The Influence of Depression Symptoms and Antidepressant Medications on Cognition and Driving Performance

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May 2015

A dissertation submitted in partial fulfillment of the requirements of the degree of Doctor of Philosophy in Clinical Psychology ©

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Abstract

Research that has examined the influence of depression symptoms and antidepressant medications on driving performance has revealed inconclusive findings (Brunnauer, Laux, Geiger, Soyka, & Möller, 2006; Bulmash et al., 2006; Ramaekers, 2003). The purpose of the present study was to elucidate the influence of depression symptoms and antidepressant medications on cognition and driving performances using self-report measures as well as an ecologically valid method measure, a driving simulator, and a clinical population. Two hundred and thirty-three drivers ranging in age from 18 to 35 years ($M = 21.88$; $SD = 3.90$ years) completed a screening measure that examined depressive and anxious symptoms, medication use, and self-reported driving behaviour on the Driving Behaviour Questionnaire (DBQ). Forty-three participants ranging in age from 18 to 35 ($M = 24.24$; $SD = 5.05$ years) also attended a laboratory session and completed a series of questionnaires designed to measure depression, driving habits, cognitive psychomotor functioning, and a diagnostic measure of MDD, two computerized tasks (one to measure attention and one to assess processing speed), and a 45 min simulated drive. In the overall sample, twenty-four (10.2%) participants were taking at least one antidepressant. Mean scores for depressive symptoms ($M = 11.09$; $SD = 9.87$) fell in the minimal range on the Beck Depression Inventory-II (BDI-II). A shortened version of the DBQ was created using this younger Canadian sample and correlation coefficients between the short and long version were excellent, ranging from .91 to .94. Overall, depressive symptoms and antidepressant use displayed little relationship to self-reported driving behaviour or driving performance on the driving simulator. However, our results do suggest that age ($B = .12$) and the cognitive/affective ($B = .12$) impairments on the BDI-II are statistically significantly related to increased self-reported absent-minded driving behaviour ($p = .03$). Overall depressive symptoms

(B = -2.48) and cognitive/affective (B = 3.45) impairments were also related to inattention on a computerized task measuring attention ($p < .05$). The cognitive and affective impairments in depression were also positively related to visual perceptual ability (B = 2.02). The overall patterns of self-report data, neuropsychological data, and behavioural data suggest that although there is some consistency between self-report measures and neuropsychological data, this does not necessarily mean these impairments in attention translate into actual driving impairments on the simulator. Future studies could conduct a similar study using on-road performance as the behavioural measure of driving performance.

Acknowledgments

I am overwhelmed with a sense of gratitude for those who have been instrumental in helping me accomplish my greatest life achievement to date.

To my supervisor, Dr. Michel Bédard, thank you for your expertise, devotion, and commitment to excellence. It was not only these qualities that made you a great supervisor, but also your ability to challenge and encourage me to think critically. I never felt judged by you even when I did not know all the answers. I appreciate your willingness to treat me as an equal and your continuous support of my efforts to meet deadlines throughout this process. I have a deep respect for you and truly appreciate the opportunity to have worked with you.

A special thank you to my co-supervisor, Dr. Mazmanian, and second reader, Dr. Oinonen. I appreciate your valuable feedback and interest in my area of research. I would also like to specially thank Bruce Weaver for frequently meeting with me to discuss analyses and for helping me to think critically about my data. I appreciate all of your knowledgeable input. I will never underestimate the value of syntax! Thanks to my research assistant, Nathan Smith, for the long hours that you put in to help me collect data. Also, I appreciate that you made my research a priority in your busy schedule.

To my parents, the gratitude that I feel for your unconditional, unrelenting support is indescribable. If there are two people in this world who have cheered me on as I ran the marathon of my dreams, it was you. Through thick and thin, you encouraged and praised my commitment, and were evidently proud of my accomplishments. It is truly an honour and life achievement to have made you proud. I hope you know without such support my dreams would have been nearly impossible.

A special thank you to my grandparents who were a great source of emotional support. I know this accomplishment would make you so proud. I will always treasure how you both supported me over the years in university and in life.

To my friends, family, and boyfriend, you have been instrumental in helping me keep perspective in life and focus on this important document. For this, I am grateful. Cheers to the upcoming memories post graduate school!

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The Influence of Depression Symptoms and Antidepressant Medications on Cognition and Driving Performance

Major Depressive Disorder (MDD) is a common mental disorder with both affective and cognitive impairments, including low mood, cognitive decline, and psychomotor retardation (Austin, Mitchell, & Goodwin, 2001; Hammar, Lund, & Hugdahl, 2003). Such impairments have the potential to interfere with an important daily function essential for functional autonomy, driving a vehicle (Brunnauer, Laux, & Zwick, 2009). Not only do mental disorders pose an increased risk for crashes because of the pathology itself (Brunnauer et al., 2009), but the pharmacological treatments that are prescribed to treat psychiatric disorders may also pose a threat to driving performance because of potential adverse side effects (Brunnauer, Laux, Geiger, Soyka, & Möller, 2006). Presently, there is a paucity of research on the influence of both MDD and antidepressant medications on driving performance. The little research that does exist reveals mixed results (Wingen, Ramaekers, & Schmitt, 2006). Therefore, it is essential to elucidate the influence of depression symptoms and antidepressant medications on driving performance.

Major Depressive Disorder

MDD is a common mental disorder that is delineated by affective, cognitive, and physiological symptoms. The *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5; American Psychiatric Association [APA], 2013) uses a non-axial categorical classification that recognises a dimensional approach to diagnosing mental disorders. The characteristics of a Major Depressive Episode include both cognitive, affective, and physiological components, including five or more of the following symptoms over the same two-week period: depressed mood most of the day, anhedonia, significant weight loss or gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy,

feelings of worthlessness or excessive guilt, inability to concentrate, and recurrent suicidal ideation (APA, 2000). The consequences of recurrent suicidal ideation can be dire as MDD is associated with high mortality, particularly for those who are male, single, live alone, or have prominent feelings of hopelessness (APA, 2013). In addition, at least one of these five symptoms must be either depressed mood or anhedonia. Finally, to meet diagnostic criteria, symptoms also have to cause significant impairment in functioning in social and/or occupational functioning. A MDD diagnosis is specified as mild (symptoms are distressing but manageable), moderate (intensity of symptoms are between mild and severe), and severe (intensity of symptoms is seriously distressing). Although symptoms can vary in severity, MDD is considered a severe psychiatric disorder (Ravnkilde et al., 2002).

Major Depressive Disorder is a prevalent mood disorder and a debilitating disorder. The World Health Organization (WHO) reports that MDD will be the second cause of disability by 2020 (Murray & Lopez, 1996). In terms of prevalence, those aged 18 to 29 have threefold higher rates of a diagnosis of MDD compared to those aged 60 and older (APA, 2013). Moreover, the APA reports that beginning in adolescence, females have 1.5 to 3-fold higher rates than males (APA, 2013). The APA also reports that the course for MDD is variable, ranging from individuals who do not experience symptoms for many years in between episodes to those who rarely experience remission. The risk of recurrence is higher in individuals who are young, experience a severe episode, and have had a previous episode (APA, 2013).

Price, Mcleod, Gleich, and Hand (2006) found that prevalence rates were higher in younger populations including university students. These researchers examined the prevalence of MDD in a first-year Canadian university sample across disciplines using a diagnostic interview. They discovered that 14% of women and 7% of men met the criteria for MDD. In addition,

Eisenberg, Gollust, Golberstein, and Hefner (2007) used the Patient Health Questionnaire-9 (PHQ-9) to assess depression and anxiety in undergraduate and graduate students. A depressive or anxiety disorder was found in 15.6% of undergraduates (n = 1,181) and 13% of graduate students (n = 166). Tomoda, Mori, Kimura, Takahashi, and Kitamura (2000) investigated MDD prevalence using a structured interview in a first-year university Introductory Psychology sample in Japan. Ten percent of males and 28% of females met criteria for MDD. Some research also shows that depression is more prevalent across different ethnicities. Young, Fang, and Zisook (2010) found that Asian-Americans and Korean-American undergraduates across psychology, biology, and medicine programs had significantly higher levels of depression compared to Caucasian undergraduates as indicated by self-report scores on the PHQ-9. Therefore, MDD can be considered a highly prevalent mood disorder among university populations.

Not only has research shown that MDD is a prevalent mood disorder, research also shows that MDD is highly comorbid with anxiety disorders such as Panic Disorder and Obsessive Compulsive Disorder (APA, 2013; Watson, 2005). Such comorbidity is considered a "...pervasive problem throughout the DSM" (Watson, 2005, p. 525). There is some contention in the literature surrounding anxiety syndromes and MDD being distinct clinical disorders (Gorman, 1996; Watson, 2005). Stulz and Crits-Christoph (2010) offer four possible explanations that may account for the comorbidity between MDD and anxiety disorders. Firstly, anxiety and depressive disorders could be distinct disorders with high comorbidity. Secondly, anxiety and depression could be distinct disorders but share symptoms of negative affect. Thirdly, anxiety and depression may be distinct disorders but the current measures of anxiety and depression do not demonstrate enough sensitivity to distinguish the constructs. Lastly, anxiety and depression may exist on a continuum. Furthermore, given that there is a great deal of overlap

between anxiety disorders and MDD, Watson (2005) argued that mood and anxiety disorders should be collapsed together into a class of emotional disorders. In addition, the Beck Depression Inventory – II and the Beck Anxiety Inventory were designed to discriminate between depression and anxiety. However, because of the comorbidity between these two constructs, these measures still overlap (r = .66; Beck, Steer, Ball, & Ranieri, 1996). Therefore, MDD and anxiety disorders are highly likely to co-occur and perhaps not always be distinct disorders.

It is also well-documented in the literature that MDD is associated with impairment in cognitive functioning (Ravnkilde et al., 2002). Neuropsychological studies have examined cognitive functioning among individuals with MDD. The cognitive components of MDD can be objectively and reliably assessed using standardized tests (Brébion, Smith, & Widlocher, 1997). Therefore, neuropsychological studies have examined the cognitive components of MDD and discovered disturbances of executive functions (Fossati, Amar, Raoux, Ergis, & Allilaire, 1999; Hill, Keshawan, Thase, & Sweeny, 2004), attentional deficits (Egeland et al., 2003), dysfunction in psychomotor skills (Brebion et al.; Hill et al., 2004), and memory deficits (Fossati et al., 1999). Researchers have found it difficult to ascertain to what extent these deficits are caused by true cognitive deficits or a lack of motivation in individuals with MDD (Ravnkilde et al., 2002).

Executive dysfunction is proposed to be a consequence of MDD in young adults (Castaneda, Tuulio-Hennriksson, Morttunen, Suvisaari, & Lönnqvist, 2008). Executive functions are the regulation of cognitive processes such as response inhibition, verbal fluency, nonverbal fluency, language comprehension, working memory, and planning (Kolb & Whishaw, 2009). For example, Egeland et al. (2003) compared individuals with MDD (n = 50) to controls (n = 50) on a measure of executive function, the Stoop Color Word Interference Test (SCWIT). Scores on the SCWIT are based on the number of seconds to name and read 48 coloured dots and words

that are not denoted by the name of the colour. Results revealed that individuals with MDD performed significantly worse across the SCWIT subtests including word (M = 19.42; SD = 5.57), colour (M = 29.88; SD = 6.42), and colour-word (M = 53.54; SD = 13.45). In contrast, healthy controls were quicker to respond on the SCWIT subtests such as word (M = 16.54; SD = 3.11), colour (M = 25.54; SD = 5.05), and colour-word (M =43.72; SD = 9.56). One potential limitation of this study was that most (n = 46) of the participants were taking psychotropic medications which makes it difficult to ascertain whether the depression or medications interfered with executive functions. However, further analyses revealed that there were no significant differences in test performance between individuals taking psychotropic medications and participants who were not taking medications. The implication is that MDD may interfere with executive functioning.

Other evidence suggests that executive dysfunction among individuals with MDD is mixed and complicated by medication prescriptions. Fossati et al. (1999) compared individuals diagnosed with MDD (n = 20) upon admission to a hospital and healthy controls (n = 20). Individuals diagnosed with MDD were treated with antipsychotic medications. Participants with an MDD diagnosis performed more poorly on tests of executive function. For example, MDD participants produced significantly fewer words on the Verbal Fluency Semantic subtest (M = 26.15; SD = 7.6) compared to healthy controls (M = 35.2; SD = 8.8). In addition, participants with MDD performed significantly worse on the Wechsler Adult Intelligence Scale – III Digit Span Forward (M = 6.05; SD = 1.3) and Backward (M = 4.65; SD = 1.1) subtests compared to healthy controls (M = 7.35; SD = 1.2) and (M = 5.95; SD = 1.1), respectively. Participants with MDD also performed significantly worse on the Visuo-Spatial Backward subtest (M = 4.63; SD = 1.2) compared to controls (M = 6.43; SD = 1.0). Lastly, MDD

participants produced significantly fewer attempted card sorts (M=17.25; SD = 3.7) and expected card sorts (M = 14.15; SD = 2.9) on the Delis Spontaneous Card Sorting Test compared to controls (M = 21.25; SD = 3.8) and (M = 16.85; SD = 2.8), respectively. However, participants with MDD did not show any deficits on the Wisconsin Card Sorting Task. Fossati and colleagues suggest that these findings are indicative of impairments in executive functions; namely, deficits in initiation ability, concept formation, and cognitive flexibility. One limitation of this study was that participants with MDD were using psychotropic medications. For example, when benzodiazepines were co-varied out of the analyses, differences in Verbal Span and the Visuo-Spatial subtests Span were no longer statistically significant suggesting that medications may be impairing executive functions rather than MDD itself. These findings suggest that executive dysfunction in individuals with MDD may be a consequence of medication.

It is unclear if MDD, antidepressant medications, or both contribute to executive dysfunction in individuals with MDD. Hill et al. (2004) examined executive functioning in individuals with MDD with and without psychosis who had not taken antidepressant medications for more than six weeks on average. Hill et al. calculated a global executive function z-score which included the Wisconsin Card Sorting Test, the Stroop Color and Word Test, the Trail Making Test B, and the Controlled Oral Word Association Test to examine any impairment in executive functioning. Results revealed that compared to controls, individuals with psychotic (M = -0.75; SD = 1.18) and non-psychotic (M = -0.38; SD = 0.80) MDD performed significantly worse on measures of executive functioning compared to controls (M = -0.04; SD = 0.72). These findings suggest that executive dysfunction may be a consequence of MDD rather than medications. It is also possible that executive dysfunction is a consequence of psychoses. In contrast, Ravnkilde et al. (2002) examined 40 severely depressed inpatients and discovered no

dysfunctions in executive functioning compared to controls. However, a major limitation of this study was that most patients were medicated which again makes these findings difficult to interpret. Additional research would be useful to ascertain whether MDD itself and/or antidepressant medications are the cause of such dysfunction.

Some researchers have also investigated whether executive dysfunction exists in individuals with MDD in remission. For example, Smith, Muir, and Blackwood (2006) discovered significant impairments on a measure of executive functioning, the Trail Making Test (TMT), in young adults ($n = 42$) whose MDD was in remission for at least one month compared to healthy controls ($n = 30$). The TMT assesses task switching and visual attention. These researchers reported that individuals whose MDD was in remission demonstrated differences on the TMT A ($M = 29.6$s; $SD = 7.83$s) and TMT B ($M = 55.9$s; $SD = 15.13$s) compared to controls who also completed the TMT A ($M = 23.0$s; $SD = 4.83$s) and TMT B ($M = 45.3$s; $SD = 10.88$s). However, the scores for both individuals whose MDD was in remission and controls all fell within the average range. Moreover, Wang et al. (2006) found no significant differences in verbal learning on the California Verbal Learning Test in individuals whose MDD was in remission ($n = 42$) compared to healthy controls ($n = 46$) and individuals who were currently depressed ($n = 57$). One explanation for these null findings may be that the participants with MDD were outpatients with mild to moderate symptoms (Castaneda et al., 2008; Wang et al., 2006). Depressive severity may be related to executive dysfunction with greater impairment seen in more severe depressives (Castaneda et al., 2008). These studies suggest that results are also mixed for executive dysfunction in individuals whose MDD is in remission.

Individuals with MDD often have difficulty with attention and difficulty focusing on several ongoing activities at one time (Ravnkilde et al., 2002). Egeland et al. (2003) found that

individuals diagnosed with MDD performed significantly worse as measured in milliseconds compared to controls on a measure of attention, the California Computerized Assessment Package (CalCAP). Egeland et al. (2003) calculated z-scores for the subtests of the CalCAP. Participants with MDD performed significantly worse on basal speed ($M = 0.86$; $SD = 1.10$) and speeded attention ($M = 0.84$; $SD = 1.10$) compared to controls ($M = 0$; $SD = 0.72$) and ($M = 0$; $SD = 0.76$), respectively. These scores were composite scores based on mean control derived z-scores. Participants with MDD did not perform significantly worse on the vigilance subtest ($M = -0.23$; $SD = 1.14$) compared to controls ($M = 0$; $SD = 1$). Therefore, evidence suggests that individuals with MDD are slow in speeded attention (Egeland et al., 2003). Furthermore, Mahurin et al. (2006) found that individuals with MDD performed slower on the TMT compared to controls. The TMT is used as both a measure of executive functioning and attention (Mitrushina, Boone, & D'Elia, 1999). Disturbances of attention appear to be a central problem for individuals with MDD.

Slowed psychomotor speed is also considered a cardinal feature of MDD by the American Psychiatric Association (APA, 2000). Psychomotor speed can be assessed by examining reaction time, speech rate, and/or motor/mental speed (Taylor et al., 2006). Nelson and Charney (1980) reported that up to 69% of individuals with MDD display symptoms of psychomotor retardation. Moreover, Brebion et al. (1997) found a significant negative relationship ($r = -.46$) between severity of psychomotor retardation and response bias on a verbal recognition memory test in 26 outpatients diagnosed with MDD. However, one limitation of this study was that 20 of the participants were taking a benzodiazepine, an antidepressant, or both, which may have influenced performance on this test. Taylor et al. (2006) also investigated psychomotor functioning in individuals who were depressed and using fluoxetine which is a

psychotropic medication that alleviates depression. Taylor et al. (2006) discovered that individuals who responded positively to fluoxetine verbalized significantly more words (M = 49.84; SD = 8.70) on the Controlled Oral Word Association Test compared to individuals who had no positive treatment response to fluoxetine (M = 38.75; SD = 4.88; η^2=1.44). Therefore, psychomotor impairments may be more evident in individuals with depression who are unresponsive to certain psychotropic medications such as fluoxetine. Taylor et al. (2006) postulated that this may reflect a dopaminergic deficit in some individuals with depression. Therefore, some evidence suggests that psychomotor impairment is associated with MDD.

Major Depressive Disorder (MDD) is associated with cognitive deficits and this may be a consequence of motivational difficulties that are characteristic of MDD. To investigate the relationship between motivation and cognitive deficits, some researchers have investigated "response bias" which is considered a cognitive-behavioural paradigm of motivation (Austin et al., 2001) For example, Elliott, Sahakian, Herrod, Robbins and Paykel (1997) found that individuals with depression show a heightened response bias to negative feedback. Response bias was measured on the Delayed Matching to Sample Test and on the Tower of London Test. These tests require participants to solve problems while simultaneously giving participants feedback on whether or not a problem was solved correctly. Individuals with depression displayed a response bias on these measures. More specifically, individuals with depression solved more problems incorrectly (69 to 79% correct) after negative feedback compared to controls (90% correct). However, individuals with depression were taking antidepressant medications. Therefore, it is unclear whether motivational difficulties or antidepressant medications impact cognitive functioning.

Hartlage, Alloy, Vázquez, and Dykman (1993) have also proposed a "cognitive effort hypothesis" which suggests that the cognitive deficits that are associated with depression are contingent on the difficulty of the task that the depressed individual is performing. The more cognitive effort the task demands, the more cognitive dysfunction a depressed individual will experience. Thus, highly demanding tasks will have a detrimental impact on depressed individuals. Other factors have been proposed that may account for cognitive deficits in individuals with MDD including: desire to please, fatigue, psychomotor retardation, anxious inhibition, and monitoring of performance (Brébion et al., 1997). There are many uncontrolled variables, such as poor motivation, that may contribute to cognitive dysfunction in individuals with MDD.

Antidepressant Medications

Given that individuals with MDD experience both affective and cognitive deficits, it is crucial to consider effective treatments to help alleviate these symptoms. Research has demonstrated that non-pharmacological treatments are just as effective as pharmacological treatments for treating some forms depression. More specifically, cognitive-behavioural therapy (CBT), problem-solving therapy, and interpersonal therapy are effective treatments for depression (Gloaguen, Cottraux, Cucherat, & Blackburn, 1998; Mynors-Walis, Gath, Lloyd-Thomas, & Tomlinson, 1995). However, one serious drawback of these therapies is that they are difficult to access and expensive in the short-term (Boyce & Judd, 1999). The APA states in the Practice Guidelines for the Treatment of Patients with MDD that pharmacological treatments are an integral part of treatment, particularly for individuals who have moderate to severe symptoms (APA, 2000).

Psychotropic medications, are chemicals that alter mood and/or behaviour by acting on the central nervous system (Julien, 2004). Psychotropic medications are widely used to treat psychiatric disorders. In Canada, 7.2% of the general population is taking at least one type of psychotropic medication (Beck et al., 2005a). Psychotropic medications are considered a general group of medications some of which can include: benzodiazepines, antidepressants, mood stabilizers, antipsychotics, opiates, cholinesterase inhibitors, and anticholinergic medications (Goodwin & Jamison, 2007). Therefore, antidepressants are a subclass of psychotropic medications and include tricyclic antidepressants (TCAs), monoamine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs), and selective norepinephrine reuptake inhibitors (SNRIs; Julien, 2004). Antidepressant medications are effective in treating depressive symptomatology (Dupuy, Ostacher, Huffman, Perlis, & Nierenberg, 2011). However, antidepressant medications have been shown to induce many side effects including impairment in cognition, attention, and motor functioning (Ramaekers, 2003).

First Generation Antidepressants.

There are two classes of antidepressants that were introduced over 40 years ago: TCAs and MAOIs. The first TCA, imipramine, was accidently discovered by Swiss scientists who thought this drug would be effective in treating schizophrenia. However, it turned out that imipramine lifted depressive symptoms in individuals with MDD instead (Lopez-Munoz, Francisco, Alamo, & Cecilio, 2009). Around the same time that imipramine was discovered, MAOIs were also discovered to be effective in alleviating depressive symptoms. TCAs are considered effective because they block the presynaptic transporter protein receptors at many transmitter receptor sites including norepinephrine and serotonin (Julien, 2004). Therefore, first

generation antidepressants increase levels of monoamine transmitters such as norepinephrine and serotonin, which in turn alleviate depressive symptoms (Delgado, 2004; Julien, 2004).

TCAs are considered the standard to which all other types of antidepressants are compared (Julien, 2004). The role of TCAs is to inhibit serotonin and noradrenaline reuptake which is important to achieve antidepressant effects (van den Broeck et al., 2009). In addition, TCAs also block postsynaptic histamine and acetylcholine receptors which can result in some adverse side effects including confusion, memory and cognitive impairments, blurred vision, and increased heart rate (Julien, 2004; Podweils & Lyketsos, 2002). In addition, impairment in motor skills has been documented (Oxman, 1996). Despite these side effects, evidence suggests that TCAs are effective at alleviating depression (Boyce & Judd, 1999).

Tricyclic antidepressants have been shown to be clinically effective for depressed patients. Nelson et al. (1999) discovered that administration of the TCA nortriptyline to a sample of 81 depressed patients was associated with a 50% improvement on the Hamilton Depression Rating Scale. In addition, Furukawa, McGuire, and Barbui (2002) conducted a large meta-analysis examining the effectiveness of low-dose (75 and 100mg/day) TCAs compared to placebo among individuals with depression. Furukawa et al. analyzed the findings of 35 studies and discovered that individuals taking a low-dose TCA of 75mg/day (OR = 1.65; 95% CI [1.36, 2.0]) and 100mg/day (OR = 1.47; 95% CI [1.12,1.94]) were at increased odds of experiencing less depressive symptoms within 8 weeks compared to placebo. Furukawa et al. conclude that treatment with a low-dose of TCAs for individuals with MDD is justified.

MAOIs were first administered in the 1950s (Julien, 2004). There are two types of monoamine oxidase (MAO) enzymes that break down neurotransmitters such as norepinephrine, dopamine, and serotonin. The first type, MAO-A is located in serotonin and norepinephrine

terminals and inhibition of MAO-A creates an antidepressant effect. The second type, MAO-B, is found in dopamine neurons and inhibition of this enzyme induces side effects including significant cardiovascular side effects with interactions with certain foods such as cold medicines, cheeses, and wines (Julien, 2004). The side effects can be severe and cause orthostatic hypotension, hypertensive crises, and in some cases can be fatal (Lofufo-Neto, Trivedi, & Thase, 1999). However, in 2003, selegiline (Eldapril), a transdermal skin patch was put on the market, which avoided the dangerous food-drug interactions that occurred with oral administration. Compared to placebo ($M = 21.26$; $SD = 9.37$), selegiline has shown to significantly lower depressive symptoms in individuals with MDD ($M = 18.67$; $SD = 9.41$) after 8 weeks of treatment (Amsterdam, 2003). These scores reflect symptoms on the Hamilton Rating Scale for Depression (HAM-D-28). A score above 20 suggests MDD. The only significant side effect that was reported was skin irritation (Amsterdam, 2003). MAOIs are used for treating anxiety, hypochondriasis, anorexia nervosa, bulimia, depressed episodes in bipolar disorder, dysthymia, depression in the elderly, and panic disorder (Julien, 2004). Although MAOIs have significant side-effects, these drugs are useful for treating MDD. Given the significant side effects of TCAs and MAOIs, in the late 1970s scientists sought to create drugs that would alleviate depressive symptoms without severe side-effects (Julien, 2004). Scientists were partially successful in their efforts; novel antidepressants were created.

Second-Generation Antidepressants.

One subclass of second-generation antidepressants is SSRIs, which are considered a first choice treatment for MDD (Anderson, 2000) and are also commonly used to treat anxiety disorders and MDD (Gorman, 2002; Ressler & Nemeroff, 2000). Serotonin is one neurotransmitter that is implicated in individuals with depression (Meyer & Quenzer, 2005).

Therefore, SSRIs help alleviate depression, at least in part, by inhibiting the reuptake of serotonin into the presynaptic cell. Serotonin is then available to bind with the postsynaptic receptor because it collects in the synaptic cleft. The six main SSRIs include: citalopram (Celexa), escitalopram (Lexapro), fluvoxamine (Luvox), paroxetine (Paxil), fluoxetine (Prozac), and sertraline (Zoloft) (Julien, 2004). In the United States between 1993 and 1995, SSRI prescriptions were on the rise (Donoghue et al., 1996). During this time, SSRI prescriptions increased by 133% compared to TCAs at 12% (Donoghue et al., 1996). In addition, in an Italian study, antidepressant consumption from 2006-2011 was found to have increased by 5% (Poluzzi et al., 2013). Atypical antidepressants such as SSRIs are commonly prescribed to treat depression and anxiety.

The efficacy of SSRIs has been compared to TCAs. MacGillivary et al. (2003) conducted an in-depth meta-analysis and examined 11 studies using samples of individuals diagnosed with MDD from 10 different countries to investigate whether SSRIs were more efficacious than TCAs. Results revealed SSRIs were no more efficacious than TCAs. However, MacGillvary et al. (2003) also discovered that SSRIs are associated with lower dropout rates, or withdrawals from treatment, compared to TCAs. MacGillvary et al. (2003) postulated that this association may be related to the milder side effects of SSRIs compared to TCAs. For instance, Wilson and Mottram (2004) conducted a meta-analysis comparing the side effects of SSRIs and TCAs in older depressed patients. Results revealed an increased withdrawal rate for classical TCAs compared with SSRIs (RR = 1.30; CI 95% [1.02, 1.64]). Common side effects of TCAs included dry mouth, drowsiness, dizziness, and lethargy. However, Wilson and Mottram reported some limitations of their study. First, there was a lack of standardization in reporting side effects among the studies examined. Second, participants were recruited from the community and

therefore these findings may not be generalizable to inpatient settings. Third, there may be a publication bias with studies that fail to find efficacy or tolerability for SSRIs compared to TCAs not being published. Despite these limitations, the literature suggests that SSRIs are considered effective for the treatment of depression and induce fewer side effects than TCAs.

