Probabilities of PTSD and Related Substance Use Among Canadian Adults

by

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A dissertation submitted to the

School of Graduate Studies

in partial fulfillment of the requirements for the degree of

Masters of Education

Faculty of Education

Memorial University of Newfoundland

December, 2018

St. John's Newfoundland

Abstract

The aim of this study was to estimate the prevalence and probabilities of comorbidities between self-reported PTSD and smoking, alcohol binge drinking, and substance use disorders (SUDs) from a national Canadian sample. Data were taken from the Public Use Microdata File of the 2012 Canadian Community Health Survey–Mental Health (N = 17 311). The prevalence of (a) smoking, (b) alcohol binge drinking, and (c) SUDs was estimated among those with a PTSD diagnosis versus those without a PTSD diagnosis. After controlling for potential socioeconomic and mental health covariates, self-reported PTSD acted as a significant predictor for group membership in the heaviest smoking, heaviest drinking, and heaviest drug usage categories. Individuals self-reporting a diagnosis of PTSD were found to have a significantly higher likelihood of engaging in smoking and alcohol binge drinking, and were more likely to meet criteria for SUDs than individuals not reporting a PTSD diagnosis. Implications of the findings are discussed.

Acknowledgements

I would like to thank my supervisor, Dr. Jackie Hesson, for her continual guidance throughout my time in graduate school. I could not have asked for a more supportive supervisor.

This dissertation is dedicated to my wife, Karla Connolly, for her profound support and endless encouragement over the course of my graduate studies.

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Posttraumatic stress disorder (PTSD) is a disabling psychiatric disorder that reflects an inability to cope with overwhelming stress caused by trauma that is followed by a distinctive pattern of symptoms. Most research pertaining to PTSD has been conducted on military veterans, however research on community populations in Canada has indicated a prevalence of 1.2% for men and 2.7% for women (Stein, Walker, Hazen, & Forde, 1997). PTSD also commonly co-occurs with other impairing psychiatric disorders, and epidemiologic survey has indicated that approximately 80% of individuals with PTSD meet criteria for at least one other psychiatric diagnosis (Brady, 1997). PTSD is particularly likely to be comorbid with affective disorders, other anxiety disorders, dissociative disorders, and substance use disorders (SUDS; Galatzer-Levy, Nickerson, Litz, & Marmar, 2013).

Drug and alcohol use is common in individuals with PTSD with research that suggests individuals with PTSD are two to three times more likely than those without PTSD to have a SUD (Kessler, Sonnega, Bromet, 1995). In addition, surveys of those seeking treatment for SUDs have revealed that between 25% and 58% of these individuals have comorbid PTSD (Brady, 1997; Galatzer-Levy et al., 2013; Mills, Teesson, Ross, & Peters, 2006), however research using population based samples is limited. Research in this area is important because it will increase our understanding of comorbid PTSD and SUDs, and improve health outcomes of those with the comorbidity through early identification and applying appropriate treatment strategies. Therefore, the current research will fill this gap in the literature by exploring the prevalence and probabilities of PTSD in relation to smoking, binge drinking, and SUDs based on self-report data from a large nationwide Canadian sample.

Post-Traumatic Stress Disorder (PTSD)

PTSD is defined as trauma from exposure to actual or threatened death, serious injury, or

sexual violation (American Psychiatric Association, 2013). The symptoms of PTSD may include disturbing thoughts and feelings, mental or physical distress to trauma-related cues, attempts to avoid trauma-related cues, alterations in how a person thinks and feels, and an increase in one's stress response (APA, 2013). A person must have been exposed to trauma and experienced symptoms for at least a month after exposure, however, the functional impairments of PTSD can last throughout one's entire lifetime (Friedman, 2015).

Exposure to stressful events such as failure, disappointment, rejection, and loss are events that normally occur during the course of one's lifetime, and most people have the psychological capacity to cope with stressful events such as these and to continue life normally. However, a catastrophic event, or series of events, in which individuals have been personally exposed to threatening situations, or witnessed death, physical harm, or sexual violence can be traumatic for an individual.

There is a significant minority of people who will be unable to cope effectively with the severe stress that they experience as a result of being confronted with a severely traumatic event (Feldner, Babson, & Zvolensky, 2007). Trauma can also include indirect exposure such as a loved one who is exposed to trauma or in which an individual is repeatedly exposed to consequences of trauma in the line of professional duties, such as police officers who are regularly exposed to the trauma of accidents, crime, and suicide (Foa, Kean, & Friedman, 2000; Kessler, 2000). Those who are unable to psychologically cope with exposure to trauma can develop serious and potentially incapacitating symptoms that are characterized as PTSD.

Avoidance of reminders of the trauma is a core symptom of PTSD (Reddy, Anderson, Liebschutz, & Stein, 2013). These reminders include people, situations or circumstances resembling or associated with the event. Individuals with PTSD often try to push memories of

the event out of their mind and avoid thinking or talking about it in detail, while others with PTSD may ruminate excessively about the event. Individuals with PTSD also describe symptoms of emotional numbing meaning that they have an inability to experience any emotional feelings (Reddy et al., 2013). In addition, impaired sleep is one of the most common symptoms among individuals diagnosed with PTSD (Ohayon & Shapiro, 2000) and has been described as universal problem across the PTSD population (Gellis & Gehrman, 2011). In addition to these negative symptoms, those with PTSD often abuse substances as a means of temporarily escaping from the insurmountable distress they experience, which further exacerbates the severity of PTSD symptoms (Bremner, Southwick, Darnell, & Charney, 1996). Due to the negative impact that substance abuse and dependence has on individuals with a diagnosis of PTSD, the relationship between these comorbid disorders will be the focus of the current study.

PTSD Diagnostic Criteria

The DSM-5 (American Psychiatric Association, 2013) defines PTSD as trauma from exposure to actual or threatened death, serious injury, or sexual violation. Diagnoses are derived from patients meeting eight criteria. The first criterion is exposure to the traumatic event, which signifies the importance of aetiology regarding this disorder. In order to be considered a valid diagnosis of PTSD, the DSM-5 acknowledges exposure to four forms of trauma: direct experience, witnessing an event, learning of a close family member or friend who experienced the event, or by being exposed to the harmful effects of the traumatic event.

The second criterion involves intrusion symptoms, and only one of these symptoms is necessary to fulfil the requirements of a PTSD diagnosis. Intrusion symptoms can include distressing memories, dreams, or flashbacks and can be psychological or physiological in nature. The third criterion is avoidance of particular stressing stimuli, of which one symptom is

necessary to meet a PTSD diagnosis. These stimuli can involve the memory of an event or the external reminders of an event.

The fourth criterion is negative alterations in cognition or mood, of which two symptoms are necessary in order to meet a PTSD diagnosis. Examples of these include an inability to recall events, negative beliefs about one's self, self-blame, anhedonia, disinterest, and detachment. The fifth criterion is increased arousal, of which two symptoms are necessary in order to meet a PTSD diagnosis. Increased arousal could take the form of irritable behaviour, hyper-vigilance, reckless behaviour, difficulty sleeping, difficulty concentrating, or an exaggerated startle response. The disturbance must cause significant impairment in functioning socially or in the workplace, and the symptoms cannot be attributed to substance abuse.

