Health Study. *Journal of the American Geriatrics Society*, 52(2):195–204.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini-Mental State: A practical method for grading the state of patients for the clinician. *Journal of Psychiatric Research*, 12, 189-198.
- Foster, J. R., Sclan, S., Welkowitz, J., Boksay, I., & Seeland, I. (1988). Psychiatric assessment in medical long-term care facilities: Reliability of commonly used rating scales. *International Journal of Geriatric Psychiatry*, 3, 229-233.
- Friedland, R. P., Koss, E., Kumar, A., Gaine, S., Metzler, D., Haxby, J. V., et al. (1988). Motor vehicle crashes in dementia of the Alzheimer type. *Annals of Neurology*, 24, 782-786.
- Frittelli, C., Borghetti, D., Iudice, G., Bonanni, E., Maestri, M., Tognoni, G., et al. (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, 232–238.
- Fuller, R. (2005). Towards a general theory of driver behaviour. *Accident Analysis & Prevention*, 37(3), 461-472.

- Galasko, D., Klauber, M. R., & Hofsetter, C. R. (1990). The Mini-Mental State Examination in the early diagnosis of Alzheimer's disease. *Archives in Neurology*, 47, 49-52.
- Galski, T., Bruno, R., & Ehle, H. (1992). Driving after cerebral damage: A model with implications for evaluation. *American Journal of Occupational Therapy*, 46, 324-332.
- Galton, C. J., Patterson, K., Xuereb, J. H., Hodges, J.R. (2000). Atypical and typical presentations of Alzheimer's disease: a clinical, neuropsychological, neuroimaging and pathological study of 13 cases. *Brain*, 123, 484-498.
- Galvin, J. E., Lee, V. M-Y., & Trojanowski, J. Q. (2001). Synucleinopathies. *Archives in Neurology*, 58, 186-190.
- Ganguli, M., Dodge, H. H., Shen, C., Pandav, R. S., DeKosky, S. T. (2005). Alzheimer disease and mortality: A 15-year epidemiological study. *Archives of Neurology*, 62(5):779-84.
- Gibb, W. R. G., Esiri, M. M., & Lees, A. J. (1987). Clinical and pathological features of diffuse cortical Lewy body disease (Lewy body dementia). *Brain*, 110, 1131-1153.
- Gilley, D. W., Wilson, R. S., Bennett, D. A., Bernard, B. A., & Fox, J. H. (1991). Cessation of driving and unsafe motor vehicle operation by dementia patients. *Archives of Internal Medicine*, 5, 941-945.
- Giovannetti, T., Schmidt, K. S., Gallo, J. L., Sestito, N., & Libon, D. J. (2005). Everyday action in dementia: evidence for differential deficits in Alzheimer's disease versus

- subcortical vascular dementia. *Journal of the International Neuropsychological Society*, 12, 45-53.
- Godley, S. T., Triggs, T. J., & Fildes, B. N. (2002). Driving simulation validation for speed research. *Accident Analysis and Prevention*, 34, 589-600.
- Gogia, P. P., & Rastogi, N. (2009). *Clinical Alzheimer Rehabilitation*. New York: Springer Publishing Company LLC.
- Grady, C. L., Grimes, A. M., Patronas, N., Sunderland, T., Foster, N. L., & Rapoport, S. I. (1989). Divided attention, as measured by dichotic speech performance, in dementia of the Alzheimer type. *Archives of Neurology*, 46, 317-320.
- Grady, C. L., Haxby, J. V., Horwitz, B., Sundaram, M., Berg, G., Schapiro, M. B., et al. (1988). Longitudinal study of the early neuropsychological and cerebral metabolic changes in dementia of the Alzheimer type. *Journal of Clinical and Experimental Neuropsychology*, 10, 576-596.
- Graeber, M. B., & Müller, U. (2003). Dementia with Lewy bodies: disease concept and genetics. *Neurogenetics*, 4, 157-162.
- Graham, N. L., Emery, T., & Hodges, J. R. (2004). Distinctive cognitive profiles in Alzheimer's disease and subcortical vascular dementia. *Journal of Neurology, Neurosurgery, and Psychiatry*, 75, 61-71.
- Grandenigo, B. (2002). Assessing and rehabilitation of car driving ability: A holistic approach. *Europa Medicophysica*, 38, 33-37.

- Greenaway, M. C., Duncan, N. L., Hanna, S., & Smith, G. E. (2012). Predicting functional ability in mild cognitive impairment with the Dementia Rating Scale-2. *Journal of International Psychogeriatrics*, 24, 987-993.
- Gurland, B. J., Wilder, D. E., Lantigua, R., Stern, Y., Chen, J., Killeffer, E. H., et al. (1999). Rates of dementia in three ethnoracial groups. *International Journal of Geriatric Psychiatry*, 14(6):481-93.
- Hakamies-Blomqvist, L. E. (1993). Fatal accidents of older drivers. *Accident Analysis and Prevention Journal*, 25, 19-27.
- Hall, J. R., Vo, H. T., Johnson, L. A., Barber, R. C., O'Bryant, S. E. (2011). The Link between Cognitive Measures and ADLs and IADL Functioning in Mild Alzheimer's: What Has Gender Got to Do with It? *International Journal of Alzheimer's Disease*, 2011, 1-6.
- Hamilton, R. L. (2000). Lewy bodies in Alzheimer's disease: a neuropathological review of 145 cases using alpha-sinuclein immunohistochemistry. *Brain Pathology*, 10, 378-384.
- Hancock, P. A., & Verwey, W. B. (1997). Fatigue, workload and adaptive driver systems. *Accident Analysis & Prevention*, 29(4), 495-506.
- Hansen, I., Slamon, D., & Galasko, D. (1990). The Lewy body variant of Alzheimer's disease: a clinical and pathological entity. *Neurology*, 40, 1-8.
- Harding, A. J., Broe, G. A., & Halliday, G. M. (2002). Visual hallucinations in Lewy body disease relate to Lewy bodies in the temporal lobe. *Brain*, 125, 391-403.

- Hatfield, J., & Fernandes, R. (2009). The role of risk-propensity in the risky driving of younger drivers. *Accident Analysis & Prevention*, 41(1), 25-35.
- He, W., Sengupta, M., & Velkoff, V. A. (2005) *65+ in the United States*. U. S. Census Bureau, Current Population Reports (P23-209). Washington, DC: U.S. Government Printing Office.
- Health Canada. (2002). Canada's Aging Population. Ottawa: Health Canada.
- Hebert, L. E., Scherr, P. A., Bienias, J. L., Bennett, D. A., & Evans, D. A. (2003). Alzheimer's disease in the U.S. population: Prevalence estimates using the 2000 consensus. *Archives of Neurology*, 60, 1119-1122.
- Hills, B. L. (1980). Vision, visibility, and perception in driving. *Perception*, 9, 183-216.
- Hodges, J. R., Patterson, K. (1995). Is semantic memory consistently impaired early in the course of Alzheimer's disease? Neuroanatomical and diagnostic implications. *Neuropsychologia*, 33, 441-459.
- Hopkins, R., Kilik, L., Day, D. J., Rows, C., & Tseng, H. (2004). Driving and Dementia in Ontario: A Quantitative Assessment of the Problem. *Canadian Journal of Psychiatry*, 49, 434-438.
- 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.

- Human Resources and Skills Development Canada (2011). Canadians in Context-Aging Population. Retrieved on June 3rd, 2013, from <http://www4.hrsdc.gc.ca/.3ndic.1t.4r@-eng.jsp?iid=33>.
- Hunt, L., Morris, J., Edwards, D. & Wilson, B. (1993). Driving performance in persons with mild senile dementia of the Alzheimer type. *Journal of the American Geriatric Society*, 41, 747-753.
- Insurance Institute for Highway Safety. (2003). Who poses the greater risk, older or younger drivers? *Status Report*, 38 (3), March 15.
- Iverson, D. J., Gronseth, G. S., Reger, M. A., Classen, S., Dubinsky, R. M., Rizzo, M. (2010). Practice Parameter update : Evaluation and management of driving risk in dementia. *American Academy of Neurology*, 74, 1316-1324.
- Jaeschke, R., Guyatt, G. H., & Sackett, D. L. (1994). User's guides to the medical literature: III. How to use an article about a diagnostic test: B. What are the results and will they help me in caring for caring for my patients? *The Journal of the American Medical Association*, 271, 703-707.
- Janke, M. (1991). Accidents, Mileage and the exaggeration of risk. *Accident Analysis and Prevention*, 23, 1983-1988.
- Jendroska, K. (1997). The relationship of Alzheimer-type pathology to dementia in Parkinson's disease. *Journal of Neural Transmission. Supplementum*, 49, 23-31.
- Joachim, C. L., Morris, J. H., & Selkoe, D. J. (1988). Clinically diagnosed Alzheimer's disease: autopsy results in 150 cases. *Annals of Neurology*, 24, 50-56.