Although SSRIs have different side effects compared to TCAs, SSRIs do induce some side effects including: insomnia, anxiety, agitation, cognitive impairment, and sexual dysfunction (Julien, 2004; Targum, 2000; Wadsworth, Moss, Simpson, & Smith, 2005). Furthermore, hyponatremia, or low sodium concentration in the blood, is considered a potentially dangerous side effect of SSRIs (De Picker, Van Den Eede, Dumont, Mookens, & Sabbe, 2014). In addition, Julien (2004) describes three common side effects: serotonin syndrome, serotonin withdrawal syndrome, and SSRI-induced sexual dysfunction. Serotonin syndrome occurs when SSRIs are consumed with other drugs. In this case, individuals can experience disorientation, agitation or restlessness, changes in autonomic nervous system functioning, changes in neuromuscular functions, and visual hallucinations can occur. Serotonin withdrawal syndrome occurs in 60% of individuals who are long term users of SSRIs. Symptoms include: disequilibria, gastrointestinal upset, fatigue, lethargy, chills, and sensory and sleep disturbances. Lastly, SSRI-induced sexual dysfunction occurs in up to 80% of individuals with depression who are taking an SSRI. As the name suggests, symptoms can include problems with physiological arousal, orgasm, erection, and sexual interest (Julien, 2004; Michelson, Bancroft, Targum, Kim, & Tepner, 2000).

Another class of second-generation antidepressants is Selective Norepinephrine Reuptake Inhibitors (SNRIs), which treat depression and anxiety by blocking the reuptake of norepinephrine and serotonin (Julien, 2004). For example, venlafaxine, an SNRI, is considered efficacious in reducing depressive symptoms. Davidson, Meoni, Haudiquet, Cantillon, and

Hackett (2002) reported that remission rates for venlafaxine were superior to placebo and to an SSRI, fluoxetine. More specifically Davidson and colleagues conducted a pooled analysis across 5 randomized controlled trial studies which examined weekly remission rates up to 6 weeks in individuals diagnosed with Major Depression. Venlafaxine showed a statistically significant increase ($p < .01$) in remission rates of depressive symptoms compared to fluoxetine and placebo at weeks 3 and 6 for patients with severe anxiety symptoms at baseline. In contrast, for individuals taking fluoxetine, a statistically significant increase in remission rates was not seen until week 4. In addition, Baldwin (2006) concluded that three SNRIs, including venlafaxine, milnacipran, and duloxetine were efficacious in alleviating anxiety symptoms that often coincide with depression and certain anxiety disorders. Side effects associated with SNRIs include increased blood pressure and heart rate, sweating, and dry mouth. However, research suggests that one SNRI, reboxetine, is effective in improving attention and cognitive functioning in individuals with depression (Ferguson, Wesnes, & Schwartz, 2003). Therefore, SNRIs have more recently become another treatment choice for individuals with depressive and/or anxious symptoms.

Antidepressant side effects can make complex tasks difficult for individuals taking antidepressant medications. One such complex task includes driving. For example, Brunnauer et al. (2006) conducted a naturalistic nonrandomized clinical study to investigate the effects of antidepressants on psychomotor function and potential driving ability. These researchers investigated visual perception, reaction time, selective attention, vigilance, and stress tolerance in 100 inpatients who met the DSM-IV criteria for MDD and who were taking antidepressant medications. These domains were investigated because German guidelines propose that these components are critical for assessment of ability to drive in Germany. A failure is considered

scoring 1 standard deviation below the mean of normative data in any of these domains (Brunnauer et al., 2006). Results revealed that mild to moderate impairments in psychomotor functions that relate to driving an automobile were present in 60% of patients. Mild to moderate impairments were classified as failing in less than 40% of the test domains. Severe impairments were found in 16% of patients. Severe impairments were classified as failing in more than 40% of the test domains. In addition, patients using SSRIs displayed better test performance compared to those using TCAs. Twenty-eight percent of patients using SSRIs passed the tests without impairments compared to 10% of patients taking TCAs. However, patients who were taking a newer antidepressant, mirtazapine, displayed significantly better global driving ability scores in which 50% passed the tests without impairments. This suggests that mirtazapine may be associated with less severe deficits in psychomotor speed and integration of acoustic and visual stimuli. These results suggest that some antidepressants may interfere with driving performance. However, there is a paucity of research that has attempted to tease apart the effect of MDD and antidepressant medications on driving performance.

More research examining the impact of medications on driving would be valuable considering that between 5% and 25% of drivers are taking psychotropic medications such as benzodiazepines (Kelly, Darke, & Ross, 2004). Furthermore, Beck et al. (2005a) examined The Canadian Community Health Survey: Mental Health and Well-Being (CCHS), a cross-sectional survey conducted by Statistics Canada between May and December 2002 which includes a sample of 36,984 Canadians aged 15 years and older, to look at prevalence rates for psychotropic medications in Canada. Among the general population, 7.2% of Canadians used psychotropic medications (Beck et al., 2005a). Usage was higher for women, the elderly, and SSRIs were the most commonly used. Beck et al. (2005b) examined antidepressant use in Canada using the

CCHS and discovered that 5.8% of Canadians used antidepressant medications. More specifically, among those diagnosed with MDD, over 40% reported using antidepressant medications. Given these prevalence rates and the potential impact that the side effects induced by medications can have on an individual, research examining the impact of antidepressant medications on driving ability is warranted.

Antidepressant Medications and Driving

Although medications can be beneficial to individuals with MDD and ultimately can improve their quality of life, antidepressant medications can also produce a range of side effects. Research suggests that side effects of psychotropic medications are well-documented (Barker, Greenwood, Jackson, & Crowe, 2004; Mishara & Goldberg, 2004). These side effects can include: sedation, lethargy, and impairment of human motor skills such as slower reaction times and reduced alertness (Ramaekers, 2003; Rapoport & Baniña, 2007). Given that these side effects generally affect the central nervous system and thus human motor skills, it is likely that antidepressant medications influence driving ability. Operating a motor vehicle is a complex task that demands attention, alertness, and coordination (Tanida & Poeppel, 2006). Both experimental and epidemiological studies have documented the effects of alcohol on driving ability (Jones & Lacey, 2001; Moskowitz & Robinson, 1988). However, less attention has been paid to the impact of antidepressant medications on driving abilities (Brunnauer et al., 2006).

Although there is a paucity of research dissociating the influence of antidepressant medications and MDD on driving performance, there are some epidemiological and experimental studies that have begun to explore this topic. Epidemiological and experimental studies have examined driving ability associated with antidepressant medications in the real world and using driving simulators or on-road tests, respectively (Rapoport et al., 2009). However, findings are

mixed and many of the studies have a number of limitations. For example, Brunnauer and Laux (2013) conducted a systematic literature review of second-generation antidepressants and driving performance and reported a lack of controlled patient studies. Moreover, there are no data to date examining some SSRIs such as agomelatine, duloxetine, bupropion, and viloxazine. Furthermore, Hetland and Carr (2014) conducted a literature review of study of psychotropic medications, including antidepressants, and driving from 1973 to 2013. These researchers examined the influence of second-generation antidepressants and driving performance and reported "inconsistency in the literature" (p. 500). In addition, Ravera, Ramaekers, de Jong-van den Berg, and de Gier (2012) conducted a literature review of epidemiological and experimental studies examining the influence of SSRIs on driving performance. Results suggest significant inconsistencies across the 15 studies that were selected. Ravera et al. suggest that future research is essential to uncover the relationship between MDD, antidepressant medications, and driving performance.

Epidemiological studies

Researchers have begun to investigate the relationship between MDD, antidepressant medications, and driving outcomes; the results have been mixed. Some evidence suggests that depression and/or antidepressants are associated with increased risk for a crash. For instance, Selzer, Rogers, and Kern (1968) found that 21% of drivers who were responsible for a fatal crash were considered clinically depressed. Furthermore, Ray, Fought, and Decker (1992) conducted a retrospective cohort study and discovered that elderly individuals who were taking TCAs were two times more at risk of being involved in a traffic collision compared to controls. In addition, Leveille et al. (1994) conducted a case-control study examining whether antidepressants and/or benzodiazepines were associated with risk of an injurious crash in older drivers. Results revealed

that antidepressants were associated with increased risk for injurious collisions among older adults (RR = 2.3, 95% CI [1.1, 4.8]). Moreover, Hu, Trumble, Foley, Eberhard, and Wallace (1998) conducted a panel data analysis on crash data from the Iowa 65+ Rural Health Study and found that use of antidepressants significantly increased risk of a crash in men (OR = 2.4, CI not reported). Therefore, some evidence suggests that both MDD and antidepressant medications may impair driving performance.

Comparing first and second-generation antidepressants side effects to driving impairments reveals contradictory results. For example, Walsh, de Gier, Christopherson, and Verstraete (2004) reported in a review paper that "Newer generation antidepressants do not seem to interfere with performance, except when used in higher doses" (p. 246). Furthermore, Walsh et al. (2004) conclude that first generation antidepressants may pose a risk to driving impairment but that new generation antidepressants are not a major problem for traffic safety. However, contrary to the conclusions of Walsh et al. (2004), Barbone et al. (1998) examined both pharmacy and police records to determine the association between antidepressant use and traffic crashes between 1992 to 1995 in the United Kingdom and found no increased risk of a traffic collision in a sample of adults taking TCAs; the OR was 0.93, 95% CI [0.72, 1.21]. In addition, adults using SSRIs were not at increased odds of a traffic collision; OR was 0.85, 95% CI [0.55, 1.33]. Therefore, research findings on the influence of first and second generation antidepressants on driving performance appears mixed.

More recent evidence also suggests mixed results for the influence of older and newer generation antidepressants on driving performance. Rapoport, Zagorski, Seitz, Hermann, Molarn, and Redelmeier (2011) examined crash risk in adults aged 65 and older who were treated with antidepressants. Crash data was obtained from healthcare and transportation databases. Five

percent (n = 7, 393) of the total sample (N = 159,678) of older adults were taking an antidepressant medication one month prior to being involved in a crash. Results were calculated using hazard ratios (HR) and revealed that the greatest risk was the association between at fault crashes and antidepressants (adjusted HR = 1.09, 95% CI [1.05, 1.12]). This finding contrasts Leveille et al. (1994) who did not find significant effects for drivers who were at-fault for a crash (n = 103), regardless if they were taking benzodiazepines or antidepressants. Paradoxically, Rapoport et al. also found that the risk of a crash for participants who were taking a first-generation antidepressant was not significant but there was a significant risk for participants taking second-generation antidepressants (adjusted HR = 1.10, 95% CI [1.07, 1.13]). However, concurrently prescribed medications mediated the relationship between crashes and antidepressants. Participants who were taking an antidepressant and benzodiazepine (adjusted HR = 1.23, 95% CI [1.17, 1.28]) displayed a significant increased risk of a crash. This risk, however, was no longer significant when a concomitant benzodiazepine was not taken (adjusted HR = 1.01, 95% CI [0.98, 1.04]).

Similarly, Rapoport et al. (2008) conducted a case-crossover study examining psychotropic medications and crashes among individuals with dementia. Results revealed that first-generation antidepressants were associated with less risk (OR = 1.31, 95% CI [1.07, 1.61]) than second-generation antidepressants (OR = 2.15, 95% CI [1.78, 2.60]). Likewise, Ravera, van Rein, de Gier, and de Jong-van den Berg (2011) also found an increased risk of being in a crash with exposure to SSRIs, a second-generation antidepressant (OR = 2.03, 95% CI [1.31, 3.14]). In addition, Gibson, Hubbard, Smith, Tatta, Britton, and Fogarty (2009) analyzed a primary care database in the United Kingdom to examine psychotropic medications and crash risk; short term (less than four weeks) use of SSRIs and use of first generation TCAs were not associated with a

significant risk of a crash. However, extended use of SSRIs increased risk (IRR = 1.16, 99% CI [1.06, 1.28]).

Potential explanations for the findings that second generation antidepressants induce more driving impairment than first generation antidepressants include: rationale for prescribing the first-generation antidepressants (i.e., for insomnia rather than depression), more individuals who were prescribed first-generation antidepressants stopped driving because they were incapacitated, and/or the possibility that physicians prescribe newer antidepressants to frailer patients (Rapoport et al., 2011). These studies provide some evidence that suggests that second-generation antidepressants may be associated with more impairment in driving performance. The literature suggests that the connection between first and second generation antidepressant medications and driving impairment does not appear to be clear cut. Therefore, rigorous experimental designs are essential.

Experimental studies

Both driving simulators and on-road tests have been used for the purpose of exploring the influence of medications on driving performance. The published findings in this area are also mixed. The primary performance measures in simulated experiments and on-road tests include the Standard Deviation of Lateral Position (SDLP) and the Standard Deviation of Speed (SDS). SDLP refers to the degree of side-to-side movements of the car when it is in the correct lane and SDS is defined as speed variability (Verster, Volkerts, & Verbaten, 2002).

Driving simulators. Driving simulators have been used as a tool to investigate psychotropic medications and driving performance. Driving simulators have some advantages over on-road tests as they can provide a safer method for evaluating driving performance, offer the opportunity for investigating risky road situations, and provide the opportunity for testing all

participants under the same conditions (Bédard, Parkkari, Weaver, Riendeau, & Dalhquist, 2010). Moreover, Bédard et al. (2010) supported the validity and reproducibility of simulator driving evaluations with several findings; for instance, there is a moderate to strong (*r*s = .44 to .83) relationship between simulator performance and neuropsychological tests that predict crashes. Furthermore, there is a relationship between actual assessment of driving performance and number of errors recorded by a driving simulator (*r* = .74), suggesting that the simulator may be as accurate as a driving evaluator. Additionally, a different evaluator can reproduce the number of demerit points reported when using the play-back function of the driving simulator suggesting that a driving evaluator does not need to be present during the driving simulation further enhancing the ecological validity of driving simulators (ICC = .73-.87). In addition, evidence suggests that simulators are useful in predicting future crash risk. More specifically, Lee and Lee (2005) found that the frequency of the use of the indicator, or signalling to change lanes, is significantly inversely related to the incidence of traffic violations among individuals aged 60 and older, incidence rate ratio = 0.77, 95% CI [0.62, 0.94]. Furthermore, Lee, Lee, Cameron, and Li-Tsang (2003) found that a driving simulator can identify inflated risk of a crash in older adults (OR = 1.13, 95% CI [1.00, 1.27]). Therefore, driving simulators can be considered a useful tool to assess driving performance.

Many studies utilizing driving simulators have examined the impact of medications in healthy controls rather than clinical populations (see Rapoport & Baniña, 2009; Sagberg, 2006). For example, Iwamoto et al. (2008) examined the impact of two antidepressants, 25 mg of amitriptyline and 10 mg of paroxetine on three driving tasks including SDLP, harsh braking, and car following in 17 healthy Japanese male participants. Iwamoto et al. discovered that acute doses of amitriptyline but not paroxetine significantly impaired SDLP and car following on the

driving simulator. More specifically, participants taking 25mg of amitriptyline had significantly more ($M = 51.3$; $SD = 12.67$) SDLP errors compared to those taking paroxetine ($M = 38.9$; $SD = 10.11$). These researchers explained that these findings are not surprising as amitriptyline has antagonistic effects on the cholinergic, adrenergic, and histaminergic receptors. This can cause cognitive impairment, disruptions in balance, and sedation, respectively (Iwamoto et al., 2008). Some limitations of this study include use of healthy participants and acute dosing rather than long-term.

Another study also investigated the impact of benzodiazepines on simulated driving performance in a clinical population. Partinen, Hirvonen, Hublin, Halavaara, and Hiltunen (2003) investigated the impact of 10 mg of zolpidem, 20 mg of temazepam, or placebo in 19 women who were diagnosed with primary insomnia. Findings were akin to Staner et al. (2005); there were no significant differences in driving performance on a driving simulator between participants receiving zolpidem compared to placebo. Moreover, there were no significant differences between temazepam and placebo in driving performance. Partinen et al. reported that two explanations are possible for these null findings. Firstly, there were substantial individual differences in driving performance among the participants which may be responsible for the lack of differentiation between zolpidem and temazepam in this study. Secondly, participants may have been somewhat tolerant to zolpidem and temazepam as some participants may have previously used a benzodiazepine. Another limitation of this study is that the sample only included women. Thus, these findings may not generalize to men. The results of studies examining clinical populations such as participants with insomnia are valuable. However, examining a clinical population, such as participants with MDD, and antidepressant use would

help to elucidate the influence of antidepressants and MDD on driving performance (Rapoport & Baniña, 2007).

The symptoms associated with depression may impair driving ability. Bulmash et al. (2006) used a driving simulator to investigate the driving ability of 18 outpatients who met the DSM-IV-TR criteria for MDD but were free of antidepressant medications and compared these participants to 29 control participants. Compared to controls, participants diagnosed with MDD displayed significantly slower reaction times ($\eta^2 = 0.08$) and increased crashes ($\eta^2 = 0.10$). More specifically, reaction time was slower for participants with MDD ($M = 1.30$ s; $SE = 0.09$ s) and number of crashes was higher ($M = 3.83$ s; $SE = 0.87$ s) compared to controls ($M = 1.04$ s; $SE = 0.07$ s) and ($M = 1.14$ s; $SE = 0.66$ s), respectively. In addition, Brunnauer et al. (2008) conducted a pre-and-post study design and sampled participants who met DSM-IV-TR criteria for Major Depression and randomly assigned participants to receive a selective noradrenergic reuptake inhibitor (NARI), reboxetine ($n = 20$), and a noradrenergic and specific serotonergic antidepressant (NaSSA), mirtazapine ($n = 20$). Results suggest significant improvements in selective attention ($p < .01$), reactivity ($p < .01$), and a significant decrease in accidents on the driving simulator ($p < .05$). Given these findings, it would be reasonable to expect that depressed patients who are on antidepressant medications may have an improved driving performance.

Shen et al. (2009) sampled 28 individuals who met the DSM-IV criteria for MDD and administered 30 mg of mirtazapine, a sedating antidepressant, for 30 days to half of participants. Participants completed a simulated drive on days 2, 9, 16, and 30. Just as Shen et al. (2009) hypothesized, participants who were given mirtazapine displayed greater driving safety as measured by an individual's ability to adjust to lane position while driving. A score of 25 is considered the safest point in the lane. Lane position scores were significantly higher in the

untreated group ($M = 30.2$; $SE = 7.4$) compared to the treated group ($M = 27.2$; $SE = 2.5$) suggesting that the untreated group were less safe. Shen et al. (2009) provide several reasons for this finding, namely, a depressed mood may lead to decreased attention to the consequences of an accident, depressed individuals may have decreased concentration and cognitive functioning, and depressed individuals may have increased anxiety, irritability, sleep disturbances, fatigue which may all be detrimental to driving.

In summary, the results of simulator studies reveal mixed findings for the effect of psychotropic medications on driving performance. While the results of studies using healthy participants suggest that some psychotropic medications impair driving ability, other research using depressed individuals suggests that antidepressants may actually improve performance. Therefore, simulator studies thus far have not established a consistent pattern and cogent explanation for the impact of psychotropic medications on driving.

On-road driving tests. Experimental studies using on-road driving tests also have been utilized to investigate the impact of psychotropic medications on driving ability and psychomotor performance. Many of these studies utilize healthy volunteers to examine psychotropic medications and driving and have found mixed results. For example, Wingen, Bothmer, Langer, and Ramaekers (2005) examined the effect of two antidepressants, escitalopram and mirtazapine, on on-road driving performance. Wingen et al. conducted a 3-way crossover design study and administered the drugs in a 15-day series. Participants were administered 10 mg/day of escitalopram or 30 mg/day of mirtazapine in the evening on days 1 to 7 followed by 20 mg/day of escitalopram or 45 mg/day in the evening on days 8-15, or placebo. Participants engaged in an on-road test and psychomotor tests on the computer on days 2, 9, and 16 as these days were considered the acute period, dose increase, and a steady state, respectively. During the acute

period, participants in the mirtazapine group performed significantly worse on SDLP (M = 21.8 cm; SE = 1.366 cm) compared to placebo (M = 17.9 cm; SE = 0.72 cm). Moreover, participants in the mirtazapine group displayed significantly more errors (M = 19.1; SE = 1.14) on a divided attention task compared to placebo (M = 17.0; SE = 0.96). A tracking error was considered to be the distance between the midpoint of the scale and the position of a cursor (measured in milometers). Mirtazapine did not impact performance during the dose increase or steady state phase. In addition, escitalopram did not impact driving or psychomotor performance in healthy volunteers.

The findings of the Wingen et al. (2005) study are not that surprising considering that mirtazapine has been shown to have more sedative side effects than escitalopram (Aronson & Delgado, 2004; Kasper, Praschak-Rieder, Tauscher, & Wolf, 1997). However, Ramaekers et al. (2011) also found that a 1.5 mg of esmirtazepine did not significantly impact SDLP on an on-road test but a 4.5 mg dose of esmirtazapine produced a rise in SDLP that decreased following repeated doses. Therefore, studies utilizing healthy volunteers have revealed some mixed results.

To obtain a better sense of the effect of antidepressant medications on driving, Ramaekers (2003) conducted a review of the major results of published randomized, double-blind studies from 1983 to 2000 examining the effects of antidepressants on on-road driving performance. They found 9 studies utilizing healthy participants and 1 study utilizing depressed participants. Results revealed that SDLP was significantly elevated compared to placebos and the effect was comparable to drivers with a blood alcohol concentration of 0.8 milligrams per millilitre. Ramaekers also suggested that using healthy volunteers possibly limits the clinical utility of the findings.

A limited amount of research has investigated on-road driving performance among individuals taking antidepressant medications. Wingen, Ramaekers, and Schmitt (2006) examined the effects of long-term antidepressant treatment on driving performance. Participants included 24 depressed patients who received a SSRI including citalopram, sertraline, paroxetine, or venlafaxine for 6-52 weeks and 24 healthy volunteers. All participants completed two standardized on-road driving tests and tests of cognition and attention in the laboratory. Results revealed significantly higher SDLP in depressed medicated participants (M = 20.5 cm; SE = 0.7 cm) compared to healthy controls (M = 18.0 cm; SE = 0.6 cm), suggesting poorer driving performance in depressed medicated participants. There were no significant differences between the different types of antidepressants and no significant group differences in the laboratory tests of cognition and attention and driving. Tests of cognition included: the visual verbal learning task, the change blindness task, the left-right test, the continuous performance test, the critical flicker fusion threshold, and the digit symbol substitution task. Wingen et al. concluded that depressive symptoms may have impacted driving performance rather than the SSRIs. Adding an additional group of depressed patients who are not taking antidepressant medications to this study would have best elucidated this question.

There are many limitations in the current research examining the impact of medications on driving performance. Most studies utilised healthy volunteers. It can be argued that healthy participants may react differently to antidepressant medications compared to depressed patients and thus the findings of studies using healthy participants may not generalise to clinical populations (Ramaekers, 2003). In addition, few studies have investigated the effect of antidepressants using driving simulators and most studies have very small sample sizes

(Rapoport & Banina, 2007). Hence, more research is needed to tease apart the influence of antidepressant medications and depression on driving performance.

Driving Behaviour

Driver behaviour is a key factor in traffic collisions (Elander, West, French, 1993; Kontogiannis, Kossiavelou, & Marmaras, 2002). The Driving Behavior Questionnaire (DBQ; Reason, Manstead, Stradling, Baxter, & Campbell, 1990) was designed to measure conscious aberrant behaviours or human-made causes of traffic collisions. Reason et al. (1990) conducted a factor analysis (N = 520) on the DBQ and found three-factors that accounted for 33% of the variance collectively. These included errors (6.5%), violations (22.6%), and lapses (3.9%). Errors are related to perceptual, attention, and information processing errors. These often include misjudgements when driving (e.g., underestimating the speed of another vehicle; Lajunen & Summala, 2003). In contrast, violations reflect a driver's style and driving habits, and have a motivational component (Özkan, Lajunen, & Summala, 2006b; Reason et al.). Violations can include speeding, running a red light, or tailgating (Lajunen & Summala, 2003). Lapses are similar to errors and include difficulties with memory such as forgetting where the car is parked.

The DBQ has been used in many different countries such as: Australia (Blockey & Hartley, 1995; Lawton, Parker, Manstead, & Stradling, 1997; Dobson et al., 1999), China (Xie & Parker, 2002), Denmark (Martinussen, Hakamies-Blomqvist, Møller, Özkan, & Lajunen, 2013), Greece (Kontogiannis, Kossiavelou, & Marmaras, 2002), The Netherlands (Lajunen, Parker, & Summala, 1999), the United Kingdom (Parker et al., 1995; Lawton, Parker, & Stradling, 1997), United Arab Emirates (Bener, Özkan, & Lajunen, 2008), United States of America (Owsley, McGwin, & McNeal, 2003), Spain (Gras et al., 2006), Sweden (Rimmö 2002), and Turkey

(Özkan & Lajunen, 2005). However, to our knowledge, only one study has been published using a Canadian sample (see Cordazzo, Scialfa, Bubric, & Ross, 2014).

Many versions of the DBQ exist and both the content and number of factors tend to vary across studies. In addition, some versions of the DBQ tend to be very long. DBQ versions have included 9-items (Martinussen, Lajunen, Møller, & Özkan, 2013), 16-items (Lawton, et al., 1997), 24-items (Parker et al., 2000), 36-items (Cordazzo, Scialfa, Bubric, & Ross, 2014), 50-items (Reason et al., 1990), 104-items (Åberg & Rimmö, 1998), and 112-items (Kontogiannis, Kossiavelou, & Marmaras, 2002). The number of items tend to vary as a result of country-specific variations (Özkan , Lajunen, Chliaoutakis, Parker, & Summala, 2006a).

Longer versions of the DBQ have displayed different factorial structures. Longer item questionnaires can also be considered overwhelming for participants and can jeopardize results as long questionnaires can lower completion rates (De Leeuw, Hox, & Dillman, 2008). Kontogiannis, Kossiavelou, and Marmaras (2002) distributed a 112-item version of the DBQ to individuals (n = 1,425) in 18 cities in Greece. Unfortunately, a response rate was not generated in this study. Results revealed a 7-factor solution, with five of the factors representing a distinction between errors and violation as in the original DBQ structure. Furthermore, Åberg and Rimmö (1998) added additional items that captured driver errors to the original DBQ. The new 104-item measure was completed by 1,400 drivers in Sweden, with a response rate of 69%. The original DBQ items confirmed the original three-factor solution.

Shorter versions of the DBQ also apply different factorial structures and are validated across different countries. For example, Özkan and Lajunen (2005) administered a Turkish translation of the original 50-item version DBQ with four additional items to measure aggressive violations to Turkish drivers aged 18 to 67 years. This version included a positive driver scale

and demonstrated a three-factor structure (violations, positive driver behaviours, and errors). In contrast, a 27-item version of the DBQ was administered cross-culturally to drivers in Britain, Finland, and The Netherlands and results supported a four-factor and two-factor solution (Lajunen, Parker, & Summala, 2004). Similarly, Cordazzo, Scialfa, Bubric, and Ross (2014)'s 36-item version of the DBQ, which was adapted for North American drivers, supported a two-factor solution consisting of errors and violations but not lapses. Moreover, Parker, McDonald, Rabbitt, and Sutcliffe (2000) administered a 24-item version of the DBQ to elderly drivers (aged 49 to 90 years) in England. This study had a good response rate (89%) and supported a five-factor solution that preserved the original DBQ three-factor solution.

Although the DBQ has been extensively researched, few studies have examined the applicability of the DBQ across different age groups, particularly with younger drivers. The studies that have examined age groups have found mixed findings for the fit of the DBQ. One recent study conducted by Martinussen and colleagues (2013) investigated the original 50-item DBQ structure across seven age subgroups using both exploratory and confirmatory factor analyses in a Danish sample (N = 11,004). The results of the exploratory analyses supported a distinction between errors/lapses and violations, suggesting a distinction between unintentional and intentional aberrant driving behaviours. The EFA also supported a four-factor solution, which Martinussen et al. labelled confused errors/lapses, unfocused errors/lapses, emotional violations, and reckless violation/lapses. However, the exploratory analyses also revealed that the best fit was with the original DBQ structure, as well as the four-factor structure compared to the two-factor structure for the whole sample. Older drivers (ages 50-80 years) also showed a better fit (CFI = .859; 4-factor) than younger groups (ages 18-29 years; CFI = .804; 4-factor). Contradictory to these findings, Rimmö (2002) conducted a confirmatory factor analysis on data

from four different studies with four subsamples including new drivers (n = 2,248; aged 19 years), inexperienced drivers (n =1,296; aged 21 years), young drivers (n =744; aged 22 to 27 years), and experienced drivers (n = 976; aged 28 to 70 years). Results supported a four-factor solution and a better fit among new drivers ($p < .05$) compared to experienced drivers ($p < .08$). One explanation for these results may be that the age range for experienced drivers was broad which may lead to significant differences between the groups. Furthermore, Cordazzo et al. (2014) conducted a principal components analysis of the 36-item DBQ using an older sample from the Alberta Motor Association (N = 2,839; M = 60.65 years; SD = 13.81). Given that this sample did not include younger participants, Cordazzo et al. included a sample of University students (n = 456; M = 20.95 years; SD = 2.16 years) in the analyses. However, Cordazzo et al. did not compare groups. The only reported finding pertaining to age was that age was negatively related to violations (β = -0.40, $p < .01$).