Theoretical Models of PTSD

There are multiple theories that have been used to explain the mechanisms of PTSD. Two theories used to help explain PTSD were rooted in basic behavioural and cognitive psychology, while another theory stems from research in the area of neuropsychology (Ehlers & Clark, 2000; Diamond, Campbell, Park, Halonen, & Zoladz, 2007; Lissek & Van Meurs, 2015).

Classical Learning Theory

Classical learning theory of PTSD is consistent with classical conditioning paradigms. From this learning perspective, symptoms of PTSD stem largely from maladaptive learning occurring during and after a traumatic encounter. Such learning manifests in both associative and non-associative forms. Through associative fear-conditioning, neutral stimuli associated with the aversive trauma acquire the capacity to trigger and maintain anxiety well after the occurrence of the traumatic episode. This conditioning process is thought to contribute centrally to the re-experiencing and avoidance symptoms that are often triggered by exposure to stimuli that

resemble aspects of the trauma, but are in fact indicative of no genuine danger (Lissek & Van Meurs, 2015). There is ongoing research in this area that suggests PTSD can be prevented through fear extinction learning (Lommen, Engelhard, Sijbrandij, Van Den Hout, & Hermans, 2013).

Classical learning models posit that conditioned behaviours can be extinguished (Schmajuk, 2008), however PTSD is extremely resistant to extinction, which cannot be accounted for by the model. PTSD is particularly resistant because it supported by a network of second-order conditionings that maintain behavioural strength in spite of the absence of the original unconditioned trauma (Wessa & Flor, 2007). So while it may be possible to prevent the development of the disorder, once the disorder has developed it is difficult to extinguish.

Cognitive Model

The cognitive model of PTSD focuses primarily on the maintenance of PTSD symptoms (Ehlers & Clark, 2000). The model posits that most individuals develop at least below-threshold symptoms of PTSD following exposure to a traumatic event, and that most of these individuals will see a reduction of symptoms over the course of a few months. However, for many individuals these symptoms can persist for years following exposure to trauma (Foa, Stein, & McFarlane, 2006).

PTSD symptoms are thought to persist after the traumatic event ends because people continue to experience a perceived sense of threat based on problematic appraisals of the event or the symptoms following the event. Individuals who develop persistent PTSD appraise the event in such a manner that they no longer view the traumatic experience as a time-limited event, but instead view it as having global implications about their future. In addition to problematic cognitive appraisals, problems with trauma-related memories also contribute to a continued sense

of threat. Problems in memory are typically observed in the findings that while individuals typically have a difficult time intentionally recalling details about the traumatic event, involuntary recall, a primary symptom cluster of the disorder, is often vividly detailed (Ehlers & Clark, 2000).

The Cognitive model is consistent with the main clinical features of PTSD and it can help explain several phenomena related to the disorder (Ehlers & Clark, 2000). The model also provides a framework for treatment by identifying three key targets for change, which have been supported by recent studies examining several aspects of the model (Ehlers, Mayou & Bryant, 1998; Dunmore et al.,1998).

Temporal Dynamics Model of Emotional Memory Processing Theory

The Temporal Dynamics Model of Emotional Memory Processing Theory describes PTSD as temporally sequenced patterns of long-term potentiation (LTP) and long-term depression (LTD) in the hippocampus, amygdala, and prefrontal cortex as part of a generalized response to stress.

LTP is the process whereby short-term memory is converted into long-term memory, which includes physical changes in the structure of neurons at the synapse. In contrast, LTD refers to processes that inhibit the creation of memories. Under extreme stress conditions the hippocampus switches function from integrating time and place to a focus on the immediate context. Typically, this period isolates the memory of the dangerous circumstance, which would naturally be used for protecting the organism from similar threats presented in the future. However, when this process is disrupted, the individual is prone to experience a more generalized stress response, rather than stress towards a particular stimulus (Diamond et al., 2007).

The result of this impairment is emotionally driven and inaccurate memories, which may present as an inability to make sense of the traumatic event. The individual can also experience inappropriate psychological and physiological responses, which can impair normal functioning and contribute to the development of psychiatric comorbidities (Foa, Stein, & McFarlane, 2006).

Impact of PTSD on Normal Functioning

Symptoms of PTSD cause considerable distress and can significantly interfere with an individual's social, educational and occupational functioning. Certain symptoms of PTSD, such as difficulties concentrating and problems sleeping, make it difficult for an individual with PTSD to pay attention, concentrate on a task, or keep organized. Not surprisingly, people with PTSD have higher rates of unemployment than people without PTSD, due to an inability to function effectively in the work place (Mills, Lynskey, Teesson, Ross, & Darke, 2005). The resulting financial problems are an additional source of stress, and have the potential to lead to extreme hardship such as homelessness.

The disorder can also have adverse effects on the individual's social relationships, leading to social withdrawal. People with PTSD are also more likely to have problems in their marriages than people without PTSD due to problems managing the negative symptoms of the disorder, financial strain caused by an inability to work, an inability to deal with crises, and a loss of intimacy (Dekel & Monson, 2010).

PTSD and Comorbid Disorders

It has been consistently shown that individuals with PTSD are more likely than those without PTSD to have substantial psychiatric comorbidity (Hernandez et al., 2013; Mills, Teesson, Ross, & Peters, 2006). Population-based surveys have demonstrated that the lifetime prevalence of PTSD with any other mental health (MH) disorder is approximately 80% (Helzer,

Robins, & Mcevoy, 1987), and have indicated that PTSD is most commonly associated with anxiety disorders, major depression disorder, bipolar disorder and SUDs (Cerimele, Bauer, Fortney, & Bauer, 2017; Galatzer-Levy, Nickerson, Litz, & Marmar, 2013).

Approximately 16% of patients with PTSD have one other psychiatric diagnosis, 17% have two other psychiatric diagnoses, and nearly 50% have three or more additional psychiatric diagnoses, which indicates that comorbidity between PTSD and other MH diagnoses is the rule rather than the exception (Brady, 1997). It is difficult to tell whether the development of PTSD contributes to the development of MH disorders, or whether MH disorders contribute to the development of PTSD. One possible explanation is that a prior history of MH disorders might be associated with an increased probability of trauma exposure resulting in an increased risk of developing PTSD. The other possible explanation is that PTSD might be associated with increased risk of subsequent disorders (Kessler, 2000; Sharkansky, Brief, Peirce, Meehan, & Mannix, 1999). Regardless of which disorder causes the development of the other, the co-occurrence of PTSD with other psychiatric disorders can be complex. At least a third of the individuals who develop PTSD remain symptomatic for three years or longer, and are at risk of secondary problems such as substance misuse (Kessler et al, 1995).

PTSD and Comorbid SUDs

PTSD and SUDs frequently co-occur and the overlap between these disorders has been an area of increasing clinical, research, and public health interest (Kaysen et al., 2014; Kline et al., 2014; Miller, Vogt, Mozley, Kaloupek, & Keane, 2006; Ouimette, Moos, & Finney, 2000; Read, Wardell, & Colder, 2013). A problem stemming from substance use related to PTSD symptoms is the development of SUDs with repeated use.