- Johansson, K., & Lundberg, C. (1997). The 1994 International Consensus Conference on Dementia and Driving: A brief Report. *Alzheimer Disease and Associated Disorders*, 11, 62-69.
- Johansson, B., & Zarit, S. H. (1995). Prevalence and incidence of dementia in the oldest old: A study of a population based sample 84-90-year-olds in Sweden. *International Journal of Geriatric Psychiatry*, 12, 53-59.
- Jones, J.G., McCann, J., & Lassere, M.N. (1991). Driving and arthritis. *British Journal of Rheumatology*, 30, 361-364.
- Jurica, P. J., Leitten, C. L., & Mattis, S. (2001). *Dementia Rating Scale—2 professional manual*. Lutz, FL: Psychological Assessment Resources.
- Kaplan, E. F., Goodglass, H., & Weintraub, S. (1983). The Boston Naming Test (2nd ed.) Philadelphia, PA: Lea & Febiger.
- Kapust, L. R. & Weintraub, S. (1992). To drive or not to drive: preliminary results from road testing of patients with dementia. *Journal of Geriatric Psychiatry and Neurology*, 5, 210-216.
- Karlikaya, G., Yukse, G., Varlibas, F., & Tireli, H. (2005). Caregiver Burden In Dementia: A Study In The Turkish Population. *The Internet Journal of Neurology*, 4, 123-154.
- Kausler, D.H. (1991). *Experimental Psychology, Cognition, and Human Aging*. New York, NY: Springer- Verlag.

- Kaszniak, A. W., Keyl, P. M., & Albert, M. S. (1991). Dementia and the older driver. *Human Factors, 33*, 527-537.
- 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.
- Kosaka, K., Yoshimura, M., Ikeda, K., & Budka, H. (1984). Diffuse type of Lewy body disease. Progressive dementia with abundant cortical Lewy bodies and senile changes of varying degree: a new disease? *Clinical Neuropathology, 3*, 185-192.
- Korner, A., Lauritzen, L., & Abelskov. K. (2006). The Geriatric Depression Scale and the Cornell Scale for Depression in Dementia. A validity study. *Nordic Journal of Psychiatry, 60*, 360-364.
- Kruger, H.-P., Kazenwadel, J. and Vollrath, M. (2000). Grand Rapids Effects Revisited: Accidents, Alcohol & Risk. Röntgenring, Germany, Centre for Traffic Sciences, University of Wuerzburg,.
- Kukull, W. A., Higdon, R., Bowen, J. D., McCormick, W. C., Teri, L., Schellenberg, G. D., et al. (2002). Dementia and Alzheimer disease incidence: A prospective cohort study. *Archives of Neurology, 59*(11):1737-46.
- Lafleche, G., Albert, M. S. (1995). Executive function deficits in mild Alzheimer's disease. *Neuropsychology, 9*, 313-20.

Lambon, R. M. A., Powell, J., Howard, D., Whitworth, A. B., Garrard, P., Hodges, J. R. (2001).

Semantic memory is impaired in both dementia with Lewy bodies and dementia of Alzheimer's type: A comparative neuropsychological study and literature review. *Journal of Neurology, Neurosurgery, and Psychiatry*, 70, 149–156.

Langford, J., & Koppela, S. (2006). Epidemiology of older driver crashes – Identifying older

driver risk factors and exposure patterns. *Transportation Research Part F: Traffic Psychology and Behaviour*, 9(5), 309-321.

Langford, J., Methorst, R., & Hakamies-Blomqvist, L. (2006). Older drivers do not have a high

crash risk—A replication of low mileage bias. *Accident Analysis and Prevention*, 38(3), 574–578.

Larsen, L., & Kines, P. (2002). Multi-disciplinary in-depth investigations of head on and left turn

road collisions. *Accident Analysis and Prevention*, 34, 367–380.

Lesikar, S. E., Gallo, J. J., Rebok, G. W., & Keyl, P. M. (2002). Prospective study of brief

neuropsychological measures to assess crash risk in older primary care patients. *The Journal of the American Board of Family Medicine*, 15(1), 11-19.

Lezak, M. D., Howieson, D. B., & Loring, D. W., with Hannay, H. J., & Fischer, J. S. (2004).

Neuropsychological assessment (4th ed.). New York: Oxford University Press.

Lindstrom-Forneri, W., Tuokko, H. A., Garrett, D., & Molnar, F. (2010). Driving as an Everyday

Competence: A Model of Driving Competence and Behavior. *Clinical Gerontologist*, 33(4).

- Liu, D., Macchiarella, N. D., & Vincenzi, D. A. (2009). Simulation fidelity. In : P.A. Hancock, D. Vincenzi, J. Wise, & M. Mouloua, *Human Factors in Simulation and Training*. Boca Ralton: Taylor & Francis, LLC, 61-73.
- Logsdon, R. G., Teri, L., Larson, E. B. (1992). Driving and Alzheimer's disease. *Journal of General Internal Medicine*, 7, 583-585.
- Lonie, J. A., Parra-Rodrigues, M. A., Tierney, K. M., Hermann, L. L., Donaghey, C., O'Carroll, R. E., et al. (2010). Predicting outcomes in mild cognitive impairment: 4-year follow-up study. *The British Journal of Psychiatry*, 197, 135-140.
- Lovestone, S., & Gautier, S. (1998). *Management of dementia*. London: The Livery House.
- Loy-English, I. (2004). The emerging spectrum of Parkinsonian Dementias. *The Canadian Alzheimer Disease Review*, 4, 16-22.
- Lucus-Blaustein, M. J., Flipp, C. I., Dungan, C., Tune, L. (1988). Driving in patients with dementia. *Journal of American Geriatric Society*, 36, 1087-1091.
- Lundberg, C., Johansson, K., Ball, K., Bjerre, B., Blomqvist, C., Braekhus, A., et al. (1997). Dementia and Driving: An Attempt at Consensus. *Alzheimer Disease and Associated disorders*, 11, 28-37.
- Lyman, J. M., McGwin, G., & Sims, R.V. (2001). Factors related to driving difficulty and habits in older drivers. *Accident Analysis and Prevention*, 33, 413-21.
- Mack, W. J., Freed, D. M., Williams, B. W., & Henderson, V. W. (1992). Boston naming test: Shortened versions for use in Alzheimer's disease. *Journal of Gerontology*, 47, 154-158.

- Maj, M., & Sartorius, N. (2003). *Dementia*. New York: Wiley.
- Manly, J. J., Mayeux, R. (2004). Ethnic differences in dementia and Alzheimer's disease. In: Anderson N, Bulatao R, Cohen B, eds. *Critical perspectives on racial and ethnic differentials in health in late life*. Washington, D.C.: National Academies Press; 95–141.
- Man-Son-Hing, M., Marshall, S. C., Molnar, F. J., & Wilson, K, G. (2007). Systematic review of driving risk and the efficacy of compensatory strategies in persons with dementia. *Journal of the American Geriatrics Society* 55(6):878-84.
- Marottoli, R. A., Richardson, E. D., Stowe, M. H., Miller, E. G., Brass, L. M., Cooney, L., et al. (1998). Development of a test battery to identify older drivers at risk for self-reported adverse driving events. *Journal of the American Geriatric Society*, 46, 562-568.
- Martin, A., & Fedio, P. (1983). Word production and comprehension in Alzheimer's disease: The breakdown of semantic knowledge. *Brain and Language*, 19, 124–141.
- Martin-Ruis, C., Court, J., Lee, M., Piggott, M., Johnson, M., Ballard, C., et al. (2000). Nicotinic receptors in dementia of Alzheimer, Lewy body and vascular type. *Acta Neurologica Scandinavica Supplements*, 176, 34-41.
- Mayo Clinic (2012). Mild Cognitive Impairment. Retrieved on June 3rd, 2012, from <http://www.mayoclinic.com/health/mild-cognitive-impairment/DS00553>.
- McCracken, P., J. Caprio Triscott, & Dobbs, A. (2001). Driving with dementia. *The Canadian Review of Alzheimer's Disease and Other Dementias*, 12, 14-20.

- McGwin, G. & Brown, D. B. (1999). Characteristics of traffic crashes among young, middleaged, and older drivers. *Accident Analysis and Prevention*, 31, 181-198.
- 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: 102 Monash University Accident Research Centre, A population-based case-control study. *American Journal of Epidemiology*, 152(5), 424-431.
- McKeith, I. G. (1995). Lewy Body Disease. *Current Opinions in Psychiatry*, 8, 252-257.
- McKeith, I. G., Ballard, C. G., Perry, R. H., Ince, P. G., O'Brien, J. T., Neill, D., et al. (2000). Prospective validation of Consensus criteria for the diagnosis of dementia with Lewy Bodies. *Neurology*, 54, 1050-1058.
- McKeith, I. G., Burn, D. J., Ballard, C. G., Collerton, Jaros, E., Morris, C. M., et al. (2003). Dementia with Lewy bodies. *Seminars in Clinical Neuropsychiatry*, 8, 46-57.
- McKeith, I. G., Dickson, D. W., Lowe, J., Emre, M., O'Brien, J. T., Feldman, H., et al. (2005). Diagnosis and management of dementia with Lewy bodies: Third report of the DLB consortium. *Neurology*, 65, 1863-1872.
- McKeith, I. G., Fairbairn, A., Perry, R., Thompson, P., & Perry, E. (1992). Neuroleptic sensitivity in patients with senile dementia of Lewy body type. *British Medical Journal*, 305, 673-676.