Given that driver behaviour can contribute to traffic collisions (Elander, West, French, 1993) and that the DBQ measures driver behaviours, one application of the DBQ is to predict crashes. Parker, West, Stradling, and Manstead (1995) examined DBQ scores (N = 1,373) and crash record data, over a 6-year period (1987-1993), from the United Kingdom Driver and Vehicle Licensing Agency. Results revealed that high violation scores were associated with collisions including active loss-of-control collisions and passive right-of-way collisions. These researchers hypothesized that this may suggest that individuals who have high violation scores fail to adjust their speed to specific conditions (Parker et al., 1995). Furthermore, de Winter and Dodou (2010) conducted a meta-analysis of 70 studies and reported that DBQ errors (r = .10) and DBQ violations (r = .13) were associated with self-reported crash involvement. In addition, there was a significant negative relationship between age and the violations-crash correlation

(standardized β = -.48, p = .008) suggesting that younger drivers tend to have increased violation scores. Moreover, Cordazzo et al. (2014) found that violations significantly predicted self-reported collisions (β = .25; $p < .001$). However, the r-squared value (R^2 = .01) indicated that this predictor accounted for less than 1% of variance.

Since the publication of the DBQ in 1990 by Reason et al., there has been extensive research on the structure of this instrument and the applicability of the DBQ across cultures. Less research has been conducted with the DBQ in young Canadian samples.

The Present Study

A principal feature of MDD is that it can induce cognitive and psychomotor disturbance including disturbances in executive functions, restlessness, attentional deficits, and slowed thought processes (APA, 2013; Brebion et al., 1997; Egeland et al., 2003; Hill et al., 2004). In addition, evidence suggests that individuals who are diagnosed with MDD often have motivational deficits (Ravnkilde et al., 2002). Both MDD and the antidepressant medications that are prescribed to treat MDD have the potential to cause cognitive and psychomotor disturbances. Antidepressant medications can impact the central nervous system causing deficits to psychomotor skills such as lethargy, slower reaction times, and reduced alertness (Ramaekers, 2003; Rapoport & Banina, 2007). However, antidepressants may also improve cognition by lifting mood and improving attention and executive functions in the short term (Impey & Baldwin, 2013). Given the cognitive and psychomotor disturbances induced by both MDD and the antidepressant medications that are prescribed to treat MDD, it is important to consider how these disturbances might impact other areas of functioning. One such area is operating a motor vehicle. In 2009, there were 2,011 individuals fatally injured in a motor vehicle collision in Canada (Transport Canada, 2011a). While in 2008, approximately 38% of fatally injured drivers

in Canada tested positive for alcohol consumption (Transport Canada, 2011b), no similar statistics are available for antidepressant medication use and vehicle collisions. Given that driving is a complex task that demands attention, alertness, and coordination (Tanida & Poeppel, 2006) and that psychotropic medication use is prevalent in the general population, more research is needed to tease apart the effects of MDD and antidepressant medications on driving performance.

To date, the literature on this topic is mixed. Both epidemiological and experimental studies report contradictory results with respect to the relationship between MDD, antidepressant medications, and driving performance (see Barbone et al., 1998; Leveille et al., 1994; Wingen et al., 2005). In addition, most of the current research has utilised healthy controls rather than clinical populations which limits the generalizability of these findings (Rapoport & Baniña, 2009). Therefore, the present study seeks to elucidate the influence of MDD and antidepressant medications on driving performances using an ecologically valid method, a driving simulator, and a clinical population. In addition, given that data using a Canadian sample to validate the DBQ is sparse, and that the factor structure of the DBQ has been inconsistent across younger age groups, our objective was to create a shorter version of the DBQ and to examine its psychometric properties in a younger Canadian sample.

Hypotheses

Given that the DBQ has only been validated on one Canadian sample and that the factor structure of the DBQ has been inconsistent across younger age groups, we aimed to explore the psychometric properties of a shorter version of the DBQ. We hypothesized that a shorter version of the DBQ would demonstrate good internal consistency and validity in a young adult Canadian sample. Furthermore, psychomotor disturbance is a cardinal feature of MDD (APA, 2000) and

research suggests that both MDD and antidepressant medications can interfere with attention and cognitive processing (Brebion et al., 1997; Ramaekers, 2003). Therefore, our secondary hypotheses were that participants who are taking antidepressant medications and/or are experiencing depressive symptoms would report more driving impairments on the DBQ and demonstrate deficits on measures of executive functioning (Trail Making Test [TMT]), visual perceptual ability (Motor-Free Visual Perception Test: Third edition [MVPT-3]), attention (Centre for Research on Safe Driving Attention Network Test [CRSD-ANT]), and visual information processing (Useful Field of View [UFOV]) compared to participants with fewer depressive symptoms.

To restate, there were four main hypotheses:

(1) We aimed to create a shortened version of the DBQ in a younger Canadian sample. We expected to observe good to excellent psychometric properties in this shorter version of the DBQ. Good to excellent psychometric properties are defined by Murphy and Davidshofer (2005) and Streiner (2003) to be Cronbach's α of .80 as excellent and Cronbach's α above .90 are considered redundant.

(2) We expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would be associated with higher levels of self-reported unsafe driving behaviour on the subtests of the shortened version of the DBQ.

(3) We expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would also be associated with higher impairments on measures of executive functioning (Trail Making Test [TMT]), visual perceptual ability (Motor-Free Visual Perception Test: Third edition [MVPT-3]), attention (Centre for Research on Safe

Driving Attention Network Test [CRSD-ANT]), and visual information processing (Useful Field of View [UFOV]).

(4) We also expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would be associated with poorer driving performance on the driving simulator.

Method

Participants

Participants were recruited from undergraduate classes at Lakehead University and from members of the community. Recruitment methods used included newspaper advertisements, flyers, Thunder Bay television, speaking directly to undergraduate classes, and announcements in the Lakehead University Communications Bulletin. Participants who were enrolled in Introductory Psychology received up to five bonus points upon completion of the present study. Participants received one bonus point for completing the online screener, two bonus points for the laboratory portion, and two bonus points for the laboratory portion of this study. All other participants received a gift-card valued at $25 for each laboratory session ($50 total) for participation in this study. In addition, all participants were entered into a draw for a $100 gift-card for completing the online portion of this study.

Inclusion criteria for this study included holding a valid General class (5) driver's license and being between the ages of 18 and 65 (Bulmash et al., 2006). Based on a previous study using a clinical population with depression and a driving simulator, participants were excluded if they self-reported a serious head injury in the past, or psychotic disorder, or self-reported neurological or medical condition. In addition, participants were excluded if they were currently receiving treatment from a psychotherapist or counsellor for depression or anxiety.

A research assistant provided the investigator with a list of eligible participants from the online screening portion of this study who indicated that they were interested in participating in the laboratory sessions. Potential participants were contacted according to their preferred method of contact (email or phone). A laboratory appointment was scheduled, which required approximately 1.5 to 2.5 hours. Participants were instructed to bring their prescription medication bottles (if applicable) and visual-correcting glasses, if they required them, to the appointment. Research Ethics Board approval was obtained for this study and signed informed consent was also obtained from each participant.

Materials

Demographic questionnaire (Appendix A). A demographic questionnaire consisting of items pertaining to basic identifying information (e.g., age, ethnicity, sex) was administered to all participants.

Driving Behaviour Questionnaire (DBQ; Reason, Manstead, Stradling, Baxter, & Campbell, 1990; Appendix B). The DBQ is a 50-item self-report measure of safe driving behaviours. Each item is scored on a 6-point Likert-type scale ranging from "0" (*never*) to "5" (*nearly all the time*). One item was excluded because it pertained to manual drivers only (i.e., "*Attempt to drive away from traffic lights in third gear.*"). The original 50-item DBQ has been found to have three factors including errors (information processing errors), lapses (errors due to difficulties with memory), and violations (poor driving habits; Özkan, Lajunen, & Summala, 2006; Reason et al., 1990). The DBQ demonstrates good internal consistencies for errors, violations, and lapses (Cronbach's α = .78, .79, and .64, respectively; Parker, Lajunen, & Stradling, 1998) In addition, Parker et al. (1995) found good test-retest reliabilities for errors (r = .69), violations (r = .81), and lapses (r = .75) over a 7-month interval. Özkan, Lajunen, and

Summala (2006) conducted a longitudinal study to examine the test-retest reliability of the errors and violations subtests of the DBQ. Good test-retest correlations were reported after an interval of three years (n = 622) for errors (r = .50) and violations (r = .76). Furthermore, the DBQ demonstrates strong correlations with the Driving Behaviour Inventory, a measure of driving stress and performances (rs = .45 to .56; Westerman & Haigney, 2000).

Driving history/habits questionnaire (Appendix C). This measure includes nine items that explore past driving information and is frequently used in research at the Centre for Research on Safe Driving. More specifically, this measure gathers information on number of kilometres driven in a week, number and time of any past collisions, and whether participants restrict their driving (e.g., daylight hours only). In addition, this measure gathers information on whether participants typically exceed the speed limit.

Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item assessment instrument that examines the intensity of depression in adolescents and adults. Each item includes a list of four statements that reflect the severity of a symptom of depression. Higher scores are indicative of more intense depressive symptoms. Item scores are totalled for a total depression score, which can range from minimal (0-13), mild (14-19), moderate (20-28) to severe (29-63) (Beck et al., 1996). Whisman, Perez, and Ramel (2000) conducted a factor analysis and reported that the BDI-II is composed of a cognitive-affective factor and a somatic factor. The items that pertain to the cognitive-affective factor correspond to sadness, past failures, loss of pleasure, feelings of guilt, feelings of punishment, self-dislike, feelings of being critical of oneself, suicidal thoughts, crying, agitation, loss of interest, feelings of worthlessness, and irritability. In contrast, the somatic factor corresponds to items such as loss of energy, disrupted sleep, changes in appetite, difficulty with concentration, and fatigue.

BDI-II scores are strongly correlated (r = .83) with the Structured Clinical Interview for the Diagnostic and Statistical Manual for Axis I Mental Disorders (SCID-I) Major Depressive Disorder (MDD) scores in a university population suggesting that the BDI has excellent criterion validity (Sprinkle et al., 2002). Furthermore, a cut-off score of 16 corresponds to a sensitivity rating of 84% for detecting depressed mood. Sprinkle et al. (2002) also investigated the test-retest reliability of the BDI-II by administering this questionnaire to 46 university students (28 women) at two time-points. The time interval between administrations was between 1 and 12 days. Results showed that the BDI-II displays excellent (r = .96) test-retest reliability (Sprinkle et al., 2002). In addition, Dozois, Dobson, and Ahnberg (1998) conducted a psychometric evaluation of the BDI-II and reported that this measure as a whole has excellent internal consistency (α = .91). Therefore, the BDI-II can be considered a good instrument for measuring the severity of depression.

Beck Anxiety Inventory (BAI; Beck & Steer, 1990). The BAI is a 21-item self-report measure of anxiety. Each item is rated on a 4-point Likert-type scale ranging from 0 (*not at all*) to 3 (*severely – I could barely stand it*). Total scores from 0 to 9 are indicative of a normal level of anxiety; a score from 10 to 18 suggests mild to moderate anxiety; scores from 19 to 29 indicate moderate to severe anxiety; and total scores between 30 and 63 reflect severe anxiety. The BAI has good internal consistency (Cronbach's α = .92) and high test-retest reliability after 1-week (r = .75; Beck, Epstein, Brown, & Steer, 1988). Furthermore, the BAI demonstrates good content validity (r = .85; Beck et al., 1988). Moreover, the BAI shows good concurrent validity with the Hamilton Anxiety Rating Scale – Revised (r = .51; Beck et al., 1988). The BAI and Beck Depression Inventory (BDI) demonstrate good discriminant validity; a moderate correlation between the BAI and BDI has been reported (r = .50; as cited in Beck & Steer, 1990).

However, Hewitt and Norton (1993) factor analysed the BAI and BDI after administering these measures to a heterogeneous clinical population and found that these two scales have good discriminant validity. The factor analyses revealed a two-factor solution in which the BDI loaded highest on factor 1 with loadings between .27 and .75. In contrast, BAI items loaded highest on factor 2 with loadings between .41 and .69. The BAI is considered a good measure of anxiety.

Medication History Questionnaire (Appendix D). A follow-up questionnaire consisting of items pertaining to treatment was administered to all participants. In addition, all participants were asked if they have seen a therapist in the past or presently (e.g., Are you currently seeing a therapist? Are you on any medications?).

The Trail Making Test, A and B (TMT; Spreen & Strauss, 1991). The TMT is a paper and pencil task that asks participants to connect randomly distributed circles in a stated order. The TMT contains two components including TMT part A (TMT-A) wherein participants sequentially connect 25 numbers on paper and TMT part B (TMT-B) in which participants alternate between numbers and letters (e.g., 1, a, 2, b, 3, c). The TMT is scored based on the amount of time it takes the participant to complete the task. The TMT measures cognitive performance using measures of visual search, psychomotor speed, divided attention, cognitive flexibility, sequencing, and conceptual tracking (Mitrushina, Boone, & D'Elia, 1999). Research reveals that individuals with MDD perform significantly slower on the TMT compared to healthy controls (Mahurin, et al., 2006). Furthermore, Atkinson et al. (2010) have found good construct validity of three variants meaning that the TMT measures what it purports to measure.

The Motor-Free Visual Perception Test: Third edition (MVPT-3; Colarusso & Hammill, 2003). The MVPT-3 is a paper and pencil test that measures visual perceptual ability independent of motor abilities and takes approximately 20 to 30 min to complete. More

specifically, this test measures perceptual processes such as spatial relationships, visual discrimination, figure-ground, visual closure, and visual memory (Colarusso & Hammill, 2003). The standard test scores range from 55 to 145 and have a mean of 100 and a standard deviation of 15. Cronbach's alphas range from .69 to .90. The MVPT-3 was administered at two time-points with an interval of 34 days to 2,005 student participants from regular classrooms across the United States. Test-retest reliabilities were good (r = .87-.92; Colarusso & Hammill, 2003). The MVPT-3 also has demonstrated good content and construct validity (Colarusso & Hammill, 2003).

The Useful Field of View® Test (UFOV®; Ball & Owsley, 1993; Edwards et al., 2005). The concept of useful field of view originated as a measure of visual acuity to diagnose eye disease which later progressed to a standard version of a computerized task to measure visual information processing and cognitive aging (Edwards et al., 2005). Today, the short version of the UFOV® is a computerized task that measures processing speed and attention. This task asks participants to complete three subtests including: processing speed, selective attention, and divided attention. The UFOV® short-form takes approximately 15 minutes to complete and the subtests increase in complexity as the test progresses and displays moderately high test-retest reliabilities. For example, Edwards et al. (2005) administered the UFOV® short-form to 66 older adults at two time-points, with an average interval of 10 days, and reported correlation coefficients of .68 to .88. Furthermore, Edwards et al. (2005) assessed the validity of the UFOV® short-form with the original UFOV®. Participants were 364 older adults who completed the standard version and short-version of the UFOV®. Results revealed a moderate correlation (r = 0.77) suggesting that the short-form of the UFOV® has validity.

Centre for Research on Safe Driving Attention Network Test (CRSD-ANT; Weaver, Bédard, & McAuliffe, 2011). The CRSD-ANT is a shorter version of the Attention Network Test (ANT). The ANT is a computerized reaction time test that combines a flanker task with arrows developed by Eriksen and Eriksen (1974) and a cued reaction time task created by Posner (1980). It takes approximately 20 min to complete and measures attention such as: alerting efficiency, orienting efficiency, and conflict efficiency. Alerting efficiency is considered a state in which an individual can achieve and maintain attentiveness. Orienting efficiency is defined as shifting attention from one location to the next while conflict efficiency, or executive function, is concerned with detecting and resolving any conflict in mental operations (Mahoney, Verghese, Goldin, Lipton, & Holtzer, 2010). MacLeod et al. (2010) collected data from 15 studies (n = 1,129) to investigate the reliability of the ANT. This analysis resulted in low split-half reliabilities for the alerting and orientating indices, Spearman-Brown rs = .38 and .55, respectively. However, a moderately high Spearman-Brown correlation (r = .81) was discovered for conflict efficiency. The ANT has also demonstrated very good concurrent validity with the UFOV® in predicting simulated driving performance (Weaver, Bédard, McAuliffe, & Parkarri, 2009). Therefore, the ANT is a useful tool for measuring attention. However, for researchers who are conducting driving research, the 20 minute version is too long. Therefore, Weaver, Bédard, and McAuliffe (2011) created a shorter 10 minute version called the CRSD-ANT. To create the shorter version, the neutral target condition of the original ANT was removed, the time intervals in the trial sequence were decreased, and the stimulus was changed from an arrow to a clip-art truck. The CRSD-ANT has 32 practice trials and 124 test trials. The test trials are in blocks of 64 and include a rest break in between blocks. The ordering of the trials is random. The CRSD-ANT demonstrates good agreement, or convergent validity, with the ANT (r = .92).

Wechsler Adult Intelligence Scale, Fourth Edition, Digit Span Subtest (WAIS-IV; Wechsler, 2008). The Digit Span subtest of the WAIS-IV is composed of a forward and backward digit strings task and a sequencing task. This subtest measures working memory, attention, and concentration (Wechsler, 2008) and was used as a measure of effort in the present study. Past research has used the Wechsler Adult Intelligence Scale – Revised (WAIS-R) and Wechsler Adult Intelligence Scale - third edition (WAIS-III) versions of the WAIS Digit Span subtest to assess effort among participants (Young, Sawyer, Roper, & Baughman, 2012). These versions differ from the WAIS-IV in that the earlier versions do not include a sequencing task. The Reliable Digit Span (RDS; Greiffenstein, Baker, & Gola, 1994) is a procedure that includes summing the longest string of digits that are repeated by the participant without error over both trials on the forward and backward conditions. RDS scores are used to assess suboptimal effort. Greiffenstein, Baker, and Goal (1994) used RDS scores with a cut-off score at ≤7 RDS to successfully distinguish individuals with persistent post-concussion syndrome from malingerers (specificity = .89; sensitivity = .68). Given that the WAIS-IV includes a sequencing task, the Reliable Digit Span-Revised (RDS-R) was developed to include this sequencing task (Young et al., 2012). The RDS-R is calculated by summing the digits repeated from the Sequencing trial to the RDS trial. Young, Sawyer, Roper, and Baughman (2012) conducted a retrospective review of the RDS and RDS-R in 277 patients in which 26% of patients had a diagnosis of a mood disorder. Results revealed that both the RDS and RDS-R displayed concurrent validity. The RDS (OR = 1.38, 95% CI [1.20, 1.59]) and RDS-R (OR = 1.25, 95% CI [1.13, 1.37]) differentiated groups on a pass/fail basis on the Word Memory Test. Last, this study found that RDS (cut-off = ≤7 and ≤6) had a specificity of .81 and .92 and sensitivity of .49 and .24, respectively. The RDS-R (cut-off = ≤11 and ≤10) had a specificity of .78 and .89 and sensitivity of .48 and .32,

respectively. Although additional research needs to be conducted on the RDS-R, it can be considered a good measure of effort.

Structured Clinical Interview for the Diagnostic and Statistical Manual-IV for Axis I Disorders Research Version, Non-Patient Edition (SCID-I-RV; First et al., 2002). The SCID-I-RV is a clinician-administered semi-structured interview that is composed of nine diagnostic modules that assess psychopathology (First et al., 2002). The SCID-I-RV has been considered the gold standard for assessing a valid self-reported diagnosis (Sanchez-Villegas et al., 2008). The Psychotic and Associated Symptoms module of the SCID-I-RV was administered to identify any participants who are experiencing a psychotic episode. The SCID-I-RV uses probe questions, follow-up questions, and skip-out questions to arrive at the correct diagnosis. Diagnoses are made during the interview and there is no scoring guide or algorithm (Sanchez-Villegas et al., 2008).

The Manitoba Road Test (MRT; Appendix E). The MRT uses a road examination demerit-based scoring system to determine acceptable driving safety practices. This test was used to evaluate performance in the driving simulator task. Participants were given demerit points when they did not perform safe driving practices in five general categories including: starting/stopping, signal violations/right of way/inattention, moving on the roadway, passing/speed, and turning. Five or 10 demerit points were given for each infraction. More demerits were given for more serious mistakes (e.g., cutting off a vehicle) and fewer demerits for minor mistakes (e.g., drives at an uneven speed). A total of demerit points was calculated. The reliability and validity of this measure have not been assessed using driving simulators. However, the MRT has been used in previous research (see Weaver, Bédard, McAuliffe, Parkkari, 2009).

Driving Simulator. The STISIM DRIVE™ simulator (Systems Technology, Inc, Hawthorne, CA) has three networked computers that are connected to three 17" monitors, a steering unit, and a foot pedal unit (see Figure 1). The steering unit includes a steering wheel, signal light, horn, speedometer, and odometer. The foot pedal unit includes both an accelerator and brake pedal. The researcher controls the simulator via a fourth computer. The simulator is designed to record input from the driver such as: speeding, lane excursions, collisions, and illegal turns. Using these measures, an overall index of driving performance can be calculated. Driving simulators are considered valid and safe for measuring driving performance (Bédard et al., 2010; Lee, Cameron, & Lee, 2003).

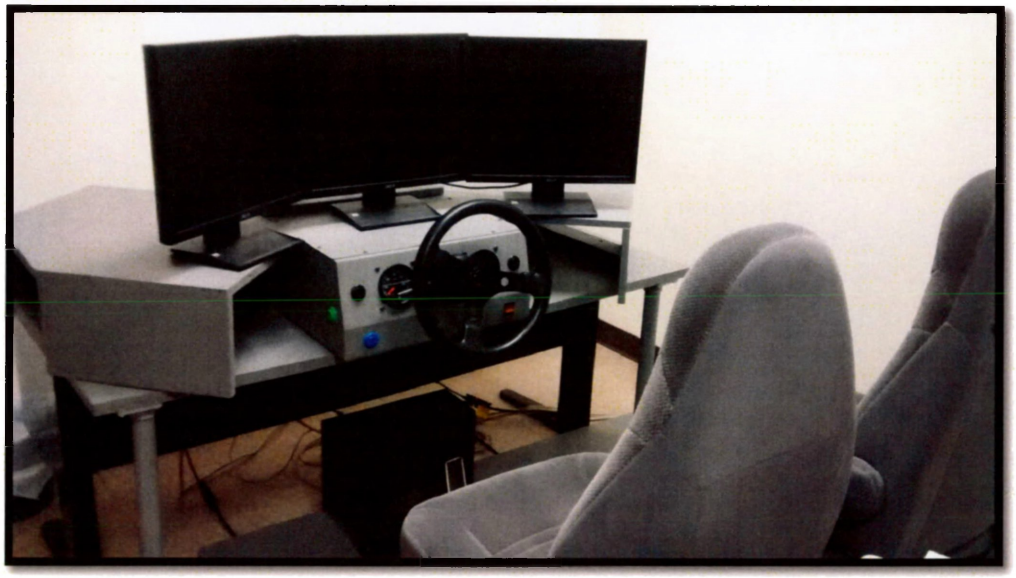

Figure 1. Driving Simulator

Patient Health Questionnaire, Ninth Edition (PHQ-9; Kroenke, Spitzer, & Williams, 2001). The PHQ-9 was derived from the Patient Health Questionnaire (PHQ), which is a 3-page self-report measure that assesses 8 DSM-IV diagnoses (Kroenke, et al., 2001). The PHQ-9 is a 9-item inventory designed to screen for MDD. Each item is rated on a 4-point Likert-type scale that includes 0 (*not at all*), 1 (*several days*), 2 (*more than half the days*), and 3 (*nearly every*

day). A diagnosis of MDD is warranted if 5 or more of the 9 items occur on "*more than half the days*" and one of the items endorsed is either low mood or anhedonia. Item 9 which states: "*Thoughts that you would be better off dead or hurting yourself in some way*" counts if it is endorsed as a 1 or more. The PHQ-9 also includes a severity item which asks "*How difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?*" The severity item ranges from "*not difficult at all*" to "*extremely difficult.*" In addition, scores range from 0 to 27; a score from 0 to 4 suggests minimal severity; scores from 5-9 indicate mild severity; scores between 10 and 14 suggest moderate severity; moderately severe scores range from 15 to 19; and total scores between 20 and 27 reflect severe MDD. Kroenke et al. (2001) conducted a reliability analysis of the PHQ-9 and found that the PHQ-9 demonstrated excellent internal consistency (Cronbach's $\alpha = .89$) in a primary care sample ($n = 3{,}000$) and in a obstetrics-gynecology sample (Cronbach's $\alpha = .86$; $n = 3, 000$). Furthermore, the PHQ-9 demonstrated high test-retest reliability after 48 hours ($r = .84$; Kroenke, et al., 2001). A PHQ-9 score of ≥ 10 demonstrated a specificity and sensitivity of .88 and likelihood ratio of 7.1 (Kroenke, et al., 2001). This measure also displays strong construct validity as it is highly correlated with the Medical Outcomes Study Short-Form General Health Survey (SF-20) mental health scale ($r = .73$). In addition, Titov, Dear, McMillan, Anderson, Zou, and Sunderland (2011) conducted a psychometric evaluation of the PHQ-9 and report adequate internal consistency (Cronbach's $\alpha = .74$; $n = 172$). Moreover, the PHQ-9 displayed convergent validity with the BDI-II ($r = .72$). The PHQ-9 is considered a good diagnostic tool for assessing MDD.

Procedure

A mass E-mail was sent out to students who were enrolled in the Introductory Psychology course at Lakehead University and undergraduate courses at Lakehead University.

The E-mail included a link to SurveyMonkey in which interested participants were directed to complete the screening questionnaire. The link was also available to members of the Thunder Bay community on Facebook. The first page of the SurveyMonkey webpage explained to participants that the initial online questionnaire would take up to 1 hour to complete and that participation was completely voluntary. Participants were then instructed to read the Information Letter A (see Appendix F), complete the Consent Form A (see Appendix G), and check a box online to indicate their consent to complete the online screening questionnaire. The screening questionnaire required participants to complete the demographics questionnaire (Appendix A), the DBQ (Reason et al., 1990; Appendix B), the driving habits and history questionnaire (Appendix C), the BDI-II (Beck et al., 1996), and the BAI (Beck & Steer, 1990) and the medical history questionnaire (Appendix D) which included items that gather information on medication use, previous treatments for depression, history of serious head injuries, and neurological conditions. After completion of the screening questionnaire, the next page of the online survey asked participants if they would be willing to be contacted via E-mail or phone to participate in a laboratory portion of the study. Next, participants were thanked for their participation. Subsequently, a research assistant scored the online screening questionnaire measures to determine eligibility for the laboratory portion of the study. Based on a previous study using a clinical population with depression and a driving simulator, participants were excluded if they self-reported a serious head injury in the past or psychotic disorder as measured using the psychotic screening module of the Structured Clinical Interview for the Diagnostic and Statistical Manual-IV for Axis I Disorders (SCID-I-RV; First, Gibbon, Spitzer, & Williams, 2002), or self-reported neurological or medical condition (Bulmash et al., 2006). In addition, participants were excluded if they were currently receiving treatment from a psychotherapist as seeking

psychotherapist could confound restuls. Inclusion criteria included holding a valid General class (5) driver's license and being between the ages of 18 and 65 (Bulmash et al., 2006). Eligible participants who indicated that they would be willing to participate in the laboratory portion of this study were contacted by the researcher (Loretta Patterson) by their preferred method of contact (E-mail or phone). The researcher invited these eligible participants to attend two laboratory sessions. Eligible participants were scheduled for an appointment, which took approximately 1.5 to 2.5 hours to complete.