Although substance use can induce sensations of euphoria and well-being, these sensations are generally outweighed by negative consequences. These negative consequences include accidents leading to physical harm of self and others, impaired social relationships, and engaging in law-breaking behaviours and incurring subsequent difficulties with the law (Symmes et al., 2015). Specific drugs may also have other detrimental effects on one's physical well-being, including sleep disturbances and irregular sleeping patterns (Ogeil, Rajaratnam, Phillips, Redman, & Broadbear, 2011), acute memory problems, psychomotor impairment, and bronchial or pulmonary illnesses (Fischer et al., 2012). Substances are often abused as means of coping with the negative symptoms of PTSD and as a result, the individual can become vulnerable to developing SUDs, which could exacerbate other issues and make treatment of the disorder less effective (Bremner, et al., 1996).

Regardless of which substance has contributed to the problem, by the time an individual has developed a diagnosable SUD, the substance use will have adversely affected their overall level of functioning and their physical and mental health. Among individuals with both PTSD and SUDs, the symptoms of PTSD and SUDs tend to be more severe, which is supported by evidence that the comorbidity is associated with a higher rate of psychosocial and medical problems and higher utilization of inpatient hospitalization (Trivedi et al., 2015). Additionally, individuals with comorbid PTSD and SUDs are more prone to substance use relapse than individuals without PTSD (Brown, Stout, & Mueller, 1996). Once comorbid PTSD and SUDs develop, it is difficult to treat them separately because there is a great deal of overlap between the symptoms of both disorders (Mills, 2013).

Substances associated with PTSD. The following section describes specific substances that are commonly abused by individuals with PTSD.

Nicotine. Nicotine is a naturally derived substance of the tobacco plant, and it is nicotine that gives the tobacco plant its reinforcing and addictive properties (Berne, 2006). While nicotine can be self-administered via chewing or intranasally inhaling tobacco, the most common route of administration is by smoking, which is a profound contributor to mortality, bronchial or pulmonary illnesses, and various forms of cancer (Sellman, Wootton, Stoner, Deering, & Craig, 1999; Peto, 1994).

The prevalence of smoking in individuals with PTSD is twice that observed in the general population (Feldner et al., 2007; Lasser, Boyd, Woolhandler, Himmelstein, McCormick, & Bor, 2000). People with PTSD are more likely to be heavy smokers than the general population, with research suggesting that nearly 75% of individuals with PTSD smoke 20 cigarettes or more per day (Buckley, Susannah, Bedard, Dewulf, & Greif, 2004). Additionally, the rate of successfully quitting smoking among individuals with PTSD is lower than the rate among individuals without a mental health diagnosis (Hapke, Schumann, Rumpf, John, Konerding, & Meyer, 2005; Lasser et al., 2000).

Alcohol. Alcohol is a depressant with addictive properties that can contribute to or exacerbate medical and psychological conditions and therefore can negatively impact the lives of those who drink and the lives of others around them (Myers, Isralowitz, & Ebrary, 2011). Alcohol can have severe physical, personal, and societal consequences that include acute memory problems, liver disease, psychomotor impairment, increased risk-taking behaviours, accidents leading to physical harm, violent behaviours, and crime leading to imprisonment (Schuckit, 2006). Generally, alcohol has the effect of depressing the CNS, which is most evident with high levels of consumption though anxiolytic effects are noted at lower levels of

consumption (Schuckit, 2006). Alcohol is not only abused due to its ease of access, but also for its dampening effects on stress (Hefner & Curtin, 2012).

Approximately half of individuals seeking treatment for alcohol dependence meet current criteria for PTSD (Brown, Stout, & Mueller, 1999). Among individuals with co-occurring PTSD and alcohol dependence, the symptoms of PTSD tend to be more severe, and there is evidence that these individuals are more prone to alcohol use relapse than non-co-morbid individuals (Mills 2013). Co-morbidity of PTSD and alcohol dependence is also associated with a higher rate of psychosocial and medical problems and higher utilization of inpatient hospitalization than either disorder alone (McCarthy & Petrakis, 2010).

Other drugs. There are numerous illicit and misused prescribed substances that have varying physical and psychological effects. Some of the most widely abused drugs among those with PTSD include cannabinoids and opioids (Kevorkian, Bonn-Miller, Belendiuk, Carney, Roberson-Nay, & Berenz, 2015; Tull, Gratz, Aklin, & Lejuez, 2010).

Cannabinoids. Cannabis is one of the most commonly abused illegal substances with approximately 42.5% of the Canadian population report having ever used cannabis (Rotermann & Langlois, 2015). The availability and ease of access to cannabis has been increasing as government regulations change towards legalization of the substance, and thus much of the reported use is recreational. However, epidemiological research in the United States has identified that a diagnosis of PTSD is associated with a 21% prevalence of meeting comorbid cannabis use disorder criteria (Kevorkian et al., 2015), compared to 9.1% among the general population (Grucza, Agrawal, Krauss, Cavazos-Rehg, & Bierut,2016).

A cannabinoid is a classification of diverse chemical compounds that act on cannabinoid receptors throughout the nervous system. There have been 113 different cannabinoids isolated

from the cannabis plant, however tetrahydrocannabinol (THC) is the primary psychoactive compound in cannabis (Aizpurua-Olaizola et al. 2016). It has been suggested that the reinforcing and rewarding effects of cannabinoids occur as a result of cannabinoid receptor-mediated activation of the mesolimbic dopamine system in a manner similar to that observed with stimulants such as cocaine (Wickelgren, 1997). Recent research from the basic sciences (Covey, Mateo,Sulzer, Cheer & Lovinger, 2017) and clinical studies (Murray, Eisner, Obsuth, & Ribeaud, 2017) supports this idea.

Cannabis is frequently smoked to allow for the absorption of delta-THC into the lungs where it then passes quickly through the blood-brain barrier (Aldington et al., 2007). THC can also be administered through ingestion when dissolved in lipids, and *transdermal* patches able to deliver cannabinoids into the bloodstream (Borgelt, Franson, Nussbaum, & Wang, 2013). The user experiences a mild euphoria, a sense of relaxation, heightened sensory acuity, and a distorted perception of time (Cumo, 2013).

Opioids. Approximately one-third of individuals with an opioid use disorder will meet criteria for PTSD during their lifetime (Mills, Teesson, Ross, Darke, & Shanahan, 2005). Additionally, research on heroin and psychiatric diagnoses has suggested that those dependent on heroin with a comorbid diagnosis of PTSD have worse treatment outcomes, and have worse occupational functioning that those without a PTSD diagnosis (Teesson, Ross, & Darke, 2007).

The term opioids applies to any substance, whether endogenous or synthetic, peptidic or non-peptidic, that produces morphine-like effects through an action on opioid receptors (Katzung & Trevor, 2015). Opiods are all derived or synthesized from the opium poppy, Papaver somniferum. Each of these drugs has different characteristics, uses, and effects on the body. Whereas raw opium comes directly from the poppy plant, opiates such as morphine and heroin

are refined from opium (Scott, 1969). Other opioids such as OxyContin and methadone are synthetic derivatives of morphine. Many opioids are used medically and are prescribed for anxiety or for pain management. However, opioids are also used illegally and are often abused (Canadian Agency for Drugs Technologies in Health, 2012).