- McKeith, I. G., Galasko, D., & Kosaka, K. (1996). Consensus guidelines for the clinical and pathological diagnosis of dementia with Lewy Bodies (DLB): Report of the consortium on DLB international workshop. *Neurology*, 47, 1113-1124.
- McKeith, I. G., Grace, J. B., Walker, Z., Byrne, E. J., Wilkinson, D., Stevens, T., et al. (2000). Rivastigmine in the treatment of dementia with Lewy bodies: preliminary findings from an open trial. *International Journal of Geriatric Psychiatry*, 15, 387-392.
- McKeith, I. G., Perry, E. K., & Perry, R. H. (1999). Report of the second dementia with Lewy body international workshop: Diagnosis and treatment. *Neurology*, 53, 902-905.
- McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, E. M. (1984). Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA work group under the auspices of the Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology*, 34, 939-944.
- Meng, A., Siren, A., & Teasdale, T. W. (2013). Older drivers with cognitive impairment: Perceived changes in driving skills, driving-related discomfort and self-regulation of driving. *European Geriatric Medicine*, 4, 154-160.
- Michon, J. A. (1985). A critical view of driver behavior models: What do we know what should we do? In L. Evans & R. C. Schwing (Eds.), *Human Behavior and Traffic Safety* (pp.485-524). New York: Plenum Press.

- Monsch, A. U., Bondi, M. W., Butters, N., Salmon, D. P., Katzman, R., Thal, L. J. (1992). Comparisons of verbal fluency tasks in the detection of dementia of the Alzheimer type. *Archives of Neurology*, 49, 1253–1258.
- Mori, E., Shimomura, T., Fujimori, M., Hirono, N., Imamura, T., Hashimoto, M., et al. (2000). Visuo-perceptual impairment in dementia with Lewy bodies. *Archives of Neurology*, 57, 489-493.
- 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 and Motor Skills*, 91(1), 279-290.
- National Center for Statistics and Analysis (1996). Traffic Safety Facts 1995, National Highway Traffic Safety Administration.
- Nebes, R. D., & Brady, C. B. (1989). Focused and divided attention in Alzheimer's disease. *Cortex*, 25, 305–315.
- NIH National Institute on Aging (2012). Alzheimer's disease fact sheet. Retrieved on October 5th, 2012 from <http://www.nia.nih.gov/alzheimers/publication/alzheimers-disease-fact-sheet>.
- O'Brien, J., McKeith, I., Ames, D., & Chiu, E. (2006). Dementia with Lewy Bodies and Parkinson's Disease Dementia. Oxon: Taylor and Francis Group.
- Oda, H., Yamamoto, Y., Maeda, K. (2009). The neuropsychological profile in dementia with Lewy bodies and Alzheimer's disease. *International Journal of Geriatric Psychiatry*, 24, 125-131.

- Odenheimer, G. L. (1993). Dementia and the older driver. *Journal of Clinical Geriatric Medicine*, 9, 865-869.
- Odenheimer, G. L., Beaudet, M., Jette, A. M., Albert, M. S., Grande, L., & Minaker, K. L. (1994). Performance-based driving evaluation of the elderly driver: Safety, reliability, and validity. *Journal of Gerontology* 49, M153-M159.
- O'Neill, D., Neubauer, K., Boyle, M., Gerrard, J., Surmon, D., Wilcock, G. K. (1992). Dementia and driving. *Journal of the Royal Society of Medicine*, 85, 199-202.
- Organisation for Economic Co-Operation and Development (OECD) (2001). Ageing and Transport: Mobility Needs and Safety Issues. Paris: OECD Publications.
- 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. and Nestor, P.G. (1991). Attention and driving skills in aging and Alzheimer's disease. *Human Factors*, 33, 539-557.
- Perry, R., & Hodges, J. R. (1999). Relationship between functional and neuropsychological performance in early Alzheimer disease. *Alzheimer Disease and Associated Disorders*, 14, 1-10.
- Petersen, R. C., Stevens, J. C., Ganguli, M., Tangalos, E. G., Cummings, J. L., DeKosky, S. T., et al. (2001). Practice parameter: Early detection of dementia: mild cognitive

- impairment (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*, 56, 1133-1142.
- Petersen, R. C., Smith, G. E., Waring, S. C., Ivnik, R. J., Tangalos, E. G., & Kokmen, E. (1999). Mild cognitive impairment: clinical characterization and outcome. *Archives of Neurology*, 56, 303-308.
- Petersen, R. C. (2004). Mild cognitive impairment as a diagnostic entity. *Journal of Internal Medicine*, 256, 183-194.
- Plassman, B. L., Langa, K. M., Fisher, G. G., Heeringa, S. G., Weir, D. R., & Ofstedal, M. B., et al. (2007). Prevalence of dementia in the United States: The aging, demographics, and memory study. *Neuroepidemiology*, 29(1-2):125-132.
- Preobrazhenskaya, I. S., Mkhitarian, E. A., & Yakhno, N. N. (2006). Comparative analysis of cognitive impairments in Lewy Body dementia and Alzheimer's disease. *Neuroscience and Behavioral Physiology*, 36, 1-6.
- Ransmayr, G (2000). Dementia with Lewy bodies: Prevalence, clinical spectrum, and natural history. *Journal of Neural Transmission Supplementary*, 60, 303-314.
- Rebok, G. W., Keyl, P. M., Bylsma, F. W., Blaustein, M. J., & Tune, L. (1994). The effects of Alzheimer disease on driving-related abilities. *Alzheimer Disease and Associated Disorders*, 8, 228-240.
- Reger, M. A., Welsh, R. K., Stenis Watson, G., Cholerton, B., Baker, L. D., & Craft, S. (2004). The relationship between neuropsychological functioning and driving ability in dementia: A meta-analysis. *Neuropsychology*, 18, 85-93.

- Reisberg, B., Ferris, S.H., de Leon, M.J., & Crook, T. (1982). The global deterioration scale for assessment of primary degenerative dementia. *American Journal of Psychiatry*, 139, 1136-1139.
- Reisberg, B., Ferris, S. H., Steinberg, G., Shulman, E., & deLeon, M. J. (1989). Longitudinal study of dementia patients and aged controls: An approach to methodological issues. In M. P. Lawton & A. R. Herzog (Eds.), *Special Research Methods for Gerontology* (pp.195-231). Amityville, NY: Baywood.
- Roberts, S. E., Vingilis, E., Wilk, P., & Seeley, J. (2008). A comparison of self-reported motor vehicle collision injuries compared with official collision data: An analysis of age and sex trends using the Canadian National Population Health Survey and Transport Canada data. *Accident Analysis & Prevention*, 40(2), 559-566.
- Robertson, I. H., Ward, T., Ridgeway, V., & Nimmo-Smith, I. (1994). The test of everyday attention. Bury St. Edmunds, UK: Thames Valley Test Company.
- Roe, C. M., Xiong, C., Miller, J. P, Morris, J. C. (2007). Education and Alzheimer disease without dementia: Support for the cognitive reserve hypothesis. *Neurology*, 68(3):223–228.
- Rosvold, H. E., Mirsky, A. F, Sarason, I., Bransome, E. D., Beck, L. H. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology*, 20, 343–50.
- Salvucci, D. D. (2006). Modeling driver behavior in a cognitive architecture. *Human Factors: The Journal of the Human Factors and Ergonomics*, 48(2), 362-380.

- Schinka, J., Loewenstein, D. A., Raj, A., Schoenberg, M. R., Banko, J. L., Potter, H., et al. (2010). Defining mild cognitive impairment: impact of varying decision criteria on neuropsychological diagnostic frequencies and correlates. *American Journal of Geriatric Psychiatry, 18*, 684-691.
- Schoenberg, M. R., & Scott, J. G. (2011). *The little black book of neuropsychology: a syndrome-based approach*. New York: Springer.
- Schultz, J. L., Davis, L. E., & King, M. K. (2005). *Fundamentals of Neurologic Disease*. New York: Demos Medical Publishing.
- Shaheen, S. A., & Niemeier, D. A. (2001). Integrating vehicle design and human factors: minimizing elderly driving constraints. *Transportation Research Part C: Emerging Technologies, 8*, 155-174.
- Sheahan, S. L., Coons, S. J., Robbins, C. A., Martin, S. S., Hendricks, J., & Latimer, M. (2005). Psychoactive medication, alcohol use, and falls among older adults. *Journal of Behavioral Medicine, 18*, 127-140.
- Sheridan, T. B. (2004). Driver distraction from a control theory perspective. *Human Factors: The Journal of the Human Factors and Ergonomics, 46*(4), 587-599.
- Shimomura, T., Mori, E., Yamashita, H., Imamura, T., Hirono, N., Hashimoto, M., et al. (1998). Cognitive loss in dementia with Lewy bodies and Alzheimer disease. *Archives of Neurology, 55*, 1547-1552.
- Shinar, D. & Scheiber, F. (1991). Visual requirements for safety and mobility of older drivers. *Human Factors, 33*, 507-519.