Upon arrival to the Lakehead University Driving Laboratory (BB 1024), participants were instructed to read the Information Letter B (see Appendix H) and participants were asked if they had any questions about the study. After all questions had been answered, participants were asked to sign the Consent Form (see Appendix I). Next, participants completed the Trail Making Test (TMT; Spreen & Strauss, 1991) and the Motor Free Visual-Perception Test – 3 (MVPT-3; Colarusso & Hammill, 2003). Next, participants completed two computerized tests including a test of visual processing speed (UFOV; Ball & Owsley, 1993; Edwards et al., 2005) and a test of attention (CRSD-ANT; Weaver, Bédard, & McAuliffe, 2011). As a screening measure of low effort, the Digit Span subtest of the Wechsler Adult Intelligence Scale – Fourth Edition (WAIS-IV; Wechsler, 2008) was administered to participants in the laboratory. Last, the Psychotic and Associated Symptoms module of the Structured Clinical Interview for the Diagnostic and Statistical Manual-IV for Axis I Disorders Research Version, Patient Edition (SCID-I-RV; First et al., 2002) was administered to identify any participants who were experiencing a psychotic episode.

Given that some individuals may experience simulator sickness, which is physical discomfort while using the driving simulator (Classen, Bewernitz, & Shechtman, 2011),

participants completed the driving simulation after the other neuropsychological tests. Our goal was to prevent any potential simulator sickness from interfering with any of the other laboratory measures. Participants were verbally instructed on how to use the simulator and completed a 10 min practice session to help them adapt to the simulator. Next, participants completed a 40 min route that included both city and highway driving on the STISIM DRIVE^{TM} simulator (Systems Technology, Inc, Hawthorne, CA). If participants experienced any physical discomfort due to the simulator, the drive was paused until the participant was ready to resume or the session was ended. The researcher was trained by another researcher at the Centre for Research on Safe Driving on specific Manitoba Road Test (MRT) scoring guidelines and scored the participants performance on the simulator using the MRT Demerit point system (Appendix E). Last, participants were asked to complete the Patient Health Questionnaire – 9 (PHQ-9; Kroenke, Robert, Spitzer, & Williams, 2001) to determine if participants met the DSM-IV-TR criteria for Major Depressive Disorder. Participants completed the PHQ-9 subsequent to the simulated drive to keep the experimenter blind to whether or not the participants met the diagnostic criteria for MDD. The participants also completed the medical history questionnaire (Appendix D) again. Last, participants were thanked for their participation and informed that they would be contacted in three months for a follow-up session in the laboratory. All participants were given a list of psychological services for psychological treatment (see Appendix J). Participants who met the diagnostic criteria for MDD were given a letter recommending they seek psychological treatment (see Appendix K).

Participants were contacted three months following participation in the laboratory session. Each participant was invited to attend a second laboratory session to complete the measures a second time and in the same order. Participants who participated in the second

laboratory session completed the TMT (Spreen & Strauss, 1991), the MVPT-3 (Colarusso & Hammill, 2003), the UFOV (Ball & Owsley, 1993; Edwards et al., 2005) and the CRSD-ANT (Fan et al., 2002). The researcher then administered the Psychotic and associated symptoms module of the SCID-I-RV (First et al., 2002) to participants. Participants then completed the orientation drive and the 40 min driving route again. Last, participants completed the PHQ-9 (Kroenke et al., 2001) and the medical history questionnaire (Appendix D). Participants were thanked for their participation and given a list of psychological services (see Appendix J). Participants who again met the diagnostic criteria for MDD were given a recommendation letter to seek psychological treatment (see Appendix K). All participants who were enrolled in psychology 1100 were awarded up to five bonus points towards their Introductory Psychology final grade for participation in this study. One point was awarded for the online screener, two points for the initial laboratory portion, and two points for the follow-up portion of this study. All other participants were awarded up to $50 in gift-cards for participation ($25 per lab session).

Preliminary data screening. Data screening was performed to investigate missing values, accuracy of data entry, normality, and outliers. Accuracy checks on the data file were visually performed. Frequencies and descriptive statistics were run on the main variables to identify odd or extreme values. Histograms and scatterplots were also generated to identify atypical or abnormal distributions. Outliers were examined using the Cook's distance statistic (D), which is a measure of the change in regression coefficients after deletion of a case. Cook's distance assesses the overall influence of a case on a model. In the screener data, one case was visually identified on both the residual plots and histograms for factor 1 ($D = 0.11$) and factor 4 ($D = 0.07$). This case was identified as the same female participant aged 19 years. Given that, Tabachnick and Fidell (2007) have suggested that Cook's distance of greater than one may be

problematic and are suspect of being an outlier, this case was not considered problematic and was retained within the data set. No outliers were identified in the laboratory data.

Statistical Analyses

All analyses were performed with PASW Statistics version 21. To investigate hypothesis 1, an exploratory factor analysis (using principal axis factoring for extraction and *oblimin* rotation) was generated on the longer version (49-item) DBQ (DBQ-LV) for the total sample. Cronbach's alphas for the factors were also generated to investigate the internal consistency of the factors. Based on high factor loadings (greater than .300; Streiner, 1994) and the highest five item-total correlations per factor, a shortened version of the DBQ (DBQ-SV) was created. Next, a forced four-factor exploratory factor analysis (using principal axis factoring for extraction and *oblimin* rotation) was generated on the DBQ-SV in the younger Canadian sample. A forced-four factor EFA was used because factors 5 and 6 only contained 2 items each with factor loadings greater than .300. Cronbach's alphas for the shortened version were also generated. Correlations between the DBQ-LV and DBQ-SV were also explored.

To investigate hypothesis two, two standard multivariable regression models were used to examine the presence of possible relationships between age, sex, depressive symptoms, anxiety symptoms, and antidepressant use and self-reported unsafe driver behaviour. Participant age, sex, depressive symptoms, and anxiety symptoms were entered into Model 1. Model 2 included the variables listed in Model 1; however, the anxiety variable was removed as it is strongly correlated with depressive symptoms. In addition, Model 2 also included variables capturing the linear component of the age by cognitive/affective depressive variable interaction, age by somatic depressive variable interaction, and age by antidepressant use interaction.

To investigate hypotheses three and four, ordinary least squares regressions were run to investigate possible relationships between depressive symptoms and the CRSD-ANT response time, CRSD-ANT alerting score, CRSD-ANT orienting score, CRSD-ANT conflict score, TMT-A score, TMT-B score, MVPT-3 standard score, MVPT-3 errors, UFOV sum score, and UFOV divided attention score. The PHQ-9, BDI-II Cognitive/Affective score, BDI-II Somatic score were the predictor variables in these models. To investigate hypothesis four, an ordinary least squares regression was also run to investigate relationships between the PHQ-9, BDI-II Cognitive Affective, and BDI-II Somatic scores and the simulated drive (MRT score).

Missing data. A formal analysis of missing values was completed for the online screening data and laboratory session data. For the online screening data, analyses revealed that, 3.56% of the values and 14.55% of all cases of interest were incomplete. Eight participants were excluded because their missing values exceeded 10. Given that there the data were not 100% complete, a Missing Value Analysis (MVA) using the expectation-maximization (EM) algorithm was employed to correct for missing data (Graham, 2009). For the laboratory data, only one participant (female; age 21) had two missing data points on the BAI. This score was prorated for analyses. All other data were complete.

The data were examined to investigate whether the assumptions of multiple regression were met. To examine multicollinearity and singularity, a correlation matrix of all the predictor variables for the screener data and laboratory data was examined. With the expected exception of the BDI-II, BAI, and PHQ-9, there were no substantial bivariate correlations between predictors. These variables were expected to be significantly correlated as depression and anxiety symptoms are highly related (APA, 2013; Stulz & Crits-Christoph, 2010). Therefore, the assumptions were considered to be met. In addition, conditioning indices and variance

proportions (collinearity diagnostics) were also examined. Evidence that multicollinearity is suggested to exist if the condition index approaches 30 and the variance proportion is greater than .50 (Tabachnick & Fidell, 2007). Examination of collinearity diagnostics suggests that there is no evidence for multicollinearity and demonstrated that the assumption was met. To test the assumptions of normality, linearity, and homoscedasticity, histograms of the residuals and scatterplots of residuals against fitted values were examined. Visual inspection of the plots revealed that the data met the assumptions.

Results

Sample characteristics.

Overall, a total of 275 participants completed the online screener. Only participants ranging in age from 18 to 35 years were included in the analyses (n = 236). However, one individual was excluded due to missing data across several measures resulting in a final sample size of 235. The average age of participants was 21.84 years (SD = 3.90 years); the majority of participants were women (79.1%). Four participants did not specify their age and were excluded from analyses. Participants' characteristics are presented in Table 1. Descriptive statistics for the main variables of interest are displayed in Tables 2 to 6. Intercorrelations among all online screener variables are displayed in Table 7.

Overall, a total of 124 participants agreed to be contacted for further participation. These participants were invited to complete the laboratory portion of this study. Forty-three participants participated in the laboratory session. Two participants were considered ineligible; one due to a having a neurological disorder and the other began seeing a therapist at the time of participation. The average age of participants was 24.24 years (SD = 5.05 years) and the majority were women (81.4%; n = 35). Only 2 participants reported taking an antidepressant medication; both were

taking an SSRI. No participant was psychotic. All participants scored in the minimal range on the BDI-II except one participant who scored in the moderate range. Descriptive statistics and intercorrelations for the main variables of interest are displayed in Tables 8 to 12.

The WAIS-IV digit span subtest was administered as a measure of effort. Reliable digit span (RDS) was originally used with the WAIS – Revised and WAIS – III. RDS is calculated by summing the longest digit forward score and the longest digit backward score. Reliable digit span – revised (RDS-R) can be calculated for the WAIS-IV as it sums the digits repeated from the longest digit sequencing trial to the RDS trial. Young, Sawyer, Roper, and Baughman (2012) report that an RDS cut-off = ≤6 (sensitivity = .24; specificity = .92) and an RDS-R cut-off = ≤10 (sensitivity = .32; specificity = .89) are recommended to achieve acceptable specificity. Both RDS and RDS-R were calculated. One female participant scored an RDS of 7. This same participant scored higher than the RDS-R cut-off and was therefore included in analyses. All other participants also scored 11 or greater on the RDS-R suggesting that these participants met the cut-off for putting in full effort. Only twelve of the 43 participants, who completed the laboratory portion of this study, completed the 3-month follow-up. Analyses were not conducted on these 12 participants, as the sample size was too small.

Main Hypotheses

Hypothesis 1. We aimed to create a shortened version of the DBQ in a younger Canadian sample. We expected to observe good psychometric properties in this shorter version of the DBQ. An exploratory factor analysis (EFA) analysis (using principal axis factoring for extraction and *oblimin* rotation) was conducted on the original 49 items of the DBQ. After excluding participants with greater than 10 variables missing, an EFA was conducted for the remaining 233 participants. Streiner (1994) argues that, when conducting an EFA, "there should

Table 1

Participant demographics and the DHQ: Km driven and crash history (n = 236)

Characteristic	Total
Age	
Range	18-35
Mean (SD)	21.84 (3.9)
Men, No. (%)	45 (19.1)
Approximately how many **kilometers (miles)** do you drive per week? (n = 234)	No. (%)
0-20km (0-12 miles)	44 (18.7)
21-50km (13-31 miles)	74 (31.5)
51-100km (32-62 miles)	73 (31.1)
Over 100 km (over 62 miles)	43 (18.3)
When driving, how many accidents (involving a person, car, or fixed object) have you been involved in? (n = 225)	
# *At fault*	
Range	0 – 5
Mean (SD)	1.4 (0.8)
# *Not at fault*	
Range	0 – 3
Mean (SD)	1.4 (0.6)
How long ago was your last **at fault** car accident involving a person, car, or fixed object? (n = 233)	No. (%)
Less than 1 year	26 (11.0)
1-2 years	19 (8.1)
2-3 years	8 (3.4)
3-4 years	4 (1.7)

4-5 years	6 (2.5)
5-10 years	8 (3.4)
More than 10 years	3 (1.3)
Never had an accident	159 (67.4)

How long ago was your last **not at fault** car accident involving a person, car, or fixed object? (n = 232)	No. (%)
Less than 1 year	20 (8.5)
1-2 years	17 (7.2)
2-3 years	9 (3.8)
3-4 years	6 (2.5)
4-5 years	5 (2.1)
5-10 years	7 (3.0)
More than 10 years	2 (0.8)
Never had an accident	166 (69.9)

DHQ = Driving Habits/History Questionnaire; Km = kilometers

Table 2

Descriptive statistics for the DHQ: Purposes for driving in a week

Items	*No. (%)*	M (*SD*) # of times per week
For what purposes do you drive in a typical week?		
Groceries	148 (62.7)	1.7 (0.9)
Attending health-related appointments	80 (33.9)	1.3 (0.8)
Attending social events	186 (78.8)	3.0 (2.1)
Worship	23 (9.7)	1.6 (0.8)
Hobbies	126 (53.4)	3.8 (2.5)
Work/school	191 (80.9)	5.9 (2.7)
Family events	108 (45.8)	1.6 (1.0)
Other	57 (24.2)	2.8 (1.9)

DHQ = Driving Habits/History Questionnaire; *M* = Mean; *SD* = Standard Deviation; N = 235

Table 3

Descriptive statistics for the DHQ: Stressful driving situations, restricting driving, and speed

Items	
Which driving situation(s) do you find stressful, uncomfortable, or avoid when possible?	No. (%)
Turning left at intersections	34 (14.4)
Navigating parking lots	37 (15.7)
Driving at night	77 (32.6)
Changing lanes	14 (5.9)
Backing up	63 (26.7)
Maintaining the speed limit	17 (7.2)
Parallel parking	144 (61.0)
Driving in bad weather	166 (70.3)
Driving in unfamiliar areas	120 (50.8)
Driving in heavy traffic	117 (49.6)
Driving with passengers in the car	20 (8.5)
Other	19 (8.1)
Driving alone	10 (4.2)
None of the above	19 (8.1)
Some people restrict their driving to certain situations. Do you restrict your driving to:	
Daytime	17 (7.3)
When accompanied by a passenger	7 (3.0)
Outside of rush hour	16 (6.9)
Local routes	19 (8.2)

Fair weather	40 (17.2)
None of the above	159 (68.2)
Other	224 (96.1)
What speed do you typically drive on local streets?	
35 km/hr or less	3 (1.3)
36-45 km/hr	7 (3.0)
46-55 km/hr	99 (42.1)
56-65 km/hr	114 (48.5)
66 km/hr or more	12 (5.1)
What speed do you typically drive on major highways?	
85 km/hr or less	3 (1.3)
86-95 km/hr	32 (13.6)
96-105 km/hr	132 (56.2)
106-115 km/hr	63 (26.8)
116 km/hr or more	5 (2.1)

DHQ = Driving Habits/History Questionnaire; N = 235

Table 4

Descriptive statistics for depressive and anxiety symptoms for the screening data by total sample and sex

Characteristic	Total Sample ($N = 235$[a])		Men ($n = 45$)		Women ($n = 186$)	
	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
BDI-II Total	0-59.85[b]	11.09 (9.87)	0-59.85	11.18 (12.01)	0-48.63	11.10 (9.33)
BDI-II C/A	0-45	7.62 (7.33)	0-45	7.98 (9.20)	0-33	7.56 (6.82)
BDI-II S	0-13	2.61 (2.36)	0-12	2.50 (2.59)	0-13	2.63 (2.31)
BAI Total	0-60	10.34 (9.71)	0-42	8.96 (10.42)	0-60	10.73 (9.52)
BDI-II Severity	Range	No. (%)	Range	No. (%)	Range	No. (%).
Mild	0-19.95	201 (85.53)	0-19.95	39 (86.67)	0-19.95	158 (84.95)
Moderate	21-27.30	20 (8.51)	25.20-25.20	1 (2.22)	21-27.30	18 (9.68)
Severe	29.40-59.85	14 (5.96)	32.55-59.85	5 (11.11)	29.40-48.63	10 (5.38)

Note. BDI-II Total = Beck Depression Inventory – II; BDI-II C/A = Beck Depression Inventory – II Cognitive/Affective; BDI-II S = Beck Depression Inventory – II Somatic; BAI = Beck Anxiety Inventory; SD = Standard Deviation; [a] Four participants did not specify their sex; [b] The BDI-II total excluded one item (suicide item) and scores were prorated to adjust for this.

Table 5

Descriptive statistics for medication use by total sample and sex

	Total ($N = 235$[a])	Men ($n = 45$)	Women ($n = 186$)
Medication	No. (%)		
SSRI	13 (5.5)	2 (4.4)	11 (5.9)
SNRI	10 (4.3)	0	10 (5.4)
TCA	1 (0.4)	0	1 (0.5)
Anxiolytic	4 (1.7)	1 (2.2)	3 (1.6)
Mood stabilizer	1 (0.4)	0	1 (0.5)
Antipsychotic	2 (0.9)	0	2 (1.1)
Psychostimulant	4 (1.7)	0	4 (2.2)
Any medication	29 (12.3)	2 (4.4)	27 (14.5)
Any antidepressant	24 (10.2)	2 (4.4)	22 (11.8)
Number of medications			
None	206 (87.7)	43 (95.6)	159 (85.5)
1	24 (10.2)	1 (2.2)	23 (12.4)
2	4 (1.7)	1 (2.2)	3 (1.6)
3	1 (0.4)	0	1 (0.5)

Note. SSRI = Selective Serotonin Reuptake Inhibitor; SNRI = Selective Norepinephrine Reuptake Inhibitor; TCA = Tricyclic Antidepressant; [a] Four participants did not specify their sex.

Table 6

Descriptive statistics for the DBQ-LV by total sample and sex

Characteristic	Total ($N = 235$[a])		Men ($n = 45$)		Women ($n = 186$)	
	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
DBQ Total	0-66	16.28 (10.45)	0-43	15.69 (11.35)	0-66	16.32 (10.16)
DBQ Lapses	0-53	15.80 (8.67)	0-40	14.23 (9.39)	0-53	16.07 (8.33)
DBQ Violations	0-50	13.41 (9.94)	0-38	12.80 (10.86)	0-50	13.44 (9.73)
DBQ Errors	0-50	8.33 (6.61)	0-28	7.57 (6.94)	0-50	8.55 (6.58)

Note. DBQ-LV = Driver Behaviour Questionnaire – Long Version; SD = Standard Deviation;
[a] Four participants did not specify their sex.

Table 7

Intercorrelations among all screener variables

Variables	1	2	3	4	5	6
1. Age[a]	-					
2. Sex[b]	.15*					
3. BDI-II C/A[c]	.15*	.03				
4. BDI-II S[d]	.07	-.04	.74**			
5. BAI[e]	-.04	-.06	.65**	.56**		
6. AntiDEP[f]	.23**	-.10	.28**	.16*	.21**	

Note. BDI-II C/A = Beck Depression Inventory – II Cognitive Affective component; BDI-II S= Beck Depression Inventory – II Somatic component; BAI = Beck Anxiety Inventory; AntiDEP = Anti-depressant medications.

[a] $N = 235$. [b] $N = 231$. [c] $N = 225$. [d] $N = 234$. [e] $N = 225$. [f] $N = 235$.

*$p < .05$. ** $p < .01$

Table 8

Descriptive statistics for the UFOV by laboratory sample and sex

Characteristic	Total ($N = 43$)		Men ($n = 8$)		Women ($n = 35$)	
	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
UFOV Sum (ms)	50.10-160.40	87.49 (27.94)	50.10-126.70	84.74 (29.98)	50.10-160.40	88.12 (27.87)
UFOV-1 (ms)	16.70-23.40	17.01 (1.22)	16.70-23.40	17.54 (2.37)	16.70-20.0	16.89 (0.78)
UFOV-2 (ms)	16.70-60.00	20.03 (9.08)	16.70-30.10	18.38 (4.74)	16.70-60.0	20.41 (9.82)
UFOV-3 (ms)	16.70-107.0	50.45 (27.94)	16.70-86.60	48.82 (26.84)	16.70-107.0	50.82 (25.08)

Note. UFOV = Useful Field of View; SD = Standard Deviation; UFOV-1 = Processing Speed; UFOV-2 = Divided Attention; UFOV – 3 = Selective Attention. All scores are in milliseconds.

Table 9

Descriptive statistics for the CRSD-ANT by laboratory sample and sex

Characteristic	Total (N = 43)		Men (*n* = 8)		Women (*n* = 35)	
	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
RT Median	498-719	601.07 (53.43)	517-690	616.88 (53.38)	498-719	596.46 (53.55)
RT Mean	503-732	614.07 (55.60)	524-729	628.63 (61.43)	503-732	610.74 (54.60)
RT SD	72.52-184.91	108.48 (22.22)	72.52-173.55	114.44 (31.55)	86.47-184.91	107.12 (19.87)
Alerting	-42-61	17.14 (26.76)	-20-61	20.63 (31.55)	-42-61	16.34 (26.01)
Orienting	-12-149	68.44 (35.65)	17-99	64.63 (27.98)	-12-149	69.31 (37.47)
Conflict	44-184	95.98 (30.22)	62-184	108.75 (40.90)	44-146	93.06 (27.13)

Note. CRSD-ANT = Centre for Research on Safe Driving - Attention Network Test; SD = Standard Deviation; RT = Response Time. All scores are in milliseconds.

Table 10

Descriptive statistics for depressive and anxiety symptoms for laboratory sample by total sample and sex

Characteristic	Total Sample ($N = 43$)		Men ($n = 8$)		Women ($n = 35$)	
	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
BDI-II Total	0-22.05	7.59 (5.99)	2.10-19.95	7.74 (7.36)	0-22.05	7.56 (5.76)
BDI-II C/A	0-14	4.37 (6.86)	1-12	5.13 (4.22)	0-14	4.20 (3.68)
BDI-II S	0-11	2.86 (2.63)	0-8	2.25 (3.06)	0-11	3.00 (2.56)
BAI Total	0-25	7.67 (7.36)	0-23	7.50 (9.37)	0-25	7.70 (6.99)
PHQ-9	0-20	4.16 (4.06)	0-20	5.63 (6.59)	0-14	3.83 (3.29)

Note. BDI-II C/A = Beck Depression Inventory – II Cognitive/Affective; BDI-II S = Beck Depression Inventory – II Somatic ; BAI = Beck Anxiety Inventory; SD = Standard Deviation; PHQ-9 = Patient Health Questionnaire – 9.

Table 11

Descriptive statistics for MRT, TMT, MVPT, and WAIS-IV for laboratory sample by total sample and sex

Characteristic	Total ($N = 43$)		Men ($n = 8$)		Women ($n = 35$)	
	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
MRT Total	15-170	80.23 (33.31)	30-170	78.75 (41.73)	15-160	80.57 (31.80)
TMT A*	11-69	23.72 (11.19)	11-30	21.50 (6.07)	13-69	24.23 (12.07)
TMT B*	18-175	54.95 (27.40)	24-83	49.00 (20.40)	18-175	56.31 (28.84)
MVPT						
Errors	1-19	9.58 (4.98)	1-18	8.13 (6.66)	1-19	9.91 (4.57)
Standard Score	73-143	98.70 (18.18)	76-137	106.88 (22.64)	73-143	96.83 (16.84)
WAIS-IV						
RDS	7-17	12.00 (1.99)	10-16	12.13 (1.89)	7-17	11.97 (2.04)
RDS-R	11-23	17.26 (2.47)	15-21	17.25 (2.12)	11-23	17.26 (2.57)

Note. MRT = Manitoba Road Test; TMT = Trail Making Test; MVPT = Motor Free Visual Perception Test-4; *scored in seconds; RDS = Reliable Digit Span; RDS-R = Reliable Digit Span – Revised

Table 12

Intercorrelations among all laboratory predictor variables

Variables	1	2	3
1. BDI-II C/A			
2. BDI-II S	.59**		
3. PHQ-9	.62**	.47**	

Note. BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S= Beck Depression Inventory – II Somatic; PHQ-9 = Patient Health Questionnaire - 9
$N = 43$.
*$p < .05$. ** $p < .01$

be an absolute minimum of five subjects per variable, with the proviso that there are at least 100 subjects" (p.140). After excluding participants with greater than 10 missing variables, our sample was 12 participants shy of meeting this criterion. Scree plots and interpretability of the factors were used to determine the number of factors to be extracted (Streiner, 1994). The EFA initially supported a 6-factor solution. However, Streiner (1994) argues that a factor should be comprised of at least three variables. In the overall sample, factors 5 and 6 only contained 2 items each with factor loadings greater than .300. In addition, Martinussen, Hakamies-Blomqvist, Mθller, Özkan,and Lajunen (2013) reported that it is not necessary to apply different DBQ structures to different driver groups, with the exception of different age groups. Martinussen et al. also suggest that a three and four-factor model was acceptable across subgroups but found a lower fit among younger groups. These researchers highlight that additional research needs to be conducted examining a factor model for younger groups. Therefore, a forced four-factor EFA was conducted on individuals aged 18 to 35. The total number of cases in the data file was 233. Together the four-factor solution explained 43.12% of variance. Factor 1 explained 29.17% of the variance, while factors 2, 3, and 4, explained 5.89 %, 4.16%, and 3.90%, of the variance, respectively. See table 13 for correlations between factors and eigenvalues. See table 14 for DBQ items and factor loadings. The same items loaded on each factor as with the whole sample, with some variation in the factor loadings for each item.

Reliability Analyses. Reliability analyses for the four-factor solution for participants aged 18 to 35 (n = 235) also demonstrated good to excellent internal consistency, which is considered to fall in the good to excellent range (Murphy & Davidshofer, 2005; see table 13). Cronbach's alphas for factors 1, 2, 3, and 4 were .92, .84, .76, and .78, respectively. This suggests that our items within each factor measure are likely measuring the same construct.

Next, given that there has yet to be a shortened version of the DBQ has been applied to a Canadian sample, our goal was to condense the DBQ items to a shortened version (DBQ-SV) with 5 items per factor and a total of 20 items. Items were selected based on the five highest item-total correlations. For factor 1, item-total correlations ranged from .45 to .68. Items DBQ24, DBQ27, DBQ41, DBQ45, and DBQ49 were included for factor 1. These items can be considered "Errors" and delineated as perceptual, attention, and information processing errors, including misjudgements when driving. Items DBQ3, DBQ4, DBQ8, DBQ15, and DBQ30 had the highest item-total correlations for factor 2 and item-total correlations ranged from .44 to .65. These items can be considered "Emotional Violations" or violations that reflect a driver's style and driving habits due to an emotional response. For factor 3, items DBQ12, DBQ13, DBQ16, DBQ32, and DBQ37 were included and item-total correlations ranged from .37 to .68. These items can be considered "Absent-Mindedness" and delineated as being forgetful or having a low level of attention while driving. Lastly, factor 4 items included DBQ17, DBQ21, DBQ25, DBQ28, and DBQ43 with item-total correlations ranging from .40 to .53. These items can be considered "Reckless Violations" or disrespectful violations. See Table 15 for descriptive statistics for the DBQ-LV and DBQ-SV and table 16 for item-total statistics of the items of the DBQ-SV and DBQ-LV. Cronbach's alphas for short version of factors 1, 2, 3, and 4 were .82, .79, .74, and .71, respectively (see Table 13).

Validity Analyses. To investigate validity, the DBQ-SV was correlated to the DBQ-LV (49-items). Correlation coefficients between the short and long version were excellent and ranged from .91 to .94 (see Table 17). A t-test was calculated to investigate any significant differences between the DBQ-LV and DBQ-SV. Results suggested no significant differences on the factor scores between the two versions. Each factor, including the total score, for both

versions was also correlated with the BDI-II as a measure of discriminant validity. All factors, except factor 2, were significantly related to the BDI-II and both versions were consistent. Correlation coefficients ranged from .12 to .22 (see Table 18). Correlation coefficients were also generated for men and women. Both versions were also consistent across the factors and the total scores for men. Correlation coefficients ranged from .16 to .51 (see Table 18). Factor 3 was the only factor that was significantly related to the BDI-II for women across both the DBQ-LV and DBQ-SV. The total score for the DBQ-SV was also significantly related to the BDI-II for women; however, the DBQ-LV total score was not significantly related to the BDI-II for women. Scatterplots were generated to depict the relationship between the DBQ-LV and DBQ-SV and BDI-II across the total sample, men, and women (see Figure 2).