Over time, repeated use of opiates can desensitize the brain's natural opioid system, by inhibit the production of the body's natural opioid neuropeptides. This builds a tolerance, so the individual no longer responds to the drug as strongly and requires a higher dose to achieve the desired effect. Tolerance fuels addiction because the addict will need to continue to increase his dosage in order to experience the desired high. Tolerance can also contribute to overdose as higher doses are administered to reach the desired effect. Compared to all other illicit substance use, opiates are associated with the largest increase in deaths by overdose (Calcaterra et al., 2013). Because opiates are effective at reducing anxiety and pain, while providing a feeling of euphoria, they are commonly used as a form of self-medication among those with PTSD (Cottler, Compton, Mager, Spitznagel, & Janca, 1992; Tull et al., 2010).

Self-Medication of PTSD Symptoms

Individuals may use psychoactive substances to ‘‘self-medicate’’ for physical or psychological ailments or neurological imbalances that would otherwise seem overwhelming or insurmountable (Khantzian, 1985; Lagoni, Crawford, & Huss, 2011; Posner, Kass, & Hulvershorn, 2014). The emphasis on medicating for these painful feelings and emotional states is central to the self-medication hypothesis (Mariani, Khantzian, & Levin, 2014). The main aspects of the self-medication hypothesis includes drug use to relieve suffering and distress, and some degree of drug preference (Khantzian, 1997; Khantzian, 1985). From a self-medication perspective, it is important to consider what co-occurring psychiatric symptoms might be

targeted to alleviate the distress that individual is intentionally or unintentionally attempting to relieve with their drug-of-choice (Mariani, Khantzian, & Levin, 2014).

Self-medication is often cited as a possible explanation for the association between PTSD and SUDs (Kaysen et al., 2014; Kline et al., 2014; Miller, Vogt, Mozley, Kaloupek, & Keane, 2006; Ouimette, Moos, & Finney, 2000; Read, Wardell, & Colder, 2013). Proponents of self-medication assert that substance use occurs as an effort to cope with trauma and serves as a means of avoidance from distress among sufferers of PTSD (Brown & Wolfe, 1994; Khantzian, 1996). Applied to SUD-PTSD comorbidity, the theory suggests that an individual is exposed to trauma, develops PTSD, and then uses substances to cope with PTSD symptoms. The use of nicotine, alcohol and other substances may provide temporary relief for PTSD symptoms, although with cessation of substance use PTSD symptoms re-emerge. With continual substance use, a diagnosable SUD then develops over time. From this perspective, substance use can be understood as a learned response to internal or environmental affect-related cues (Bandura, 1991; Khantzian, 1997; Lang, Patrick, & Stritzke, 1999; Stewart, 1996).

While some literature suggests that pre-existing SUDs are related to increased odds of subsequent PTSD, the findings are inconsistent (Berenz & Coffey, 2012). More often, studies indicate PTSD predicts subsequent SUD, as it is consistent with the self-medication hypothesis, or the theory that individuals use substances to cope with psychiatric distress (Khantzian, 1999). PTSD precedes SUD in retrospective (Kessler, 1995) and prospective studies (Chilcoat & Breslau, 1998). Cross-sectional studies have demonstrated links between PTSD and using substances to cope with negative affect (Waldrop, Back, Verduin, & Brady, 2007), and that self-medication motives for use is a potential mediator of the relationship between PTSD and problem drinking/drug use (O'Hare & Sherrer, 2011). Taken together, there is a larger body of

literature supporting PTSD as a potential causal mechanism for the development of SUD rather than vice versa; however, causality has not been determined.

It is important to recognize that once an individual meets the criteria for both disorders, they influence each other. For example, past studies have shown that within-individual increases in PTSD symptoms are associated with increases in subjective cravings to administer substances (Simpson, Stappenbeck, Varra, Moore, & Kaysen, 2012), while improvements in PTSD symptoms are related to decreased substance use (Back, Brady, Jaanimagi, & Jackson, 2006). Research has shown that patients perceive a strong interrelationship between symptoms of PTSD and SUD in that when symptoms of one condition worsen, symptoms of the other condition also worsen, and that when symptoms of one condition improve, symptoms of the other condition improve (Back, Brady, Sonne, & Verduin, 2006; Brown, Stout, & Gannon-Rowley, 1998).

Consistent with the self-medication hypothesis, research has indicated that drug abuse is more strongly associated with avoidance and re-experiencing symptom clusters while alcohol use is associated with hyperarousal symptom clusters (McFall, Mackay, & Donovan, 1992). Essentially, these substances are used to avoid or dampen the negative symptoms that individuals with PTSD experience. Self-medication has been proposed as an explanation for the high levels of cannabis use reported by those with PTSD because the psychoactive properties of cannabis have the potential to alleviate some of the negative symptoms of PTSD, specifically those related to sleep and nightmares (Bonn-Miller, Babson, & Vandrey, 2014).

Self-medication could also account for the strong association between PTSD and smoking. It is possible that individuals with PTSD smoke to regulate their experience of PTSD symptoms (Beckham et al., 1997; Thorndike, Wernicke, Pearlman, & Haaga, 2006). This is supported by people with PTSD who smoke reporting that they smoke to reduce negative affect

and to increase stimulation (Feldner et al., 2007), which may reflect an attempt to avoid aversive emotions elicited by re-experiencing symptoms and to counteract numbing symptoms (Baschnagel, Coffey, Schumacher, Drobes, & Saladin, 2008).

While a specific mechanism to explain why individuals with a diagnosis of PTSD abuse substances is uncertain, it is known that substance use can exacerbate PTSD symptoms and lead to worse outcomes than the disorder alone. It is essential that health professionals are aware of comorbid substance abuse or dependence among those with PTSD because of the severity of these co-occurring disorders and the special attention that the comorbidity requires.

The Current Study

While there is a body of research investigating the associations between PTSD and smoking, alcohol use, and substance abuse or dependence, there has been no comprehensive investigation of these relationships using a population based study within a Canadian context. The aim of this study will be to estimate the prevalence and probabilities of PTSD and smoking, binge drinking, and SUDs from a nationwide Canadian sample. A series of hypotheses are made regarding expected relationships:

Hypothesis 1: Increased levels of smoking will be observed more frequently among individuals who report a diagnosis of PTSD than among individuals without a PTSD diagnosis.

Hypothesis 2: Increased levels of alcohol binge drinking will be observed more frequently among individuals who report a PTSD diagnosis than among individuals without a PTSD diagnosis.

Finally,

Hypothesis 3: SUDs will be observed more frequently among individuals who report a PTSD diagnosis than among individuals without a PTSD diagnosis.

Method

Participants

Data from the Public Use Microdata File of the 2012 Canadian Community Health Survey (CCHS; Statistics Canada, 2013) were analyzed. During this year, the CCHS assessed the mental health (MH) status of Canadians in terms of selected MH disorders, assessed functioning in relation to MH, and examined potential links between MH and sociodemographic variables. Respondents for the survey were selected in three stages. First, geographical areas were selected, followed by households within each geographical area. Finally, one respondent from each household was randomly selected. The national response rate for the survey was 68.9% with the CCHS-MH providing cross-sectional data from 25,113 Canadians aged 15 years or older who were residing in private residences in the 10 provinces at the time of the survey. Statistics Canada (2013) estimates the total number of individuals excluded from the survey to represent less than 3% of the target population. In the survey database, age is recorded categorically and ranges from "15 to 19 years" to "80 years or older." As the focus of the present study was on adults, individuals in the age category "15 to 19 years" (n = X) and age categories 65 years and older (n = X) were not included in the analyses. The sample size for each outcome measure is recorded individually in each "Results" subsection of this article.