- Silverstein, N.M., Flaherty, G., & Tobin, T.S. (2002). *Dementia and Wandering Behavior: Concern for the Lost Elder*. New York, NY: Springer.
- Skyving, M., Berg, H.-Y., & Laflamme, L. (2009). A pattern analysis of traffic crashes fatal to older drivers. *Accident Analysis & Prevention*, 41 (2), 253-258.
- Snowdon, D. A. (2003). Healthy aging and dementia: findings from the Nun study. *Annals of Internal Medicine*, 139, 450-454.
- Spieler, D. H., Balota, D. A., & Faust, M. E. (1996). Stroop performance in healthy younger and older adults and in individuals with Dementia of the Alzheimer's type. *Journal of Experimental Psychology: Human Perception & Performance*, 22, 461-479.
- Staplin, L., & Lyles, R. W. (1992). Age differences in motion perception and specific traffic maneuver problems. *Transportation Research Record*, 1325, 23-33.
- Statistics Canada. (2005). General Social Survey. Retrieved from http://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&SDDS=4503&Item_Id=62269&lang=en on October 9th, 2009.
- Statistics Canada. (2006). *Canadian Vehicle Survey: Annual, 2000* and *Canadian Vehicle Survey: Annual, 2006*. Ottawa, Statistics Canada.
- Stamatiadis, N. (1996). Gender effect on the accident patterns of elderly drivers. *Journal of Applied Gerontology*, 15, 8-22.
- Stamatiadis, N., & Deacon, J. A. (1997). Quasi-induced exposure: methodology and insight. *Accident Analysis and Prevention*, 29, 37-52.

- Steg, L., & Gifford, R. (2005). Sustainable transport and quality of life. *Journal of Transport Geography* 13, 59-69.
- Stern Y. (2006). Cognitive reserve and Alzheimer disease. *Alzheimer Disease & Associated Disorders*, 20(2):112–117.
- Stern, Y., Gurland, B., Tatemichi, T. K., Tang, M. X., Wilder, D., Mayeux, R. (1994). Influence of education and occupation on the incidence of Alzheimer's disease. *Journal of the American Medical Association*, 271(13), 1004–10.
- Sunderland, T., Jeste, D. V., Baiyewu, O., Sirovatka, P. J., & Regier, D. A. (2007). *Diagnostic Issues in Dementia: Advancing the Research Agenda for DSM-5*. Arlington, VA: American Psychiatric Association.
- Templeton, V. H., & Kirkman, D. N. (2007). Fraud, vulnerability, and aging: Case studies. *Alzheimer's Care Today*, 8, 265-277.
- Transport Canada. (2001). *Trends in motor vehicle traffic collision statistics 1988 - 1997*. Ottawa: Transport Canada.
- Transport Canada. (2010). Canadian Motor Vehicle Traffic Collision Statistics: 2010. Retrieved on June 3rd, 2013, from <http://www.tc.gc.ca/eng/roadsafety/tp-1317.htm#2>.
- Tuokko, H., Tallman, K., Beattie, B. L., Cooper, P., Weir, J. (1995). An examination of driving records in a dementia clinic. *Journal of Gerontology*, 550B, S173-181.
- 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. *Journal of Neurology, Neurosurgery & Psychiatry*, 76, 764-768.
- Uc, E.Y., Rizzo, M., Anderson, S.W., Shi, Q., & Dawson, J.D. (2004). Driver route-following and safety errors in early Alzheimer's Disease, *Neurology*, 63, 832-837.

- Ulleberg, P. (2001). Personality subtypes of young drivers. Relationship to risk-taking preferences, accident involvement, and response to a traffic safety campaign. *Transportation Research Part F: Traffic Psychology and Behaviour*, 4(4), 279-297.
- University of California Davis, Alzheimer's Disease Centre (2007). Mild cognitive impairment. Retrieved May 30, 2007 from <http://alzheimer.ucdavis.edu/faq/mci.php>.
- Unsworth, C. A., Lovell, R. K., Terrington, N. S., & Thomas, S. A. (2005). Review of tests contributing to the occupational therapy off-road driver assessment. *Australian Occupational Therapy Journal*, 52, 57-74.
- Vaez, M., & Laflamme, L. (2005). Impaired driving and motor vehicle crashes among Swedish youth: An investigation into drivers' sociodemographic characteristics. *Accident Analysis & Prevention*, 37(4), 605-611.
- Valcour, V. G., Masaki, K. H., & Blanchette, P. L. (2002). Self-reported driving, cognitive status, and physician awareness of cognitive impairment. *Journal of the American Geriatric Society* 50(7), 1265-1267.
- Van der Flier, W. M., Scheltens, P. (2005). Epidemiology and risk factors of dementia. *Journal of Neurology, Neurosurgery and Psychiatry*, 5, v2-7.
- van Zomeren, A., Brouwer, W., & Minderhoud, J. (1987). Acquired brain damage and driving: A review. *Archives of Physical Medicine and Rehabilitation*, 68, 697-705.
- Vernon, D., E., & Diller, C. L. (2002). Evaluating the Crash and Citation Rates of Utah Drivers Licensed with Medical Conditions 1992-1996. *Accident Analysis and Prevention*, 34, 237-246.
- Vision Australia Foundation (2002). Statistics on eye health in Australia. Retrieved

May 24, 2007, from <http://www.visionaustralia.org.au/index>.

- Vodden, K., Smith, D., Eaton, F., & Mayhew, D. (2007). *Analysis and estimation of the social cost of motor vehicle collisions in Ontario*. Ottawa: Transport Canada.
- Walker, J. E., & Howland, J. (1991). Falls and fear of falling among elderly persons living in the community: Occupational therapy interventions. *American Journal of Occupational therapy*, 45, 119-122.
- Walker, M. P., Ayre, G. A., Cummings, J. L., Wesne, K., McKeith, I. G., O'Brien, J. T., & Ballard, C. G. (2000). Quantifying fluctuation in dementia with Lewy bodies, Alzheimer's disease and vascular dementia. *Neurology*, 54, 1616-1624.
- Walker, J. A. (1967). Cardiovascular disease, aging and traffic accidents. *Journal of Chronic Disease*, 20, 615-620.
- Warrington, E. K. & James, M. J. (1991). The visual object and space perception battery. Bury St. Edmunds, UK: Thames Valley Test Company.
- Welsh, K. A., Butters, N., Hughes, J. P., Mohs, R., Heyman, A. (1991). Detection of abnormal memory decline in mild cases of Alzheimer's disease using CERAD neuropsychological measures. *Archives of Neurology*, 48(3), 278-281.
- Welsh, K. A., Butters, N., Hughes, J. P., Mohs, R. C., Heyman, A. (1992). Detection and staging of dementia in Alzheimer's disease: use of the neuropsychological measures developed for the Consortium to Establish a Registry for Alzheimer's disease. *Archives of Neurology*, 49, 448-452.
- Williams, A. F. (2003). Teenage drivers: patterns of risk. *Journal of Safety Research*, 34(1), 5-15.

- Williams, V. G., Bruce, J. M., Westervelt, H. J., Davis, J. D., Grace, J., Malfoy, P. F., & Tremont, G. (2007). Boston naming performance distinguishes between Lewy body and Alzheimer's dementias. *Archives of Clinical Neuropsychology*, 22, 925-931.
- Wilson, S., & Pinner, G. (2013). Driving and dementia: a clinician's guide. *Advances in Psychiatric Treatment*, 19, 89-96.
- Yesavage, J. A., Brink, T. L., Rose, T. L., Lum, O., Huang, V. S., Adey, M., et al. (1983). Development and validation of a geriatric depression screening scale: A preliminary report. *Journal of Psychiatric Research*, 17, 37-39.
- Zarit, S & Zarit, J. (2007). *Mental Disorders in Older Adults: Fundamentals of Assessment and Treatment*. (2nd Ed.). New York: The Guilford Press.

Appendix A

Revised criteria for the clinical diagnosis of dementia with Lewy bodies (DLB)

1. *Central feature* (essential for a diagnosis of possible or probable DLB)

Dementia defined as progressive cognitive decline of sufficient magnitude to interfere with normal social or occupational function. Prominent or persistent memory impairment may not necessarily occur in the early stages but is usually evident with progression. Deficits on tests of attention, executive function, and visuospatial ability may be especially prominent.

2. *Core features* (two core features are sufficient for a diagnosis of probable DLB, one for possible DLB)

Fluctuating cognition with pronounced variations in attention and alertness

Recurrent visual hallucinations that are typically well formed and detailed

Spontaneous features of parkinsonism

3. *Suggestive features* (If one or more of these is present in the presence of one or more core features, a diagnosis of probable DLB can be made. In the absence of any core features, one or more suggestive features is sufficient for possible DLB. Probable DLB should not be diagnosed on the basis of suggestive features alone.)