As a measure of concurrent validity, the DBQ-LV and DBQ-SV were correlated with the simulated drive total score for participants who completed the laboratory session ($N = 43$). A t-test was also generated to investigate any significant differences between the DBQ-LV, DBQ-SV, and MRT total score. Results suggested no significant differences on the factor scores between the two versions and the MRT scores (see Table 19). Both versions were consistent. Correlation coefficients for both versions and the MRT ranged from .03 to .26 (see Table 19).

Hypothesis 2. We expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would predict higher levels of self-reported unsafe driving behaviour on the subtests of the shortened version of the DBQ. Multivariable (multiple predictor variables) regression models were employed to examine the presence of possible relationships between sex, age, depressive symptoms (BDI-II Cognitive/Affective and Somatic symptoms), anxiety symptoms (BAI), antidepressant use, and driving errors (Factor 1), emotional violations (Factor 2), absent-mindedness (Factor 3), and the sum of the four factors. Factor 4 (Reckless

Table 13

Correlation coefficients between factors, eigenvalues, and Cronbach's alphas for the 4-factor EFA for ages 18-35 ($n = 235$)

	Factor 1	Factor 2	Factor 3	Factor 4
Factor 1 (Errors)	**1.00**			
Factor 2 (Emotional Violations)	.360	**1.00**		
Factor 3 (Absent-mindedness)	.476	.290	**1.00**	
Factor 4 (Reckless Violations)	.399	.348	.241	**1.00**
Eigenvalues	14.29	2.89	2.04	1.91
Cronbach's α (DBQ-LV)	.92	.84	.76	.79
Cronbach's α (DBQ-SV)	.82	.79	.74	.71

Note. EFA = Exploratory Factor Analysis; DBQ-LV = Driving Behaviour Questionnaire – Long Version; DBQ-SV = Driving Behaviour Questionnaire – Short Version

Table 14

Factor structure and factor loadings for the 4-factor EFA for ages 18-35

DBQ items (AGES 18-35)	Factors			
	1	2	3	4
Dbq5 Attempt to drive away without first having switched on the ignition.	.556			
Dbq9 Intend to switch on the windshield wipers, but switch on the lights instead, or vice versa.	.366			
Dbq10 Turn left on to a main road into the path of an oncoming vehicle that you hadn't seen or whose speed you had misjudged.	.493			
Dbq11 Misjudge the gap between parked cars and nearly (or actually) hit the adjacent vehicle.	.592			
Dbq19 Try to overtake without first checking in your mirror, and then get honked at by the car behind you which has already begun its overtaking maneuver.	.432			
Dbq23 On turning right, nearly hit a cyclist who has come up on your inside.	.666			
Dbq24 In a line of vehicles turning right on to a main road, pay such close attention to the traffic approaching from the left that you nearly hit the car in front.	.571			
Dbq27 Lost in thought or distracted, you fail to notice someone waiting at a crosswalk or a pedestrian crosswalk light that has just turned red.	.610			
Dbq29 Misjudge the speed of an oncoming vehicle when overtaking.	.435			
Dbq30 Hit something when backing up that you had not previously seen.	.333			
Dbq31 Fail to notice someone stepping out from behind a bus or parked vehicle until it is nearly too late.	.678			
Dbq35 Cut the corner on a left-hand turn and have to swerve violently to avoid an oncoming vehicle.	.675			
Dbq36 Get into the wrong lane to make a left or right hand turn at an intersection.	.414			
Dbq38 Fail to give right of way when a bus is signaling its intention to pull out.	.401			
Dbq39 Ignore yield signs and narrowly avoid colliding with traffic that has the right of way.	.601			
Dbq40 Fail to check your mirror before pulling out, changing lanes, turning etc.	.385			
Dbq41 Attempt to overtake a vehicle that you hadn't noticed was signaling its intention to turn left.	.679			
Dbq45 Fail to notice pedestrians crossing when turning into a side-street from a main	.578			

road.				
Dbq49 Misjudge your crossing interval when turning left and narrowly miss colliding with an oncoming vehicle.	.640			
Dbq1 Check your speedometer and discover that you are unknowingly traveling faster than the legal limit.		.524		
Dbq3 Become impatient with a slow driver in the outer lane and overtake on the inside.		.798		
Dbq4 Drive as fast along country roads at night with headlights on low as on high beam.		.578		
Dbq6 Drive especially close or 'flash' the car in front as a signal to drive faster or get out of your way.		.460		
Dbq8 Get distracted or preoccupied, realize belatedly that the vehicle ahead has slowed, and have to slam on the brakes to avoid a collision.		.504		
Dbq15 Stuck behind a slow moving vehicle on a two-lane highway, you are driven by frustration to try to over-take in risky circumstances.		.617		
Dbq20 Deliberately disregard the speed limits late at night or very early in the morning.		.438		
Dbq34 Overtake a slow-moving vehicle on the inside lane or hard shoulder of a motorway.		.369		
Dbq44 Drive with only 'half-an-eye' on the road while looking at a roadmap, dialing/text messaging on a cell phone, changing a cassette/CD or radio channel.		.366		
Dbq7 Forget where you left your car in a multi-level car park.			.404	
Dbq12 "Wake up" to realize that you have no clear recollection of the road along which you have just traveled.			.337	
Dbq13 Miss your exit on a motorway and have to make a lengthy detour.			.577	
Dbq16 Intending to drive to destination 'A', you "wake up" to find yourself on route to 'B', where the latter is more the usual journey.			.521	
Dbq22 Lost in thought, you forget that your lights are on high beam until 'flashed' by another vehicle.			.382	
Dbq32 Plan your route badly, so that you meet traffic congestion you could have avoided.			.555	
Dbq 37 Fail to read the signs correctly and exit from a highway on the wrong road.			.437	
Dbq2 Lock yourself out of your car with the keys still inside.				.368

Dbq17 Take a chance and cross on lights that have turned red.	.409
Dbq21 Forget that your license/plates have expired and discover that you are driving illegally.	.464
Dbq25 Drive back from a party, restaurant, or pub, even though you realize that you may be over the legal blood-alcohol limit.	.433
Dbq28 Park in a no parking area and risk a fine.	.410
Dbq42 Deliberately drive the wrong way down a deserted one-way street	.401
Dbq43 Disregard red lights when driving late at night along empty roads.	.532
Dbq46 Get involved in unofficial 'races' with other drivers.	.413
Dbq47 'Race' oncoming vehicles for a one-car gap on a narrow or obstructed road.	.451

Note. $n = 235$

Table 15

Descriptive statistics for the DBQ-LF and DBQ-SV

Factors	Range	Mean (*SD*)
Factor 1 – DBQ-LV	0-68	11.09 (9.29)
Factor 1 – DBQ-SV	0-19	2.65 (2.75)
Factor 2 - DBQ-LV	0-34	12.50 (6.96)
Factor 2 - DBQ-SV	0-21	6.87 (4.40)
Factor 3 – DBQ-LV	0-26	7.16 (4.64)
Factor 3 – DBQ-SV	0-19	4.67 (3.45)
Factor 4 - DBQ-LV	0-21	3.86 (4.23)
Factor 4 - DBQ-SV	0-14	2.30 (2.80)

* DBQ-LV = Driving Behaviour Questionnaire – Long Version; DBQ-SV = Driving Behaviour Questionnaire – Short Version; SD = Standard Deviation; $n = 235$.

Table 16

Item-total statistics for the DBQ-SV

Factor (items)	Item-total correlation	Cronbach's α if item deleted
1. Errors		
DBQ24 "Nearly hit car in front"	.67	.91
DBQ27 "Fail to notice pedestrian"	.68	.91
DBQ41 ""Overtake a vehicle"	.65	.91
DBQ45 "Fail to notice pedestrian"	.67	.91
DBQ49 "Misjudge crossing interval"	.65	.91
2. Emotional Violations		
DBQ3 "Impatient with slow driver"	.64	.82
DBQ4 "Drive fast with headlights on low at night"	.61	.82
DBQ8 "Preoccupied and slam on brakes"	.57	.83
DBQ15 "Frustration causing you to over-take in risky situation"	.65	.82
DBQ20 "Deliberately disregard speed limit"	.57	.83
3. Absent-Mindedness		
DBQ12 "Realize no clear recollection of road"	.45	.80
DBQ13 "Miss your exit"	.59	.78
DBQ16 "Wake up and find self on wrong route"	.68	.76
DBQ32 " Plan route badly	.52	.78
DBQ37 "Exit from highway on wrong road"	.50	.79
4. Reckless Violations		
DBQ17 "Cross lights on red"	.53	.74
DBQ21 "Drive when license/plates expired"	.52	.75

DBQ25 "Drive home after BAC over limit"	.46	.76
DBQ28 "Park in no parking area"	.52	.75
DBQ43 "Disregard red lights"	.49	.75

Note. DBQ-SV = Driving Behaviour Questionnaire – Short Version; $N = 235$.

Table 17

Correlations (*r*) and t-tests between the DBQ-LV and DBQ-SV

Factors	*r* [95% CI]	*t-score*	*df*	*p*
Factor 1	.91 [.89, .93]	0.14	214	.887
Factor 2	.94 [.93, .95]	-0.59	223	.559
Factor 3	.94 [.92, .95]	-0.21	227	.831
Factor 4	.93 [.91, .94]	1.55	225	.121

Note. DBQ-LV = Driving Behaviour Questionnaire – Long Version; DBQ-SV = Driving Behaviour Questionnaire – Short Version; CI = Confidence Interval

Table 18

Correlations (*r*) between the DBQ-LV and DBQ-SV with the BDI-II total score

Factors	Total sample[1] *r* [95% CI]	Women[2] *r* [95% CI]	Men[3] *r* [95% CI]
Factor 1 – DBQ-LV	**.17*** [.04, .30]	.07 [-.08, .22]	**.51**** [.23, .71]
Factor 1 – DBQ-SV	**.17*** [.04, .30]	.10 [-.05, .24]	**.41**** [.12, .63]
Factor 2 - DBQ-LV	.12 [-.01, .24]	.10 [-.05, .24]	.18 [-.13, .46]
Factor 2 - DBQ-SV	.13 [.00, .25]	.12 [-.02, .27]	.16 [-.14, .44]
Factor 3 – DBQ-LV	**.21**** [.09, .33]	**.19**** [.05, .33]	.21 [-.09, .48]
Factor 3 – DBQ-SV	**.22**** [.09, .34]	**.22**** [.08, .35]	.16 [-.14, .44]
Factor 4 - DBQ-LV	**.21**** [.08, .33]	.12 [-.03, .26]	**.42**** [.12, .64]
Factor 4 - DBQ-SV	**.18**** [.05, .30]	.09 [-.06, .23]	**.33*** [.03, .58]
Total Score - DBQ-LV	**.19**** [.07, .33]	.11 [-.04, .26]	**.48**** [.18, .70]
Total Score - DBQ-SV	**.21**** [.08, .33]	**.16*** [.01, .30]	**.34*** [.03, .59]

Note. DBQ-LV = Driving Behaviour Questionnaire – Long Version; DBQ-SV = Driving Behaviour Questionnaire – Short Version; BDI-II = Beck Depression Inventory II; [1]N = 233; [2]*n* = 45; [3]*n* = 186

*$p < .05$; ** $p < .01$

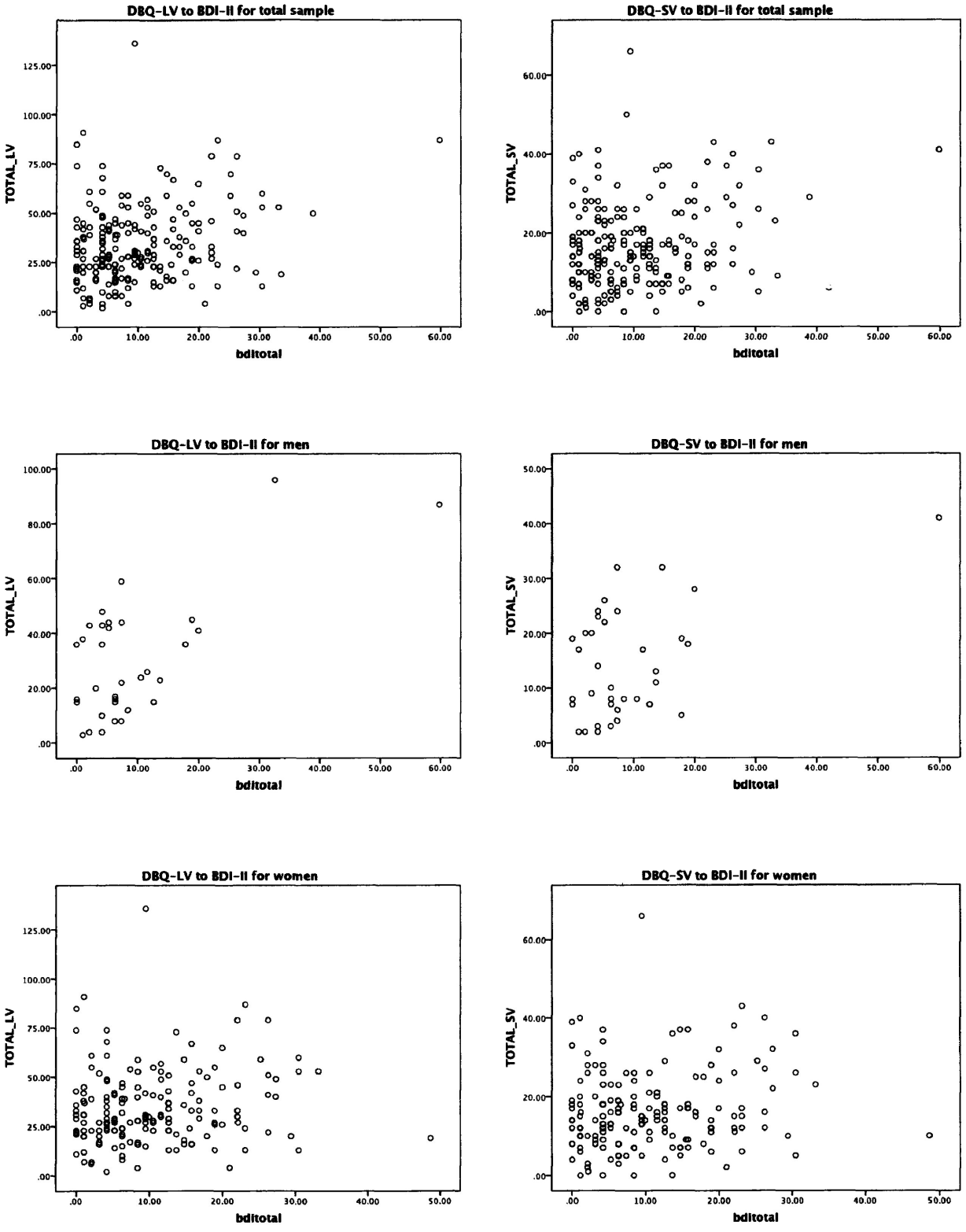

Figure 2. Scatterplots of the DBQ- LV and DBQ-SV and BDI-II total scores across the total sample (Total N =229), men (n = 45), and women (n = 184).

Table 19

Correlations (*r*) and t-tests between the DBQ-LV and DBQ-SV and MRT

Factors	*r* [95% CI]			*t-score*	*df*	*p*
	DBQ-LV & MRT	*DBQ-SV & MRT*	*DBQ-LV & DBQ-SV*			
Factor 1	.04 [-.26, .34]	.03[-.27,.33]	.91 [-.11, .14]	.181	40	.857
Factor 2	.07 [-.24, .37]	.10[-.21,.39]	.94 [-.13, .08]	-.526	39	.602
Factor 3	.06 [-.25, .35]	.05[-.25,.35]	.94 [-.11, .11]	.053	40	.958
Factor 4	.25 [-.05, .52]	.23[-.08,.50]	.93 [-.09, 14]	.423	39	.674
Total	.25 [-.07, .51]	.26[-.06,.52]	.96 [-.11, .09]	-.239	38	.812

Note. DBQ-LV = Driving Behaviour Questionnaire – Long Version; DBQ-SV = Driving Behaviour Questionnaire – Short Version; MRT = Manitoba Road Test; CI = Confidence Interval

Violations) was not included as it was not hypothesized that depressive and anxious symptoms would be related to reckless behaviour. Centering, the practice of subtracting a constant from predictors before fitting the model, was applied to age. The intercept is a representation of the value of the outcome when all of the predictors are valued at zero. Centering the predictors is important as it changes the meaning of an intercept due to the fact that some predictors do not logically have a value of zero (e.g., an age of zero; Tabachnick & Fidell, 2001). Therefore, age was centered at 18, which was the minimum value. See figure 3 for residual plots of predicted value of factors 1 to 3 and DBQ total with unstandardized residuals. See Figure 4 for histograms of unstandardized residuals.

Factor 1 (Errors). Tables 20 and 21 display results of two regression models predicting Factor 1 (Errors). The first model, which includes the BAI as a predictor, predicting Factor 1 (Errors) was not statistically significant, $F(6, 228) = 1.64$, $p = .14$, and accounted for 4% (2% adjusted) of the variability in Factor 1. No individual variables significantly predicted self-reported unsafe driving behaviour on factor 1 (see Table 20). Therefore, a second model was performed without the BAI as a predictor variable. The second model predicting Factor 1 (Errors) was not statistically significant, $F(8, 228) = 1.62$, $p = .12$, and accounted for 6% (2% adjusted) of the variability in Factor 1 (see Table 21).

Factor 2 (Emotional Violations). Tables 22 and 23 display results of two regression models predicting emotional violations. The first model includes the BAI as a predictor variable, and was not statistically significant, $F(6, 228) = .98$, $p = .44$. This model accounted for 3% (0% adjusted) of the variability in Factor 2 (see Table 22). A second model was performed without the BAI as a predictor variable. The second model was also not statistically significant, $F(8, 228) = 1.08$, $p = .38$ and accounted for 4% (0% adjusted) of the variability in Factor 2 emotional

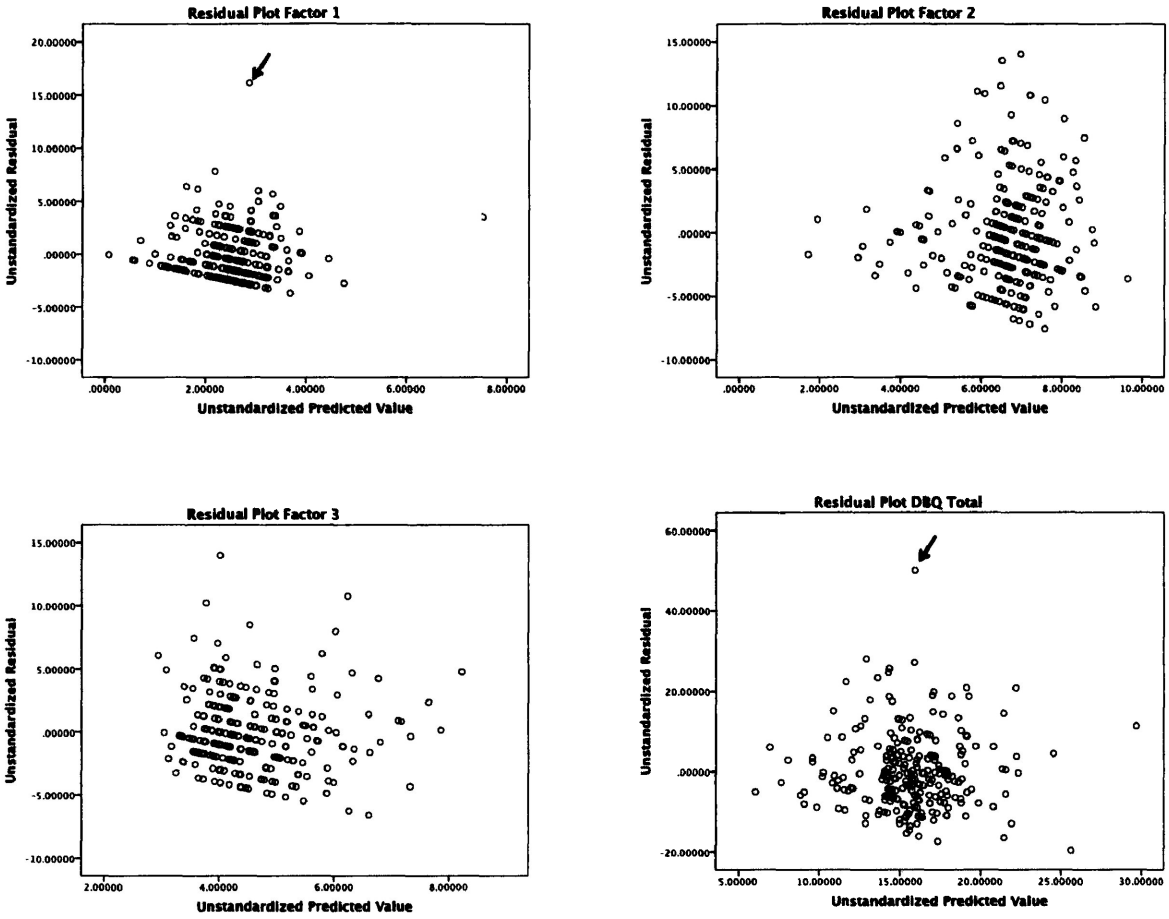

Figure 3. Plots of the predicted value of factors 1, 2, 3, and DBQ total with unstandardized residuals. The arrows represent the same participant (Factor 1 D = 0.11; DBQ total D = 0.07)

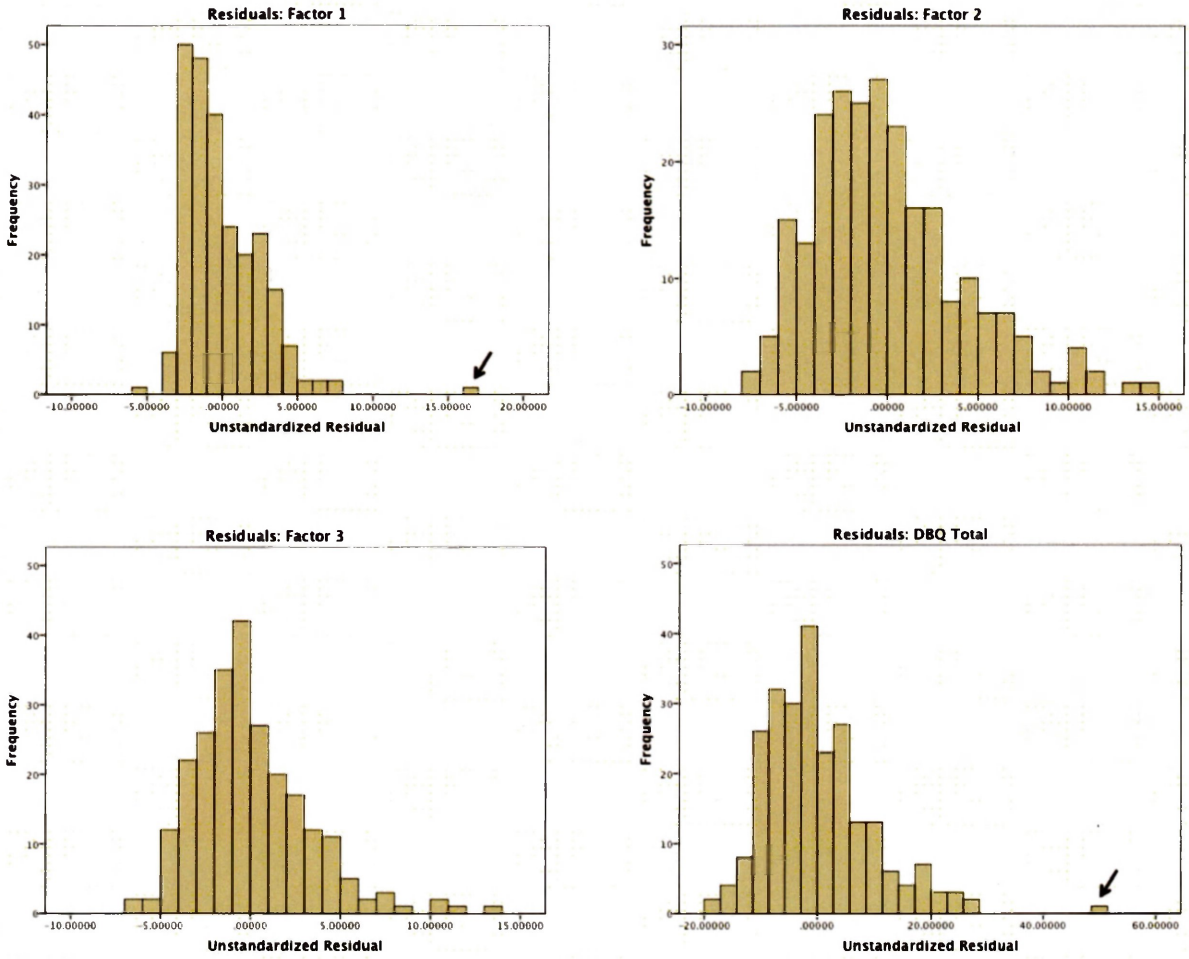

Figure 4. Histograms of unstandardized residuals for factors and DBQ total. The arrows represent the same participant (Factor 1 D = 0.11; DBQ total D = 0.07).

Table 20

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, BAI Total, and Antidepressant use on Factor 1 (Errors; N = 229) pooled data

Variable (Pooled data)	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	0.01	0.05	[-0.10, 0.10]	0.03	.98
sex (Male = 1, Female = 0)	-0.27	0.47	[-1.19, 0.64]	-0.58	.56
BDI-II C/A	0.02	0.04	[-0.07, 0.11]	0.46	.65
BDI-II S	0.09	0.10	[-0.10, 0.29]	0.95	.34
BAI Total	0.02	0.03	[-0.03, 0.07]	0.88	.38
Antidepressant use	-0.96	0.63	[-2.20, 0.29]	-1.51	.13

Note. R^2 = .04; Adjusted R^2 = .02; [a] BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; BAI = Beck Depression Inventory. The raw score for age was centered on a value of 18.
*p <.05. ** p <.01.

Table 21

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, and Antidepressant use on Factor 1 (Errors; N = 229) pooled data

Variable (Pooled data)	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	-0.01	0.05	[-0.11, 0.09]	-0.21	.84
sex (Male = 1, Female = 0)	-1.12	0.70	[-2.48, 0.24]	-1.61	.11
BDI-II C/A	0.02	0.04	[-0.02, 0.07]	0.55	.63
BDI-II S	0.07	0.11	[-0.04, 0.17]	0.64	.49
antidepressant use	-0.95	0.66	[-1.61, -0.29]	-1.45	.15
sexBYBDI-II C/A	0.07	0.11	[-0.15, 0.30]	0.66	.51
sexBYBD-II S	0.06	0.29	[-0.51, 0.62]	0.19	.85
sexBYantidep	1.80	2.11	[-2.37, 5.89]	0.83	.40

Note. R^2 = .06; Adjusted R^2 = .02; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; antidep = any antidepressant; [a]The raw score for age was centered on a value of 18.

Table 22

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, BAI Total, and Antidepressant use on Factor 2 (Emotional Violations; N = 229) pooled data

Variable (Pooled data)	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	-0.09	0.08	[-0.24, 0.07]	-1.07	.28
sex (Male = 1, Female = 0)	-0.52	0.76	[-2.01, 0.98]	-0.68	.50
BDI-II C/A	-0.01	0.07	[-0.14, 0.14]	-0.02	.99
BDI-II S	0.20	0.16	[-0.12, 0.52]	1.22	.22
BAI Total	0.01	0.04	[-0.07, 0.09]	0.25	.81
Antidepressant use	-0.36	1.04	[-2.39, 1.67]	-0.35	.73

Note. $R^2 = .03$; Adjusted $R^2 = 0$; [a] BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; BAI = Beck Depression Inventory. The raw score for age was centered on a value of 18.

violations (see Table 23). Similar to the model predicting Factor 1, no significant individual predictors of interest emerged.

Factor 3 (Absent-Mindedness). The results of two regression models predicting absent-mindedness (Tables 24 and 25) were generated. The first model was statistically significant, $F(6, 228) = 2.73, p < .01$, and accounted for 7% (4% adjusted) of the variability in Factor 3. Age ($B = .12$) was a significant predictor of absent-mindedness, such that increasing age was associated with an increase in absent-minded driving behaviour. The cognitive/affective component of the BDI-II ($B = .12$) was also a significant predictor of absent-mindedness suggesting that increased cognitive/affective impairments were associated with absent-minded driving behaviour. The second model predicting Factor 3 (Absent-mindedness) was also statistically significant, $F(8, 228) = 2.15, p = .03$, and accounted for 7% (4% adjusted) of the variability in Factor 3 (Absent-mindedness). Similar to model 1, Age ($B = .12$) and the cognitive/affective component of the BDI-II ($B = .12$) were significant predictors of absent-mindedness. These results suggest increasing age and increased cognitive/affective impairments are associated with absent-mindedness on the DBQ.

