Incident Traumatic Brain Injury in Precariously Housed Persons

by

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Abstract

Persons living in precarious housing face numerous mental and physical health risks, including disproportionally higher incidence of traumatic brain injury (TBI) compared with the general population. A number of challenges hamper the existent literature on incident TBI in this population, potentially attenuating estimates of TBI occurrence. In precariously housed persons, this study (1) captured TBI events in a prospective design that included participant education regarding injury sequelae and the use of a comprehensive and validated screening tool deployed repeatedly and proximate (i.e., monthly) to incident TBI, (2) characterized the types of TBI events that occurred through detailed assessment of injury details (i.e., count, severity, mechanism, acute intoxication), with test-retest reliability analyses on self-reported injury characteristics, and (3) identified specific risk factors for incident TBI, amongst broad predictor categories (i.e., substance dependence, psychiatric illness, prior brain injury, psychological functioning), through detailed pre-injury assessment, in order to inform targeted assessment and prevention strategies. Three hundred and twenty six participants were recruited from single-room occupancy hotels and screened monthly for incident TBI. Observed and estimated rates of TBI were obtained, and logistic and poisson regression identified pre-injury risk factors for TBI occurrence, severity, and count. Across TBI definitions and approaches to missing data, incidence proportion ranged from 18.7 to 50.7 percent, event proportion ranged from 27.9 to 91.1 percent, incidence rate ranged from 30,086 to 50,674 per 100,000 person-years, and event rate ranged from 44,882 to 91,104 per 100,000 person-years. Education, role functioning, schizophrenia spectrum disorder, opioid dependence, lifetime number of TBI, and lifetime history of TBI were significant predictors of TBI occurrence. This study makes four important contributions: (1) screening for brain injury at repeated proximal assessments (i.e., monthly) obtains a considerably higher self-reported rate of TBI in precariously housed persons, (2) this multimorbid population suffers from remarkably high rates of self-reported brain injury, and (3) several key and specific risk factors for TBI occurrence and (4) TBI severity were identified. Harm reduction strategies targeting those most vulnerable are imperative to improve functioning and prevent further injury and associated consequences.

Keywords: precarious housing; homelessness; traumatic brain injury; incidence; multimorbidity; risk factors

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Chapter 1. Introduction

1.1. Outline

Traumatic brain injury (TBI) is increasingly considered a critical public health and socio-economic problem throughout the world. TBI is a major cause of death and lifelong disability, commonly leading to neurocognitive deficits, behavioural changes, and mental health symptoms. Associated impairments have also been linked to further disruption of interpersonal relationships, as well as community, social and vocational integration (Roozenbeek, Maas, & Menon, 2013). Even still, TBI has been considered a silent epidemic, as the magnitude of the problem is thought to be largely unseen (Langlois & Sattin, 2005). This is particularly true for traumatic brain injury in the context of homelessness and precarious housing, which receives considerably less media and research attention than other populations with high rates for TBI (e.g., veterans, athletes). While much of the research on homeless and precarious housed persons has focused on other factors associated with poor cognitive and daily functioning, this environment also provides numerous risk factors for TBI occurrence and worse outcomes following TBI at both a biological and environmental level.

This study aims to highlight the pervasiveness of incident TBI in precariously housed persons. As an outline of the introduction to this study, ecological theory (Bronfenbrenner, 1979) and the ecology of homelessness (Nooe & Patterson, 2010) are described to outline the bidirectional relationship between homelessness, biopsychosocial risk factors, and health status, including TBI occurrence and outcomes. A description of precarious housing in Canada will be provided to highlight the numerous risk factors for TBI occurrence present in this environment, as well as the context within which TBI are acquired. The vulnerability of this population to sustain TBI and face worsened functioning is highlighted through these contextual factors of living in this setting, including altered levels of brain (Satz, 1993) and cognitive (Stern, 2002) reserve, combined with reduced access to medical care (Honer et al., 2017). Within this environment which is highly prone to injury, the rate and impact of TBI in precariously housed persons will be described, along with methodological challenges to measuring TBI in this population. Lastly, study objectives and hypotheses will be defined.

1.2. Theoretical Framework

Providing a foundation for the bidirectional relationship between housing environment and health status, which includes TBI occurrence and outcomes, modern theories propose that a complex process unfolds involving a system of interactions within the individual, and between the individual and the environmental contexts of which they are a part (Ettekal & Mahoney, 2017). Bronfenbrenner's (1979) seminal ecological systems theory described human experience across the lifespan with a set of nested levels of the environment. There are four interrelated types of environmental systems including, the micro-, meso-, exo-, and macro-systems. These levels range from proximal settings that directly influence individuals to more distal settings that are thought to indirectly influence human experience. The levels within ecological systems theory are often presented graphically as a series of four systems nested around a focal individual like a set of concentric circles, each impacting health status at multiple levels (Ettekal & Mahoney, 2017). Continuing this work, Bronfenbrenner's (2005) bioecological systems theory emphasized the active role that people play in their experience. Rather than passive observers, there is a transactional relationship between people and the systems within which they function. Within the bioecological systems theory, health status is viewed as the joint product of the person, context, process, and time. Person factors include individual demographics and characteristics that interact with the setting to influence outcomes. Context factors include the four systems of Bronfenbrenner's original ecological systems theory (i.e., the micro-, meso-, exo-, and macro-systems). As the hypothesized primary mechanism of experience, process factors represent the reciprocal connections between person and context factors. These bidirectional relationships are thought to cyclically influence health status and outcomes. Lastly, and describing the dynamic nature of development and experience, processes occur in a place (i.e., microtime), over a period of time (i.e., mesotime), and within a specific historical context (i.e., macrotime; Ettekal & Mahoney, 2017). Thus, people can be seen as both the cause and the effect of their situation, such that each change they make causes a reactive change in the larger dynamic system. (Segev, Levinger, & Hochman, 2017). Poor physical and mental health must be assessed at multiple levels of analysis, viewing negative life experience as the result of the interaction between individual vulnerabilities and unfortunate contextual factors (Toro, Trickett, Wall, & Salem, 1991).

From an ecological perspective, TBI occurrence and outcomes can be understood as the result of multi-directional connections between individual risk factors, socioeconomic structures, and circumstances of the homeless and precariously housed environment (Toro, Trickett, Wall, & Salem, 1991). Recognizing the complexity of this environment, Nooe and Patterson's (2010) ecology of homelessness model attempts to analyze the transactional natural of factors contributing to homelessness. Rather than implying unidirectional causation, individual risk factors are thought to increase vulnerability for homelessness and health status while being integrated with many other interacting factors. Specifically, the model examines the relationship between individual and structural biopsychosocial risk factors, individual and social outcomes, temporal course, and housing status/outcomes. Biopsychosocial risk factors comprise a range of factors that may make an individual more apt to acquire brain injuries and suffer worse associated consequences, including individual biology, developmental experience, and social circumstances such as housing availability, housing stability, and poverty. On an individual level, factors such as one's demographics, personality, developmental experiences, health, and mental health interact within each other and between different levels. On a structural level, factors such as societal culture including discrimination, social practices including public policies, social situations, and resource availability including health care. Through both direct and indirect multi-directional relationships, individual and structural biopsychosocial risk factors influence each other. Thus, while poor health status may lead an individual towards homelessness, the reverse also applies, that homelessness may lead to worsened health outcomes. Understanding the environment within which individuals live is critical to obtaining a complete understanding of factors leading to poor health outcomes including TBI.

As an ecological approach to public health, Rhodes and colleagues (Rhodes, 2002; Rhodes, Singer, Bourgois, Friedman, & Strathdee, 2005) introduced the conceptual framework of the 'risk environment,' defined as the space in which a variety of factors interact to increase the chances of drug-related harm, or health and vulnerability in general. Within the basic framework, the risk environment comprises two dimensions, types of environment and level of environmental influence. Physical, social, economic, and policy make up the four types of environment, while there are micro and macro levels of environmental influence. There is also consideration of two environmental factors that account for the mechanisms of how environments structure

risk. Susceptibility factors are those that determine the rate at which an epidemic is propagated, while vulnerability factors make it more or less likely that morbidity will have deleterious impacts (Barnett et al., 2000). Under this framework, the risk environment paradigm has been applied to identify ways in which housing is linked to health risk (McNeil et al., 2015).

Precarious housing functions as a risk environment for poor health outcomes, such that substance use and multimorbidity may lead to susceptibility for further pathology and adverse effects. For example, features of the physical environment (e.g., unsanitary living quarters, presence of used syringes in common areas) have been associated with substance use in order to “tune out” the environment (Knight et al., 2014; Lazarus et al., 2011). The risk for both TBI occurrence and associated outcomes is also increased through both individual and structural risk factors inherent to the environment. In this way, homelessness and TBI are bidirectional associated, such that homelessness is a known cause of and consequence of TBI (Hwang et al., 2008). Based on ecological theory (Bronfenbrenner, 1979), research on risk and protective factors for TBI occurrence emerged in an attempt to reduce the number of individuals suffering from brain injury and the associated negative consequences, including homelessness. Similarly to the ecology of homelessness (Nooe & Patterson, 2010), home, school, religious, and social influences are conceptualized as biopsychosocial risk and/or protective factors acting as main effects or interactions in the relationship between these variables and TBI outcomes (Wilde et al., 2010).

1.3. Precarious Housing as Risk Environment

Having a clear understanding of the precarious housing environment highlights the associated biopsychosocial risk and/or protective factors for TBI occurrence. As the centre of our social, emotional, and often economic lives, housing is the basis of stability and security for an individual or family (United Nations, 2001). Safe and affordable housing is both necessary for human health and a means to reduce systemic health inequities and lower associated long-term healthcare costs. Housing is directly linked to physical safety (Canada Mortgage and Housing Corporation, 2014) and health status (McNeil et al., 2015). A good home is critical in allowing persons to fully participate in the economic, social, and cultural lives of their community and their country (Wellesley Institute, 2010). Within Canada, acceptable housing is defined as housing that is

adequate, suitable, and affordable. Specifically, acceptable housing is that which is not in need of major repair, has sufficient bedrooms for the size and make-up of the household, and can be obtained without spending 30 percent or more of before-tax household income (Canada Mortgage and Housing Corporation, 2014). While increasingly viewed as a commodity, acceptable housing is most importantly an internationally recognized human right (United Nations, 2001) and the Canadian government has acknowledged its obligation to ensure all Canadians are well-housed (United Nations, 1976).

Even still, at least 235,000 Canadians experience the risk environment of homelessness in a given year, and over 35,000 on a given night (Gaetz, Dej, Richter, & Redman, 2016). Furthermore, a large portion of homelessness and housing insecurity in Canada is hidden from view. For every person experiencing homelessness an estimated 3.5 people experience hidden homelessness, temporarily staying with friends, relatives, or others, because they have nowhere else to live and no immediate prospect of permanent housing (Eberle, et al., 2009). Of the 12 million households in Canada, approximately 1.6 million households are in core housing need, being unable to access acceptable housing and living in precarious housing that is unaffordable, overcrowded, and/or unfit for habitation (Canada Mortgage and Housing Corporation, 2014). As rental and ownership costs rise, housing affordability continues to diminish, and approximately 1.5 million households are involuntarily paying 30 percent or more of their income on shelter (Wellesley Institute, 2010). An estimated 3.3 million households live in homes that require repairs, and 1.3 million of those households report the need for major repairs that affect the health and safety of the people living in the home. Even more prevalent than homelessness, precarious housing in impoverished environments may create comparably severe consequences for its occupants (Argintaru at al., 2013; Fazel, Geddes, & Kushel, 2014; Honer et al., 2017; Hwang, Wilkins, Tjepkema, O'Campo, & Dunn, 2009).