Data Collection

The majority of interviews (87%) for the CCHS-MH (Statistics Canada, 2013) were conducted in person with the remaining interviews completed via telephone. No proxy interviews were allowed. Interviews were conducted by lay people trained by representatives from Statistics Canada's Collection Planning and Management Division. Interviews were completed using the computer-assisted personal inter- viewing (CAPI) method, which allows for

customs interviews for each respondent based on his or her individual characteristics and survey results, ensuring that interviewers do not ask questions that do not apply to the respondent (Statistics Canada, 2013). Data were collected during the period from January to December 2012. The survey sample does not include individuals living in the three Canadian territories, individuals living on reserves, full-time members of the Canadian Forces, or individuals who are institutionalized.

Materials

Self-report of PTSD. As part of the CCHS-MH interview, respondents were asked to indicate whether they had been diagnosed by a health professional with a variety of mental health conditions that had lasted or were expected to last for 6 months or longer. One of these questions was, "Do you have post-traumatic stress disorder?" (no/yes). Prevalence of PTSD was determined based on the number of individuals in the entire sample of respondents who responded "yes" to the question of whether they had been diagnosed by a health professional with PTSD.

Substance abuse behaviours. Alcohol abuse and dependence and substance use, abuse, and dependence modules do not require screening questions as all respondents are administered a minimum set of questions on smoking, alcohol, and substance use. The questions used for the CCHS-MH modules on nicotine use, alcohol abuse and dependence, and substance abuse and dependence are based on a recognized WHO version of the *Composite International Diagnostic Interview* (CIDI) modified for the needs of CCHS-MH (Statistics Canada, 2013). The WHO-CIDI is a standardized instrument for the assessment of mental disorders and conditions based on the definitions and criteria of *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; APA, 1994) and *International Classification of Diseases and Related Health Problems*

(*ICD-10*; WHO, 1992). Mental conditions or problems found in the CCHS-MH are partially coded to *DSM-IV* (Statistics Canada, 2013).

Computer-based algorithms were used to calculate criteria for each disorder based on respondents' answers to the questions within each disorder module. For each disorder, 12-month criteria included meeting the criteria for a lifetime diagnosis of the disorder, experiencing an episode of the disorder within the previous 12 months, and experiencing a marked impairment in occupational and social functioning. Smoking habits, frequency of alcoholic beverage consumption, and criteria for substance abuse or dependence (including cannabis) were examined over a 12-month period.

Type of smoker. Type of smoker assessed smoking habits (daily smoker, occasional smoker, or not at all). Type of smoker was assessed with the question, "At the present time, do you smoke cigarettes daily, occasionally or not at all?"

Alcohol binge drinking. Alcohol binge drinking is assessed by the frequency of consuming more than five drinks at one occasion within the previous 12 months (*never*, *less than once a month*, *once a month*, *2 to 3 times a month*, *once a week*, *more than once a week*). Alcohol binge drinking was assessed with the question, "How often in the past 12 months have you had 5 or more drinks on one occasion?"

Substance abuse or dependence. Substance abuse or dependence is a composite variable that identifies whether the respondent meets CCHS-MH/WHO-CIDI criteria for any drug abuse or drug dependence for the past 12 months. The respondents' use of illegal drugs or nonmedical use of prescription drugs during the past 12 months was assessed with questions such as, "The second group of medicines is stimulants, sometimes called speed, ice, glass, crystal, crank, pep pills, or uppers. These are medicines that people sometimes use to stay awake,

to improve their low mood, or to lose weight. Examples include dexamyl, methamphetamine, Adderall, and Ritalin. Have you ever used a stimulant non-medically?" and "Have you used it in the past 12 months?" Substances included in this variable are cannabis, cocaine/crack, club drugs, hallucinogens, heroin/opium, inhalants/solvents, or any other substances.

Sociodemographic covariates. A battery of sociodemographic covariates were used within the regression analyses, which included sex (male/female), age (5-year increments from 20 to 64), race (White/non-White), marital status (married, common-law, widowed, divorced/separated, single), education (less than high school, high school, some post-secondary, post-secondary graduation), income in Canadian dollars (less than $10,000; $10,000-$19,999; $20,000-$29,999; $30,000-$39,999; $40,000-$49,999; and $50,000 and above), and province (see Table 1 for descriptive statistics).

Mental health covariates. Mental health covariates were also used within the regression analyses. These covariates included diagnoses of generalized anxiety disorder, major depressive disorder, and/or bipolar disorder. These mental health variables are included because they are known to be comorbid with PTSD (Hernandez, 2013; Price & Van Stolk-Cooke, 2015), and it is important to account for their effect on the relationships between PTSD and smoking, alcohol binge drinking, and SUDs.

The effect of anxiety on the relationships between PTSD and smoking, binge drinking, and SUDs by including General Anxiety Disorder as an MH covariate was examined. Respondents met the CCHS-MH/WHO-CIDI criteria for lifetime Generalized Anxiety Disorder, if they reported having an episode of generalized anxiety lasting at least 6 months in the 12 months prior to the interview, and clinically significant distress or impairment in social, occupational, or other important areas of functioning. Next, the effect of depression on the

relationships between PTSD and smoking, binge drinking, and SUDs by including Major Depression as an MH covariate was examined. Respondents who met the criteria reported meeting the criteria for lifetime major depressive episode, having a major depressive episode in the 12 months prior to the interview, and clinically significant distress or impairment in social, occupational, or other important areas of functioning. We examined the effect of Bipolar Disorder as a covariate on the relationships between PTSD and smoking, binge drinking, and SUDs. The criteria for bipolar disorder 12-month episode are met when the respondent has met the criteria for lifetime bipolar disorder and has had a 12-month episode of Bipolar I, Bipolar II, or hypomania (see Table 1 for descriptive statistics).

Data Analysis

Hierarchical multinomial logistic regression (cigarette smoking and binge drinking) and hierarchical binary logistic regression (drug abuse or dependence) were used to investigate whether PTSD predicted substance abuse or disorders. Odds ratios (ORs) or relative risk ratios (RRRs) were used for each model to remain consistent with previous research (Saunders, Lambert-Harris, McGovern, Meier, & Xie, 2015; Sripada, Pfeiffer, Valenstein, & Bohnert, 2014).

In each model, demographic covariates (age, sex, race, marital status, household income, education, and province of residence) was assessed in Block 1, and mental health covariates were assessed in Block 2. Mental health covariates were chosen because of their relationships with either PTSD or SUDs. Finally, PTSD was assessed as a predictor in Block 3. Data were weighted in accordance with instructions from CCHS-MH user guide (Statistics Canada, 2013).

Researchers used a weighting variable provided by Statistics Canada (2013) to ensure that the analyses were representative of the broader Canadian population. All regression models

used heteroscedastic-consistent corrections in their estimation of standard error. All significant results of interest to the researchers were discussed in the context of their effect sizes, because small differences between groups may become statistically significant at high levels of statistical power. Significant effect sizes that were less than small (i.e., Cohen's $d < .20$) were not discussed substantively as they failed to meet the cut-off point. All data analysis was performed with Stata 15.

Results

Type of Smoker (n = 16,557). Using hierarchical multinomial logistic regression, researchers regressed smoking categories (using "Daily Smoker" as the base category) onto demographic covariates in Block 1, $\chi^2(40) = 524.29$, $p < .001$. Smoking categories were regressed onto mental health covariates in Block 2, $\chi^2(6) = 30.33$, $p < .001$. Anxiety, depression, and bipolar disorder predicted a greater likelihood of being in the base category than the "Never" and "Occasional Smoker" category.