Sum of factors. The results of two regression models predicting the sum of the four factors (Tables 26 and 27) were generated. The first model includes the BAI as a predictor variable, which was not statistically significant, $F(6, 228) = 1.85, p = .09$, and accounted for 5% (2% adjusted) of the variability in the sum of the four factors. The second model predicting the sum of the four factors was also not statistically significant, $F(8, 228) = 1.52, p = .15$, and accounted for 5% (2% adjusted) of the variability in the sum of the four factors. Similar to the models predicting Factors 1 and 2, no significant individual predictors of interest emerged.

Hypothesis 3. We expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would be related to increased impairments on measures of attention (CRSD-ANT), executive functioning (TMT), visual perceptual ability (MVPT-3), and visual information processing (UFOV).

CRSD-ANT. The results of four regression models predicting the CRSD-ANT median response time (RT), alerting, orienting, and conflict scores (Tables 28 to 31) were generated. Median RT was used instead of mean RT because raw RT distributions are known to be positively skewed (Williams & Zimmerman, 1996). None of the overall models with ANT variables as outcomes were statistically significant (See *F*-ratios plus R^2 and adjusted R^2 values in Tables 28 to 31). However, the PHQ-9 (B = -2.48) and BDI-II Cognitive Affective (B = 3.45) scores were significantly associated with the alerting score from the CRSD-ANT. See figure 5 for histograms of unstandardized residuals. See figure 6 for residual plots of CRSD-ANT median response time (RT), alerting, orienting, and conflict scores with unstandardized residuals.

TMT. The results of regression models predicting the TMT-A or TMT-B total score (see Table 32 and 33) were generated. The models with the TMT-A or TMT-B total score as an outcome were not statistically significant (See *F*-ratios plus R^2 and adjusted R^2 values in Tables 32 and 33). See figures 7 and 8 for histograms of unstandardized residuals and residual plots of the TMT-A and TMT-B with unstandardized residuals, respectively.

MVPT-3. None of the models with MVPT-3 variables as outcomes were statistically significant (See *F*-ratios plus R^2 and adjusted R^2 values in Tables 34 and 35). The BDI-II Cognitive Affective scores were significantly associated with the standard score (B = 2.02) and errors (B = -.65) score from the MVPT-3. See Figure 9 for histograms of unstandardized residuals.

Table 23

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, and Antidepressant use on Factor 2 (Emotional Violations; N = 229) pooled data

Variable	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	-0.09	0.08	[-0.25, 0.06]	-1.16	.25
sex (Male = 1, Female = 0)	-0.96	1.14	[-3.18, 1.26]	-0.85	.40
BDI-II C/A	0.04	0.07	[-0.03, 0.11]	0.64	.67
BDI-II S	0.12	0.17	[-0.05, 0.30]	0.71	.55
Antidepressant use	-0.87	1.08	[-1.90, 0.21]	-0.81	.42
sexBYBDI-II C/A	-0.24	0.19	[-0.62, 0.14]	-1.22	.22
sexBYBDI-II S	0.53	0.48	[-0.41, 1.47]	1.10	.27
sexBYantidep	4.94	3.44	[-1.81, 11.69]	1.43	.15

Note. $R^2 = .04$; Adjusted $R^2 = 0$; [a] BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; antidep = any antidepressant; The raw score for age was centered on a value of 18.

Table 24

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, BAI Total, and Antidepressant use on Factor 3 (Absent-Mindedness; N = 229) pooled data

Variable (Pooled data)	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	0.12*	0.06	[0.01, 0.24]	1.97	.05
sex (Male = 1, Female = 0)	-0.81	0.57	[-1.88, 0.26]	-1.42	.16
BDI-II C/A	0.12*	0.05	[0.01, 0.23]	2.21	.03
BDI-II S	-0.05	0.12	[-0.29, 0.19]	-0.42	.68
BAI Total	-0.03	0.03	[-0.07, 0.06]	-0.10	.92
Antidepressant use	-0.71	0.78	[-2.23, 0.81]	-0.92	.36

Note. R^2 = .07; Adjusted R^2 = .04; [a] BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; BAI = Beck Depression Inventory. The raw score for age was centered on a value of 18.
*p <.05. ** p <.01.

Table 25

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, and Antidepressant use on Factor 3 (Absent-mindedness; N = 229) pooled data

Variable	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	0.12*	0.06	[0.01, 0.24]	2.04	.04
sex (Male = 1, Female = 0)	-0.41	0.86	[-2.08, 1.27]	-0.47	.64
BDI-II C/A	0.12*	0.05	[0.07, 0.18]	2.38	.02
BDI-II S	-0.03	0.13	[-0.17, 0.10]	-0.26	.79
Antidepressant use	-0.64	0.81	[-2.29, 1.00]	-0.79	.43
sexBYBDI-II CA	-0.02	0.14	[-0.30, 0.26]	-0.16	.87
sexBYBDI-II S	-0.05	0.35	[-0.74, 0.64]	-0.14	.89
sexBYantidep	-1.35	2.60	[-6.44, 3.74]	-0.52	.60

Note. R^2 = .07; Adjusted R^2 = .04; [a] BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; antidep = any antidepressant; The raw score for age was centered on a value of 18.
*p <.05. ** p <.01.

Table 26

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, BAI Total, and Antidepressant use on the sum of the four factors (DBQ-Total; N = 229) pooled data

Variable (Pooled data)	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	0.19	0.19	[-0.17, 0.55]	1.02	.31
sex (Male = 1, Female = 0)	-1.08	1.76	[-4.52, 2.36]	-0.61	.54
BDI-II C/A	0.18	0.17	[-0.14, 0.51]	1.10	.27
BDI-II S	0.28	0.37	[-0.46, 1.01]	0.74	.46
BAI Total	0.03	0.10	[-0.16, 0.23]	0.35	.73
Antidepressant use	-2.87	2.38	[-7.55, 1.80]	-1.20	.23

Note. R^2 = .05; Adjusted R^2 = .02; [a] BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; BAI = Beck Depression Inventory. The raw score for age was centered on a value of 18.

Table 27

Standard multivariable regression of age, sex, BDI-II Cognitive/Affective, BDI-II Somatic, and Antidepressant use on the pooled data of the sum of the four factors (N = 229) pooled data

Variable	*B*	*SE B*	*95% CI*	*t-score*	*p-value*
age[a]	0.18	0.18	[-0.18, 0.54]	0.99	.32
sex (Male = 1, Female = 0)	-3.11	2.62	[-8.24, 2.03]	-1.19	.24
BDI-II C/A	0.25	0.16	[0.09, 0.41]	1.55	.32
BDI-II S	0.09	0.40	[-0.31, 0.49]	0.23	.82
Antidepressant use	-3.32	2.49	[-8.42, 1.76]	-1.34	.18
sexBYBDI-II CA	-0.31	0.45	[-1.18, 0.57]	-0.69	.49
sexBYBDI-II S	1.10	1.11	[-1.07, 3.27]	1.00	.32
sexBYantidep	5.89	7.96	[-9.70, 21.49]	0.74	.46

Note. $R^2 = .05$; Adjusted $R^2 = .02$; [a] BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; antidep = any antidepressant; The raw score for age was centered on a value of 18.

Table 28

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the CRSD-ANT median response time

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-sore*	*p*
PHQ-9	-1.76	2.67	-0.13	[-7.16, 3.65]	*-0.66*	*.52*
BDI-II C/A	2.11	3.16	0.15	[-4.27, 8.49]	*0.67*	*.51*
BDI-II S	-3.70	3.99	-0.18	[-11.77, 4.36]	*-0.93*	*.34*

Note. $F(3, 39) = 0.54$, $p = .66$; $R^2 = .04$; Adjusted $R^2 = -.03$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

Table 29

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the CRSD-ANT alerting score

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	-2.48	1.26	-0.38*	[-5.04, .08]	-1.96	.05
BDI-II C/A	3.45	1.49	0.48*	[0.43, 6.47]	2.31	.03
BDI-II S	-1.75	1.89	-0.17	[-5.57, 2.07]	-.93	.36

Note. $F(3, 39) = 2.15$, $p = .11$; $R^2 = .14$; Adjusted $R^2 = .07$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

Table 30

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the CRSD-ANT orienting score

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	-1.97	1.68	-0.22	[-5.37, 1.44]	-1.17	.25
BDI-II C/A	2.38	1.99	0.25	[-1.64, 6.40]	1.49	.24
BDI-II S	3.74	2.51	0.23	[-1.34, 8.82]	1.20	.15

Note. $F(3, 39) = 2.17, p = .11$; $R^2 = .14$; Adjusted $R^2 = .08$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

Table 31

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the CRSD-ANT conflict score

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	1.98	1.49	0.27	[-1.04, 5.00]	1.33	.19
BDI-II C/A	0.31	1.76	0.04	[-3.26, 3.87]	0.17	.86
BDI-II S	-1.70	2.23	-0.15	[-6.21, 2.80]	-0.77	.45

Note. $F(3, 39) = 0.88$ $p = .46$; $R^2 = .06$; Adjusted $R^2 = 0$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

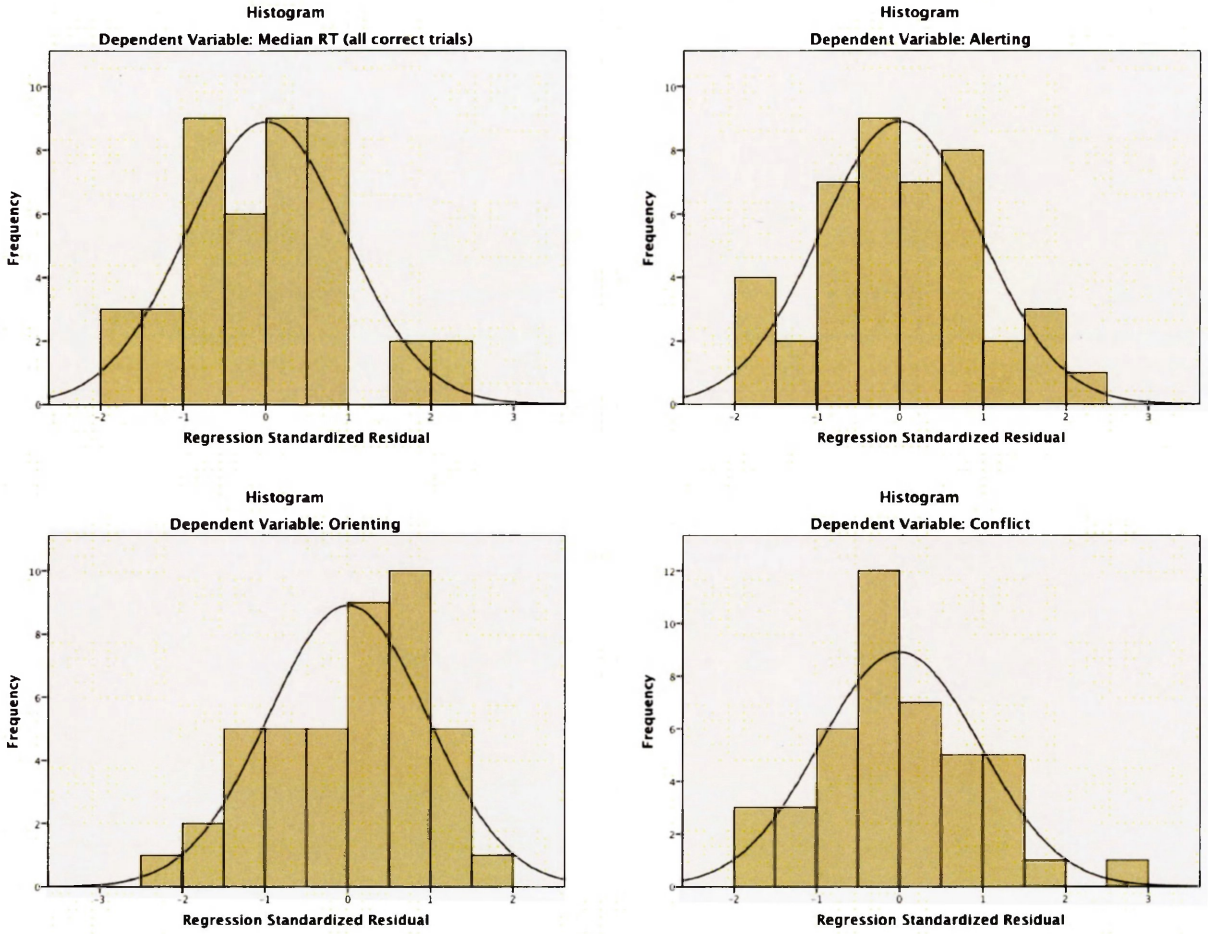

Figure 5. Histograms of unstandardized residuals for the CRSD-ANT median response time (RT), alerting, orienting, and conflict scores

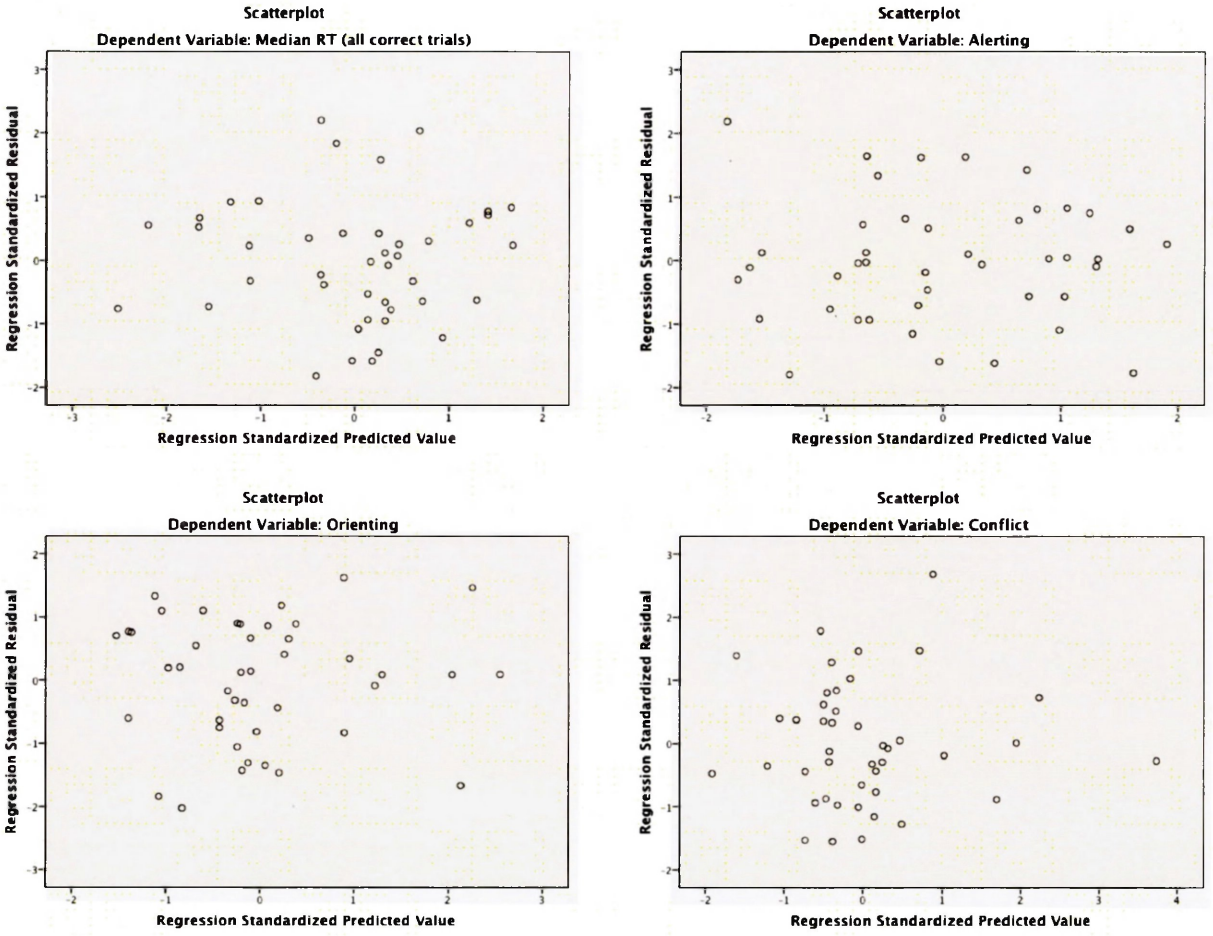

Figure 6. Plots of the predicted value of the CRSD-ANT median response time (RT), alerting, orienting, and conflict scores with unstandardized residuals.

Table 32

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the TMT-A score

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	-0.36	0.57	-0.13	[-1.51, 0.79]	-0.64	.53
BDI-II C/A	0.28	0.67	0.09	[-1.08, 1.63]	0.41	.68
BDI-II S	0.17	0.85	0.04	[-1.54, 1.88]	0.20	.84

Note. $F(3, 39) = 0.15$ $p = .93$; $R^2 = .01$; Adjusted $R^2 = -.06$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

Table 33

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the TMT-B score

Variable	*B*	*SE B*	*β*	*95% CI*	*t-score*	*p*
PHQ-9	-1.97	1.31	-0.29	[-4.61, 0.68]	-1.50	.14
BDI-II C/A	2.17	1.55	0.30	[-0.96, 5.30]	1.40	.17
BDI-II S	2.13	1.95	0.21	[-1.82, 6.08]	1.09	.28

Note. $F(3, 39) = 1.83$ $p = .16$; $R^2 = .12$; Adjusted $R^2 = .06$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

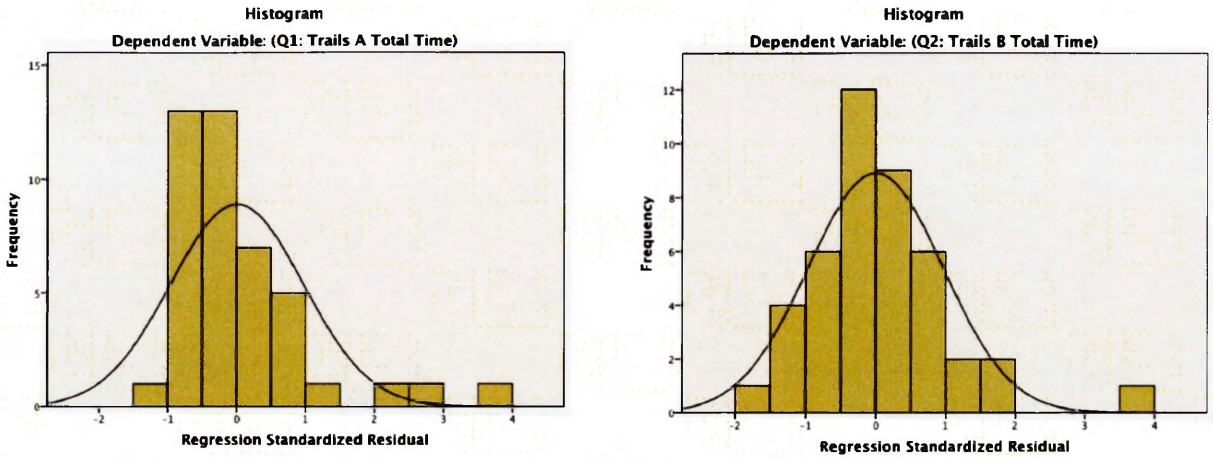

Figure 7. Histograms of unstandardized residuals for the TMT-A and TMT-B.

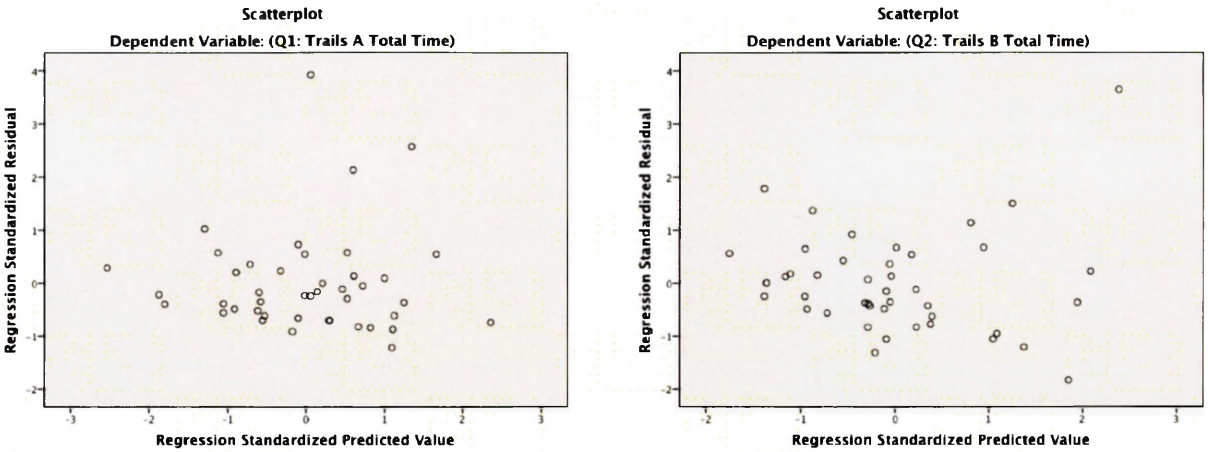

Figure 8. Plots of the predicted value of TMT-A and TMT-B with unstandardized residuals.

Table 34

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the MVPT-3 standard score

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	-1.36	0.86	-0.30	[-3.10, 0.38]	-1.58	.12
BDI-II C/A	2.02	1.02	0.42*	[-0.04, 4.08]	1.98	.05
BDI-II S	-2.06	1.29	-0.30	[-4.66, 0.54]	-1.60	.12

Note. $F(3, 39) = 2.06$, $p = .12$; $R^2 = .14$; Adjusted $R^2 = .07$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic; MVPT-3 = Motor-Free Visual Perception Test: Third edition

*$p < .05$. ** $p < .01$.

Table 35

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the MVPT-3 errors.

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	0.44	0.23	0.36	[-0.03, 0.91]	1.89	.07
BDI-II C/A	-0.65	0.28	-0.49*	[-2.35, 0.02]	-2.35	.02
BDI-II S	0.52	0.35	0.28	[-0.18, 1.23]	1.50	.14

Note. $F(3, 39) = 2.47$, $p = .08$; $R^2 = .16$; Adjusted $R^2 = .10$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

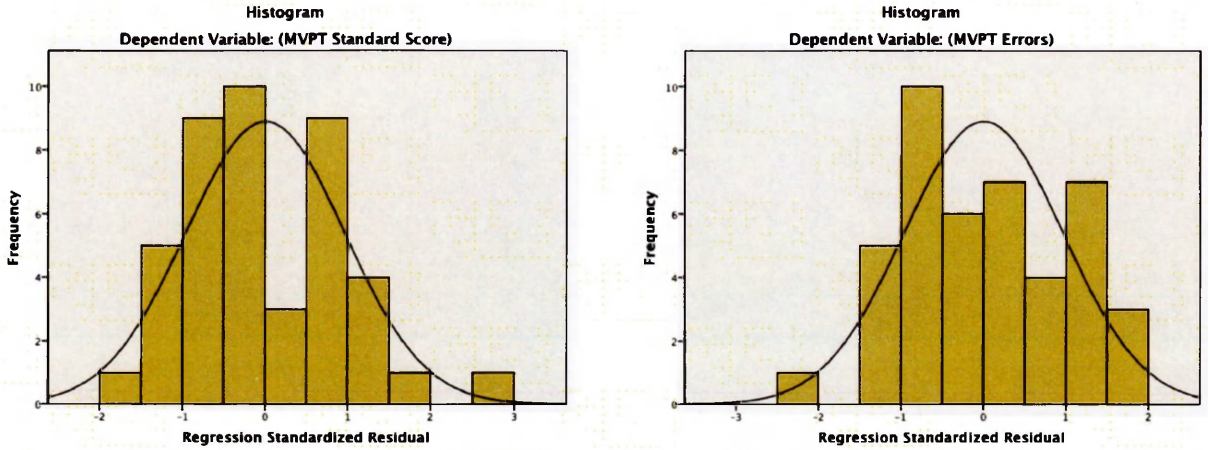

Figure 9. Histograms of unstandardized residuals for the MVPT standard score and MVPT errors.

See Figure 10 residual plots of MVPT-3 errors and MVPT-3 standard scores with unstandardized residuals.

UFOV. The results of two regression models predicting the UFOV sum score and UFOV divided attention score (Tables 36 and 37) were generated. None of the models with UFOV variables as outcomes were statistically significant (See *F*-ratios plus R^2 and adjusted R^2 values in Tables 36 and 37). See Figures 11 and 12 for histograms of unstandardized residuals and residual plots, respectively.

Hypothesis 4. We expected to observe that depressive symptoms would predict poorer driving performance on the driving simulator. However, given that only 2 participants who participated in the laboratory session were taking an antidepressant (both were taking an SSRI), we excluded antidepressant use from these analyses.

MRT. The results of one regression model predicting the MRT demerit points score (Table 38) was generated. The model with the MRT demerit points score as an outcome was not statistically significant (See *F*-ratios plus R^2 and adjusted R^2 values in Table 38). See also figures 13 and 14 for histograms of unstandardized residuals and residual plots, respectively.

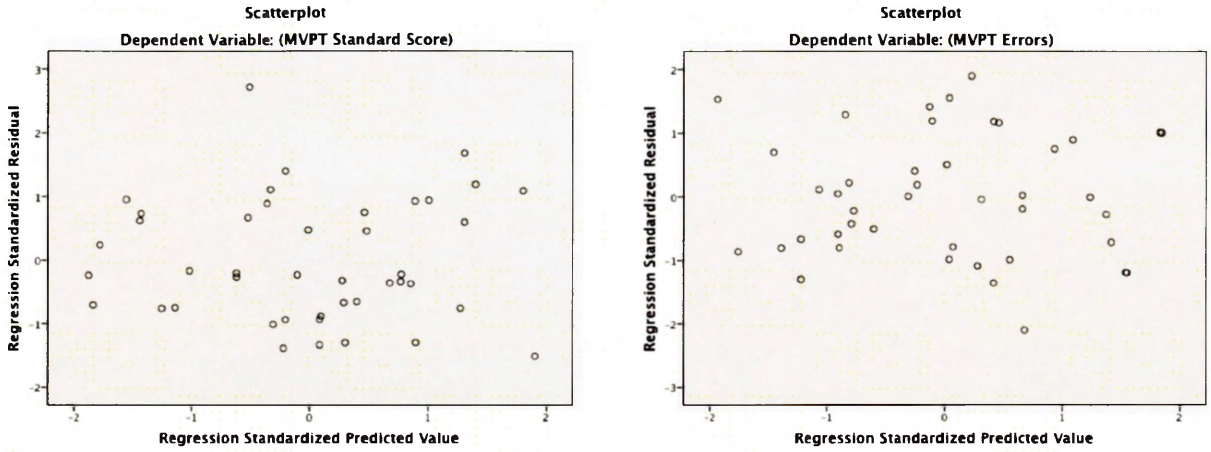

Figure 10. Plots of the predicted value of MVPT standard score and MVPT errors with unstandardized residuals.

Table 36

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the UFOV sum score

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	1.22	1.40	0.18	[-1.60, 4.04]	0.88	.39
BDI-II C/A	-1.32	1.65	-0.18	[-4.66, 2.01]	-0.80	.42
BDI-II S	1.61	2.08	0.15	[-2.61, 5.82]	0.77	.45

Note. $F(3, 39) = .56$, $p = .65$; $R^2 = .04$; Adjusted $R^2 = -0.03$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
*$p < .05$. ** $p < .01$.

Table 37

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the UFOV divided attention score

Variable	*B*	*SE B*	*β*	*95% CI*	*t-score*	*p*
PHQ-9	-0.34	0.45	-0.15	[-1.24, 0.56]	-0.76	.45
BDI-II C/A	-0.28	0.51	-0.12	[-1.34, 0.79]	-0.53	.60
BDI-II S	1.12	0.67	0.32	[-0.23, 2.46]	1.68	.10

Note. $F(3, 39) = 1.02, p = .39$; $R^2 = .07$; Adjusted $R^2 = .01$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
$^*p < .05$. $^{**}p < .01$.