Across major cities within Canada, single room occupancy (SRO) hotels make up a large portion of precarious housing, sometimes referred to as the "housing of last resort" (Bowen & Mitchell, 2016). Initially, SRO hotels were built in the early 1900s as temporary living quarters for transient laborers in the local resource industries. During the 1960s, levels of poverty and substance use grew among residents. As injectable cocaine was introduced into the area, rates of human immunodeficiency virus (HIV) rose

to epidemic levels. From this history, the Downtown Eastside of Vancouver, British Columbia, remains an area with high numbers of low-income and unemployed residents, with low-cost SRO rooms often the only alternative to homelessness. More recently, the Downtown Eastside, where many of the SRO hotels are placed, has been given the designation of the poorest postal code in Canada. Each SRO tenant lives in a room that is 8 to 12 square metres in size, including a sink and possible hotplate to prepare food. Washroom and shower facilities are shared by 10 to 15 tenants. This precarious housing often fails to meet one or more of the criteria for acceptable housing, as many SROs rarely meet housing affordability standards, are infested with pests, have fire safety concerns, and have washroom and shower facilities that raise questions concerning suitability (Canada Mortgage and Housing Corporation, 2014; Jones et al., 2013; Linden, Mar, Werker, Jang, & Krausz, 2013; Vila-Rodriguez et al., 2013). While some SROs are owned and/or managed through partnerships with public housing or non-profit housing providers, many SRO buildings are privately operated and run as businesses (Bowen & Mitchell, 2016). SRO units are generally the least costly independent housing option in cities, excluding room sharing, yet still constitute a significant financial strain on their residents (Bowen & Mitchell, 2016). Within the Downtown Eastside, SRO residents typically pay 60 percent or more of their income on rent, despite available government assistance (Shannon et al., 2006), double the rent burden (i.e., the ratio of income to rent; Belsky, Goodman, & Drew, 2005) associated with affordable housing and above the 50 percent cut off establishing “severe” or high rent burden (Joint Center for Housing Studies of Harvard University, 2013).

Within this environment, homeless and precariously housed persons experience numerous biopsychosocial risk factors for poor health outcomes and TBI occurrence. Of those experiencing homelessness in Canada, over 72 percent are male and over half are made up of adults between the ages of 25 to 49. Homeless and precariously housed young and older adults experience a mortality rate over eight times that of age- and sex-matched Canadians (Jones et al., 2015). In fact, the probability that a 25-year-old living in shelters, rooming houses, or single room occupancy hotels will survive to the age of 75 is only 32 percent for men and 60 percent for women (Hwang, Wilkins, Tjepkema, O’Campo, & Dunn, 2009). As a potential mechanism of TBI events, those experiencing homelessness in Canada are also 35 times more likely to be the victims of physical assaults than those housed. Approximately 35 percent of homeless persons in Toronto,

Canada, report being physically assaulted within the previous year, with two thirds of those reporting more than one assault within the year (Khandor, & Mason, 2007; Zakrison, Hamel, & Hwang, 2004). Through bidirectional relationships with TBI, 25 percent of homeless adults experience cognitive impairment, with the mean overall intellectual functioning falling approximately one standard deviation below average (Depp, Vella, Orff, & Twamley, 2015). This vulnerable population faces poor social and occupational functioning, including work productivity, independent living, and social relationships (Vila-Rodriguez et al., 2013). Worsened TBI outcomes in this population are likely given the substantial neurocognitive burden attributable to high rates of multimorbidity (Gicas et al., 2014), with individuals experiencing a median of three co-morbid illnesses (Vila-Rodriguez et al., 2013). In a precariously housed population, substance dependence affected nearly all individuals, with approximately 61 percent injection drug use within the previous year. Acute intoxication may increase the likelihood of falls and impulsive behaviours resulting in TBI events. The majority experience mental illness, with psychotic illness being the most common (Vila-Rodriguez et al., 2013). Medically, precariously housed persons experience high rates of physical illness. Positive viral serology is common for human immunodeficiency virus and hepatitis C virus. Approximated 45 percent of individuals experience neurological illness, with 28 percent of individuals having pathological findings on neuroimaging. The most common findings include brain aneurysm, infarction, hemorrhage, and TBI (Vila-Rodriguez et al., 2013), with more than half of homeless and precariously housed individuals having a history of TBI (Stubbs et al., 2020). With this, precariously housed persons demonstrate higher rates of seeking emergency department care for substance use disorders, and mental and neurological illness (Honer et al., 2017). However, while access to medical care is high among homeless and marginally housed persons (Hwang et al., 2013), clinical treatment effectiveness is variable (i.e., highest for HIV/AIDS, intermediate for opioid dependence, and lowest for psychosis; Honer et al., 2017). While many suffer from TBI, the availability and access to appropriate treatment, and the likelihood of sufficient recovery from TBI in the precarious housing environment, is questionable.

1.4. Traumatic Brain Injury in Precariously Housed Persons

While the impact of multimorbidity is considerable in precariously housed persons, many of these comorbidities also act as risk factors for further brain injury. In fact, many of the risk factors for TBI occurrence that have been highlighted in research examining TBI in the general population are highly prevalent in precariously housed persons. First off, precariously housed persons experience many of the same common risk factors for TBI faced in the general population. Considering demographic factors, TBI occurs most frequently in males compared to females, across all age groups (Faul, 2010). In a population based sample, the male to female rate ratio was found to be 1.67 to 1 (Feigin et al., 2013). Age has also been found to be a significant predictor of TBI occurrence, with TBI rates highest among young children age 0 to 4 (1337.3 per 100,000), older adolescents ages 15 to 19 (896.2 per 100,000), and older adults age 75 and above (932 per 100,000; Faul, 2010; Hyder, Wunderlich, Puvanachandra, Gururaj, & Kobusingye, 2007). Education and socioeconomic status have also been linked to TBI occurrence, with lower levels of each associated with an increased likelihood for recurrent TBI (Lasry et al., 2017).

Additional less common risk factors for TBI occurrence in the general population are much more prevalent in precariously housed persons, making this already vulnerably population uniquely at risk for incident TBI. When considering substance use and psychiatric illness, pre-injury alcohol and drug abuse are well-established risk factors for TBI (Corrigan, 1995; Parry-Jones, Vaughan, & Miles Cox, 2006; Taylor, Kreutzer, Demm, & Meade, 2003). In a meta-analysis of TBI in those with and without a history of alcohol/substance abuse (Unsworth & Mathias, 2017), those who sustained a TBI had a 37 to 79 percent prevalence of pre-injury alcohol abuse (Parry-Jones et al., 2006; Taylor et al., 2003) and an 18 to 37 percent prevalence of pre-injury drug abuse (Soderstrom et al., 1997; Taylor et al., 2003). Individuals with diagnoses of schizophrenia, bipolar disorder, and depression have been found to report higher rates of lifetime TBI (Malaspina et al, 2001). Lastly, having a history of TBI has been associated with TBI occurrence at follow-up (Lasry et al., 2017).

While these factors remain important considerations in predicting TBI occurrence in the general population, given that precariously housed persons experience high levels of substance dependence, psychiatric illness, and neurological illness including lifetime

history of TBI, along with lower levels of education and socioeconomic status, this population is at particularly high risk for TBI. These risk factors for TBI occurrence are inherent to the homeless and precariously housed environment, providing a risk environment for TBI in this already vulnerable population. Of the limited research examining risk factors for incident TBI in homeless and vulnerably housed persons, Nikoo and colleagues (2017) found that, in line with the general population, lifetime history of TBI, endorsing a history of mental health diagnoses at baseline, problematic alcohol and drug use, younger age, and poorer mental health were associated with increased risk of incident TBI during the follow-up period. Additionally, residential instability was associated with incident TBI occurrence, highlighting the relationship between housing and TBI risk.

In addition to these known risk factors, precariously housed persons may experience risk for TBI through bidirectional associations. Separate from health-related risk factors, psychological risk factors for TBI have been largely ignored, despite a handful of studies that have found related factors (i.e., cognition, impulsivity) to precede TBI occurrence (Nordstom, Edin, Lindstrom, & Nordstom, 2013; Olson-Madden, Forster, Huggins, & Schneider, 2012). TBI is known to deleteriously impact role functioning (Temkin, Corrigan, Dikmen, & Machamer, 2009); however, this relationship may be bidirectional such that many of the areas impacted may also act as further risk factors for TBI occurrence. Role functioning refers to an individual's capacity to perform the tasks of daily life typical to one's age and natural environment (Anatchkova & Bjorner, 2010; Goodman, Sewell, Cooley, & Leavitt, 1993). Role functioning can be examined within the various domains in which most adults operate, including personal self-care, cognitive and affective functioning, social and familial relationships, and vocational and/or educational achievement. High levels of role functioning can be demonstrated by the ability to maintain working productivity, live and care for oneself independently, and be a part of safe and stable personal relationships. Low levels of role functioning can involve minimal skills or adaptability to function at work or home, the inability to practice appropriate self-care, and/or demonstrating deviant or avoidant social behaviour resulting in poor integration into the community (Goodman, Sewell, Cooley, & Leavitt, 1993). Role functioning is thought to represent the dynamic interaction between health conditions, and personal and environmental contextual factors (World Health Organization, 2002). Thus, examining role functioning may provide a more individualized

measure of functioning than physical or psychiatric diagnoses alone because, while these factors can be closely related, role functioning can help to identify how a well a person is functioning within their personal and social environment while accounting for the variability in adjustment and response to personal circumstances (Goodman, Sewell, Cooley, & Leavitt, 1993). Currently, role functioning is considered a Traumatic Brain Injury Common Data Elements recommended psychosocial risk factor measure (Wilde et al., 2010) conceptualized under Bronfenbrenner's (1979) ecological systems theory (Gerring, & Wade, 2012), yet further research is needed in adult populations.

Given the high prevalence of numerous of risk factors for TBI present in precariously housed persons, it is unsurprising that TBI is increasingly understood to be highly prevalent in precariously housed populations. A recent systematic review and meta-analysis found that more than half of homeless and precariously housed individuals report a history of TBI, with one quarter reporting a history of moderate or severe TBI (Stubbs et al., 2020). Homeless and precariously housed persons have also been found to experience a disproportionally higher incidence of TBI compared with the general population. The annual incidence proportion of TBI in homeless or vulnerable housed persons have ranged from 17.1 to 19.4 percent, or at least 17,100 per 100,000 (Nikoo et al., 2017), which is considerably higher than the pooled North American annual incidence proportion of 331 per 100,000 (.331 percent) in the general population (Nguyen et al., 2016). Among individuals with available data, 37.2 percent of individuals reported at least one incident TBI during a three year follow-up period (Nikoo et al., 2017). Considering this vulnerable population experiences high rates of cognitive impairment, neurocognitive burden, poor social and occupational functioning, and multimorbidity including neurological illness, high rates of TBI necessitates consideration of the impact of subsequent brain injury in these individuals.

Generally, while some individuals experience minimal acute effects of TBI and quickly return to pre-injury functioning, others experience significant deficits that persist despite rehabilitation attempts. In fact, there is significant variability in outcomes between individuals with similar types and severity of injuries (Lingsma, Roozenbeek, Steyerberg, Murray, & Maas, 2010). To better predict outcomes in cognition and functioning following brain injury, the theories of brain (Satz, 1993) and cognitive (Stern, 2002) reserve have been applied to TBI populations (Leary et al., 2018; Steward et al., 2018). Broadly, brain reserve is a passive model which posits that brain size or neuronal

count directly impact the amount of brain insult required before clinical deficits emerge (Katzman, 1993), and that there is individual variability in the threshold of vulnerability to clinical symptoms in those with equal brain tissue or neuronal loss (Satz, 1993). Cognitive reserve refers to an individual's ability to actively cope with brain damage by using pre-existing cognitive processes or by enlisting compensatory processes (Stern, 2002). Cognitive reserve is often measured using proxies, such as estimated premorbid intelligence and length of exposure to cognitively stimulating life events (Steward et al., 2018). Higher levels of reserve are thought to lead to better functional outcomes by moderating the effects of TBI, impacting both the extent of acute impairments and the speed of recovery over time (Bigler & Stern, 2015). The theory of cognitive reserve has been further divided into "neural reserve" and "neural compensation" models (Stern, 2009). Broadly, the neural reserve model posits that the capacity and efficiency of one's cognitive processing systems protect brain networks from disruption following injury to the brain, altering acute cognitive functioning following brain injury. Neural compensation model posits that existing brain networks may be capable of adaptation and network reorganization following injury, allowing individuals to recover more rapidly from pathological disruption of pre-existing networks by enlisting compensatory networks (Stern, 2009; Steward et al., 2018). From an ecological perspective, brain and cognitive reserve act as risk factors for worsened functioning, interacting with other factors to moderate outcomes.