In Block 3, researchers entered PTSD into the overall model, $\chi^2(2) = 13.38$, $p = .001$, and it significantly improved the overall prediction of smoking categories. Overall, PTSD was not helpful in differentiating between "Occasional Smoker" and "Daily Smoker"; however, persons who had PTSD were 2.1 times more likely to be in the "Daily Smoker" group than in the "Never" group (see Table 3).

Alcohol Binge-Drinking (n = 13,615). Using a hierarchical multinomial logistic regression, the prediction of binge drinking using "More than once a week" as a reference category was explored. Binge drinking categories were regressed onto demographic covariates in Block 1, $\chi^2(100) = 1150.93$, $p < .001$, which significantly improved the prediction of binge drinking. Mental health covariates were entered in Block 2, $\chi^2(100) = 26.98$, $p = .029$, which also

significantly improved the overall model. Generally, when persons reported they had bipolar disorder it was associated with a reduced likelihood of being in categories other than the base categories. Anxiety was generally non-significant, and depression was only sporadically associated with an increased likelihood of being in the base category. Framed differently, mental health covariates were inconsistently related to binge-drinking categories, although bipolar disorder tended to have a more consistent relationship.

Post-traumatic stress disorder was entered in Block 3, $\chi^2(5) = 13.47$, $p = .019$, which significantly improved the overall model. Participants who were identified as having PTSD were 3.7 times *more likely* to be in the base category ("Binge drink more than once a week") than the "Never binge drink" category, 5 times *more likely* to be in the base category than the "Binge-drink once a month" category, and 3.2 times *more likely* to be in the base category than the "Binge-drink 2-3 times a month" category. These significant findings represented medium-to-large effects sizes. Please note that while there were non-significant comparisons in "Binge drink once a week" and "Binge drink less than once a month", the directionality of the findings were the same (~2 times *more likely* to be in the base category). Overall, the hypothesis that PTSD would predict membership in the more severe binge-drinking category, was supported (see Table 2).

Substance Abuse or Dependence (*n* = 16,356). Using hierarchical binary logistic regression, researchers regressed drug abuse onto demographic covariates in Block 1, $\chi^2(20) = 203.45$, $p < .001$, which significantly improved the overall model. Researchers added mental health covariates in Block 2, $\chi^2(3) = 89.18$, $p < .001$; depression, bipolar disorder, and anxiety were all associated with scoring more positively for drug abuse or dependence.

Post-traumatic disorder was entered in Block 3, $\chi^2(1) = 6.87$, $p = .009$, and was a significant, positive predictor of drug abuse or dependence that was in the medium-to-large effect size range, OR = 2.98, 95% CI [1.31, 6.78], $z = 2.62$, $p = .009$. These results were consistent with the hypothesis that PTSD would be associated with substance abuse.

Discussion

Research indicates that co-occurring PTSD and substance abuse or dependence can have more severe negative consequences than either disorder alone, and the problems that individuals with PTSD experience are compounded with the addition of substance abuse or dependence symptoms (Mills, 2013). Therefore, the purpose of the present research was to estimate the prevalence and probabilities of smoking, alcohol binge drinking, and substance abuse or dependence among adults reporting a PTSD diagnosis within the general Canadian population.

Generally, the results from the current study suggest that having PTSD was associated with higher levels of binge-drinking, smoking, and substance abuse or dependence than not having PTSD. Results indicated that individuals who reported a diagnosis of PTSD were more likely to smoke on a daily basis, binge drink on a weekly basis, and to meet criteria for substance abuse or dependence than people who did not report a diagnosis of PTSD. These results were significant while including comorbid psychiatric variables for generalized anxiety, major depression, and bipolar disorder. Framed differently, these results suggest that while controlling for the shared variance between PTSD and generalized anxiety, major depression, and bipolar disorder, PTSD substantively and meaningfully contributed to the overall model. The significant effects associated with PTSD were in the medium-to-large effect size range, suggesting that there was practical significance to the current study's findings.

Although this analysis did not test the role of self-medication within these models, the

results do suggest that individuals with PTSD could be using substances to medicate for the negative symptoms that they experience as a form of coping. In self-medication models of substance abuse, drugs are thought to serve a coping function whereby they facilitate general mood regulation (Khantzian, 1997). There is reason to believe that some people use a diverse array of psychoactive drugs, including tobacco (Kassel, Stroud, & Paronis, 2003), alcohol (Cooper, Russell, Skinner, & Windle, 1992), cannabis (Schafer & Brown, 1991), and opioids (Calhoun et al., 2000) as a means of coping with stressors and regulating mood. Those with PTSD are known to experience high levels of distress brought on by symptoms of the disorder, which makes it is highly plausible that the high levels of substance use observed among this population is a result of self-medication.

Virtually all smokers attribute their smoking, at least in part, to its anxiolytic properties (Spielberger, 1986). Smokers report that they smoke more when they are stressed, angry, anxious, or sad (Shiffman, 1993), and they hold the expectation that smoking will alleviate these negative moods (Copeland, Brandon, & Quinn, 1995). Additionally, much of the research on comorbid PTSD and alcohol dependence suggests that individuals with PTSD are more likely than those without PTSD to endorse using alcohol to cope with negative affect (O'Hare & Sherrer, 2011; Waldrop, Back, Verduin, & Brady, 2007). This is likely because of the depressant properties of alcohol and the effect of decreasing stress and anxiety associated with PTSD. In terms of other drugs that are abused among those with a diagnosis of PTSD, cannabis and opioids are among the most commonly reported.

There is evidence that cannabis may be used by those with PTSD for affective vulnerability as a short-term emotion regulation strategy to reduce or manage perceived aversive psychological and mood states (Metrik, Kahler, McGeary, Monti, & Rohsenow, 2011). Cannabis

is often abused to decrease distress (Potter, Vujanovic, Marshall- Berenz, Bernstein, & Bonn-Miller, 2011), or to cope with symptoms of anxiety among the general population (Bonn-Miller, Vujanovic, & Zvolensky, 2008). Among those with PTSD, coping to relieve PTSD-related negative affect has been established as a powerful motivator for cannabis use (Bonn-Miller, Vujanovic, Boden, & Gross, 2011; Bujarski et al., 2012; Potter et al., 2011). Opioids, such as heroin, are also described by PTSD patients as having benefits for coping with negative affect and distress due to their calming effects and ability to relieve stress and ease physical tension (Calhoun et al., 2000).

Although many individuals with PTSD smoke, binge drink alcohol, and abuse other drugs in an attempt to cope with their negative symptoms, the development of SUDs can worsen their symptoms by leading to greater psychological distress (Najavits, Weiss, & Shaw, 1999), and worse psychosocial adjustment (Riggs, Rukstalis, Volpicelli, Kalmanson, & Foa, 2003). Therefore, comorbid PTSD should be considered when an individual does not have an adequate response to the standard treatment for a diagnosed SUD (Benton, Deering, & Adamson, 2012). Health professionals working with individuals diagnosed with PTSD need to be aware of concomitant substance use issues and should tailor their assessments to screen for substance use among individuals with PTSD.