REM sleep behavior disorder

Severe neuroleptic sensitivity

Low dopamine transporter uptake in basal ganglia demonstrated by SPECT or PET imaging

4. *Supportive features* (commonly present but not proven to have diagnostic specificity)

Repeated falls and syncope

Transient, unexplained loss of consciousness

Severe autonomic dysfunction, e.g., orthostatic hypotension, urinary incontinence

Hallucinations in other modalities

Systematized delusions

Depression

Relative preservation of medial temporal lobe structures on CT/MRI scan

Generalized low uptake on SPECT/PET perfusion scan with reduced occipital activity

Abnormal (low uptake) MIBG myocardial scintigraphy

Prominent slow wave activity on EEG with temporal lobe transient sharp waves

5. A diagnosis of DLB is *less likely*

In the presence of cerebrovascular disease evident as focal neurologic signs or on brain imaging

In the presence of any other physical illness or brain disorder sufficient to account in part or in total for the clinical picture

If parkinsonism only appears for the first time at a stage of severe dementia

6. *Temporal sequence* of symptoms

DLB should be diagnosed when dementia occurs before or concurrently with parkinsonism (if it is present). The term Parkinson disease dementia (PDD) should be used to describe dementia that occurs in the context of well-established Parkinson disease. In a practice setting the term that is most appropriate to the clinical situation should be used and generic terms such as LB disease are often helpful. In research studies in which distinction needs to be made between DLB and PDD, the existing 1-year rule between the onset of dementia and parkinsonism DLB continues to be recommended. Adoption of other time periods will simply confound data pooling or comparison between studies. In other research settings that may include clinicopathologic studies and clinical trials, both clinical phenotypes may be considered collectively under categories such as LB disease or alpha-synucleinopathy.

Appendix B

The Global Deterioration Scale for Assessment of Primary Degenerative Dementia

Level	Clinical Characteristics
1 No cognitive decline	No subjective complaints of memory deficit. No memory deficit evident on clinical interview.
2 Very mild cognitive decline (Age Associated Memory Impairment)	Subjective complaints of memory deficit, most frequently in following areas: (a) forgetting where one has placed familiar objects; (b) forgetting names one formerly knew well. No objective evidence of memory deficit on clinical interview. No objective deficits in employment or social situations. Appropriate concern with respect to symptomatology.
3 Mild cognitive decline (Mild Cognitive Impairment)	Earliest clear-cut deficits. Manifestations in more than one of the following areas: (a) patient may have gotten lost when traveling to an unfamiliar location; (b) co-workers become aware of patient's relatively poor performance; (c) word and name finding deficit becomes evident to intimates; (d) patient may read a passage or a book and retain relatively little material; (e) patient may demonstrate decreased facility in remembering names upon introduction to new people; (f) patient may have lost or misplaced an object of value; (g) concentration deficit may be evident on clinical testing. Objective evidence of memory deficit obtained only with an intensive interview. Decreased performance in demanding employment and social settings. Denial begins to become manifest in patient. Mild to moderate anxiety accompanies symptoms.
4 Moderate cognitive decline (Mild Dementia)	Clear-cut deficit on careful clinical interview. Deficit manifest in following areas: (a) decreased knowledge of current and recent events; (b) may exhibit some deficit in memory of one's personal history; (c) concentration deficit elicited on serial subtractions; (d) decreased ability to travel, handle finances, etc. Frequently no deficit in following areas: (a) orientation to time and place; (b) recognition of familiar persons and faces; (c) ability to travel to familiar locations. Inability to perform complex tasks. Denial is dominant defense mechanism. Flattening of affect and withdrawal from challenging situations frequently occur.

5 Moderately severe cognitive decline (Moderate Dementia)	Patient can no longer survive without some assistance. Patient is unable during interview to recall a major relevant aspect of their current lives, e.g., an address or telephone number of many years, the names of close family members (such as grandchildren), the name of the high school or college from which they graduated. Frequently some disorientation to time (date, day of week, season, etc.) or to place. An educated person may have difficulty counting back from 40 by 4s or from 20 by 2s. Persons at this stage retain knowledge of many major facts regarding themselves and others. They invariably know their own names and generally know their spouses' and children's names. They require no assistance with toileting and eating, but may have some difficulty choosing the proper clothing to wear.
6 Severe cognitive decline (Moderately Severe Dementia)	May occasionally forget the name of the spouse upon whom they are entirely dependent for survival. Will be largely unaware of all recent events and experiences in their lives. Retain some knowledge of their past lives but this is very sketchy. Generally unaware of their surroundings, the year, the season, etc. May have difficulty counting from 10, both backward and, sometimes, forward. Will require some assistance with activities of daily living, e.g., may become incontinent, will require travel assistance but occasionally will be able to travel to familiar locations. Diurnal rhythm frequently disturbed. Almost always recall their own name. Frequently continue to be able to distinguish familiar from unfamiliar persons in their environment. Personality and emotional changes occur. These are quite variable and include: (a) delusional behavior, e.g., patients may accuse their spouse of being an impostor, may talk to imaginary figures in the environment, or to their own reflection in the mirror; (b) obsessive symptoms, e.g., person may continually repeat simple cleaning activities; (c) anxiety symptoms, agitation, and even previously nonexistent violent behavior may occur; (d) cognitive abulia, i.e., loss of willpower because an individual cannot carry a thought long enough to determine a purposeful course of action.
7 Very severe cognitive decline (Severe Dementia)	All verbal abilities are lost over the course of this stage. Frequently there is no speech at all –only unintelligible utterances and rare emergence of seemingly forgotten words and phrases. Incontinent of urine, requires assistance toileting and feeding. Basic psychomotor skills, e.g., ability to walk, are lost with the progression of this stage. The brain appears to no longer be able to tell the body what to do. Generalized rigidity and developmental neurologic reflexes are frequently present.

Appendix C

Participant Health Profile Questionnaire**DEMOGRAPHIC INFO**

Date: _____

Reference number: _____

Sex: _____

Mother Tongue: _____

Age: _____ Birth Date: _____

Handedness: _____

a) Has this always been your dominant hand? _____

b) Ambidextrous (i.e., both hands): what task do you accomplish with each?

Number of school years completed: _____

Previous occupation(s): _____

Daily activities (physical activities, work, hobbies):

Have you been assessed for legal competency and if so what was the outcome (only for participants with DLB or AD): _____

Are you usually accompanied to your regular appointments at the Memory Disorders Clinic (only for participants with DLB or AD): _____

Number of studies participated in (only for participants with DLB or AD): _____

MEDICAL INFO

Vision problems (myopic, presbyotic, glaucoma, cataracts etc.):

Glasses: Y N

Colour blindness: Y N

Hearing Impaired: Y N

Hearing Aid: Y N

Have you ever lost consciousness, or been in a coma following a head injury (indicate circumstances)?

Do you suffer from:

Headaches: _____

Epilepsy: _____

Apoplexy (i.e., Hemorrhagic stroke which is uncontrolled bleeding into the brain due to a cerebrovascular accident (CVA), resulting in sudden loss of consciousness and paralysis of various parts of the body): _____

Heart attack: _____

High blood pressure: _____

Diabetes (type 1 or 2): _____

Thyroid insufficiency: _____

Sleep Apnea (i.e., Pauses in breath during sleep): _____

Heart congestion (symptoms, duration): _____

TIA's (i.e., mini-stroke that causes no lasting damage): _____

Major surgeries: _____

Other serious illnesses (e.g. at birth): _____

Exposure to toxins: _____

Medication:

Amount

Duration

Alcoholism: (present): _____
(past): _____

Tobacco: _____

Other drugs: _____

Have you ever taken medication because you were anxious, depressed, or unhappy?

BACKGROUND INFO

Sleep:

Typically, what is your best period of sleep during the day (morning, afternoon, evening or overnight)?

Have you ever experienced problems with insomnia? _____

Typically, how many hours do you sleep per night?

Schools and Studies:

Can you recall having any problems learning? Do you have problems with basic academic skills such as reading, mathematics, and/or writing?

As a student: _____

Now: _____

Have you ever dropped or skipped a school year? Why?

Other information:

Over the course of a normal day, how much tea, coffee, or other stimulants do you consume?

Have you ever had difficulty with your orientation, that is, finding your way in a familiar or an unfamiliar environment?

Do you have a valid driver's licence? _____

FAMILY HISTORY

Yes

At what age?

Dementia:

Parkinson's disease:

Heart disease:

High blood pressure:

Stroke:

Diabetes:

Psychiatric disorder:

Neurological disease:

Others:

Are there other matters concerning your family history or your own health, which you think are important and relevant, that I have not asked you?

Source of reference: _____

Appendix D

Telephone Script for Control Participants – Research Assistant

“Hello [potential participant’s name], my name is [first and surname] and I am a Research Assistant calling from Dr. Sylvain Gagnon’s lab in the School of Psychology at the University of Ottawa. You’ve expressed an interest in participating in one of our research studies. We are presently conducting a study on the mental abilities associated with early stage dementia in comparison to healthy elderly individuals. Are you still interested in learning more?”

[IF NO] Thank you, goodbye.