In a meta-analysis of the contribution of brain and cognitive reserve to outcomes following TBI, Mathias and Wheaton (2015) found that proxy measures of brain (i.e., age, sex) and cognitive (i.e., education, premorbid intelligence) reserve have consistently been found to moderate outcomes after TBI. Low brain and cognitive reserve leave individuals less able to shoulder greater levels of neuropathology before neurobehavioral manifestations occur (Patel et al., 2013). As proxies for lower levels of cognitive reserve, older age, lower education, lower premorbid intelligence, and lower occupational attainment have been associated with worse post-TBI cognitive outcomes across a broad range of cognitive abilities (i.e., verbal and visuospatial learning and memory, verbal fluency, working memory, executive functioning, information processing and visuomotor speeds; Green et al., 2008; Kesler, Adams, Blasey, & Bigler, 2003; Leary et al., 2018; Rassovsky, Levi, Sela-Kaufman, Sverdlik, & Vakil, 2015). After controlling for injury severity, cognitive reserve has been associated with post-TBI mood,

vocational and status, and social and daily functioning (Rassovsky, Levi, Sela-Kaufman, Sverdlik, & Vakil, 2015; Salmond, Menon, Chatfield, Pickard, & Sahakian, 2006). As proxies of low brain and cognitive reserve, our research group previously characterized the multimorbidity of a large sample of precariously housed persons and found high rates of neurological illness, including brain infarction and history of head injury, and low educational and occupational achievement, making this population apt to suffer worse consequences following TBI (Vila-Rodriguez et al., 2013).

Although brain and cognitive reserve theories have helped in accounting for much of the variability in functioning following brain injury, conceptually, low reserve captures only one set of the many possible risk factors for neurocognitive burden. The aggregate impact of multiple risk factors, including proxies for brain and cognitive reserve, results in neurocognitive burden, which is the total level of burden on one's neurocognitive functioning. Beyond direct proxies for brain and cognitive reserve, the multitude of risk factors that precariously housed populations often face across the lifespan (e.g., developmental, substance use, viral infection, psychiatric illness, neurological illness and brain injury) impose a substantial neurocognitive burden (Gicas et al., 2014). From this perspective, many precariously housed persons experience continuous insults to the brain, leaving individuals with less capability to deal with further brain insult. For instance, this population has been found to experience high rates of viral infection, substance dependence, mental illness including psychosis, history of head injury, and other neurological illness including brain infarction, with a median number of three multimorbid illnesses (Vila-Rodriguez et al., 2013).

Our research group examined the impact of these risk factors and lifetime prevalence of TBI on cognitive functioning in a precariously housed sample and found that the burden of aggregate risk factors accounted for more than one third of the variance in cognitive functioning (O'Connor, 2016). Various risk factors for neurocognitive burden have been found to interact with the effects of TBI, eliciting a synergistic deleterious impact on cognition and functional outcomes (Monti et al., 2013; Moretti et al., 2012). Compared to those with TBI alone, additional neurocognitive deficits have been found in persons with TBI and comorbid depression (Chamelian & Feinstein, 2006) and substance abuse (Unsworth & Mathias, 2017). Pre-injury alcohol use has been found to predict TBI outcomes in some studies (Barker et al., 1999; Dikmen, Donovan, Lberg, Machamer, & Temjkin, 1993; Ponsford, Tweedly, & Taffe,

2013; Wilde et al., 2004), but not others (Allen, Goldstein, Caponigro, & Donohue, 2009; De Guise et al., 2009; Lange et al., 2014; O'Dell et al., 2012; Turner, Kivlahan, Rimmele, & Bombardier, 2006; Vickery et al., 2008). Of note, studies reporting a significant relationship between pre-injury alcohol use and outcomes use samples that include more severe TBI, which is not the case for milder spectrum TBI (Silverberg et al., 2016). Similarly, alcohol intoxication at the time of injury has been associated with worse short-term outcomes in one study with mild to severe TBI (Vickery et al., 2008), but not others examining mild to moderate TBI (Lange et al., 2007, 2014; Scheenen et al., 2016; Silverberg et al., 2016), with mixed neuropsychological and functional recovery (Joseph et al., 2014; Lange et al., 2014, 2007; Lange, Iverson, & Franzen, 2008; O'Dell et al., 2012; Scheenen et al., 2016; Schutte & Hanks, 2010; Tate, Freed, Bombardier, Harter, & Brinkman, 1999). Cognitive deficits have been associated with psychotic disorders (Saykin et al., 1994), viral infection (Simioni et al., 2010), neurological illness or insult (Vermeer, 2003), and poor vascular health (Virta et al., 2013), which may moderate outcomes following TBI.

As homeless and precariously housed persons face high rates of both TBI and comorbidities adding to neurocognitive burden, these individuals are apt to suffer worsened recovery and outcomes following brain injuries. For instance, having a history of substance abuse has been found to be associated with poorer neuropsychological recovery following TBI, increased chance of obtaining a second head injury, and higher mortality rates following TBI. Alcohol intoxication at the time of injury has been found to be associated with acute post-TBI complications, longer hospital stays, and poorer discharge status (Corrigan, 1995). In TBI inpatients, diagnoses of psychosis and schizophrenia are associated with increased likelihood for adverse discharge disposition (Brandel et al., 2017). In fact, while a major moderator of immediate and long-term outcome following TBI is injury severity (i.e., measured by loss of consciousness, post-traumatic amnesia, daze and confusion, and/or Glasgow Coma Scale; Dikmen et al., 2009), more accurate prediction of worse neuropsychological and functional outcomes have been found from TBI severity in combination with pre-injury alcohol abuse (Dikmen, Donovan, Løberg, Machamer, & Temkin, 1993), lower cognitive reserve (Donders & Stout, 2018; Rassovsky et al., 2015), lower educational attainment, worse cognitive functioning, and higher levels of anxiety (Ponsford, Draper, & Schonberger, 2008). Unsurprisingly, given the ubiquity of these comorbid risk factors, TBI in precariously

housed persons has been found to be associated with wide-ranging deleterious outcomes. Similar to findings in other community samples (Silver, Kramer, Greenwald, & Weissman, 2001), our research group has previously reported that persons with a history of TBI had a higher rate of current mood disorder, worse current mental and physical health, and more ongoing neurological symptoms (Schmitt et al., 2017). In a recent systematic review and meta-analysis, TBI was found to be broadly associated with poorer health and functioning in this population (Stubbs et al., 2020), which is likely influenced by the interaction of TBI with other health risks that also impact cognitive and functional outcomes, thus worsening the effects of multimorbidity in this vulnerable population (Monti et al., 2013; Moretti et al., 2012; Schimitt et al., 2017; O'Connor, 2016).

1.5. Limitations of Current Research

Given the deleterious impacts and worsened outcomes of TBI in precariously housed persons, having a clear understanding of the pervasiveness of TBI in this vulnerable population is imperative. While generally understood that TBI disproportionally impacts precariously housed persons compared to the general population, a number of challenges hamper the existent literature aiming to determine the rate of TBI in this population, resulting in potentially attenuated estimates of TBI. First, while the incidence of TBI in precariously housed persons is known to be disproportionally higher than that of the general population (17.1 to 19.4 percent compared to .331 percent; Nguyen et al., 2016; Nikoo et al., 2017), there is insufficient research examining the epidemiology of TBI in precariously housed populations. Other populations facing relatively high rates of TBI (e.g., 170 per 100,000 incidence proportion of sport-related TBI; Theadom et al., 2014) are given a high degree of attention compared to the relatively underdeveloped literature on TBI in precariously housed persons.

Second, like the broader TBI literature, the ascertainment methods of TBI in precariously housed persons chiefly rely upon self-report. However, the accuracy of self-report in samples of precariously housed persons may be lower due to the extensive multimorbidities commonly faced by these individuals, which can negatively affect cognition and memory (Ennis, Roy, & Topolovec-Vranic, 2015; Gicas et al., 2014). This population may be predisposed to forget minor injuries and have recall bias surrounding

more severe injuries, particularly across longer follow-up periods. Adding to this concern, participants are often asked to retrospectively recall TBI events over extensive timeframes where accurate recollection is improbable. Since a high percentage of marginally housed persons experience multiple TBI throughout their lifetime (Barnes et al., 2015; Topolovec-Vranic et al., 2012), TBI would be best captured through ongoing repeated assessments.

Third, the majority of studies in the area have not aimed to primarily address TBI (Topolovec-Vranic et al., 2012), but rather have focused on overall health and housing status. Consequently, while important contributions have been made, studies have not typically adopted comprehensive and well-validated measurement tools (Bailie et al., 2017). With this, important injury details including the mechanism of injury and TBI severity are often missed, leading to a less than comprehensive understanding of the pervasiveness of TBI in precariously housed persons. Few studies have comprehensively characterized the types of injuries occurring in this population and personal factors that predict injury occurrence. In order to best acquire this information, TBI screening in research with similar populations would also benefit from participant education, considering that the public's understanding of TBI-associated symptoms has consistently been found to be inadequate (Gouvier, Prestholdt, & Warner, 1988; Guilmette, & Pagloa, 2004; Willer, Johnson, Rempel, & Linn, 1993).

Fourth, the assessment of risk factors for TBI occurrence in marginally housed persons has been limited to broad predictive categories (e.g., substance dependence) despite considerable variation within these groupings and their relationship to TBI (e.g., TBI outcomes following alcohol versus drug use history; Unsworth & Mathias, 2017). In this complex and multimorbid population, further granulation is needed to identify specific risk factors within the precarious housing environment for incident TBI to improve outcomes and help target prevention strategies. Bidirectional associations between psychological risk factors for TBI have been largely ignored, representing an under-recognized set of factors that take into consideration the individual variation in cognition, behaviour, and functioning, despite physical and psychiatric diagnoses.

1.6. Study Objectives

The objectives of the present study were threefold. First, in precariously housed persons, we captured TBI events in a prospective design that included participant education regarding injury sequelae and the use of a comprehensive and validated screening tool deployed repeatedly and proximate (i.e., monthly) to incident TBI. Second, we characterized the types of TBI events that occurred in this marginalized sample through detailed assessment of injury details (i.e., count, severity, mechanism, acute intoxication). In addition to these methodological improvements that are thought to improve the reliability of reports, test-retest reliability analyses were conducted on self-reported injury characteristics. Third, we identified specific risk factors for incident TBI, amongst broad predictor categories (i.e., substance dependence, psychiatric illness, prior brain injury, psychological functioning), through detailed pre-injury assessment, in order to inform targeted assessment and prevention strategies.

1.7. Hypotheses

The pervasiveness of head injury in this population was examined by first determining the annual incidence proportion, in order to understand the risk of acquiring a TBI over a one person-year period. Individuals who are homeless or living in precarious housing have been found to have high lifetime prevalence (Hwang et al., 2008; O'Brien et al, 2015) and incidence rates (Nikoo et al., 2017) of TBI. Previous research screened for self-reported TBI yearly; however, given that memory problems are commonly found in marginally housed populations (Ennis, Roy, & Topolovec-Vranic, 2015), more proximal, consistent screening is needed in this population. This research screened for incident TBI monthly, which was predicted to reduce the number of missed head injuries and improve the accuracy of reports. With this, we hypothesized that the annual incidence rate of TBI in this study would be higher than the 19.4% (highest of reports) previously found in homeless and vulnerably housed samples across three Canadian cities (Nikoo et al.; **Hypothesis 1**).

As a supplementary analysis, in order to understand the types of TBI most often acquired in this population, the mechanism and severity of injuries were characterized. In our previous research examining history of TBI in a similar cohort of homeless and vulnerably housed individuals, injury severity was 45% mild and 55% moderate to

severe (Schmitt et al., 2017). Considering our previous study inquired about participants' most *serious* past head injury, incident TBI were expected to have a higher proportion of mild injuries. Given the somewhat overlapping sample, the most common mechanisms of incident TBI were predicted to be related to those previously demonstrated (i.e., assault, motor vehicle accidents, falls, and biking/sport-related incidents), with higher proportions falls and biking/sport-related incidents, as the most frequent mechanisms of injury (Rao, McFaull, Thompson, & Jayaraman, 2017).

Next, pre-injury risk factors for incident traumatic brain injury were examined to identify demographic characteristics and comorbid factors that make individuals more apt to acquire a TBI. Given the high rate of incident TBI found in homeless and marginally housed persons (Nikoo et al., 2017), as well as the high rate of comorbidity in this population (Jones et al., 2015; Ludwig et al., 2012; Patel & Burke, 2009; Shannon, Ishida, Lai, & Tyndall, 2006; Vila-Rodriguez et al., 2013), it is necessary to identify which factors make an individual more apt to acquire a TBI in the future, in order to best target preventative strategies and TBI screening measures. It was hypothesized that sex, lifetime history of TBI, alcohol dependence, diagnosis of depression, diagnosis of primary psychotic disorder, cognition, impulsivity, and role functioning would be significant predictors of TBI occurrence (**Hypothesis 2**). First off, this hypothesis was based on research of the general population, whereby TBI occurs most frequently in males (Faul, 2010), those with pre-injury alcohol and drug abuse (Corrigan, 1995; Parry-Jones, Vaughan, & Miles Cox, 2006; Taylor, Kreutzer, Demm, & Meade, 2003), as well as diagnoses of schizophrenia, bipolar disorder, and depression (Malaspina et al, 2001). Although age has been found to be a significant predictor of TBI occurrence (Faul, 2010; Hyder, Wunderlich, Puvanachandra, Gururaj, & Kobusingye, 2007), our sample is largely made up of young adults (mean age 44) who are not within the age ranges more apt to acquire a TBI and, as a result, age was not hypothesized to be a significant predictor in our sample. This hypothesis was also based on the limited research examining risk factors for incident TBI in homeless and vulnerably housed persons, where having a lifetime history of TBI, endorsing a history of mental health diagnoses at baseline, problematic alcohol and drug use, younger age, poorer mental health, and residential instability were associated with increased risk of incident TBI during the follow-up period (Nikoo et al., 2017). Amongst possible psychological risk factors, lower cognition (Nordstom, Edin, Lindstrom, & Nordstom, 2013) and impulsivity (Olson-Madden, Forster,

Huggins, & Schneider, 2012) have been found to precede TBI occurrence in the general population and high TBI risk populations, respectively. Role functioning is commonly impacted following TBI (Temkin, Corrigan, Dikmen, & Machamer, 2009) but may also act as a risk factor for TBI occurrence, such that individuals with lower role functioning are more apt to sustain TBI.