Current treatments for PTSD primarily include Cognitive Behavioural Therapy and pharmacological interventions. CBT is a time-limited therapy that involves setting specific treatment goals and working towards the remediation of problematic symptoms. CBT treatments have been specifically designed for PTSD symptoms, and have demonstrated particularly strong empirical support and, subsequently, have been designated as evidence-based interventions for PTSD (Bernardy & Friedman, 2015). These treatments include, but are not limited to, exposure

therapy (Foa & Rothbaum, 1998), cognitive processing therapy (Resick, Monson, & Chard, 2010), and Imagery Rehearsal Therapy (Moore & Krakow, 2010).

In addition to CBT treatments, research in pharmacotherapy for PTSD has indicated that antidepressant and anxiolytic medications have been effective, specifically the selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs; Bernardy & Friedman, 2015; Friedman, Davidson, & Stein, 2009). The efficacy of SSRIs and other antidepressant medications is due primarily to the overlap of PTSD symptoms with those of depression and many anxiety disorders, and it has been suggested that SSRI or SNRI treatments are especially effective for PTSD symptoms such as irritability, anger, anxiety, and depression (Shiromani, Keane, & LeDoux, 2012). Treatment for PTSD symptoms will help reduce the level of substance use, but if substance dependence has developed it will need to be treated before the patient can benefit from treatments for PTSD (Berenz & Coffey, 2012). While the nature of PTSD and SUD comorbidity is largely unknown, it is clear that there are negative implications for comorbid PTSD and SUDs and that the individuals with this comorbidity represent a group with unique treatment needs.

Limitations

There are several limitations to the study. First, the PTSD was assessed using self-report, which is in itself a limiting factor. Individuals may not have had the opportunity to be diagnosed by a health professional. Without access to medical files or a diagnostic interview from the respondents, it was not possible to confirm the accuracy of respondents' self-reports. This lack of verification of PTSD diagnosis may have also affected the association between self-reported PTSD and related SUDs. Second, a significant limitation is that full-time military personnel were not included in the CCHS database. Military personnel have higher rates of PTSD than the

general populations due to exposure to traumatic events that take place in combat (Misca, & Forgey, 2017). Additionally, the database does not include prison populations. Research indicates an interlink between traumatic experiences and criminal behaviour revealing that offenders present a higher prevalence of PTSD and associated symptoms when compared with the general population (Wright, Borrill, Teers & Cassidy, 2006). Third, there was no information in the data to indicate symptom severity or the nature of current PTSD symptoms. Fourth, there was no information available in the data to identify whether participants had received any form of treatment for PTSD. Without treatment information, the researchers could not determine whether treatment mitigated the relationship between PTSD and SUD. As the focus of the present study was adults aged 20 to 64, these findings cannot be generalized to teen or senior populations. These age groups should be a focus of future research.

Generalized anxiety disorder, major depressive disorder, and/or bipolar disorder were included as covariates in the analyses to control for the co-occurrence of these disorders. However, the literature suggests that these disorders can precede the development of PTSD, and that having a diagnosis of these disorders is predictive of developing PTSD following exposure to a traumatic event (Yehuda, Halligan, & Bierer, 2001). Additionally, anxiety and depression are symptoms of PTSD, and by controlling for generalized anxiety disorder, major depressive disorder, and/or bipolar disorder as covariates in the analyses, the analyses may have also partially controlled for PTSD and attenuated the degree to which PTSD was shown to predict smoking, alcohol binge drinking, and substance abuse or dependence. This pattern of findings could be considered evidence for the self-medication model, although it is important to note that causal inferences cannot be made.

Although the aforementioned issues do present some degree of ambiguity within interpreting the results, it is important to note the strength of the study: a nationally representative sample of Canadians. Although researchers excluded groups from analyses, the retained sample still offers a high degree of external validity. Canadians from every province between the age of 20 and 64 were selected in a representative fashion. This level of external validity is unusual and was only possible because the CCHS-MH database was accessible. Future research would benefit from using a more comprehensive measure of PTSD that includes classification of symptoms, rather than relying on a self-reported diagnosis from a medical professional.

Conclusion

Limitations notwithstanding, this study improves our understanding of the relationship between PTSD and SUDs in a nationally representative sample. The results add to the literature suggesting that an individual with a diagnosis of PTSD is at an increased risk for developing a SUD. Further, the findings from this research highlight the importance of screening individuals with PTSD for comorbid SUDs, given the high rate of comorbidity between the two disorders.

Exposure to trauma cannot be prevented, therefore the prevention of PTSD is virtually impossible. Given that it is not possible to prevent traumatic events from occurring, the next best approach would be to promote resilience and provide behavioural strategies that provide individuals exposed to trauma the tools necessary to cope effectively with severely stressful situations. Therefore, future research should investigate the impact of improving resiliency and teaching behavioural strategies on reducing comorbid PTSD and SUDs symptom development.

The comorbidity of substance use disorders is a significant problem among people with PTSD. This study demonstrates that a self reported diagnosis of PTSD is associated with

increased severity of smoking, alcohol binge drinking, and drug abuse or dependence in a national Canadian sample. Routine screening and treatment of SUDs should be undertaken among those receiving treatment for PTSD, and conversely screening and treatment of PTSD may be justified in substance disorder treatment settings. Although these findings do not show cause and effect, they do support the self-medication hypothesis as they demonstrate a link both between the severity of PTSD symptoms and the severity of substance use.

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Table 1.

Descriptive Statistics for Demographic Substance Use, Demographic Covariates, and Mental Health Covariates by Post-Traumatic Stress Diagnosis (PTSD).

	No PTSD *n* = 13,210	PTSD *n* = 242
Frequency of 5+ drinks in past year		
Never	44.04%	39.74%
Less than once a month	26.02%	32.09%
Once a month	10.40%	6.06%
2-3 times a month	9.67%	5.73%
Once a week	6.21%	7.15%
More than once a week	3.48%	9.24%
Type of smoker		
Not at all	75.33%	55.06%
Occasional	6.81%	9.73%
Daily	17.85%	35.20%
Drug abuse/dependence past year		
Yes	1.75%	8.67%
No	98.25%	91.33%
Male	47.43%	73.98%
White	80.62%	76.88%
Marital Status		
Married	51.48%	30.21%
Common-law	14.84%	12.78%
Widowed	1.00%	6.75%
Divorced/Separated	8.23%	22.92%
Single	24.45%	27.34%
Education		
Less than high school	8.54%	17.08%
High school	15.33%	10.40%
Some post-secondary	6.79%	11.10%
Post-secondary graduate	69.34%	61.42%
Anxiety	6.01%	22.36%
Depression	6.18%	43.69%
Bipolar	2.26%	16.11%
Age (M/SD)	6.01/2.53	5.76/2.37
Income (M/SD)	6.18/2.79	4.61/3.04

Table 2.

Predicting Smoking with Demographic Covariates, Mental Health Covariates, and PTSD.