[IF YES] “This study involves completing a variety of neuropsychological tests. The tests are either administered by computer or paper-pencil format. Examples of tasks involved include visual and attention tasks.”

“Participation in this study will take approximately 5 hours of your time and will be spread out over two sessions. All fees relating to parking or public transportation will be covered and refreshments will be offered. The final decision about participation is yours. ”

“I would like to assure you that this study has been reviewed and received ethics clearance from the SCO Health Service Research Ethics Board.”

“Would you be interested in participating?”

[IF NO]: “Thank you, good-bye”.

[IF YES]: “Thank you; we appreciate your interest in our research.”

“In order to participate in this study, there is a 10-15 minute health and demographic questionnaire that you would be required to complete over the phone with me. This is simply a routine procedure in order to have a general profile of our participants’ mental and physical background. Also, there are certain inclusion criteria that must be met in order for you to be an eligible participant. Would this be a good time?”

(IF NO) (Reschedule an appropriate time)

(IF YES) “Great. Before we begin, I would like to assure you that all the information I will be collecting today is strictly confidential; in fact, none of the information will be associated with your name. Only I and Dr. Gagnon will have access to this information.”

“In addition, if there are any questions that cause you any discomfort, please let me know and we will move on to the next one, no questions asked.”

“Let us begin”

(INSERT QUESTIONNAIRE)

(If participant does not meet inclusion criteria): “Unfortunately, you do not meet all of our inclusion criteria for this particular study. However, we are conducting many studies at the lab that may require your participation. Would you be interested in learning more about these studies?”

(IF YES): Provide information

(If participant meets inclusion criteria):

“I have sessions open on [day and date] at [time, a.m. or p.m.]. Will you be available then? It is important to note that certain tests will be completed during the first session. The results from these tests might exclude from the rest of the testing, however you will still be reimbursed for your traveling time.”

[IF NO]: Offer another day and time until one is found that is mutually convenient.

[IF YES]: “This is great. Let me give you some important details about the study. Have you got a pen so that you can write this down and keep it with you?”

“This study is called “Differentiation of Cognitive Deficits in Early Dementia with Lewy bodies (DLB) from Early Alzheimer’s Disease (AD).” and my name is [first and surname]. This study will take place in two testing session both of which will be at the EBRI [provide instruction on how to get to the EBRI and the testing room]

“The day before your session, I will contact you by telephone as a reminder. However, in the meantime, if you discover you will be unable to make it, please call. Please try to provide at least 24 hours notice.”

“I look forward to meeting you on [mention day, date and time again]. Thank you very much again for helping us with our research”.

Appendix F

Telephone Script for Early Stage Dementia – Research Assistant

“Hello [potential participant’s name], my name is [first and surname] and I am a Research Assistant calling from Dr. Sylvain Gagnon’s lab in the School of Psychology at the University of Ottawa. The reason I am calling is that we are conducting a study on the mental abilities of patients with early stage dementia. You indicated your interest in learning more about future studies to Dr. Frank or Dr. Loy-English at the Bruyère Continuing Care Center at the time that you sought services at the Memory Clinic. Are you still interested in learning more?”

[IF NO] Thank you, goodbye.

[IF YES] “This study involves completing a variety of neuropsychological tests. The tests are either administered by computer or paper-pencil format. Examples of tasks involved include visual and attention tasks.”

“Participation in this study will take approximately 5 hours of your time and will be spread out over two sessions. All fees relating to parking or public transportation will be covered and refreshments will also be offered. The final decision about participation is yours.”

“I would like to assure you that this study has been reviewed and received ethics clearance from the SCO Health Service Research Ethics Board. However, the final decision about participation is yours. In addition, your participation in no way affects future treatment at the Bruyère Continuing Care Centre.”

“Would you be interested in participating?”

[IF NO]: “Thank you, good-bye”.

[IF YES]: “Thank you; we appreciate your interest in our research.”

“In order to participate in this study, there is a 10-15 minute health and demographic questionnaire that you would be required to complete over the phone with me. This is simply a routine procedure in order to have a general profile of our participants’ mental and physical background. Also, there are certain inclusion criteria that must be met in order for you to be an eligible participant. Would this be a good time?”

(IF NO) (Reschedule an appropriate time)

(IF YES) “Great. Before we begin, I would like to assure you that all the information I will be collecting today is strictly confidential; in fact, none of the information will be associated with your name. Only I and Dr. Gagnon will have access to this information.

“In addition, if there are any questions that cause you any discomfort, please let me know and we will move on to the next one, no questions asked.”

“Let us begin” (INSERT QUESTIONNAIRE)

(If participant does not meet inclusion criteria): “Unfortunately, you do not meet all of our inclusion criteria for this particular study. I thank you for your time. Have a good day. Good bye”

(If participant meets inclusion criteria):

“I have sessions open on [day and date] at [time, a.m. or p.m.]. Will you be available then? (If participant is usually accompanied to all regular appointments at the Memory Disorders Clinic make sure to take an appointment that is convenient for both individuals). It is important to note that certain tests will be completed during the first session. The results from these tests might exclude from the rest of the testing, however you will still be reimbursed for your traveling time.”

[IF NO]: Offer another day and time until one is found that is mutually convenient.

[IF YES]: “This is great. Let me give you some important details about the study. Have you got a pen so that you can write this down and keep it with you?”

“This study is called “Differentiation of Cognitive Deficits in Early Dementia with Lewy bodies (DLB) from Early Alzheimer’s Disease (AD).” and my name is [first and surname]. This study will take place in two testing session both of which will be at the EBRI [provide instruction on how to get to the EBRI and the testing room]

“The day before your session, I will contact you by telephone as a reminder. However, in the meantime, if you discover you will be unable to make it, please call me. Please try to provide at least 24 hours notice.”

“I look forward to meeting you on [mention day, date and time again]. Thank you very much again for helping us with our research.

Appendix H



uOttawa

L'Université canadienne
Canada's university

Title of Project: Differentiation of Cognitive Deficits in Early Dementia with Lewy bodies (DLB) from Early Alzheimer's Disease (AD).

Principal Investigator: Sylvain Gagnon, PhD
University of Ottawa

Co-Investigator: Stephanie Yamin, Bsc.
University of Ottawa

Participation Information and Consent Form

You have been asked to take part in a research study. It is up to you to decide whether to be in the study or not. Before you decide, you need to understand what the study is for, what risks you might take and what benefits you might receive. This consent explains the study.

We are inviting you to participate in a research study that looks at the mental capabilities of individuals in the early stages of dementia. As a participant in this study, you will be given several neuropsychological tests. These tests measure intellectual functions such as memory, attention and visual perception.

The study will take about 5 hours of your time over two sessions. It is your decision to participate and you can refuse if you do not want to. If you volunteer, you will be helping research in the field of aging. Additionally, Ms. Yamin will examine your medical record at the Bruyère Continuing Care Center in order to verify your diagnosis if applicable.

There are no known risks to participating in this study. However, you may feel tired because the tests make you think a lot. The experimenters will always check up on your well-being and you will get breaks. If you want more help, you can contact the Centre for Psychological Services at 562-5289.

What's involved?

If I take part in this study:

- On the first visit, Ms. Yamin will give me some neuropsychological tests. These are tests that are normally given to patients to test their memory, attention and visual perception.
- The first session should take 2.5 hours of my time. The tests have no known risk. If I find them too much to take, I can stop them at any time.
- Ms. Yamin will ask me to return to the laboratory at a date that works for me for a second session. During this second session, I will be asked to complete more neuropsychological tests.
- The second session should take 2.5 hours of my time. The tests have no known risk. If I find them too much to take, I can stop them at any time.
- Doing the study will not affect any other tests or treatments I might have at the Bruyère Continuing Care Center.
- Participating in this study may not benefit me. However, our results may benefit other individuals with early stage dementia.
- Should you decide to withdraw from the study, you will be asked for your consent to retain all or parts of your data that has been collected.

Money Matters

- Dr. Gagnon will pay the costs of my getting to and from the Bruyère Continuing Care Center for this study. Parking fees will be paid for. The reimbursement will be provided even if I decide to withdraw from the study (up to a maximum of \$85).
- Dr. Gagnon is getting support from the University of Ottawa Research Office and Faculty of Social Sciences RIND to pay for the costs of this study.
- I should not get hurt by taking part in this study. However, if any harm does come to me during the study, Dr. Gagnon will see that I get proper treatment.

Privacy

Dr. Gagnon and Ms Yamin will protect my identity. All information will be stored at the Memory Disorders Clinic at the Bruyère Continuing Care Center.

- They will keep paper records in a locked file cabinet. They will keep electronic data on a secure computer protected by a password or on disks in the locked file cabinet.
- They will use code numbers in place of my name on all records.
- No one other than Dr. Gagnon and Ms Yamin will see my study records.
- Dr. Frank will store my study records for at least 15 years. After that, they will be destroyed.
- If they publish results of this study, they will not reveal my identity. Only group averages will be presented.

Continuing consent

During this study, Dr. Gagnon will tell me about any new information that might affect my wanting to stay in the study.

Questions?