As a supplementary analysis, pre-injury risk factors for incident traumatic brain injury severity and count were examined. Injury severity has been found to be a major moderator of cognitive functioning following TBI (Dikmen et al., 2009; Schretlen & Shapiro, 2003), and repetitive mild TBI events have been linked to chronic neuropathological and neurobehavioural changes (Mouzon et al., 2013). Given that this population faces high rates of traumatic brain injury, identifying individual characteristics associated with more severe and/or repetitive injuries, may help to identify those at increased risk of worsened outcomes. A summary of all predicted associations is presented in Table 1.

Table 1 Summary of Predicted Associations

Hypothesis	Variable	Outcome / Predictor Variable
1	TBI incidence proportion	Higher than 19.4%
2	TBI occurrence across one person-year	Sex, lifetime history of TBI, alcohol dependence, depression, primary psychotic disorder, cognition, impulsivity, role functioning

Chapter 2. Methods

2.1. Participants

Between November 2008 and May 2018, 524 individuals were recruited from four single room occupancy (SRO) hotels located in a low-income neighbourhood of Vancouver, British Columbia, as part of a longitudinal study (Hotel study; see Vila-Rodriguez et al., 2013). Briefly, persons were eligible if they were SRO residents, able to communicate in English, and provided written, informed consent. These were the only inclusion/exclusion criteria for the Hotel study. Subsequently, between December 2016 and May 2017, a total of 326 individuals were recruited to be a part of the Hotel-TBI sub-study (recruitment numbers per month: 40 December 2016, 111 January 2017, 64 February 2017, 65 March 2017, 25 April 2017, 20 May 2017), which involved going to participants SROs or waiting for participants to come to the research office for their regularly scheduled monthly meetings. Between December 2016 and May 2018, these 326 individuals completed monthly TBI screening assessments (see Figure 1 for flow diagram of participant inclusion). A description of the sample demographic and clinical characteristics is provided in Table 2. The sole inclusion criterion was English fluency. Participants provided written informed consent and received small honoraria after each completed screening assessment. Ethics approval was obtained from the University of British Columbia - Providence Health Care Research Ethics Board (H16-01310) and the Simon Fraser University Office of Research Ethics (2016s0586).

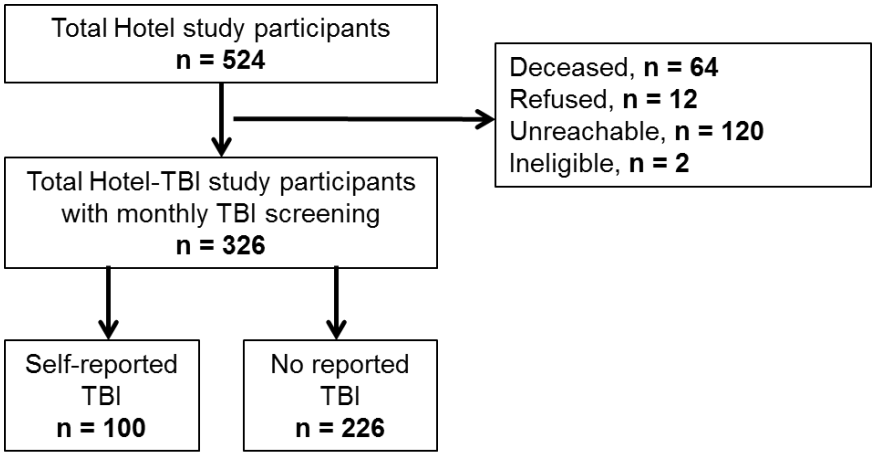


Figure 1 Flow diagram of participant inclusion

Table 2 Sample Demographic and Clinical Characteristics

Clinical Characteristic	*Total N*	*N*	*%*
		M (SD)	
Demographics			
Age (years)	326	40.5 (11.3)	
Education (years)	326	10.5 (2.3)	
Monthly Income (CAD)	322	850.3 (415.3)	
Sex			
Males	326	239	73.3
Females	326	87	26.7
Ethnicity			
Caucasian	324	180	55.6
Indigenous	324	91	28.1
Other	324	53	16.4
Alcohol & Drug Dependence			
Alcohol	297	59	19.9
Stimulant	297	232	78.1
Opioid	297	129	43.4
Cannabis	297	113	38.0
Other	294	27	9.2
Psychiatric Illness			
Depression	297	42	12.9
Bipolar spectrum disorder	296	32	10.8
Schizophrenia spectrum disorder	297	55	18.5
Substance induced psychotic disorder	297	47	15.8
Psychosis NOS	297	36	12.1
Other	295	170	57.6
Medical & Prior Brain Injury			
Lifetime number of TBI	326	Median = 3.00 (IQR = 4.0)	
Lifetime history of TBI	326	108	33.1
MRI-defined TBI	283	15	5.3
Incident TBI	326	100	30.7
With LOC	326	61	18.7
Without LOC	326	39	12.0
History of seizures/epilepsy	323	50	15.5
History of stroke	321	13	4.0
History of heart attack/disease	293	33	11.3
Viral Infection			
HIV	296	41	13.9
HepB	291	98	33.7
HCV (QPCR positive)	298	185	62.1
Cytomegalovirus	227	149	65.6
Herpes simplex virus	284	236	83.1
Psychological			
Number of criminal offenses	244	27.9 (19.1)	
Fetal alcohol spectrum disorders	297	14	4.7
Attention deficit hyperactivity disorder	322	78	24.2

Clinical Characteristic	*Total N*	*N*	*%*
		M (SD)	
Cognitive disorder NOS	297	39	13.1

Note. NOS = not otherwise specified; TBI = traumatic brain injury; LOC = loss of consciousness; HIV = human immunodeficiency virus; HepB = hepatitis B virus; HCV = hepatitis C virus.

2.2. Procedures

TBI screening assessments were completed by trained research assistants supervised by a Neuropsychiatrist (WP) and Registered Psychologist (AET). Participants were provided with an educational pamphlet outlining common causes and symptoms of TBI (see Appendix A). The pamphlet also provided contact information for a nearby hospital emergency room and local walk-in medical clinics, and participants were instructed to first seek medical attention in the event of a head injury. Participants reported TBI events at or between monthly assessments.

TBI screening occurred monthly over one person-year period (i.e., a one year period from entry into the study for each individual). A total of 2520 screening assessments were completed across 326 person-years, with 2433 unique months screened. On average, participants completed 7.73 screens across a 12 month period (*SD* = 3.63; *median* = 8.00), with a range of 1 to 14 screens. Across the possible 326 person-years, data was present across 202.75 person-years (37.81% missing data).

Following the framework outlined by Richter and colleagues (2019) for handling missing data in observational TBI research, missing data patterns were examined and the missingness mechanism (i.e., the reason why the data are missing; Rubin, 1976) was determined. To account for missing data, and understand the target population appropriately by determining the missing data mechanism, three approaches were taken to estimate rates of TBI. In approach A, a conservative estimate (i.e., likely underestimate of true rate) was obtained by imputing zeros to all the missing values (i.e., no reported TBI). In approach B (i.e., likely overestimate of the true rate), a liberal estimate was determined by treating missing values as missing completely at random (MCAR) and removing them. In approach C, presumed to be the closest to the estimated rate of TBI if there was no missing data (see discussion section), an estimate was determined by using multiple imputation based on the available observations while controlling for relevant variables found to be associated with missingness.

For approach C, longitudinal missing data patterns were first examined. Specifically, relevant demographic (i.e., age, sex, ethnicity, education), time variant (i.e., month of year, screen order, recruitment time), psychiatric (i.e., schizophrenia spectrum, psychosis not otherwise specified, depression, bipolar spectrum diagnoses; alcohol, stimulant, opioid, methadone, and cannabis dependencies), and TBI-related (i.e., acquiring a TBI any time within one's person-year, severity of TBI acquired at any time within one's person-year, number of TBI acquired within one's person-year, acquiring a TBI in month prior or following, severity of TBI in month prior or following, enrollment in additional clinical assessments) variables were examined for their association with whether the data was missing versus present across all possible time points. The dependent variable was dichotomously coded for whether the participant was present or missing at each possible monthly screening, with each participant's scores as a cluster. Generalized linear modeling determined that recruitment date (i.e., the date at which participants were recruited into the study) was the only variable found to predict missingness. Specifically, being recruited into the study at a later date was associated with more missing data. Thus, the missingness mechanism was considered to be *missing at random* (*MAR*; Rubin, 1976), since missingness was associated with an observed variable. Multiple imputation analyses were then performed to impute missing data (see Data Analysis section). Of note, comparable longitudinal analyses were also performed to determine whether relevant variables were associated with whether participants came in to report a TBI or not; no variables were found to predict reported TBI. Thus, no observed relevant variable predicted whether participants were more or less likely to come in and report a TBI.

To determine head injury rates, two TBI definitions were employed. First, the standard definition of TBI (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004) was operationalized as any self-reported trauma to the head or neck, with known cause, resulting in any length of loss of consciousness (LOC), post-traumatic amnesia (PTA), daze, and/or confusion. As a second and more conservative estimate, TBI was defined as a LOC from any self-reported trauma to the head or neck, with known cause (i.e., excluding those with only PTA, daze, and/or confusion). For both TBI definitions, when individuals had no memory of the event (i.e., retrospective PTA and/or drug or alcohol blackout), an observer report or physical evidence of trauma to the head was required.

Incidence proportion (number of individuals with TBI out of total number of individuals), event proportion (number of TBI events out of total number of individuals), incidence rate (number of individuals with TBI over total screening time), and event rate (number of TBI events over total screening time) were calculated over a person-year period. TBI count was operationalized as the number of TBI events (using standard TBI definition) that occurred over a person-year period.

To characterize the types of TBI events, participants reported the mechanism and context of their injuries, including whether their injuries occurred during a drug overdose or acute intoxication. TBI events were considered to have occurred during a drug overdose if there was (a) an observer report or observable sign of head trauma, (b) self-reported drug use (not including alcohol use only) at the time of injury, and (c) self-report of required naloxone administration. In this sample with ubiquitous substance dependence, acute intoxication at the time of injury was defined as participant self-report of intoxication by drugs or alcohol *beyond typical use*. Given the reliability results, severity classification was determined according to length of LOC only. TBI were defined as mild with no LOC or LOC less than or equal to 30 minutes; moderate/severe with LOC more than 30 minutes; or unknown with an unknown length of LOC (but known LOC occurrence; Carroll, Cassidy, Holm, Kraus, & Coronado, 2004). Finally, our previous work demonstrated sex differences in mechanism of history of TBI (Schmitt et al., 2017), so rates and characteristics of TBI events were further separated by sex.

Several categories of risk factors for TBI were evaluated (i.e., substance dependence, psychiatric illness, prior brain injury, psychological functioning), using data from the most proximal assessment prior to participants' first TBI screening assessment. Substance dependence and psychiatric illness were determined through monthly screening by trained research assistants and yearly assessment by a psychiatrist (WGH, OL). Prior brain injury was evaluated with self-reported neurological history at study entry and yearly MRI of the brain. Lastly, to determine psychological functioning, trained assistants conducted yearly neurocognitive and bi-annual health and role functioning assessments.

2.3. Traumatic Brain Injury and Risk Factor Measurement

Demographic Information

Self-reported age, sex, ethnicity, and education were ascertained through standard interview, which incorporated questions from the Canadian Community Health Survey (Statistics Canada, 2003). Regarding sex, individuals could self-report being male, female, or transgendered.