	Relative Risk Ratios/95% Confidence Intervals (from Robust SE)		
	Block 1	Block 2	Block 3
Daily Smoker [Base Category]			
Never Smoker			
Constant	2.57 [1.68, 3.92] ***	2.74 [1.79, 4.19] ***	2.74 [1.79, 4.20] ***
Anxiety		0.84 [0.61, 1.15]	0.90 [0.65, 1.24]
Depression		0.71 [0.55, 0.91] **	0.77 [0.59, 1.00] *
Bipolar		0.62 [0.42, 0.92] *	0.64 [0.44, 0.95] *
PTSD			0.47 [0.32, 0.71] ***
Occasional Smoker			
Constant	0.65 [0.28, 1.51]	0.70 [0.30, 1.64]	0.70 [0.30, 1.64]
Anxiety		0.47 [0.27, 0.84] *	0.48 [0.27, 0.86] *
Depression		0.77 [0.44, 1.33]	0.79 [0.44, 1.40]
Bipolar		0.67 [0.29, 1.56]	0.68 [0.29, 1.58]
PTSD			0.82 [0.35, 1.94]
Pseudo R^2	.069 ***	.071 ***	.073 **

Note. Due to space constraints, coefficients for Block 1 were omitted. Block 1 consisted of sex (male/female), age, ethnicity (non-white/white), marital status, education, income, and province.

$^{***}p < .001$, $^{**}p < .01$, $^{*}p < .05$

Table 3.

Predicting Frequency of Binge Drinking with Demographic Covariates, Mental Health Covariates, and PTSD.

	Relative Risk Ratios/95% Confidence Intervals (from Robust SE)		
	Block 1	Block 2	Block 3
More than once a week [Base Category]			
Never			
Constant	3.09 [1.15, 8.33] *	3.71 [1.41, 9.73] **	3.78 [1.48, 9.61] **
Anxiety		1.14 [0.45, 2.93]	1.28 [0.50, 3.24]
Depression		0.60 [0.34, 1.07]	0.68 [0.40, 1.16]
Bipolar		0.35 [0.14, 0.89] *	0.39 [0.17, 0.90] *
PTSD			0.27 [0.11, 0.65] **
Less than once a month			
Constant	5.86 [2.16, 15.87] **	7.06 [2.69, 18.56] ***	7.14 [2.78, 18.32] ***
Anxiety		0.80 [0.32, 2.04]	0.87 [0.34, 2.19]
Depression		0.70 [0.40, 1.21]	0.73 [0.44, 1.24]
Bipolar		0.31 [0.14, 0.70] **	0.33 [0.16, 0.69] **
PTSD			0.48 [0.20, 1.15]
Once a month			
Constant	2.77 [0.94, 8.19]	3.29 [1.14, 9.47] *	3.36 [1.20, 9.43] *
Anxiety		0.85 [0.32, 2.23]	0.95 [0.36, 2.47]
Depression		0.70 [0.37, 1.31]	0.80 [0.44, 1.46]
Bipolar		0.39 [0.15, 1.02]	0.44 [0.18, 1.07]
PTSD			0.20 [0.07, 0.53] **
2-3 times a month			
Constant	1.99 [0.65, 6.11]	2.43 [0.81, 7.27]	2.47 [0.84, 7.24]
Anxiety		0.94 [0.35, 2.51]	1.03 [0.39, 2.77]
Depression		0.45 [0.24, 0.86] *	0.49 [0.26, 0.93] *
Bipolar		0.41 [0.17, 0.97] *	0.45 [0.20, 1.00]
PTSD			0.32 [0.12, 0.82] *
Once a week			
Constant	2.2 [0.74, 6.57]	2.61 [0.90, 7.57]	2.63 [0.92, 7.49]
Anxiety		1.39 [0.53, 3.66]	1.48 [0.56, 3.89]
Depression		0.66 [0.34, 1.27]	0.69 [0.36, 1.34]
Bipolar		0.33 [0.13, 0.86] *	0.35 [0.14, 0.87] *
PTSD			0.54 [0.18, 1.64]
Pseudo R^2	.076 ***	.078 *	.079 *

Note. Due to space constraints, coefficients for Block 1 were omitted. Block 1 consisted of sex (male/female), age, ethnicity (non-white/white), marital status, education, income, and province.

***$p < .001$, **$p < .01$, *$p < .05$

Table 4.

Predicting Drug Abuse/Dependence with Demographic Covariates, Mental Health Covariates, and PTSD.

	Odds Ratio/95% Confidence Intervals (from Robust SE)		
	Block 1	Block 2	Block 3
Constant	0.03 [0.01, 0.09] ***	0.02 [0.00, 0.06] ***	0.02 [0.00, 0.06] ***
Sex (Male/Female)	0.35 [0.25, 0.49] ***	0.31 [0.22, 0.44] ***	0.29 [0.20, 0.42] ***
Age	0.74 [0.69, 0.80] ***	0.73 [0.67, 0.79] ***	0.73 [0.67, 0.79] ***
Race (Non-White/White)	1.90 [1.27, 2.84] **	1.68 [1.12, 2.52] *	1.71 [1.14, 2.57] *
Base (Married)			
Common-law	3.10 [1.66, 5.79] ***	2.81 [1.51, 5.22] **	2.79 [1.48, 5.23] **
Widowed	15.3 [5.29, 44.22] ***	15.93 [5.16, 49.14] ***	13.76 [4.23, 44.81] ***
Divorce	3.93 [2.14, 7.24] ***	3.34 [1.80, 6.18] ***	3.01 [1.62, 5.58] ***
Single	3.34 [1.99, 5.61] ***	2.88 [1.71, 4.86] ***	2.89 [1.71, 4.90] ***
Base (<High school)			
High School	0.88 [0.47, 1.63]	1.03 [0.54, 1.97]	1.08 [0.56, 2.07]
Some Post-Sec.	1.25 [0.64, 2.46]	1.4 [0.71, 2.79]	1.44 [0.73, 2.84]
Post-Sec. Grad.	0.65 [0.39, 1.08]	0.76 [0.45, 1.30]	0.79 [0.46, 1.34]
Income	0.96 [0.91, 1.02]	1.01 [0.95, 1.08]	1.01 [0.95, 1.08]
Base (NL)			
PE	1.07 [0.39, 2.91]	1.77 [0.47, 6.60]	1.56 [0.44, 5.57]
NS	2.33 [1.01, 5.37] *	3.45 [1.04, 11.44] *	3.31 [1.07, 10.18] *
NB	1.57 [0.66, 3.71]	2.29 [0.68, 7.75]	2.18 [0.69, 6.93]
QC	1.12 [0.50, 2.47]	1.69 [0.53, 5.40]	1.63 [0.55, 4.84]
ON	1.45 [0.67, 3.16]	1.93 [0.61, 6.10]	1.78 [0.61, 5.23]
MB	1.85 [0.78, 4.37]	2.69 [0.79, 9.13]	2.50 [0.79, 7.94]
SK	1.75 [0.62, 4.90]	2.47 [0.63, 9.73]	2.37 [0.64, 8.75]
AB	1.44 [0.62, 3.34]	2.02 [0.61, 6.73]	1.92 [0.62, 5.95]
BC	1.74 [0.78, 3.87]	2.50 [0.77, 8.07]	2.34 [0.78, 7.03]
Anxiety		2.35 [1.27, 4.34] **	2.22 [1.20, 4.11] *
Depression		3.98 [2.33, 6.82] ***	3.66 [2.07, 6.47] ***
Bipolar		2.48 [1.23, 4.98] *	2.26 [1.12, 4.56] *
PTSD			2.99 [1.32, 6.78] **
Pseudo R^2	.128 ***	.184 **	.189 *

***$p < .001$, **$p < .01$, *$p < .05$