If I have any questions later or if I want a copy of the study results, I can contact Ms. Yamin or Dr. Gagnon.

This study has been read and accepted by the SCO Health Service Research Ethics Board. If you have any comments or questions after your participation in this study, please contact the Chair of the Research Ethics Board at the Bruyère Continuing Care Center, Dr. Lisa Sweet.

Consent Form

I, _____, agree to take part in this research study being done by Dr. Sylvain Gagnon and his graduate student Stephanie Yamin.

I have read the information sheet that gives all the details about this study.

I have had enough time to ask questions I had about it.

I would like a copy of the study's finding once the study has been completed? Y / N

Signature

Signing this form gives us your consent to be in this study. It tells us that you understand the information about the research study. When you sign this form, you do not give up your legal rights. Researchers or agencies involved in this research study still have their legal and professional responsibilities.

Signed,

Participant

Date

Researcher or delegate

Date

Appendix I

Research Advertisement:

The Laboratory of Cognitive Aging at the University of Ottawa is currently recruiting participants for a study taking place at the Bruyère Continuing Care Center. We are searching for Anglophone participants in good physical and mental health that are 65+ of age, to participate in a study concerning the psychology of aging supervised by Dr. Sylvain Gagnon. Transportation costs will be reimbursed.

Appendix L

Testing Protocol

Recruitment through newspaper ads (controls) or at the Bruyère Continuing Care Center (AD & DLB)

Phone interview with demographic questionnaire

Session 1 (2.5 hours) at Bruyère Continuing Care Center:

- Consent signed
- GDS completed
- GDS depression
- MMSE
- DRS-2 (counterbalanced – original or alternate)
- BNT
- VOSP

Session 2 (2.5 hours) at Bruyère Continuing Center:

- DRS-2 (counterbalanced – original or alternate)
- TEA
- UFOV
- Stimulated driving task
- Debriefing

Appendix M

Probable AD according to NINCDS-ADRDA criteria (McKhann et al., 1984)

I. Criteria for the clinical diagnosis of PROBABLE Alzheimer's disease

- dementia established by clinical examination and documented by the MMSE; 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

II. The diagnosis of PROBABLE Alzheimer's disease is supported by:

- progressive deterioration of specific cognitive functions such as language (aphasia), motor skills (apraxia), and perceptions (agnosia);
- impaired activities of daily living and altered patterns of behavior;
- family history of similar disorders, particularly if confirmed neuropathologically; and
- laboratory results of:
 - normal lumbar puncture as evaluated by standard techniques,
 - normal pattern or non-specific changes in EEG, such as increased slow-wave activity, and
 - evidence of cerebral atrophy on CT with progression documented by serial observation

III. Other clinical features consistent with the diagnosis of PROBABLE Alzheimer's disease, after exclusion of causes of dementia other than Alzheimer's disease, include:

- plateaus in the course of progression of the illness;
- associated symptoms of depression, insomnia, incontinence, delusions, illusions, hallucinations, catastrophic verbal, emotional, or physical outbursts, sexual disorders, and weight loss;
- other neurologic abnormalities in some patients, especially with more advanced disease and including motor signs such as increased muscle tone, myoclonus, or gait disorder;
- seizures in advanced disease; and
- CT normal for age

IV. Features that make the diagnosis of PROBABLE Alzheimer's disease uncertain or unlikely include:

- sudden, apoplectic onset;
- focal neurologic findings such as hemiparesis, sensory loss, visual field deficits, and incoordination early in the course of the illness; and
- seizures or gait disturbances at the onset or very early in the course of the illness.

Appendix N

Guidelines for the Assessment of Dementia in Hospitals (assessment is done every six months)	
Medical History	Information about current mental or physical conditions, prescription drug intake, and family health history.
Physical Examination	Includes evaluation of nutritional status, blood pressure, and pulse. An evaluation of any comorbid medical conditions is done. Comorbid medical conditions can have an effect on dementia symptoms.
Neurological Examination	An magnetic resonance imaging (MRI) study of the brain is used to search for possible causes of dementia (i.e., strokes for vascular dementia).
Laboratory Tests	Blood and urine tests are conducted to verify that there is not any other condition that may be causing the dementia symptoms.
Psychiatric Evaluation	Daily Function (Activities of Daily Living; ADL): Basic activities of daily living, such as feeding and toileting are assessed. This is used to evaluate the degree of assistance received by the patient during a set period of time. Six activities are assessed including bathing, dressing, toileting, transfer, continence, and feeding.
	Cognition (MMSE): The MMSE assesses the sense of time and place and the ability to remember, understand, communicate, and do simple calculations.
	Disorders of Mood and Emotion (Neuropsychiatric Inventory Questionnaire; NPI-Q): Both agitation, psychotic and depressive symptoms are assessed. Psychosis is present in certain types of dementia and can be used as a diagnostic indicator (i.e., DLB). Depression is assessed as depressive symptoms can induce a worsening of dementia symptoms.
	Neuropsychological Battery: The battery includes tests of memory, reasoning, vision-motor coordination, and language function.
	Caregiver Status: The caregiver undertakes a structured interview to fill in the gaps of the testing. Following the caregiver's physical and emotional health is also assessed. Good physical and emotional health in the caregiver is crucial in the optimal care of the patient with Dementia.

Appendix O

Exclusion criteria for patients with AD and DLB diagnoses:

- 1- Serious visual or hearing impairments left uncorrected (e.g. cataracts and color blindness)
- 2- Serious health problems (e.g. mental illness, history of head injury, epilepsy, apoplexy, heart attacks, hypertension, and sleep apnea).
- 3- History of substance abuse.
- 4- History of learning disabilities (i.e., can they recall having any problems learning in school?)
- 5- Participation in more than 3 studies.

Title of Project: Differentiation of Cognitive Deficits in Early Dementia with Lewy bodies (DLB) from Early Alzheimer's Disease (AD).

Principal Investigator: Sylvain Gagnon, PhD
University of Ottawa

Co-Investigator: Stephanie Yamin, Bsc.
University of Ottawa

Participation Information and Consent Form for Substitute Decision Maker

An individual under your legal responsibility has been asked to take part in a research study. It is up to you to decide whether this individual should participate in this study or not. Before you decide, you need to understand what the study is for, what risks the individual under your legal responsibility might face and what benefits this individual might receive. This consent explains the study.

We are inviting the individual under your legal responsibility to participate in a research study that looks at the mental capabilities of individuals in the early stages of dementia. As a participant in this study, he/she will be given several neuropsychological tests. These tests measure intellectual functions such as memory, attention and visual perception.

The study will take about 5 hours of your time over two sessions. It is your decision whether the individual under your care should participate and you can refuse if you do not think it would be appropriate. If you determine that the individual under your legal care should volunteer, you will be helping research in the field of aging. Additionally, Ms. Yamin will examine the individual under your legal responsibility's medical record at the Bruyère Continuing Care Center in order to verify the diagnosis if applicable.

There are no known risks to participating in this study. However, the individual under your legal care may feel tired because the tests make people think a lot. The experimenters will always check up on participant's well-being and breaks will be provided. If you feel that the individual under your care needs additional help, you can contact the Centre for Psychological Services at 562-5289

What's involved?

If I take part in this study:

- On the first visit, Ms. Yamin will give the participant some neuropsychological tests. These are tests that are normally given to patients to test their memory, attention and visual perception.

- The first session should take 2.5 hours of your time and the individual under your legal care's time. The tests have no known risk. If the individual under my legal care finds them too much to take, I can withdraw him/her from the study at any time.
- Ms. Yamin will ask me and the individual under my legal care to return to the laboratory at a date that works for me for a second session. During this second session, the person under my legal care will be asked to complete more neuropsychological tests.
- The second session should take 2.5 hours of your time and the individual under your legal care's time. The tests have no known risk. If the individual under my legal care finds them too much to take, I can withdraw him/her from the study at any time.
- Doing the study will not affect any other tests or treatments that the individual under your legal care might have at the Bruyère Continuing Care Center.
- Participation in this study may not benefit the individual under my legal care. However, our results may benefit other individuals with early stage dementia.
- Should you decide to withdraw from the study, you will be asked for your consent to retain all or parts of your data that has been collected.

Money Matters

- Dr. Gagnon will pay the costs of getting to and from the Bruyère Continuing Care Center for this study. Parking fees will be paid for. The reimbursement will be provided even if I decide to withdraw the individual under my legal care from the study (up to a maximum of \$85).
- Dr. Gagnon is getting support from the University of Ottawa Research Office and Faculty of Social Sciences RIND to pay for the costs of this study.
- The individual under my legal care should not get hurt by taking part in this study. However, if any harm does come to the individual under my legal care during the study, Dr. Gagnon will see that he/she get proper treatment.

Privacy

Dr. Gagnon and Ms Yamin will protect the individual under my legal care's identity. All information will be stored at the Memory Disorders Clinic at the Bruyère Continuing Care Health Center.