Traumatic Brain Injury

For determining head injury rates, TBI occurrence was assessed using the Ohio State University TBI Identification Method Interview Form (OSU TBI-ID; Corrigan, & Bogner, 2007), which is a Traumatic Brain Injury Common Data Elements recommended measure (Hicks et al., 2013). To further characterize TBI events, additional injury details were obtained using a TBI screening questionnaire (see Appendix B).

Substance Dependence and Psychiatric Illness

Substance dependence and psychiatric illness were diagnosed through psychiatric interview according to the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision; DSM-IV-TR; American Psychiatric Association, 2000) in consensus with the Best Estimate Clinical Evaluation and Diagnosis 2 (BECED-II; Endicott, 1988). Information was collected on mental status (psychiatric interview), Axis I symptoms (Beck Depression Inventory 2nd ed.; Beck, Steer, & Brown, 1996; Mini International Neuropsychiatric Interview; Sheehan et al., 1998), psychotic symptoms (Positive and Negative Syndrome Scale; Kay, Fiszbeis, & Opler, 1987), and personality disorders (International Personality Disorder Examination - Screener; Loranger, Janca, & Sartorius, 1997). Self-reported attention-deficit/hyperactivity disorder was assessed through a structured medical history interview. A Composite Harm Score (CHS) was previously developed to index the personal risk from substances used (Jones et al., 2013). Briefly, as an extension of the Independent Scientific Committee on Drugs (ISCD) assigned quantitative scores indexing the overall harm of individual drugs, the CHS incorporates the number and type of substances used, and the frequency of use by an individual participant ($CHS = \Sigma_{1\text{-}13}$ = ICDS Harm score x Frequency).

Prior Brain Injury

Self-reported neurological illness, including history of TBI (history of "*serious* head/face injury" with associated LOC), stroke, and seizures and/or epilepsy, were reviewed through structured interview. Lifetime number of TBI was assessed using the Brain Injury Screening Questionnaire (Dams-O'Connor et al., 2014). Objective history of traumatic brain injury was determined through consensus review of anatomical brain MRI by a neuropsychiatrist and qualified neuroimaging scientist (DJL, WJP).

Psychological Functioning

Crystallized reading ability was estimated using the Wechsler Test of Adult Reading (WTAR; Wechsler, 2001) standard reading score. A composite fluid cognition measure was calculated as the standardized mean of the Hopkins Verbal Learning Test – Revised (HVLT-R; Brandt, 1991) immediate recall score, the Stroop Color and Word Test (Golden, 1978) interference score, and the signal detection (A') score from the Rapid Visual Information Processing (RVIP) subtest of the Cambridge Neuropsychological Test Automated Battery (CANTAB; Fray, Robbins, & Sahakian, 1996). Decision making was estimated using the Iowa Gambling Task mean net score (i.e., proportion of cards selected from the advantageous decks; Bechara, Damasio, Damasio, & Anderson, 1994), while self-reported impulsiveness was measured using the Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995).

A composite role functioning measure was calculated as the standardized mean of the total scores from the Role Functioning Scale (Goodman, Sewell, Cooley, & Leavitt, 1993) and the Social and Occupational Functioning Assessment Scale in the Diagnostic and Statistical Manual and Mental Disorders (4th ed., DSM-IV; American Psychiatric Association, 1994). Domains examined include working productivity, ability for independent living and self-care, and extent of immediate or extended social network relationships.

Health Risk

The Maudsley Addiction Profile physical and mental symptom scores were used to estimate physical and mental health (Marsden et al., 1998). Further details on self-

reported physical symptoms and medical illness were reviewed through structured interview.

Chapter 3. Data Analysis

3.1. Rate of Traumatic Brain Injury

To capture TBI rate, we report both observed rates using the available data and estimated rates using three approaches (i.e., approaches A, B, and C). For the estimated rates, missing data was either assumed to be zero (approach A), assumed to be missing at random and removed (approach B), or imputed (approach C), each resulting in complete datasets with no missing data. For approach C, to create the complete dataset for estimation, the Markov chain Monte Carlo (MCMC) method (Schafer, 1997) with full conditional specification was used. Data from all present months was used as the predictor for the imputation of data for missing months, while controlling for any variable associated with missingness (i.e., recruitment date). Fifty imputations were completed, with a maximum of 50 iterations in each (sets of 10, 20, 30, 40, 50, 100 were completed with no significant difference in findings). The observed dataset accounted for 202.75 person-years. After imputation, the complete dataset accounted for 326 person-years.

3.2. Risk Factors for Traumatic Brain Injury

Regression analyses were conducted to examine risk factors for three related incident TBI dependent measures: TBI occurrence, severity, and count. For all analyses, assumptions were met and the number of TBI events per number of variables in the model was not found to exceed values thought to cause bias and/or precision errors (Peduzzi, Concato, Kemper, Holford, & Feinstein, 1996). When predicting TBI occurrence, individual hierarchical binomial logistic regressions were conducted for each predictor category (i.e., substance dependence, psychiatric illness, prior brain injury, psychological functioning). Variables found to be significant predictors in any of the categorized models were then entered into a final model. Binomial logistic and Poisson regressions were used to predict TBI severity and count, respectively. Both analyses included only individuals who acquired at least one incident TBI over a one person-year period (excludes those with no TBI). Relevant risk factors were screened for their individual impact on TBI severity and count (see Appendices C and D, respectively).

Variables with a small effect size (*d* = .2; Cohen, 1992) or higher were entered into a final regression model. All statistical analyses were conducted using SPSS version 24.0 (IBM Corp., 2016). A two-tailed test was used and a p-value less than 0.05 was considered statistically significant.

Chapter 4. Results

4.1. Reliability

A subset of individuals (N = 42) repeated the TBI screening questionnaire regarding the same TBI event at a later date (*mean* = 7.88 days, *SD* = 4.78, range 4-19 days), and test-retest reliability analyses were conducted on self-reported injury details. Using Cicchetti (1994) guidelines, reliability was found to be excellent for reports of mechanism of TBI event (*ICC* = .950); excellent for occurrence of LOC (*ICC* = .909); excellent for length of LOC (*ICC* = .973); fair for occurrence of PTA, daze, and/or confusion (*ICC* = .453), and poor for length of PTA, daze, and/or confusion (*ICC* = .155). Severity classification (mild, moderate/severe) according to only LOC remained consistent for 90.3% of participants.

4.2. Rate of Traumatic Brain Injury

Critical to determining the true rate of TBI in this population, we examined the number of TBI events across the sample and observation period. During the 326 person-years period, 175 TBI events were reported across 100 participants. This indicates that 100 participants reported at least one TBI event and 226 reported no TBI events within their person-year period. With this, the observed incidence proportion (100 individuals with TBI out of 326 total individuals) was 30.7 percent, or 30,674.85 per 100,000 population. The observed event proportion (175 TBI events out of 326 total individuals) was 53.7 percent, or 53,680.98 TBI per 100,000 population. The observed incidence rate (100 individuals with TBI over 202.75 person-years) was 0.493218 persons per year, or 49,321.82 per 100,000 person-years. The observed event rate (175 TBI events over 202.75 person-years) was 0.863132 events per year, or 86,313.19 per 100,000 person-years. Of those 100 participants who acquired at least one TBI event during this time, 61 participants (61.0%) reported only one TBI event, while 39 participants (39.0%) reported two or more TBI events (range 0 to 6; see Figure 2).

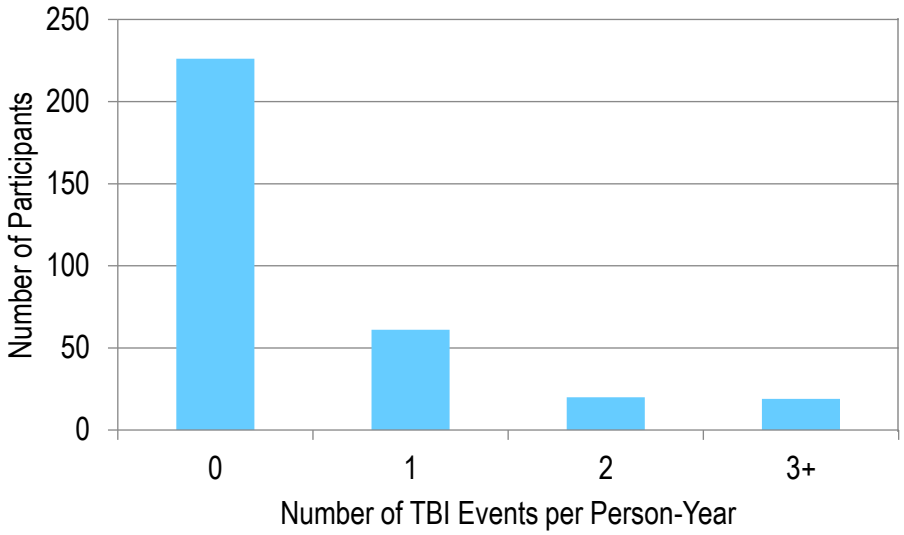


Figure 2 Frequency of Traumatic Brain Injury Count

When TBI was classified as a head injury with associated loss of consciousness (but not when only post-traumatic amnesia and/or daze and confusion present), there were 91 TBI acquired across 61 participants. With this, the incidence proportion, when defined only by loss of consciousness, was 18.7 percent, or 18,711.66 per 100,000 population. The incidence rate was 0.3008631 persons per year, or 30,086.31 per 100,000 person-years (61 persons with TBI over 202.75 person-years). The event proportion of number of traumatic brain injuries in this marginally housed sample (91 injuries in 326 total individuals) was 27.9 percent, or 27914.11 TBI per 100,000 population. The event rate was 0.4488286 injuries per year, or 44,882.86 per 100,000 person-years (91 injuries over 202.75 person-years). Of those 61 participants who acquired at least one TBI during this time, 42 participants (68.9%) reported only one TBI, while 19 participants (31.1%) reported two or more TBIs (range 0 to 4).

As seen in Table 3, rates of TBI vary according to both the definition of TBI and approach to handling missing data. Unsurprisingly, rates are higher as the definition of TBI becomes more liberal (i.e., standard versus LOC only). When missing data is imputed (approach C), estimated rates are also higher than observed rates. Across both TBI definitions, and observed and estimated rates, the incidence proportion ranges from 18.7% or 18,711.66 per 100,000 population, to 50.7% or 50,674.85 per 100,000 population. The event proportion ranges from 27.9% or 27,914.11 per 100,000 population, to 91.1% or 91,104.29 TBI per 100,000 population. The incidence rate

ranges from 30,086.31 to 50,674.85 per 100,000 person-years. The event rate ranges from 44,882.86 to 91,104.29 per 100,000 person-years.

Table 3 Rate of Traumatic Brain Injury

TBI Definition	Incidence Proportion (per 100,000 population)	Event Proportion (per 100,000 population)	Incidence Rate (per 100,000 person-years)	Event Rate (per 100,000 person-years)
Standard				
Observed	30,674.85	53,680.98	49,321.82	86,313.19
Estimated				
Approach A	30,674.85	53,680.98	30,674.85	53,680.98
Approach B	30,674.85	53,680.98	49,321.82	86,313.19
Approach C	50,674.85	91,104.29	50,674.85	91,104.29
LOC only				
Observed	18,711.66	27,914.11	30,086.31	44,882.86

Note. Standard = any LOC, PTA, daze, and/or confusion; Estimated = observed data with missing values manipulated; Observed = present data only; LOC only = any loss of consciousness (excluding PTA, daze, and/or confusion only).

4.3. Characteristics of Traumatic Brain Injury Events

To better understand the types of TBI events acquired in this population, we examined the severity (mild 0-30 mins LOC; moderate/severe > 30 mins LOC) and mechanisms of injuries. Of the 175 TBI events, 142 events were classified as mild (81.1% of all events, 81.6% of events with known severity), 32 were classified as moderate/severe (18.3% of all events, 18.4% of events with known severity), and 1 was unknown (0.6% of all events). The most common mechanisms of injury were falls, assaults, and hitting one's head on an object (Table 4). Of these events, 17 (9.7%) occurred in the context of a drug overdose (not including blackouts due to alcohol only). Acute intoxication was only assessed for 79 TBI events (45.1% of all events) as its evaluation was initiated after the study was underway. Of these 79 events, 48 were acquired when the participant was acutely intoxicated by drugs or alcohol (at least 27.4% of total events; 60.6% of events with available data).