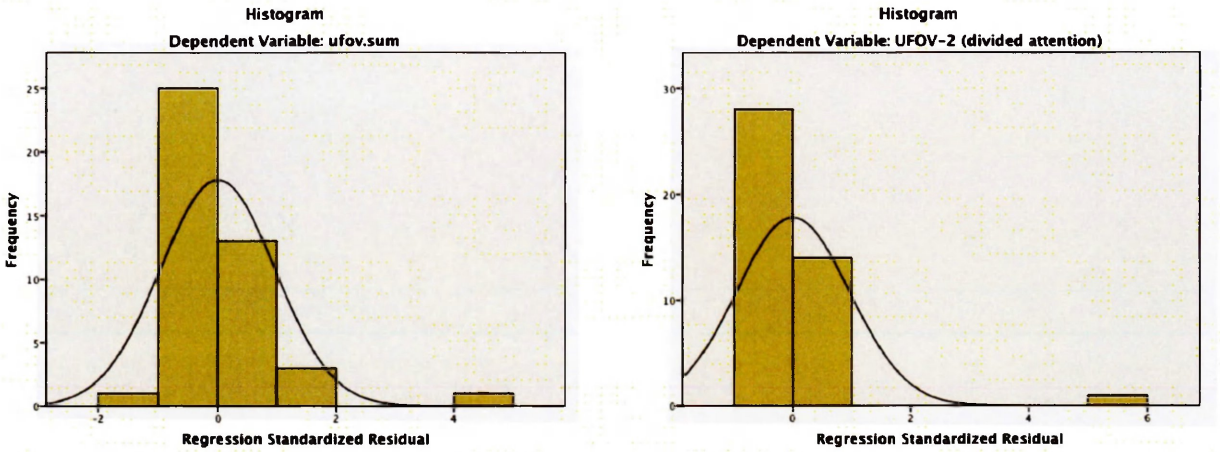

Figure 11. Histograms of unstandardized residuals for the UFOV sum and UFOV divided attention scores.

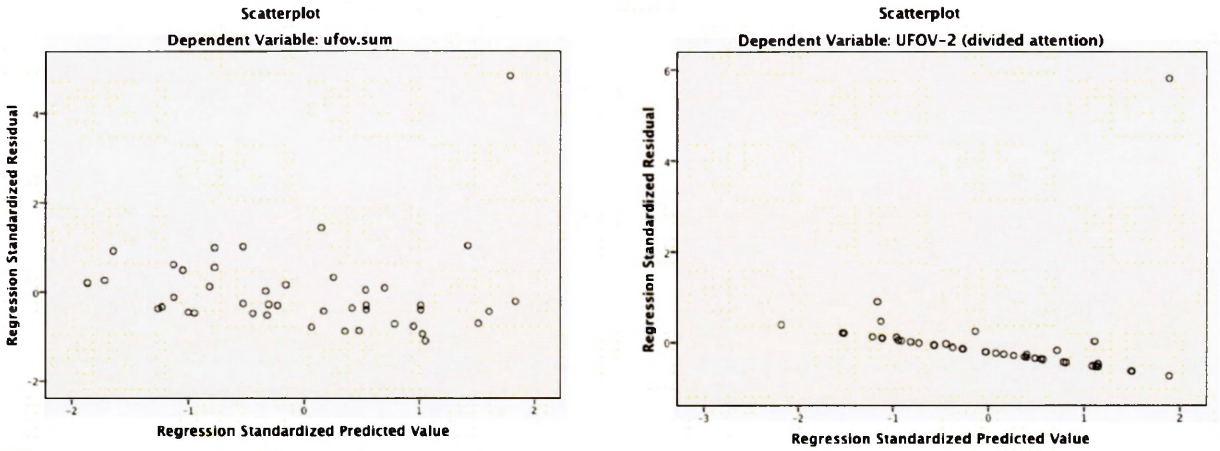

Figure 12. Plots of the predicted value of UFOV sum and UFOV divided attention score with unstandardized residuals.

Table 38

Ordinary least squares regression of PHQ9, BDI-II Cognitive/Affective and BDI-II Somatic for the MRT (Demerit points) score

Variable	*B*	*SE B*	*ß*	*95% CI*	*t-score*	*p*
PHQ-9	1.64	1.66	0.20	[-1.70, 4.99]	0.99	.33
BDI-II C/A	-2.67	1.96	-0.30	[-6.62, 1.29]	-1.36	.18
BDI-II S	0.45	2.47	0.04	[-4.55, 5.44]	0.18	.86

Note. $F(3, 39) = 0.70$, $p = .56$; $R^2 = .05$; Adjusted $R^2 = -0.02$; $N = 43$; PHQ-9 = Patient Health Questionnaire – 9; BDI-II C/A = Beck Depression Inventory – II Cognitive Affective; BDI-II S = Beck Depression Inventory – II Somatic.
$^*p < .05$. $^{**}p < .01$.

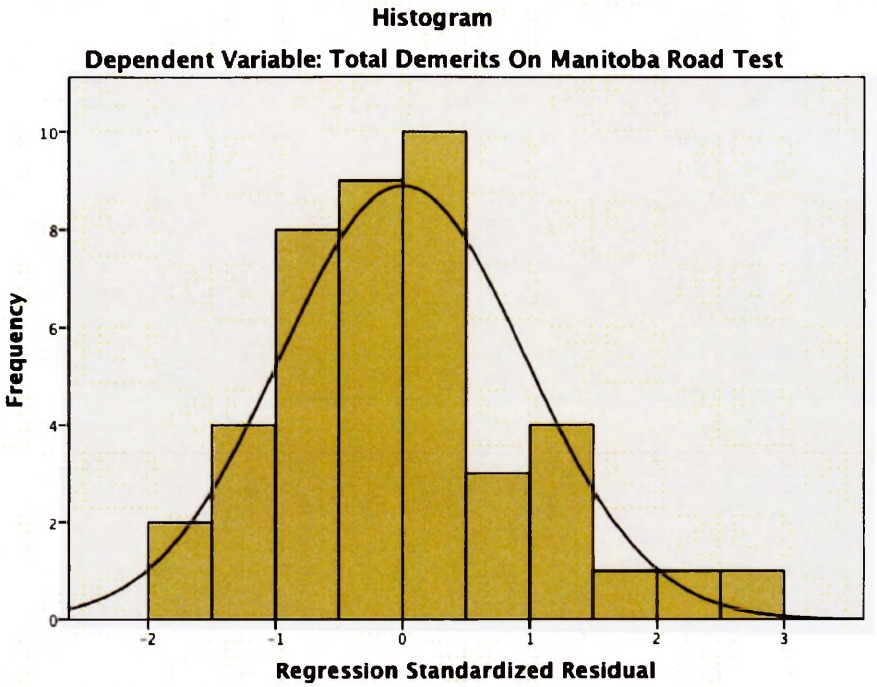

Figure 13. Histogram of unstandardized residuals for the MRT.

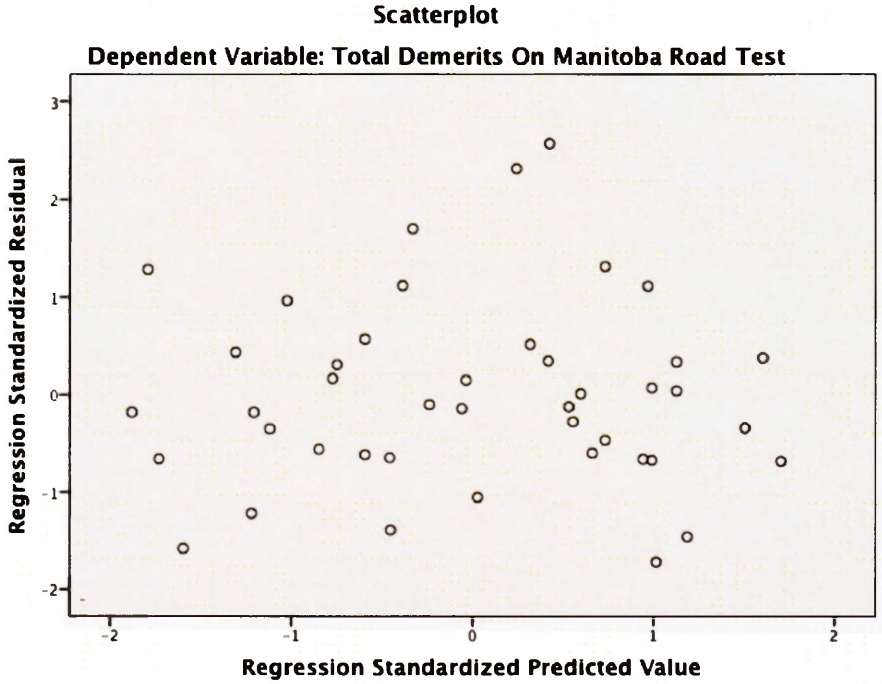

Figure 14. Plot of the predicted value of the MRT with unstandardized residuals.

Discussion

Research on the topic of the influence of Major Depressive Disorder (MDD) and the antidepressants used to treat MDD on driving performance has reported contradictory results (see Barbone et al., 1998; Leveille et al., 1994; Wingen et al., 2005). MDD can lead to cognitive and psychomotor disturbances such as impairments in executive functions, attentional deficits, restlessness, motivational deficits, and slowed thought processes (APA, 2013; Brebion et al., 1997; Egeland et al., 2003; Hill et al., 2004; Ravnkilde et al., 2002), which can be detrimental to the task of driving. Likewise, antidepressant medications can cause deficits to psychomotor skills (Ramaekers, 2003; Rapoport & Banina, 2007) but can also improve cognition by improving mood, attention, and executive functions in the short term (Impey & Baldwin, 2013). Therefore, it can be difficult to ascertain if any impairments in driving function are the result of antidepressant medications or MDD itself (Hetland & Carr, 2014). Therefore, it was logical to conduct additional research on this topic using an ecologically valid, a driving simulator. In addition, the factor structure of the Driver Behaviour Questionnaire (DBQ) has been inconsistent across younger age groups. To the best of the authors' knowledge, previous studies have not examined the psychometric properties of a shortened version of the DBQ in a younger Canadian sample.

Main Hypotheses

Hypothesis 1. ***We aimed to create a shortened version of the DBQ in a younger Canadian sample. We expected to observe good to excellent psychometric properties in this shorter version of the DBQ. Murphy and Davidshofer (2005) and Streiner (2003) recommend reliability estimates of .80, and estimates above .90 to be redundant. Furthermore, Murphy and Davidshofer (2005) recommend validity coefficients of .30 and higher.***

Previous research on the factor structure of the DBQ with younger participants has revealed contradictory findings (see Martinussen et al., 2013 & Rimmö, 2002). Similar to Martinuseen and colleagues (2013), our findings supported a four-factor solution using the 50-item DBQ among Canadian participants aged 18 to 35. All factor loadings were greater than .30 (Streiner, 1994). We also created a shortened version (DBQ-SV) by selecting the five highest item-total correlations for each factor, totalling 20 items for the shortened version. The highest item-total correlations ranged from .37 to .68 across the four factors. Each of the four factors corresponds to different aspects of driver behaviour. Factor 1 is considered "Errors" and the items are delineated by perceptual, attentional, and/or information processing errors. Factor 2 can be considered "Emotional Violations" that reflect a driver's style and habits to an emotional response to a particular situation. In contrast, factor 3 corresponds to "Absent-Mindedness" or being forgetful and having poor attention while driving. Lastly, factor 4 is considered "Reckless Violations" or disrespectful violations.

Our study adds to the literature as we examined the DBQ structure in a younger Canadian population. Cordazzo, Scialfa, Bubric, and Ross (2014) conducted a principal components analysis (using *varimax* rotation) of the 36-item DBQ using two Canadian samples including a younger (M = 20.9 years; SD = 2.1 years) and an older (M = 60.6 years; SD = 13.8) sample. To the best of our knowledge, this is the only other study examining the DBQ structure in a Canadian study. In contrast to our four-factor solution, Cordazzo et al. reported a two-factor solution that explained 27.06% of the variance for errors and violations but not lapses. However, Cordazzo et al. did not compare age groups. The only reported finding pertaining to age was that age was negatively related to violations (β = -0.4, $p < .01$). Furthermore, Cordazzo et al. (2014) found that violations for the total sample were significantly related to self-reported collisions (β

= 0.3; $p < .001$). However, they further highlight that the correlations were small in magnitude and have "no predictive utility for collision risk." (p. 103). In addition, given that Cordazzo et al. relied solely on self-report data, our study adds to Cordazzo et al.'s findings as we collected simulated drive data in addition to self-report data. In contrast to Cordazzo et al.'s self-report data, our study revealed that the simulated drive total score was not significantly related to either the DBQ-LV or DBQ-SV across all four factors, including violations (Correlation coefficients ranged from .03 to .26; see Table 19).

Martinussen and Prato (2014) also aimed to examine age and the DBQ. These researchers identified sub-groups of drivers who engage in dangerous acts while driving by using the original (50-item) 3-factor structure of the DBQ including errors, lapses, and violations. One factor that contributed to the groups was age. The group of "violating unsafe drivers" was composed mostly of younger drivers. The average age of this group was 39.3 years (SD = 14.1). These drivers self-reported the lowest safety skills (M = -0.7; SD = 0.8), and highest frequency of violations (M = 1.4; SD = 0.9) on the DBQ. In addition, this group reported the highest frequency of accidents (M = 0.6; SD = 1.0), parking fines (M = 1.1; SD = 3.9), and speeding fines (M = 0.4; SD = 0.9) over the past 3 years. This study suggests a different driving profile for younger participants. However, it is important to consider that Martinussen and Prato (2014) defined "younger drivers" as below the age of 55 and the maximum age of participants in our study was age 35. Furthermore, our study revealed a 4-factor solution and Martinussen and Prato examined the original 3-factor DBQ solution.

We also examined reliability and validity indices across the 4-factor solution. Cronbach's alpha reliability estimates fell in the good to excellent range (.71 to .82) and no estimate was above .90 and therefore this reduces the risk of redundancy. Correlation coefficients between the

DBQ-LV and DBQ-SV fell in the excellent range suggesting that the shortened version demonstrates convergent validity with the longer version. Each factor also demonstrated discriminant validity when correlated with the BDI-II. Driving errors were significantly related to the total BDI-II scores across the total sample and for men only. This suggests that for women, self-reported perceptual, attentional, and/or information processing driving errors are not significantly related to depressive symptoms. This finding is surprising given that MDD is related to slowed attentional and information processing (Brebion et al., 1997; Egeland et al., 2003). One explanation for this may be that young women tend to report less driving errors compared to men on the DBQ (Blockey & Hartley, 1995). Across both the long version and short version, Emotional Violations were not statistically related to the BDI-II, which provides evidence of discriminant validity. This finding is also surprising; however, one explanation for this is that the items composing this scale generally reflect irritability while driving. On the BDI-II, two items specifically assess agitation and irritability. A closer examination suggests that only a minority of participants felt agitated (9%). Absent-minded driving was significantly related to the BDI-II across both versions and for the total sample and women, and approached significance for men. Reckless violations were significantly and positively associated with the BDI-II across both versions for the total sample, men, and across the DBQ-SV for women. The DBQ-LV approached significance for women. In sum, these results suggest that the DBQ-LV and DBQ-LV demonstrated good discriminant validity with the BDI-II.

Hypothesis 2. ***We expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would predict higher levels of self-reported unsafe driving behaviour on the subtests of the shortened version of the DBQ, with the exception of the items pertaining to aggressive violations.***

MDD can induce both cognitive and affective symptoms and impairments in functioning such as depressed mood, weight changes, sleep difficulties, psychomotor retardation, loss of energy, feelings of worthlessness and guilt, irritability, and suicidal ideation (APA, 2013). These symptoms tend to be prevalent in young adult populations (Eisenberg et al., 2007; Price et al., 2006; Takahashi & Kitamura, 2000). MDD is also highly comorbid with anxiety disorders and there is contention in the literature as to whether or not anxiety and depression are distinct disorders as there is a large amount of overlap between symptoms (APA, 2013; Beck et al., 1996; Watson, 2005). Moreover, antidepressant medications are associated with deficits in psychomotor skills such as lethargy, slower reaction times, and reduced alertness (Ramaekers, 2003). Given that the task of driving a vehicle requires sound cognition, attention, and psychomotor functioning (Tanida & Poeppel, 2006), is it is logical to expect that MDD symptoms could also impair driving performance. To the best of our knowledge, no study has examined the influence of MDD and antidepressant medications on self-reported driving behaviour using the DBQ as a dependent measure.

Our findings suggest that Factor 1 is composed of 5-items that can be considered driving errors, which are errors in perceptual, attention, information processing, and misjudgements when driving. In addition, factor 2 is composed of 5-items and is considered "Emotional violations" which reflect driving style and habits due to an emotional response. For both factor 1 and 2, the first model which included age (centered on age 18), sex, BDI-II C/A, BDI-II S, BAI, and antidepressant use were used as predictors of driving errors. Contrary to our prediction, the first model was not statistically significant suggesting that depressive and anxious symptoms, and antidepressant medication use was not related to self-reported driving errors or emotional violations. A second model was run excluding the BAI as it highly comorbid with the BDI-II

(Beck, Steer, & Brown, 1996). This model also included three interactions such as the interaction between sex and the BDI-II CA, sex and the BDI-II S, and sex by antidepressant use. This model also did not reveal statistically significant results. These findings replicate previous studies. For example, Hindmarch (1995) conducted a literature review and reported that SSRIs and TCAs were not associated with an increased risk of a crash.. In addition, Iwamoto et al. (2008) examined the acute effects of paroxetine, an SSRI, in healthy Japanese males. Results suggested no significant differences in standard deviation of lane position (M = 38.9 cm; SD = 9.00 cm) compared to controls (M = 37.2 cm; SD = 7.7 cm).

Our findings contrast with Bulmash et al (2006) who examined driving performance of participants (N = 18) with a diagnosis of MDD. These researchers found that participants with MDD crashed significantly more frequently compared to controls. Bulmash et al used a driving simulator to measure crash rates. The difference in these findings may be attributable to the population used. Bulmash et al. used a clinical population and in the present study the majority of participants were university students who fell in the minimal range on depressive symptoms. Another explanation for insignificant findings is that women report being more compliant with traffic regulations and obeying speed limits compared to men (Bergdahl, 2005); the majority of our sample was composed of women (79.1%) and therefore there may be less variability in DBQ scores.

Factor 3 is also composed of 5-items that represent absent-mindedness which is considered being forgetful or having a low-grade attention. Attention is an important factor when driving an automobile. For instance, Weafer, Camarillo, Fillmore, Milich, and Marczinski (2008) conducted an experiment to investigate whether symptoms of attention-deficit hyperactivity disorder (ADHD) interfere with driving performance. One feature of ADHD is a persistent

pattern of inattention including failing to pay attention to details or making careless mistakes (APA, 2013). Weafer et al. found that individuals with a DSM-IV-TR diagnosis of ADHD displayed significantly greater SDLP ($M = 1.6$; $SD = 0.7$) compared to controls ($M = 1.3$; $SD = 0.4$; $p < .01$).

Both model 1 and model 2 in our study demonstrated overall statistically significant results when examining Factor 3. Age and the cognitive and affective impairments on the BDI-II emerged as significantly related to self-reported absent-mindedness on the road. More specifically, increasing age and increasing cognitive/affective impairments were significantly associated with an increase in absent-minded driving behaviour across both models. This finding makes logical sense. The cognitive/affective impairments measured by the BDI-II, which includes cognitive/affective and somatic impairments measured are also found in older individuals. A key feature of MCI and Alzheimer's disease is a decline in memory and learning (APA, 2013). Carvalho, Tan, Springate, and Davis (2013) factor analyzed the BDI-II with a sample of older individuals ($M = 74$ years) with an MCI or Alzheimer's disease and found that the cognitive/affective symptoms of depression accounted for 36% of the variance providing support for cognitive/affective impairments among older individuals. Although our sample was much younger ($M = 21.9$; $SD = 3.9$), our results support the finding that increasing age and cognitive/affective impairments is associated with driving errors due to absent-mindedness and forgetfulness. No other variables were significant in this model. Furthermore, the two regression models for the sum of the four factors did not display significant results. Factor 4, or "reckless violations", are disrespectful in nature and therefore were not examined in relation to the dependent variables as there is no theoretical basis for mood and recklessness.

Overall, the results of hypothesis two suggest that only cognitive/affective mood symptoms and age are associated with driving impairments due to absent-mindedness. Medication use, anxiety symptoms, sex, and somatic mood symptoms were not related to driving errors, emotional violations, or absent-mindedness. Previous research examining mood, medications, and driving performance is limited and has revealed mixed and inconsistent findings (see Barbone et al., 1998; Leveille et al., 1994; Wingen et al., 2005). Our findings support some previous research. For example, Hindmarch (1995) conducted a literature review and found that SSRIs were not associated with a significant increase in accidents. In addition, Barbone et al. (1998) also found that antidepressant use was not associated with increased risk of a traffic crash using police records. Similarly, Leung, Deane, Taylor, and Bliokas (2009) examined the effects of anxiety on driving performance. Regression analyses demonstrated that anxiety symptoms were not significantly related to driving outcomes on an on-road test (β = -0.3, p = .07). Taken together, our overall findings for hypothesis 2 suggest that mood impairments and age are significantly related to self-reported driving impairments due to absent-mindedness.

Hypothesis 3 and 4. ***We expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would also predict higher impairments on measures of attention (Centre for Research on Safe Driving Attention Network Test [CRSD-ANT]), executive functioning (Trail Making Test [TMT]), visual perceptual ability (Motor-Free Visual Perception Test: Third edition [MVPT-3]), and visual information processing (Useful Field of View [UFOV]). We also expected to observe that higher levels of antidepressant medication use and/or depressive symptoms would predict poorer driving performance on the driving simulator.***

Depressive symptoms can interfere with attentional processes (Egeland et al., 2003; Ravnkilde et al., 2002). Contrary to our hypothesis, our regression models as whole showed that antidepressant medications and depressive symptoms were not significantly related to disturbances in attentional processes. However, a negative relationship was observed between diagnostic depressive symptoms on the PHQ-9 and the CRSD-ANT alerting score (B = -2.48, p < .05), which is a state of achieving and maintaining attentiveness (Posner, 1980). This finding suggests that higher depressive scores are significantly related to poorer alerting performance. In contrast, Han et al. (2012) compared Attention Network Test orienting, alerting, and conflict scores across adolescents diagnosed with Major Depressive Disorder (MDD; n = 31) and healthy controls (n = 30). No significant differences between groups were found for ANT alerting scores (F[1, 58] = 0.01; p = .97) or orienting scores (F[1, 58] = 0.8; p = .36). However, Han et al. did find a significant difference between conflict response times between adolescents with MDD (M = 118.6; SD = 69.7) compared to controls (M = 92.6; SD = 22.8). These findings suggest that adolescents with MDD have poorer conflict efficiency, or take longer to detect and resolve any conflict in mental operations on the ANT conflict task (Mahoney, Verghese, Goldin, Lipton, & Holtzer, 2010). Taken together, there is evidence to suggest that depressive symptoms may interfere with attentional processes, which can be considered essential for operating a motor vehicle (Tanida & Poeppel, 2006).

The TMT measures cognitive performance by measuring divided attention, cognitive flexibility, sequencing, visual search, psychomotor speed, and conceptual tracking (Mitrushina, Boone, & D'Elia, 1999). The UFOV also measures divided attention but in a computerized format. Findings in our study suggest that depressive symptoms were not associated with significant impairments in performance on the TMT or on the UFOV. These findings are

consistent with a study conducted by Hetland et al. (2014), who examined participants taking "potentially driver impairing (PDI)" medications, including antidepressant medications, and scores on the TMT and UFOV. No significant differences on the TMT were found when comparing the whole sample ($N = 225$; $M = 61.7$s), participants taking one or more PDI medications ($n = 155$; $M = 60.0$s), and participants taking no medications ($n = 70$; $M = 65.4$s). Likewise, no significant differences were demonstrated on the UFOV when examining the whole sample (N = 225; $M = 270.4$s), participants taking one or more PDI medications ($n = 155$; $M = 261.3$s), and participants taking no medications ($n = 70$; $M = 293.5$s). A large portion (39.6%) of this sample also met diagnostic criteria for MDD. These findings suggest that mood does not significantly impair performance on tasks that demand divided attention and psychomotor speed.

Our study also examined the influence of mood on a visual perceptual task that does not involve psychomotor abilities. Cognitive/affective impairments on the BDI-II were significantly correlated with better visual perceptual abilities on the MVPT-3. Higher scores on the MVPT-3 reflect fewer deficits in visual perceptual abilities. This finding may be explained by "depressive realism" (DM), or the depressed individuals process information differently (more accurately) than non-depressed individuals (Dykman, Abramson, Alloy, Hartlage, 1989). Moreover, the DM hypothesis postulates that depressed individuals can make realistic inferences while non-depressed individuals are biased towards optimism which serves to provide them with an illusion of control (Moore & Fresco, 2012). Therefore, it is possible that individuals with higher cognitive/affective depression scores have more accurate visual perceptual abilities. Kornbrot, Msetfi, and Grimwood (2013) investigated depressive realism (measured using the BDI-I) and the judgement of time perception and psychophysical functions. Participants were asked to estimate the length of a tone in seconds while also remembering a number. Participants who

scored high on the BDI-I demonstrated more accurate judgements of time perception compared to non-depressed participants ($p < .05$). Moore and Fresco (2012) also conducted a meta-analysis of depressive realism literature. Results of over 75 studies indicate that depressed individuals exhibit slightly less perceptual/attentional bias compared to non-depressed individuals (Cohen's $d = -.07$; $SD = .46$). In contrast, non-depressed individuals demonstrated an optimism bias (Cohen's $d = .29$; $SD = 2.53$) such that these individuals tend to believe they have more control over an outcome than depressed individuals. Although it is possible that our finding of cognitive/affective impairments being associated with better visual perceptual abilities is spurious ($p = .05$), our findings may also lend some support to the hypothesis that greater cognitive/affect impairments that contribute to depression, are associated with greater visual-perceptual abilities.

To the best of our knowledge, only one study to date has examined the effects of depressive symptoms alone on driving performance. Bulmash et al. (2006) examined driving simulator performance in participants who met the DSM-IV-TR diagnostic criteria for MDD and who were not taking antidepressant medications. A diagnosis of MDD was determined by staff psychiatrists at an outpatient clinic. Severity was assessed using the BDI-II ($M = 27.4$; $SD = 11.5$). Our study also examined the relationship between depressive symptoms and driving performance on the driving simulator in participants who were not taking antidepressant medications. Bulmash et al. (2006) found that participants with MDD ($n = 18$) demonstrated a significant increase in crash rates ($\eta^2 = 0.10$) and significantly slower reaction times ($\eta^2 = 0.08$) compared to healthy controls. However, our findings contrast those found by Bulmash et al. For example, our findings suggest that depressive symptoms were not associated with impairments in driving function on the total score on the Manitoba Road Test, which is a sum score of errors in

starting, stopping, signalling, oncoming traffic, passing, intersections, speed, turning, and inattention. However, participants in the present study did not meet diagnostic criteria for MDD (scores on the PHQ-9 were low; $M = 4.2$; $SD = 4.1$) and participants in Bulmash et al.'s study met diagnostic criteria. Moreover, consistent with our study, Bulmash et al. found no significant relationship between BDI-II scores and steering response time or crashes on the driving simulator suggesting that depressive symptoms were not significantly associated with steering impairments or crashes. Overall, these findings suggest that a diagnosis of MDD, or severe depressive symptoms, may impair driving performance. However, it is important to consider that Bulmash et al. had a very small sample size ($n = 18$) suggesting that the findings may be confounded and not generalizable to the population. Given that our sample was composed of individuals who did not meet diagnostic criteria for MDD and/or reported mild depressive symptoms, there is a risk of committing a type-II error. More specifically, there is a risk of failing to reject the null hypothesis when it is true due to an underrepresentation of individuals with MDD in our sample.