- They will keep paper records in a locked file cabinet. They will keep electronic data on a secure computer protected by a password or on disks in the locked file cabinet.
- They will use code numbers in place of my name on all records.
- No one other than Dr. Gagnon and Ms Yamin will see the individual under my legal care's study records.
- Dr. Frank will store study records for at least 15 years. After that, they will be destroyed.
- If they publish results of this study, they will not reveal the individual under my legal care's identity. Only group averages will be presented.

Continuing consent

During this study, Dr. Gagnon will tell me about any new information that might affect my decision to allow the individual under my legal care to stay in the study.

Questions?

If I have any questions later or if I want a copy of the study results, I can contact Ms. Yamin or Dr. Gagnon.

This study has been read and accepted by the SCO Health Service Research Ethics Board. If you have any comments or questions after your participation in this study, please contact the Chair of the Research Ethics Board at the Bruyère Continuing Care Center, Dr. Lisa Sweet.

Consent Form

I, _____, agree that the individual under my legal care may take part in this research study being done by Dr. Sylvain Gagnon and his graduate student Stephanie Yamin.

I have read the information sheet that gives all the details about this study.

I have had enough time to ask questions I had about it.

I would like a copy of the study's finding once the study has been completed? Y / N

Signature

Signing this form gives us your consent to allow the individual under your legal care to participate in this study. It tells us that you understand the information about the research study. When you sign this form, you do not give up your legal rights. Researchers or agencies involved in this research study still have their legal and professional responsibilities.

Signed,

Participants' Legal Co-signer

Date

Researcher or delegate

Date

Appendix Q Demerit-Point Assessment

Manitoba
Highways and
Transportation
Driver and
Vehicle Licensing



Road Test in Traffic Class 4, 5 & 6

IMPORTANT PLEASE READ

On this form you find results of your road test in traffic. Please note the items checked off require improvement and you should practice them to improve yourself as a driver. The demerit marks assessed are for the department's use in assessing a driver's qualifications and the total marks may or may not have a bearing on whether you passed or failed the road test.

Note: Should you require any further explanation or comments regarding this road test, you must produce this form.

I. STARTING	DEDUCT	DEDUCTIONS
A. Fails to check traffic	5	☐ ☐ ☐ ☐ ☐
B. Fails to signal	5	☐ ☐ ☐ ☐ ☐
C. Fast or uneven get-away	5	☐ ☐ ☐ ☐ ☐
D. Rolls when on grade	5	☐ ☐ ☐ ☐ ☐
E. Starts before light turns green	5	☐ ☐ ☐ ☐ ☐

II. STOPPING	DEDUCT	DEDUCTIONS
A. Stops no reason	5	☐ ☐ ☐ ☐ ☐
B. Stops too suddenly	5	☐ ☐ ☐ ☐ ☐
C. Over-running cross walk	5	☐ ☐ ☐ ☐ ☐
D. Not at safe place	5	☐ ☐ ☐ ☐ ☐
E. Fails to stop entering roadway	5	☐ ☐ ☐ ☐ ☐

III. SIGNAL VIOLATIONS	DEDUCT	DEDUCTIONS
A. Thru on red	10	☐ ☐ ☐ ☐ ☐
B. Thru on red (enters amber)	10	☐ ☐ ☐ ☐ ☐
C. Thru on red (right turn)	10	☐ ☐ ☐ ☐ ☐
D. Does not stop	10	☐ ☐ ☐ ☐ ☐
E. Leaves when not safe	10	☐ ☐ ☐ ☐ ☐

IV. VEHICLES MOVING ON ROADWAY	DEDUCT	DEDUCTIONS
A. Straddles traffic lane	5	☐ ☐ ☐ ☐ ☐
B. Follows too closely	5	☐ ☐ ☐ ☐ ☐
C. Fails to check changing lanes	5	☐ ☐ ☐ ☐ ☐
D. Fails to signal	5	☐ ☐ ☐ ☐ ☐
E. Cuts off vehicle	10	☐ ☐ ☐ ☐ ☐
F. Drives on wrong side of street	5	☐ ☐ ☐ ☐ ☐
G. Wanders	5	☐ ☐ ☐ ☐ ☐
H. Crosses solid line	5	☐ ☐ ☐ ☐ ☐
I. Uncertain gear shifting	5	☐ ☐ ☐ ☐ ☐
J. Fails to drive in proper lane	5	☐ ☐ ☐ ☐ ☐

V. PASSING	DEDUCT	DEDUCTIONS
A. Too close to pedestrians or vehicles	5	☐ ☐ ☐ ☐ ☐
B. Passes where unlawful or unsafe	10	☐ ☐ ☐ ☐ ☐
C. Speeds up when being passed	10	☐ ☐ ☐ ☐ ☐

VI. UNCONTROLLED INTERSECTIONS/RAILWAY CROSSINGS/ YIELD SIGNS/PEDESTRIAN CORRIDORS OR CROSSWALKS	DEDUCT	DEDUCTIONS
A. Fails to slow down or check	5	☐ ☐ ☐ ☐ ☐
B. Fails to yield	10	☐ ☐ ☐ ☐ ☐

VII. SPEED	DEDUCT	DEDUCTIONS
A. Exceeds stated speed limit	10	☐ ☐ ☐ ☐ ☐
B. Too fast for conditions	5	☐ ☐ ☐ ☐ ☐
C. Slows unnecessarily before or after lane change	5	☐ ☐ ☐ ☐ ☐
D. Slows thru intersection	5	☐ ☐ ☐ ☐ ☐
E. Hinders or drives too slowly	5	☐ ☐ ☐ ☐ ☐
F. Drives at uneven rate of speed	5	☐ ☐ ☐ ☐ ☐

Date: _____ Location of Test: _____

Applicant's Signature: _____

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VIII. TURNING	LEFT	RIGHT
A. Improper signal	5 ☐ ☐ ☐ ☐ ☐	5 ☐ ☐ ☐ ☐ ☐
B. Improper approach	5 ☐ ☐ ☐ ☐ ☐	5 ☐ ☐ ☐ ☐ ☐
C. Improper during	5 ☐ ☐ ☐ ☐ ☐	5 ☐ ☐ ☐ ☐ ☐
D. Improper after	5 ☐ ☐ ☐ ☐ ☐	5 ☐ ☐ ☐ ☐ ☐
E. Improper speed	5 ☐ ☐ ☐ ☐ ☐	5 ☐ ☐ ☐ ☐ ☐
F. Slides away	5 ☐ ☐ ☐ ☐ ☐	5 ☐ ☐ ☐ ☐ ☐
G. Strikes/over curb	5 ☐ ☐ ☐ ☐ ☐	5 ☐ ☐ ☐ ☐ ☐
H. Fails to yield	10 ☐ ☐ ☐ ☐ ☐	10 ☐ ☐ ☐ ☐ ☐
I. Fails to establish	5 ☐ ☐ ☐ ☐ ☐	
J. Fails to clear intersection (assisted)	10 ☐ ☐ ☐ ☐ ☐	

IX. BACKING	DEDUCT	DEDUCTIONS
A. Fails to look rear, sides	5	☐ ☐ ☐ ☐ ☐
B. Checks mirror only	5	☐ ☐ ☐ ☐ ☐
C. Backs jerkily or fast	5	☐ ☐ ☐ ☐ ☐
D. Uncertain steering	5	☐ ☐ ☐ ☐ ☐

X. INATTENTIVE	DEDUCT	DEDUCTIONS
A. Leaves signal on	5	☐ ☐ ☐ ☐ ☐
B. Signals through intersection	5	☐ ☐ ☐ ☐ ☐
C. Hesitant	5	☐ ☐ ☐ ☐ ☐
D. Fails to yield to emergency vehicle	10	☐ ☐ ☐ ☐ ☐

XI. CONTROL	DEDUCT	DEDUCTIONS
A. Shifts on turns	5	☐ ☐ ☐ ☐ ☐
B. Starting, driving or turning in improper gear	5	☐ ☐ ☐ ☐ ☐
C. Clutch control	5	☐ ☐ ☐ ☐ ☐
D. Coasts to stop	5	☐ ☐ ☐ ☐ ☐
E. Fails to downshift	5	☐ ☐ ☐ ☐ ☐
F. Steering	5	☐ ☐ ☐ ☐ ☐
G. Fails to use horn properly	5	☐ ☐ ☐ ☐ ☐
H. Uncertain on location of controls	5	☐ ☐ ☐ ☐ ☐
I. Assisted		☐ ☐ ☐ ☐ ☐

XII. SCHOOL BUS CERTIFICATE ONLY	DEDUCT	DEDUCTIONS
A. Fails to stop at railway crossing		☐
B. Fails to look in both directions at a railway crossing		☐
C. Changes gears while crossing railway tracks		☐
D. Open/Close door		☐
E. Activates loading lights on road test		☐

ROAD TEST RESULTS	Reason for failure:
☐ PASS ☐ FAIL	☐ Accident
GRAND TOTAL _____	☐ Assisted
CLASS TEST ☐ 4 ☐ 5 ☐ 6	☐ Dangerous action
	☐ Serious violation
	☐ Graduated reasons
	☐ Test discontinued
	☐ Class 5: backing

Examiner's number: _____ Examiner's signature: _____

"Talking Point No Marks"