Table 4 Mechanisms of Traumatic Brain Injury

Mechanism	Number of TBI Events	Percentage of Total TBI Events	Number in Context of Drug Overdose	Number in Context of Acute Intoxication
Fall	79	45.1	15	32
Assault	44	25.1	0	8
Hit head on Object	23	13.1	2	4

Mechanism	Number of TBI Events	Percentage of Total TBI Events	Number in Context of Drug Overdose	Number in Context of Acute Intoxication
Hit by Object	10	5.7	0	2
Pedestrian Accident	9	5.1	0	1
Biking/Sport-Related	6	3.4	0	1
Motor Vehicle Accident	1	0.6	0	0
Unknown	3	1.7	0	0
Other	0	0	0	0
Total	**175**	**100**	**17 (of 175; 9.7%)**	**48 (of 79*; 60.6%)**

** Note: Self-reports of acute intoxication were only obtained for 79 of the total 175 injuries.*

When separated by sex, there were no statistically significant differences in number of total TBI events (χ^2 (1) = 2.559, p = .110), or those occurring in the context of a drug overdose (χ^2 (1) = .238, p = .626) or acute intoxication (χ^2 (1) = .016, p = .900). For mechanisms of TBI, females were 2.28 times more likely than males to acquire a TBI as a result of a fall (χ^2 (1) = 5.405, p = .020), while males were 3.19 times more likely than females to acquire a TBI as a result of an assault (χ^2 (1) = 5.532, p = .019; see Figure 3 for mechanisms of TBI by sex).

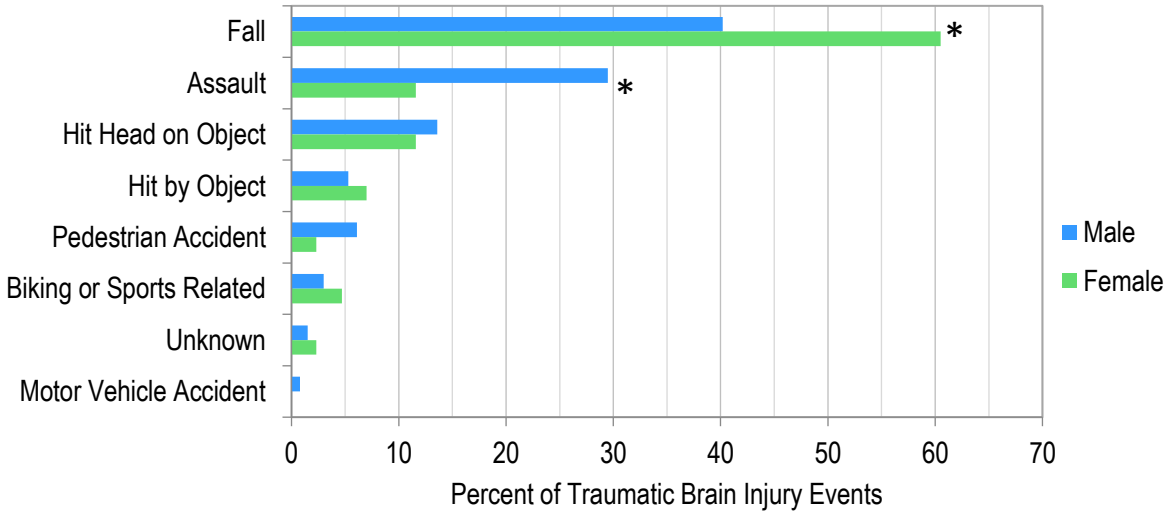


Figure 3 Mechanisms of Traumatic Brain Injury by Sex

Lastly, persons with opioid (χ^2 (1) = 6.632, p = .010) and cannabis (χ^2 (1) = 8.475, p = .004) dependence were more likely to sustain a TBI due to assault compared to any other mechanism, while those with alcohol dependence were more likely to sustain a TBI due to falls (χ^2 (1) = 3.842, p = .040).

4.4. Risk Factors for Traumatic Brain Injury Occurrence

Hierarchical binomial logistic regression revealed several risk factors for TBI occurrence (standard definition), as indicated in Table 5. For all estimated models, age, sex, and education were entered in Block 1. Further, risk variables were entered in Block 2 for models that separately evaluated each predictor category (i.e., substance dependence, psychiatric illness, prior brain injury, psychological functioning). Finally, for the final model, all significant risk variables identified by the categorized models were entered in Block 2.

The Hosmer and Lemeshow Test indicated a well fit model, χ^2 (8) = 5.886, p = .660. Block 1 accounted for approximately 3.8% of the variance (Nagelkerke R^2) in TBI occurrence, χ^2 (3) = 7.537, p = .057. Within the demographic category, education was the only significant predictor of TBI occurrence. Controlling for sex and age, for every grade decrease in education level, individuals were 1.13 times more likely to sustain TBI (B = -.125, p = .040). Within the substance dependence category, opioid dependence was the only significant predictor of TBI occurrence. Those with opioid dependence were 2.17 times more likely to sustain TBI (B = .777, p = .005). Within the psychiatric illness category, schizophrenia spectrum was the only significant predictor of TBI occurrence. Individuals with a schizophrenia spectrum disorder were 2.342 times less likely to sustain TBI (B = -.851, p = 035). Within the prior brain injury category, persons with a lifetime history of TBI were 1.992 times more likely to sustain TBI (B = .689, p = .018) and for each additional TBI event, individuals were 1.035 times more likely to sustain TBI (B = .035, p = .032). Lastly, within the psychological category, composite role functioning was the only significant predictor. As role functioning decreased by one standard deviation, persons were 1.453 times more likely to sustain TBI (B = -.375, p = .008).

The final model predicted approximately 20.7% of the variance in TBI occurrence, χ^2 (8) = 43.536, p < .001. The percentage accuracy in classification of the final model was 73.2%. Controlling for the other variables in the final model, education, composite role functioning, schizophrenia spectrum disorder, opioid dependence, lifetime number of TBI, and lifetime history of TBI were significant predictors of TBI occurrence. Using those with no TBI as a reference, as education decreased by one year, persons were 1.136 times more likely to sustain TBI (B = -.128, p = .044). Individuals with a diagnosis of schizophrenia spectrum disorder were 3.049 times less

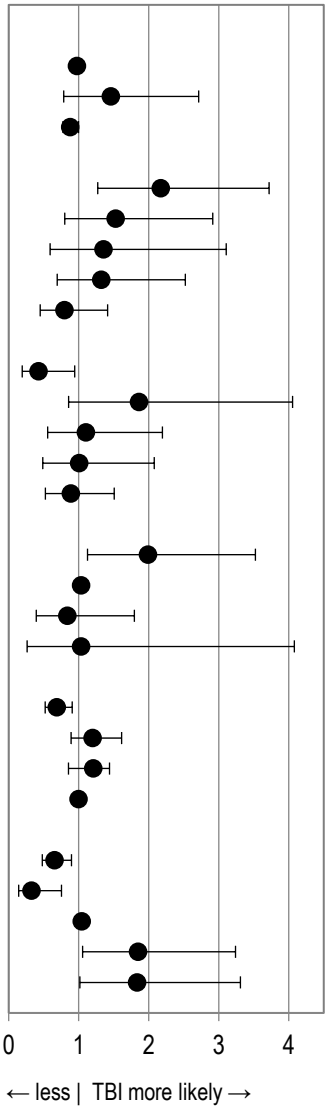
likely to sustain TBI (B = -1.114, p = .009). Persons with opioid dependence were 1.850 times more likely to sustain TBI (B = .615, p = .031). As the number of lifetime TBI increased by one injury, persons were 1.045 times more likely to sustain TBI (B = .044, p = .019). Those with a lifetime history of TBI were 1.835 times more likely to sustain TBI (B = .607, p = .044). Lastly, as composite role functioning decreased by one standard deviation, persons were 1.524 times more likely to sustain TBI (B = -.421, p = .008).

An interaction between schizophrenia spectrum disorder and CHS revealed that those with schizophrenia spectrum disorder were less likely to use harmful substances compared to those without (i.e., lower CHS; t = 2.487, p = .014), but only in those with no TBI. In those who sustained a TBI, there was no difference in harmful substance use in those with and without schizophrenia spectrum disorder. Those with schizophrenia spectrum disorder also reported less social network relationships than those without (t = 2.000, p = .046).

Again using those with no TBI as a reference, when TBI was defined according to LOC only (i.e., any LOC, excluding those with only PTA, dazed, and/or confusion), the final model remained similar in terms of the odds ratios and significance, with two differences. Lifetime number of TBI was no longer a significant predictor in the model (OR = 1.022, B = .026, p = .154), while those with alcohol dependence were 2.941 times more likely to sustain TBI with LOC (B = 1.079, p = .013).

Table 5 Binomial Logistic Regressions Predicting Traumatic Brain Injury Occurrence

Predictor Variables	Odds Ratio (95% CI)	
	Unadjusted[a]	Adjusted[b]
Block 1		
Age	.984 (.964,1.005)	.975 (.949,1.001)
Sex [c]	1.316 (.781,2.216)	1.461 (.786,2.714)
Education	.898 (.809,.998)*	.880 (.776,.997)*
Block 2a (N=290)		
Opioid dep [d]	1.903 (1.164,3.113)*	2.174 (1.272,3.719)**
Alcohol dep. [e]	1.318 (.727,2.389)	1.528 (.801,2.915)
Other drug dep. [f]	1.524 (.678,3.427)	1.355 (.591,3.107)
Stimulant dep. [g]	1.323 (.720,2.428)	1.322 (.693,2.523)
Cannabis dep. [h]	.905 (.547,1.497)	.798 (.450,1.413)
Block 2b (N = 290)		
Schiz. Spectrum [i]	.403 (.193,.840)*	.427 (.193,.943)*
Bipolar spectrum [j]	2.029 (.966,4.263)	1.863 (.856,4.055)
SIP disorder [k]	1.227 (.639,2.354)	1.106 (.557,2.196)
Depression [l]	1.193 (.602,2.365)	1.006 (.487,2.079)
Other [m]	1.125 (.684,1.850)	.889 (.524,1.507)
Block 2c (N = 262)		
History of TBI [n]	2.117 (1.297,3.454)**	1.992 (1.126,3.524)*
Number of TBI	1.039 (1.009,1.069)*	1.035 (1.003,1.068)*
Seizures/epilepsy [o]	.846 (.434,1.649)	.841 (.394,1.795)
MRI-defined TBI [p]	.731 (.226,2.362)	1.036 (.263,4.080)
Block 2d (N = 295)		
Role functioning	.754 (.586,.969)*	.688 (.521,.907)**
Fluid cognition	1.106 (.870,1.406)	1.199 (.891,1.613)
Impulsiveness	1.199 (.941,1.528)	1.209 (.854,1.440)
Reading ability	1.001 (.788,1.271)	1.001 (.980,1.022)
Final (N = 269)		
Role functioning	.754 (.586,.969)*	.656 (.480,.897)**
Schiz. spectrum	.403 (.193,.840)*	.328 (.143.754)**
Number of TBI	1.039 (1.009,1.069)*	1.045 (1.007,1.085)*
Opioid dep.	1.903 (1.164,3.113)**	1.850 (1.056,3.240)*
History of TBI	2.117 (1.297,3.454)**	1.835 (1.017,3.310)*



Note. CI = confidence interval; Dep = dependence; Schiz = schizophrenia; SIP = substance induced psychosis; TBI = traumatic brain injury; MRI = magnetic resonance imaging.

[a] Zero order association; [b] Adjusted for age, sex, and education; [c] N female = 87; [d] N = 129; [e] N = 59; [f] N = 27; [g] N = 232; [h] N = 113; [i] N = 55; [j] N = 32; [k] N = 47; [l] N = 42; [m] N = 170; [n] N = 108; [o] N = 50; [p] N = 15

2a Substance dependence; 2b Psychiatric illness; 2c Prior brain injury; 2d Psychological functioning

*p < .05. **p < .01. *** p < .001.

4.5. Risk Factors for Traumatic Brain Injury Severity

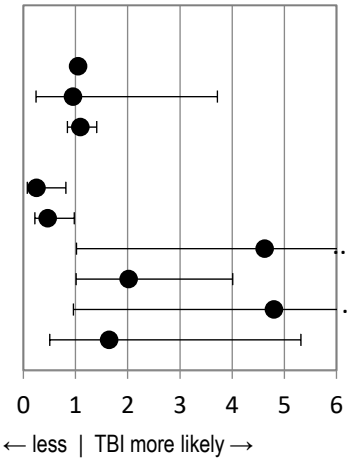
A single hierarchical binomial logistic regression further revealed several risk factors for TBI severity in individuals with at least one TBI event. Table 6 indicates that age, sex, and education were entered into Block 1, while previously screened indices of sustained attention, physical health, lifetime history of TBI, and diagnosis of a primary psychotic disorder, alcohol dependence, and cannabis dependence were entered into Block 2.