Limitations and Future Directions

There are a number of limitations and suggestions for future research that should be considered. Firstly, participants were recruited through convenience sampling from undergraduate courses at Lakehead University and from community organizations. Convenience sampling may reflect a self-selection bias and may not represent the general population and thus may not be generalizable. More specifically, our laboratory sample in particular may have differed from those who chose not to participate. It was difficult to recruit individuals who were experiencing significant depressive symptoms and/or taking antidepressant medications to complete the laboratory portion of our study. Hartlage, Alloy, Vázquez, and Dykman (1993)

hypothesized a "cognitive effort hypothesis" which proposes that the cognitive deficits associated with MDD are directly related to the difficulty of the task. Therefore, highly demanding tasks are likely to have a detrimental impact on depressed individuals and thus may cause depressed individuals to avoid these tasks. This theory may provide an explanation as to why it was difficult to recruit participants who were experiencing significant depressive symptoms and/or taking antidepressant medications. We did, however, assess effort using the WAIS-IV digit span subtest with participants who completed the laboratory portion of this study. Based on reliable digit span-revised scores, only no participants scored in a range that is indicative of sub-optimal effort. Therefore, although there is evidence that the participants who participated in the laboratory session were motivated to exert effort during participation, it is possible that a selection bias may have impacted our results. Future studies should aim to recruit participants with MDD and/or participants who are taking antidepressant medications to participate in laboratory sessions using a driving simulator. This is especially important given that most of the current research has utilised healthy controls rather than clinical populations to investigate the influence of MDD and antidepressant medications on driving performance (Rapoport & Baniña, 2007). One way to maximize the likelihood of participation may be to reduce the amount of time a participant spends in the laboratory. A 2-hour session may have been perceived as too long and cognitively demanding. Taken together, future studies should be sensitive to self-selection bias and design experiments that are shorter in duration.

An additional factor that may have influenced the generalizability of our findings was that participants were recruited from a northern community (Thunder Bay, ON.). This suggests that participants in the present study may not have had as much exposure to complex driving situations (e.g., traffic circles, complex intersections). This may have reduced the variability of

self-reported unsafe driving behaviours on the DBQ. Additionally, the online portion of this study was based solely on self-report measures, which may not translate to actual performance. "People's behaviour does not always conform to what they say they would do, so survey research will never replace direct observation" (Shaughnessy, Zechmeister, & Zechmeister, 2006; p. 188). For example, our study found no significant relationship between both versions of the DBQ and the simulated driving scores (*r*s = .03 to .26) suggesting that self-reported driving behaviours are not related to actual driving performance. Furthermore, self-reports such as the DBQ rely on accurate and reliable responses including recalling information on the DBQ pertaining to instances when an individual was forgetful (e.g., forgetting where care was left) which seems counterintuitive as it requires participants to recall information that was previously forgotten (Lajunen & Summala, 2003). In addition, previous research has documented that driver's tend to fall into the illusory superiority phenomenon wherein they tend to believe that they are better than average at the task of driving (Riendeau & Patterson, 2012; Freund, Colgrove, Burke, & McLeod, 2005; Svenson, 1981). Therefore, caution should be taken when interpreting results from self-report measures. Lastly, we included multiple comparisons in our regression analyses and this may have confounded the results.

One strength of the present study was that we did not solely rely on self-report data. Our study included a laboratory portion, which included data from neuropsychological data including the CRSD-ANT, UFOV, and MVPT and driving data from a driving simulator. Driving simulators have demonstrated validity and are a safe tool for measuring driving performance (Bédard et al., 2010; Lee, Cameron, & Lee, 2003). In addition, to the best of our knowledge this was the first study to validate the DBQ on a younger Canadian sample using a driving simulator. Another strength of this study was that we followed best practices for EFA analyses. For

example, Streiner (1994) argues that "there should be an absolute minimum of five subjects per variable, with the proviso that there are at least 100 subjects." (p.140) suggesting that our overall sample size met the minimum standard ($n = 266$). In addition, Streiner reports that eigenvalues must be reported (see Table 13 for eigenvalues) and the sum of the eigenvalues should account for at least 50% of the variance. In our study, the eigenvalues for the whole sample met this criteria (accounted for a total of 49.9%) and for participants aged 18-35 ($n = 233$; accounted for a total of 43.1%) approached the cut-off for this criteria. Additional best practice criteria that our study followed includes: reporting how many factors were retained, specifying factor rotation, and including a minimum of 3 variables per factor.

Overall, future studies could examine the influence of mood and antidepressant medications on driving performance with a clinical population using a larger sample size to increase power, reduce the risk of committing type I and II errors, and generate more generalizable results. The present study also only examined individuals aged 18 to 35 and was unable to examine the influence of antidepressant medications due to difficulty with recruitment. Future studies could examine older participants and compare the groups to investigate any differences in age groups, depressive symptoms, and medication use on driving performance. Given that age is strongly related to higher crash rates compared to other age cohorts (Owsley, 2002), and therefore can be considered a confounding variable, it will be important for future studies to control for age and/or compare age cohorts.

At present, the literature on the influence of mood and medications on driving performance continues to be mixed and sparse. However, this study adds to the literature because, to the best of our knowledge, this is the first study to examine self-report, neuropsychological, and behavioural measures, including actual driving performance in a

younger Canadian sample. Overall the main findings of our study were that cognitive affective impairments of depression (BDI-II CA) were associated with impairment in both self-report and neuropsychological measures. For example, BDI-CA impairments were positively associated with self-reported absent-mindedness on the road ($B = .12, p < .05$) and the CRSD-ANT alerting score, which requires a reflexive component and indicates whether an individual can maintain attention ($B = 3.45, p < .05$). This lends some support for depressive symptoms interfering with attentional processes on the road. The magnitude of these associations may have been stronger if our sample consisted of more individuals with severe depressive symptoms. Furthermore, in contrast to these findings, the relationship between the BDI-II CA and MVPT-3 ($B = 2.02, p = .05$) reflects fewer deficits in visual perceptual abilities. However, it is important to note that this finding may be spurious as $p = .05$. The overall patterns of self-report data, neuropsychological data, and behavioural data suggest that although there is some consistency between self-report measures and neuropsychological data (BDI-II CA; DBQ; CRSD-ANT), this does not necessarily mean these impairments in attention translate into actual driving impairments on the simulator. Further investigation is needed to provide more conclusive findings. Future studies could conduct a similar study using on-road performance as the behavioural measure of driving performance. Future research is essential on this topic specifically comparing clinically depressed Canadian populations with individuals taking antidepressant medications, healthy controls, and across age cohorts.

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Appendix A

Demographic Questionnaire

1. Name _____
2. Date of birth (dd/mm/yyyy) _____/_____/_____
3. Sex
   [ ] Male [ ] Female
4. Mailing address (including postal code)

   _____
   _____
   _____
5. Phone number: ( ) _____
6. Email address (if applicable)_____
7. How old were you when you obtained your driver's license? _____

Appendix B

No.	Driving Behaviour Questionnaire Please indicate, on average, how frequently you engage in the following behaviours using the scale below: **0 = never 1 = hardly ever 2 = occasionally 3 = quite often** **4 = frequently 5 = very frequently**						
1	Check your speedometer and discover that you are unknowingly traveling faster than the legal limit.	0	1	2	3	4	5
2	Lock yourself out of your car with the keys still inside.	0	1	2	3	4	5
3	Become impatient with a slow driver in the outer lane and overtake on the inside.	0	1	2	3	4	5
4	Drive as fast along country roads at night with headlights on low as on high beam.	0	1	2	3	4	5
5	Attempt to drive away without first having switched on the ignition,	0	1	2	3	4	5
6	Drive especially close or 'flash' the car in front as a signal to drive faster or get out of your way.	0	1	2	3	4	5
7	Forget where you left your car in a multi-level car park.	0	1	2	3	4	5
8	Distracted or preoccupied, realize belatedly that the vehicle ahead has slowed, and have to slam on the brakes to avoid a collision.	0	1	2	3	4	5
9	Intend to switch on the windshield wipers, but switch on the lights instead, or vice versa.	0	1	2	3	4	5
10	Turn left on to a main road into the path of an oncoming vehicle that you hadn't seen or who's speed you had misjudged.	0	1	2	3	4	5
11	Misjudge the gap between parked cars and nearly (or actually) hit the adjacent vehicle.	0	1	2	3	4	5
12	"Wake up" to realize that you have no clear recollection of the road along which you have just traveled.	0	1	2	3	4	5
13	Miss your exit on a motorway and have to make a lengthy detour.	0	1	2	3	4	5
14	Forget which gear you are currently in and have to check with your hand.	0	1	2	3	4	5
15	Stuck behind a slow moving vehicle on a two-lane highway, you are driven by frustration to try to over-take in risky circumstances.	0	1	2	3	4	5
16	Intending to drive to destination 'A', you "wake up"	0	1	2	3	4	5

	to find yourself on route to 'B', where the latter is more the usual journey.	
17	Take a chance and cross on lights that have turned red.	0 1 2 3 4 5
18	Angered by another driver's behaviour, you give chase with the intention of giving him/her a piece of your mind.	0 1 2 3 4 5
19	Try to overtake without first checking in your mirror, and then get honked at by the car behind you which has already begun its overtaking manoeuvre.	0 1 2 3 4 5
20	Deliberately disregard the speed limits late at night or very early in the morning.	0 1 2 3 4 5
21	Forget that your licence/plates have expired and discover that you are driving illegally.	0 1 2 3 4 5
22	Lost in thought, you forget that your lights are on high beam until 'flashed' by another vehicle.	0 1 2 3 4 5
23	On turning right, nearly hit a cyclist who has come up on your inside.	0 1 2 3 4 5
24	In a line of vehicles turning right on to a main road, pay such close attention to the traffic approaching from the left that you nearly hit the car in front.	0 1 2 3 4 5
25	Drive back from a party, restaurant, or pub, even though you realize that you may be over the legal blood-alcohol limit.	0 1 2 3 4 5
26	Have an aversion to a particular class of road user, and indicate your hostility by whatever means you can.	0 1 2 3 4 5
27	Lost in thought or distracted, you fail to notice someone waiting at a crosswalk or a pedestrian crosswalk light that has just turned red.	0 1 2 3 4 5
28	Park in a no parking area and risk a fine.	0 1 2 3 4 5
29	Misjudge the speed of an oncoming vehicle when overtaking.	0 1 2 3 4 5
30	Hit something when backing up that you had not previously seen.	0 1 2 3 4 5
31	Fail to notice someone stepping out from behind a bus or parked vehicle until it is nearly too late.	0 1 2 3 4 5
32	Plan your route badly, so that you meet traffic congestion you could have avoided.	0 1 2 3 4 5
33	Overtake a single line of stationary or slow-moving vehicles, only to discover that they were lining up to get through a one-lane gap or roadwork lights.	0 1 2 3 4 5
34	Overtake a slow-moving vehicle on the inside lane or hard shoulder of a motorway.	0 1 2 3 4 5

35	Cut the corner on a left-hand turn and have to swerve violently to avoid an oncoming vehicle.	0	1	2	3	4	5
36	Get into the wrong lane to make a left or right hand turn at an intersection.	0	1	2	3	4	5
37	Fail to read the signs correctly and exit from a highway on the wrong road.	0	1	2	3	4	5
38	Fail to give right of way when a bus is signaling its intention to pull out.	0	1	2	3	4	5
39	Ignore yield signs and narrowly avoid colliding with traffic that has the right of way.	0	1	2	3	4	5
40	Fail to check your mirror before pulling out, changing lanes, turning etc.	0	1	2	3	4	5
41	Attempt to overtake a vehicle that you hadn't noticed was signaling its intention to turn left.	0	1	2	3	4	5
42	Deliberately drive the wrong way down a deserted one-way street	0	1	2	3	4	5
43	Disregard red lights when driving late at night along empty roads.	0	1	2	3	4	5
44	Drive with only 'half-an-eye' on the road while looking at a roadmap, dialing/text messaging on a cell phone, changing a cassette/CD or radio channel.	0	1	2	3	4	5
45	Fail to notice pedestrians crossing when turning into a side-street from a main road.	0	1	2	3	4	5
46	Get involved in unofficial 'races' with other drivers.	0	1	2	3	4	5
47	'Race' oncoming vehicles for a one-car gap on a narrow or obstructed road.	0	1	2	3	4	5
48	Brake too quickly on a slippery road and/or steer the wrong way in a skid.	0	1	2	3	4	5
49	Misjudge your crossing interval when turning left and narrowly miss colliding with an oncoming vehicle.	0	1	2	3	4	5

Appendix C

Driving History/Habits Questionnaire

1. Date (dd/mm/yyyy) _____/_____/_____
2. Approximately how many **kilometres (miles)** do you drive per week?
   [ ] 0-20 km (0-12 m) [ ] 51-100 km (32-62 m)
   [ ] 21-50 km (13-31 m) [ ] over 100 km (> 62 m)
3. How long ago was your last car collision involving a person, car, or fixed object?
   [ ] Less than 1 year [ ] 4-5 years
   [ ] 1-2 years [ ] 5-10 years
   [ ] 2-3 years [ ] More than 10 years
   [ ] 3-4 years [ ] Never had an accident
4. When driving, how many collisions (involving a person, car, or fixed object) have you been involved in? (*Do not include cases where you were a passenger*) _____
   For how many of these was reporting to the police deemed unnecessary? _____
5. For what purposes do you drive in a **typical week**? *(Check all that apply to you)*

	How many times per week?
[ ] Groceries	_____
[ ] Other shopping (e.g., drug store, clothes shopping)	_____
[ ] Health-related appointments (e.g., doctor, dentist)	_____
[ ] Social events (e.g., recreation centres, friends)	_____
[ ] Worship (e.g., church, synagogue, etc.)	_____
[ ] Hobby-related (e.g., attend classes)	_____
[ ] Work, school, or volunteer activities	_____
[ ] Family events	_____
[ ] Other, please specify _____	

6. Which driving situation(s) do you find stressful, uncomfortable, or avoid when possible? *(Check all that apply to you)*
   [ ] Turning left at intersections [ ] Navigating parking lots
   [ ] Driving at night [ ] Changing lanes
   [ ] Backing up [ ] Maintaining the speed limit
   [ ] Parallel parking [ ] Driving in bad weather
   [ ] Driving in unfamiliar areas [ ] Driving in heavy traffic
   [ ] Driving with passengers in the car [ ] Other _____
   [ ] Driving alone [ ] None of the above
7. Some people restrict their driving to certain situations. Do you restrict your driving to: *(Check all that apply to you)*
   [ ] Daytime [ ] Local routes

[ ] When accompanied by a passenger
[ ] Outside of rush hour
[ ] Fair weather
[ ] Other _____
[ ] None of the above

8. With regard to the speed limit on **local streets** (posted speed limit of 50 km/hr), do you typically drive:
   [ ] 35 km/hr or less
   [ ] 36-45 km/hr
   [ ] 46-55 km/hr
   [ ] 56-65 km/hr
   [ ] 66 km/hr or more

9. With regard to the speed limit on **major highways** (posted speed limit of 90 km/hr), do you typically drive:
   [ ] 85 km/hr or less
   [ ] 86-95 km/hr
   [ ] 96-105 km/hr
   [ ] 106-115 km/hr
   [ ] 116 km/hr or more

Appendix D

Medication History Questionnaire

1. Date (dd/mm/yyyy) _____/_____/_____

2. Are you currently taking medications?

□ Yes □ No

If yes,

Please list all your current medications; write the specific name(s) as printed on the medication label(s) and then indicate the dose.

Medication Name	**Medication Dose**
1.	
2.	
3.	
4.	
5.	
6.	
7.	
8.	
9.	
10.	

3.Are you currently seeing a therapist or seeking treatment for depression and/or anxiety?

□ Yes □ No

4. Have you seen a therapist in the past for treatment for depression and/or anxiety?

□ Yes □ No

a. If yes, please indicate when you saw a therapist and for how long you saw a therapist

5.Have you ever had a serious head injury?

□ Yes □ No

b. If yes, please indicate when you experienced a serious head injury

6.Do you have any of the below conditions?

	Yes	No
Diabetes or high blood sugar	❑1	❑2
Heart disease	❑1	❑2
Stroke	❑1	❑2
Seizures or epilepsy	❑1	❑2
Parkinson's disease	❑1	❑2
Sleep apnea or sleeping sickness	❑1	❑2
Narcolepsy	❑1	❑2
Dementia (e.g., Alzheimer disease)	❑1	❑2
Physical Frailty (reduced flexibility or reduced muscle strength)	❑1	❑2
Poor hearing	❑1	❑2
Poor vision	❑1	❑2
Arthritis	❑1	❑2
Broken bones	❑1	❑2
Sudden lapses in consciousness (Syncope)	❑1	❑2
Other	❑1	❑2

Please specify for other:

_____

Appendix E

Driving Simulator Evaluation Form
Lakehead University

I. STARTING

	Deduct	Deduction
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 green	5	

II. STOPPING

	Deduct	Deduction
A. Stops for no reason	5	
B. Stops too suddenly	5	
C. Over-running crosswalk	5	
D. Not at safe place	5	
E. Leaves when not safe	5	

III. SIGNAL VIOLATIONS

	Deduct	Deduction
A. Thru on red	5	
B. Thru on red (enters amber)	5	
C. Thru on red (right turn)	5	
-- Stop Sign --		
D. Does not stop	5	
E. Leave when not safe	5	

IV. VEHICLES MOVING ON ROADWAY

	Deduct	Deduction
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 wrong side of street	5	
G. Wanders	5	
H. Crosses solid line	5	
I. Uncertain gear shifting	5	
J. Fails to drive in proper lanes	5	

V. PASSING

	Deduct	Deduction
A. Too close to pedestrians or vehicles	5	
B. Passes when unlawful Or unsafe	10	
C. Speeds up when being passed	10	

VI. UNCONTROLLED INTERSECTIONS/RAILWAY CROSSINGS/YIELD SIGNS/PEDESTRIAN CORRIDORS OR CROSSWALKS

	Deduct	Deduction
A. Fails to slow down or check	5	
B. Fails to yield	10	

VII.SPEED

	Deduct	Deduction
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. Hinder or drives too slowly	5	
F. Drives at uneven speed	5	

VIII.TURNING

	Left	Right
A. Improper signal	5	
B. Improper approach.	5	
C. Improper during	5	
D. Improper after	5	
E. Improper speed	5	
F. Shies away	5	
G. Strikes/over curbs	5	
H. Fails to yield	10	
I. Fails to establish	5	
J. Fails to clean intersection	5	

IX. INATTENTIVE

	Deduct	Deduction
A. Leaves signal on	5	
B. Signals through intersection	5	
C. Hesitant	5	
D. Fails to yield emergency vehicle	5	

PARTICIPANT #: _____ DATE: _____

ROAD TEST RESULTS:

PASS FAIL

GRAND TOTAL ___

Appendix F

Participant Information Sheet A

The Influence of Mood and Medications on Driving Performance

Dear Potential Participant:

Thank you for taking part in this research study investigating the role of mood and medications on driving performance.

During this study you will be asked to complete a series of online questionnaires. This study will take approximately 0.5 hours to complete. Following completion of this study, a research assistant may contact you to invite you to a follow-up laboratory portion of this study, if you agree to be contacted further.

This research project is being conducted under the supervision of Dr. Michel Bédard and Dr. Dwight Mazmanian. Only Dr. Bédard and Dr. Mazmanian, a research assistant, and Loretta Patterson will have access to the information you provide. Your information will be assigned a unique subject number to ensure anonymity and confidentiality. The information will be securely stored in the Centre for Research on Safe Driving (CRSD) at Lakehead University for at least five years. In addition, your identifying information will be kept completely confidential throughout reports of results and publications. This study has received support from St. Joseph's Care Group Award in Applied Health Research and the Canadian Institutes of Health Research.

Participation in this research study is completely voluntary and you may decline to answer any question or refuse to participate in any part of this study. If for any reason you wish to withdraw from the study you may do so at any time without penalty. The physical risk associated with this study will be the same as that of working with a personal computer for up to 30 minutes. In terms of psychological risk, there is a chance that you may experience some psychological discomfort subsequent to completing two questionnaires that ask about symptoms of depression and anxiety. Given that this is a risk, we will provide you with a list of psychological services. You will receive one Psychology 1100 bonus point for your participation if you are enrolled in this course.

This study has been approved by the Lakehead University Research Ethics Board, who may be contacted at 1-807-343-8934. If you have questions or concerns regarding the ethics of the project, please contact the Research Ethics Officer at 1-807-343-8283.

A report of findings will be available to those interested upon request. If you require additional information please do not hesitate to contact one of the researchers.

Thank you,

Loretta Patterson, M.A.
PhD Student, Clinical Psychology

Michel Bédard, PhD
Canada Research Chair in Aging and Health

Lakehead University
Phone: (807) 766-7256
Email: lblanche@lakeheadu.ca

Director, Centre for Research on Safe Driving
Lakehead University
Phone: (807) 343-8630
Email: mbedard@lakeheadu.ca

Appendix G

CONSENT FORM A

By providing my name, student number, and E-mail, and checking the box below, I indicate that I have read the previous "Participant Information Sheet" and that I agree to participate in this study which is conducted by Loretta Patterson in the Department of Psychology for her PhD dissertation under the supervision of Dr. Michel Bédard and Dr. Dwight Mazmanian. I understand that any questions that I might have about my participation can be answered by Loretta Patterson and/or Drs. Bédard and Mazmanian. In providing my identifying information below, I understand and agree to the following:

1. I understand the information on the "Participant Information Sheet".
2. I agree to participate in this study.
3. I fully understand what I will be required to do as a participant in this study.
4. I am a volunteer and can withdraw at any time from this study without penalty or consequence.
5. I may choose not to answer any question asked in the questionnaires without penalty or consequence.
6. My data will be confidential and stored in the Centre for Research on Safe Driving (CRSD) for a period of at least five years.
7. My information will remain anonymous should any publications or public presentations come out of this study.
8. I may receive a summary of this research study upon completion of this study.
9. I give my permission to be contacted by telephone and/or E-mail for the purpose of participation in this study.

I agree to participate in the study examining mood, medications, and driving.

_____ _____

Full Name (*please print)* Date

_____ Please check this box to consent ☐

E-mail

_____ _____

Student Number Phone

_____

Name of Psychology 1100 Professor

I agree to be contacted regarding future studies being conducted by researchers in the Centre for Research on Safe Driving (CRSD).

_____

Signature of Participant Date

Email Address: _____

Phone Number: ( )

Appendix H

Participant Information Sheet B

The Influence of Mood and Medications on Driving Performance

Dear Potential Participant:

Thank you for taking part in this research study investigating the influence of mood and medications on driving performance.

During this study you will be asked to complete two computerized tasks (one to assess processing speed and one to measure attention), a series of questionnaires designed to measure depression, driving habits, cognitive and psychomotor functioning, a structured interview with the experimenter that assesses your mood and substance use, and a 40 minute simulated drive. If you wear glasses, we ask that you bring them with you to the laboratory. In addition, if you are taking any medications, we ask that you bring your prescription bottles with you to the laboratory session. This study will take approximately 2 hours to complete.

This research project is being conducted under the supervision of Dr. Michel Bédard and Dr. Dwight Mazmanian. Only Dr. Bédard and Dr. Mazmanian, a research assistant, and Loretta Patterson will have access to the information you provide. Your information will be assigned a unique subject number to ensure anonymity and confidentiality. The information will be securely stored in the Centre for Research on Safe Driving (CRSD) at Lakehead University for at least five years. In addition, your identifying information will be kept completely confidential in reports of results and publications. This study has received support from the St. Joseph's Care Group Award in Applied Health Research and the Canadian Institutes of Health Research.

Participation in this research study is completely voluntary and you may decline to answer any question or refuse to participate in any part of this study. If for any reason you wish to withdraw from the study you may do so at any time without penalty. There is a chance that you may experience simulator discomfort while on the driving simulator. If this occurs, the simulation can be paused or terminated. Other than that, the physical risk will be the same as that of working with a personal computer for up to 60 minutes and driving under normal conditions for up to 45 minutes. Given that this study is investigating mood, a list of local psychological services will be provided to you should you want to contact them. In addition, we may provide you with a letter recommending that you contact these services. We may also contact you in 3 months to invite you to participate in a follow-up portion of this study. You will receive up to four Psychology 1100 bonus points for your participation. More specifically, you will receive two points for the first laboratory portion of this study and two for the 3-month follow-up portion.

This study has been approved by the Lakehead University Research Ethics Board, who may be contacted at 1-807-343-8934. If you have questions or concerns regarding the ethics of the project, please contact the Research Ethics Officer at 1-807-343-8283.

A report of findings will be available to those interested upon request. If you require additional information please do not hesitate to contact one of the researchers.

Thank you,

Loretta Patterson, M.A.
PhD Student, Clinical Psychology
Lakehead University
Phone: (807) 766-7256
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Michel Bédard, PhD
Canada Research Chair in Aging and Health
Director, Centre for Research on Safe Driving
Lakehead University
Phone: (807) 343-8630
Email: mbedard@lakeheadu.ca

Appendix I

CONSENT FORM B

By providing my name, student number, and E-mail, I indicate that I have read the previous "Participant Information Sheet" and that I agree to participate in this study which is conducted by Loretta Patterson in the Department of Psychology for her PhD dissertation under the supervision of Dr. Michel Bédard and Dr. Dwight Mazmanian. I understand that any questions that I might have about my participation can be answered by Loretta Patterson and/or Drs. Bédard and Mazmanian. By providing my identifying information below, I understand and agree to the following:

1. I understand the information on the "Participant Information Sheet".
2. I agree to participate in this study.
3. I fully understand what I will be required to do as a participant in this study.
4. I am a volunteer and can withdraw at any time from this study without penalty or consequence.
5. I may choose not to answer any question asked in the questionnaires without penalty or consequence.
6. I understand that some participants may experience simulator discomfort while on the driving simulator, and that the simulation may be paused or terminated should I experience simulation discomfort. Other than that, the physical risk will be the same as that of working with a personal computer for up to 60 minutes and driving under normal conditions for up to 45 minutes.
7. I am aware that I may receive a letter recommending that I seek psychological services.
8. I am aware that I will receive a list of psychological services and that these can be used should I experience distress during my participation in this study.
9. My data will be confidential and stored in the Centre for Research on Safe Driving (CRSD) for a period of at least five years.
10. My information will remain anonymous should any publications or public presentations come out of this study.
11. I may receive a summary of this research study upon completion of this study.
12. I give my permission to be contacted by telephone and/or E-mail for the purpose of participation in this study.

I agree to participate in the study examining mood and driving.

Full Name *(please print)*	Date
Signature *(please sign)*	E-mail
Student Number	Phone

Name of Psychology 1100 Professor

I agree to be contacted regarding future studies being conducted by researchers in the Centre for Research on Safe Driving (CRSD).

_____

Signature of Participant Date

Email Address: _____

Phone Number: ( )

Appendix J

Below is a list of agencies that offer mental health services for your information. Thank you again for your participation in this study.

1. **Lakehead University Student Health and Counselling Centre** (807-343-8361)
   Located across from Security, near the Agora and University Centre Theatre. Personal counselling for students covering a wide variety of issues.

2. **Family Services Thunder Bay** (807-684-1880)
   A not-for-profit organization providing confidential counselling, advocacy, education, and support for individuals and families in Thunder Bay. Counsellors provide comprehensive help for a wide variety of issues such as grief and coping, substance use, credit and financial problems, anger, anxiety, depression, and past experiences of violence. Fees are based upon individual circumstances and no person will be denied service due to an inability to pay.

3. **Personal Development Centre** (St. Joseph's Care Group). (807-343-2400)
   An adult out-patient program which offers and innovative, multi-disciplinary approach to treating a variety of mental health issues such as anxiety, depression, stress related problems, self-esteem issues, and compromised coping strategies. A physician's referral is required for admission to the program.

4. **Thunder Bay Crisis Response Service** (807-346-8282) (Toll free 1-888-269-3100)
   This is a community based crisis response support program for individuals experiencing a mental health crisis in the Thunder Bay District.

5. **Canadian Mental Health Association** (807-345-5564; 200 Van Norman St. Thunder Bay, ON. P7A 4B8; http://www.cmha-tb.on.ca)
   CMHA is a Canada-wide organization that promotes mental health of all and supports the recovery of people who are experiencing a mental illness. The CMHA has programs that assist with employment, interventions for youth, housing, peer support, recreation services for individuals with mental illness, and public education for the community.

Appendix K

Participant Recommendation Letter

Dear Participant,

We would like to thank-you again for your participation in this study examining mood and medications on driving performance.

As part of this study, the experimenter asked you some questions pertaining to how you have been feeling over the past two weeks. Your answers to these questions suggested that you may benefit from seeking psychological and medical support. We have provided you with a list of psychological services should you wish to contact them.

You may also contact Loretta Patterson and/or Dr. Bédard with any questions you may have regarding seeking psychological support.

Thank you,

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