The Hosmer and Lemeshow Test indicated that the model fit well, χ^2 (8) = 7.663, p = .467. Block 1 (demographics) accounted for approximately 3.8% of the variance (Nagelkerke R^2) in TBI severity, χ^2 (3) = 2.168, p = .538, while Block 2 (combined model) predicted approximately 36.5% of the variance in TBI severity, χ^2 (9) = 23.979, p = .004. Using those with mild TBI as a reference, those with a diagnosis of a primary psychotic disorder were 4.049 times less likely to sustain moderate/severe TBI (msTBI; B = -1.399, p = .022). Persons who were alcohol dependent were 4.621 times more likely to sustain msTBI (B = 1.531, p = .047). As sustained attention decreased by one standard deviation, persons were 2.155 times more likely to sustain msTBI (B = -.768, p = .043). As self-reported physical health deceased by one standard deviation, persons were 2.013 times more likely to sustain msTBI (B = -.699, p = .047).

A similar interaction between primary psychotic disorder and CHS revealed that those with a primary psychotic disorder were less likely to use harmful substances compared to those without (i.e., lower CHS; t = 3.339, p = .001), but only in those with mild TBI. In those who sustained msTBI, there was no difference in harmful substance use in those with and without primary psychotic disorder.

Table 6 Binomial Logistic Regression Predicting Traumatic Brain Injury Severity (N = 78)

Predictor Variables	Odds Ratio (95% CI)	
	Unadjusted[a]	Adjusted[b]
Block 1		
Age	1.022 (.980,1.065)	1.048 (.971,1.130)
Sex [c]	.427 (.162,1.126)	.949 (.242,3.718)
Education	1.008 (.853,1.191)	1.089 (.844,1.406)
Block 2		
Psychotic dis. [d]	.422 (.199,1.037)	.247 (.075,.815)*
Sustained attention	.529 (.317,.882)*	.464 (.221,.975)*
Alcohol dep. [e]	2.836 (1.069,7.528)*	4.621 (1.018,20.979)*
Physical health	1.595 (1.013,2.511)*	2.013 (1.010,4.012)*
Cannabis dep. [f]	1.658 (.701,3.923)	4.801 (.959,24.045)
History of TBI [g]	1.781 (.777,4.084)	1.641 (.506,5.319)



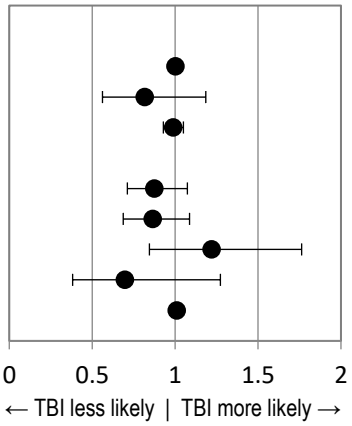
Note. OR = odds ratio; CI = confidence interval; Dis = disorder; Dep = dependence; TBI = traumatic brain injury.
[a] Zero order association; [b] Adjusted for age, sex, and education; [c] N female = 31; [d] N = 52; [e] N = 22; [f] N = 35; [g] N = 45
*p < .05. **p < .01. ***p < .001.

4.6. Risk Factors for Traumatic Brain Injury Count

In those with at least one TBI event, a single Poisson regression did not reveal any significant risk factors for the number of TBI events (see Table 7). Age, sex, and education were entered into Block 1, while previously screened indices of composite role functioning, composite fluid cognition, lifetime number of TBI, and diagnoses of schizophrenia spectrum disorder and alcohol dependence were entered into Block 2 (combined model). Given high expected cell frequencies, the deviance goodness-of-fit test indicated that the model was a good fit to the observed data, χ^2 (82) = 48.758, p = .595. The final model did not significantly predict incident TBI count, χ^2 (8) = 12.176, p = .144. Controlling for the other variables in the model, there were no significant predictors of TBI count.

Table 7 Poisson Regression Predicting Traumatic Brain Injury Count (N = 91)

Predictor Variables	Odds Ratio (95% CI)	
	Unadjusted[a]	Adjusted[b]
Block 1		
Age	1.006 (.991,1.021)	1.002 (.985,1.019)
Sex [c]	.748 (.531,1.052)	.816 (.562,1.185)
Education	.975 (.919,1.035)	.988 (.929,1.050)
Block 2		
Role functioning	.876 (.728,1.054)	.874 (.712,1.074)
Fluid cognition	.831 (.686,1.007)	.864 (.687,1.087)
Alcohol dependence [d]	1.175 (.833,1.656)	1.220 (.845,1.763)
Schiz. spectrum [e]	.712 (.404,1.254)	.697 (.382,1.273)
Lifetime number of TBI	1.009 (.996,1.023)	1.010 (.996,1.025)



Note. OR = odds ratio; CI = confidence interval; Schiz = schizophrenia; TBI = traumatic brain injury.
[a] Zero order association; [b] Adjusted for age, sex, and education; [c] N female = 28; [d] N = 21; [e] N = 10
*p < .05. **p < .01. *** p < .001.

Chapter 5. Discussion

5.1. Summary of Findings

5.1.1. Rate of Traumatic Brain Injury

Based upon a well-validated assessment regimen, as well as consistent participant education on the sequelae of TBI, we found that the rate of TBI in this precariously housed population was considerably higher than other rates reported in community samples regardless of the definition of TBI and approach to handling missing data. The observed incidence proportion indicated that 18.7% of individuals acquired at least one TBI when defined conservatively by LOC only. In line with hypotheses, when defined according to LOC, PTA, and/or daze/confusion (standard definition), the incidence proportion of 30.7% is 1.6 to 1.8 times higher than other reports in homeless and vulnerably housed persons ranging from 17.1 to 19.4% (Nikoo et al., 2017), and 92.7 times higher than the pooled North American annual incidence proportion (.331%; Nguyen et al., 2016). Even still, given a more complete dataset (i.e., approach C which used multiple imputation methods while controlling for recruitment date, the only relevant variable found to predict missingness), the true incidence proportion is estimated to be closer to 50.7%, a rate that would be 2.6 to 3.0 times higher than previous reports in precariously housed persons (Nikoo et al., 2017), and 153.2 times higher than the North American proportion (Nguyen et al., 2016). As approach C reduces missing data and thus missing TBI reports, while controlling for factors associated with missingness, 50.7% is the closest and best estimate to date of the true incidence proportion of TBI in precariously housed persons.

When the full number of TBI events is considered, the observed event proportion indicated that, on average, closer to 27.9 to 53.7% of individuals (LOC versus standard definitions, respectively) would have acquired at least one TBI, with estimated rates as high as 91.1%. Of the 100 individuals who acquired a TBI, 61% reported one event and 39% reported two or more, indicating that the distribution of TBI events was not equal across those with TBI.

Taking the screening timeframe into account, the observed incidence rate indicated that 0.30 to 0.49 individuals acquired a TBI for every person-year screened

(LOC versus standard definition, respectively). This rate is 47.5 to 77.5 times that of the pooled North American incidence rate (i.e., 632 per 100,000 person-years; Nguyen et al., 2016). Estimated rates indicated that closer to 0.51 individuals acquired a TBI per person-year, 80.7 times the North American rate (Nguyen et al., 2016). Lastly, taking both the screening timeframe and number of TBI events into account, the event rate of 0.45 to 0.86 events per person-year indicated that there was close to one acquired TBI event (standard definition) for every person-year screened. Estimated rates indicated that closer to 0.91 TBI events occurred each person-year.

5.1.2. Characteristics of Traumatic Brain Injury Events

Using comprehensive methodology to better understand the types of TBI events acquired by precariously housed persons, we found that 81.1% of all events were classified as mild, 18.3% were moderate to severe, and 0.6% were unknown. These characteristics are a novel finding as the severity of incident TBI has never been reported in homeless and marginally housed populations. As a comparison, our research group previously reported 45% mild and 55% moderate to severe TBI in an overlapping sample of individuals (Schmitt et al., 2017); however, rather than incident TBI, the past study examined lifetime history of *serious* head injury. Comparatively, this study demonstrates a considerably higher proportion of mild TBI (i.e., 81.1 versus 45 percent). The considerable increase in mild TBI reported in this study highlights the impact of ascertainment in characterizing TBI, which has also been found to impact rates of TBI (Stubbs et al., 2020). Specifically, Schmitt and colleagues (2017) screened for *serious* head injury (versus any head injury in this study), which likely resulted in a higher number of reported moderate to severe TBI. Schmitt and colleagues also examined lifetime history of TBI, requiring longer recall intervals (years versus one month), which may have resulted in missed mild and distal TBI. Thus, the characteristics of TBI events reported by Schmitt and colleagues likely represent more severe and proximal injuries. This has negative implications for obtaining accurate estimates of the rate of TBI, understanding the true characteristics of TBI events, and making unbiased comparisons of TBI versus non-TBI samples (i.e., missed TBI result in false positive errors and increased error in group comparisons). Methodological improvements made by this study provide an improved understanding of the characteristics of TBI in precariously housed adults (see below).

As a further characterization, acute intoxication by drugs or alcohol was present in at least 27.4% of events, with 9.7% occurring in the context of a drug overdose (not including blackouts due to alcohol only). The most common mechanisms of injury were falls (45.1%), assaults (25.1%), and hitting one's head on an object (13.1%). Compared to Schmitt and colleagues' (2017) work on lifetime history of *serious* head injury, falls were more than four times as common, while assaults and biking/sport-related causes were more than two times less common, and motor vehicle accidents were considerably less common. This suggests that in precariously housed persons, falls may result in mild or less memorable TBI, whereas assaults, biking/sport-related causes, and motor vehicle accidents may result in more severe TBI or those that are more memorable over longer follow-up intervals. Compared to the general population, assaults were a more common mechanism of injury (percent not reported for direct comparison), while falls (52.5 versus 45.1 percent), motor vehicle accidents (10 versus 0.6 percent), and biking/sport-related causes (36.6 versus 3.4 percent) were less common (Rao, McFaull, Thompson, & Jayaraman, 2017). Females were 2.28 times more likely than males to acquire a TBI as a result of a fall, while males were 3.19 times more likely than females to acquire a TBI as a result of an assault. Persons with opioid and cannabis dependence were more likely to sustain a TBI due to assault compared to any other mechanism, while those with alcohol dependence were more likely to sustain a TBI due to falls.

5.1.3. Risk Factors for Traumatic Brain Injury

While certain psychological risk factors have been examined as potential predictors of TBI occurrence (i.e., cognition), this is the first study in precariously housed persons to examine additional psychological risk factors under a bi-directional systems lens (i.e., factors that are impacted by TBI, may also predict future TBI occurrence). Within the psychological category, role functioning was identified as a novel predictor of TBI occurrence. Specifically, lower role functioning was associated with an increased likelihood to sustain TBI (1.52 times increase per standard deviation decrease in functioning). Lower scores on the role functioning composite can be attributed to poor working productivity, inability for independent living and self-care, and/or lack of immediate or extended social network relationships.

As a novel finding, we found differential predictive effects of different substance dependences. In line with hypotheses and prior research (Nikoo et al., 2017; Parry-

Jones et al., 2006; Taylor et al., 2003), those with alcohol dependence were 2.94 times more likely to sustain TBI when defined by LOC only (i.e., excluding PTA, daze, and/or confusion). While not hypothesized, persons with opioid dependence were 1.85 times more likely to sustain TBI regardless of TBI definition, and opioid dependence was the strongest risk factor for TBI occurrence in the final model. Exploratory analyses revealed that persons with opioid dependence were more likely to sustain TBI due to assault than any other mechanism, while those with alcohol dependence were more likely to sustain a TBI due to falls.

Within the psychiatric illness category, and as a novel finding, those with schizophrenia spectrum disorder were 3.04 times less likely to sustain TBI regardless of TBI definition. This is in contrast to hypotheses and previous research demonstrating that individuals with diagnoses of schizophrenia and bipolar disorder report higher rates of lifetime TBI compared to those with no mental illness (Malaspina et al, 2001). Even still, the rate of TBI in those with schizophrenia spectrum was 18.2 percent, which is in line with that found in other samples of persons with schizophrenia (range of 8 to 39.6%; Fujii & Fujii, 2012). Within this sample, those with schizophrenia spectrum disorder were less likely to abuse harmful substances compared to those without, but only in those with no TBI. They also reported less social network relationships, suggesting that social isolation and lower harmful substance use may be protective moderating factors for TBI in precariously housed persons with schizophrenia spectrum disorder. In contrast to hypotheses, persons with a diagnosis of depression were not more likely to sustain TBI.

As a replication of findings from other groups (Nordstom, Edin, Lindstrom, & Nordstrom, 2013; Nordstrom, & Nordstrom, 2011), we found that low education level was associated with a higher likelihood of TBI occurrence. In contrast to hypotheses, sex was not a significant predictor of TBI occurrence, highlighting that both men and women in precarious housing experience high rates of TBI (due to differing mechanisms; see above). Within the prior brain injury category, both a history of TBI and the number of lifetime TBIs predicted TBI occurrence. Specifically, those with a self-reported history of TBI were 1.83 times more likely to sustain TBI regardless of TBI definition. However, considering those with a history of TBI experienced a median of 3.00 TBI events in this sample, persons with a history of TBI may have closer to a 3.13 times increased likelihood to sustain TBI (standard definition only) than those with no history of TBI. This finding replicates that of previous research in similar populations, which found that

persons with a lifetime history of TBI were 3.10 times more likely to sustain TBI at follow-up, which was the strongest of all predictors in the model (Nikoo et al., 2017).

Adding to this critical field of research, this is the first study to also examine risk factors for TBI severity and count in precariously housed persons. Among persons with at least one TBI event, several risk factors were predictive of worse TBI severity. Alcohol dependence provided a 4.62 times increased likelihood of sustaining a moderate/severe TBI (msTBI). Conversely, those with a primary psychotic disorder had a 4.04 decreased likelihood to sustain msTBI, which may also be associated with decreased harmful substance use. As sustained attention and physical health decreased by one standard deviation, individuals were 2.15 and 2.01 times more likely to sustain msTBI, respectively. No risk factors were found to be predictive of TBI count amongst those with at least one TBI event, indicating that amongst those with risk factors for TBI occurrence, the number of acquired TBI events may depend on situational factors.

5.2. Implications

5.2.1. Rate & Characteristics of Traumatic Brain Injury

Within a sample of persons with highly prevalent substance dependence, psychiatric illness, and viral infection, we observed remarkably high rates of TBI. As a conservative estimate of the incidence proportion, 30.7% is both disproportionally higher than that of the general population (.331%; Nguyen et al., 2016), as well as the highest incidence proportion of TBI ever reported in precariously housed persons (highest prior report of 17.1 to 19.4%; Nikoo et al., 2017). Even still, the estimated incidence proportion of 50.7% given a more complete dataset using imputation methods is thought to be the closest and best estimate to the true rate of TBI in this population. This proportion is remarkably higher than that of sport-related TBI in the general population (50,674.85 compared to 170 per 100,000; Theadom et al., 2014), which receives considerable attention through media, clinical work, and research. Comparably, research examining TBI in precariously housed persons is limited and relatively underdeveloped (see Measuring TBI section below). As a consequence, this has presumably resulted in an inadequate understanding of the pervasiveness of TBI in this vulnerable population, reduced clinical screening for TBI, and fewer treatment and prevention efforts. Public education is needed on the burden of TBI in precariously housed persons. While the

issues of mental illness and substance dependence are often captured when conceptualizing this population, this study highlights that precariously housed persons can be viewed as victims of ongoing brain injury.

Considering this vulnerable population experiences high rates of cognitive impairment, neurocognitive burden, poor social and occupational functioning, and multimorbidity including neurological illness, the high rates of TBI demonstrated necessitates consideration of the impact of brain injury in these individuals. According to brain (Satz, 1993) and cognitive (Stern, 2002) reserve theories, TBI likely adds to and exacerbates the functional challenges experienced by marginalized persons, with lower levels of reserve moderating the effects of TBI (Leary et al., 2018; Steward et al., 2018). Of concern, many of the risk factors for TBI found in our study are also risk factors for neurocognitive burden and associated worsened TBI outcomes and recovery. For instance, education, lifetime history of TBI, and lifetime number of TBI predict TBI occurrence, and are also proxies for cognitive and brain reserve. Thus, those who are most likely to acquire a brain injury are also those who are more likely to experience worsened acute impairments and slowed recovery over time. Considering our research group previously found high rates of neurological illness in a large sample of precariously housed persons (Vila-Rodriguez et al., 2013), including brain infarction and history of head injury, this places a large group of individuals at increased risk for TBI, with less physical and functional neurological resources to cope with brain injuries. Considering the already reduced level of cognitive and daily functioning, this population is at risk of suffering worse consequences following TBI.

Above cognitive and brain reserve, the multitude of risk factors that precariously housed populations often face across the lifespan (e.g., developmental, substance use, viral infection, psychiatric illness, neurological illness and brain injury) impose a substantial neurocognitive burden. The aggregate impact of multimorbidity results in increased burden on one's neurocognitive functioning, with continuous insults to the brain leaving individuals with even less capability to deal with further brain insult. As a result, the high rate of TBI experienced by this population may lead to qualitatively different and worsened impacts of TBI compared to that experienced by the general population. In this sample of individuals with ubiquitous substance dependence, having a history of substance abuse has been associated with poorer neuropsychological recovery following TBI, increased chance of obtaining a second head injury, and higher

mortality rates following TBI (Corrigan, 1995). While alcohol and opioid dependence are risk factors for neurocognitive burden and the reduced ability to cope with this subsequent brain injury, persons with alcohol (when TBI defined by LOC only) and opioid (any TBI definition) dependence are also more likely to sustain TBI.

Given the high rate of incident TBI in precariously housed persons and likely poor outcomes following TBI, it is unsurprising that this population shows higher rates of seeking emergency department care (Honer et al., 2017) and health service use (Stubbs et al., 2020) for neurological illness. However, while clinical treatment effectiveness has been examined for other pathologies (i.e., HIV/AIDS, opioid dependence, and psychosis), the extent to which precariously housed persons who sustain TBI are receiving adequate clinical treatment remains unclear. Under Rhodes and colleagues' (Rhodes, 2002; Rhodes, Singer, Bourgois, Friedman, & Strathdee, 2005) conceptual framework of the risk environment, precariously housed persons experience the interaction of a variety of factors to worsen health and vulnerability. The environment is thought to increase risk for TBI and worsened outcomes through susceptibility and vulnerability factors. Specific risk factors for TBI (i.e., lower education, opioid dependence, lifetime history of TBI, higher lifetime number of TBI, and lower composite role functioning) act as susceptibility factors increasing the likelihood of TBI in the population. Risk factors for neurocognitive burden (e.g., developmental, substance use, viral infection, psychiatric illness, neurological illness and brain injury) and contextual factors (e.g., inadequate detection of TBI, poor access to clinical treatment for TBI, and injury prone environment that is not conducive to recovering from TBI) act as vulnerability factors making it more likely that TBI will have deleterious impacts (Barnett et al., 2000).

Together, the pervasiveness of TBI in precariously housed persons, in combination with the potential catastrophic impacts on cognition and daily functioning, necessitates further research on recovery from incident TBI in this population. Lifetime history of TBI has been found to be broadly associated with poorer health and functioning in this population (Stubbs et al., 2020), which is likely influenced by the interaction of TBI with other health risks that also impact cognitive and functional outcomes, thus worsening the effects of multimorbidity in this vulnerable population (Monti et al., 2013; Moretti et al., 2012; Schimitt et al., 2017; O'Connor, 2016). TBI has been associated with possible accelerated cognitive ageing in precariously housed

persons, with significant declines in verbal memory over a nine year period compared to those with no TBI (Gicas et al., 2020). Future longitudinal research is needed to determine the acute impact of TBI in this vulnerable population and whether these individuals show an appropriate or reduced recovery from TBI. Particular attention should be placed on individuals experiencing repeated and more severe TBI. Determining moderators of the response to TBI impacts and recovery from injury will also be important. Lastly, prevention initiatives will be fundamental to improving day-to-day functioning within this vulnerable population, as well as the social culture within which they live.

5.2.2. Measuring Traumatic Brain Injury

Given the high rates of TBI and vulnerability to suffer poor outcomes following brain injury, research examining TBI in precariously housed persons is limited and methodologically weak. Compared to other samples with high rates of incident TBI (e.g., athletes, veterans), there is a very limited literature in precariously housed persons, with only one of study that examined the incidence proportion of TBI in homeless and marginally housed adults (Nikoo et al., 2017). Compared to the 50.7 percent annual incidence proportion of TBI found in this study, Nikoo and colleagues (2017) found a range of 17.1 to 19.4 percent when screening for TBI at yearly intervals. In the current prospective study design, methodological improvements were made in screening for TBI that are thought to account for the higher and improved estimates of the true rate of TBI in precariously housed persons. Given the following methodological improvements, the rates and proportions of TBI presented in this study should be viewed as the most accurate estimates available of the pervasiveness of TBI in precariously housed persons.

First, a well-validated screening tool (i.e., OSU TBI-ID; Corrigan, & Bogner, 2007) was used to screen for incident TBI. This allowed for a comprehensive characterization of the types of TBI events that occurred, including the mechanism and severity of injuries, through detailed assessment of injury details. This is the first known study to conduct test-retest reliability analyses of self-reported injury details in precariously housed persons. Results suggest that this population has reliable reports (over 4 to 19 days) of the occurrence of TBI and injury details, including the injury mechanism and length of loss of consciousness. While consideration of the common limitations of self-

report measures is valid, this study design does not appear to be any less suited to precariously housed persons than other populations. Considering that analyses revealed that individuals were reliable reporters of LOC occurrence and fairly reliable reporters of the occurrence of PTA, daze, and/or confusion, the observed rate of TBI using a standard definition (i.e., LOC, PTA, and/or daze/confusion) is thought to be an accurate representation of the minimum observed rate, and the standard definition of TBI remains appropriate for future incident TBI research in similar populations over similar follow-up periods. However, considering reports were reliable for length of LOC but unreliable for length of PTA, daze and/or confusion, even over a relatively short follow-up period compared to other studies (i.e., 4 to 19 days), TBI severity classification in research of precariously housed persons is best determined according to length of LOC only. Other studies that do not solely use LOC for severity classification may be presenting unreliably classified injury details. Of note, it remains unclear if reliability of self-reported TBI occurrence and injury details in precariously housed persons generalizes to studies with longer follow-up periods (e.g., 1 year).

As additional methodological improvements, participants in this study were provided with initial and ongoing education regarding the common causes and symptoms of traumatic brain injury, in the form of a paper pamphlet that was read aloud and given to participants to keep. This may have improved participants' understanding of and ability to identify TBI-associated symptoms. Screening for TBI occurred repeatedly (i.e., monthly) and proximate to participants' potential injury. The existent research screened for head injury at yearly intervals, without the benefit of participant education on TBI sequelae, which likely resulted in missed mild and distal head injuries and an underestimation of the true rate of TBI. As this population can experience cognitive difficulties, particularly memory difficulties (Ennis, Roy, & Topolovec-Vranic, 2015; Gicas et al., 2014), repeated and proximate TBI screening may have reduced the number of missed minor injuries and potential recall bias surrounding the details of more severe injuries. Given the significant discrepancy between rates of TBI when screened yearly versus monthly, thought to be accounted for by the varied frequency of screening and duration of recall required, screening for TBI at a minimum of monthly intervals is essential in precariously housed persons. In comparison to other populations experiencing high rates of TBI, studies examining sport-related TBI report rates according to the number athletic exposures (Theadom et al., 2014). That is, even in an

otherwise healthy population, participants are asked to report TBI at most typically weekly or daily intervals, allowing for few missed TBI events. Considering granularity is reduced as the duration of recall required increases (e.g., monthly versus yearly follow-up periods), precariously housed persons should optimally be screened for TBI at daily intervals to reduce missed TBI occurrences. Together, these methodological changes likely resulted in improved TBI estimates and more reliable reports of TBI event characteristics. Related future research will benefit from participant education on TBI sequelae and repeated (i.e., monthly to daily), proximal, and comprehensive screening methodology to best capture TBI in precariously housed persons.

Even with these methodological improvements, an important consideration in longitudinal TBI research is the significant patient attrition that results in missing data, with even lower follow-up attendance among persons with lower socioeconomic backgrounds (Krellman et al., 2014; Yue et al., 2014; Zelnick et al., 2014). With this, the adequate handling of missing data is imperative, as inadequate methods can decrease power, violate analyses, and introduce bias in interpretation. Identifying the missingness mechanism (i.e., the reason why the data are missing; Rubin, 1976) informs what statistical approaches may best be used for handling missing data and can aid in the interpretation of conclusions (Richter et al., 2019). In a systematic review of the handling of missing data in traumatic brain injury research, Richter and colleagues (2019) found that only half of studies examined attempted to identify why data were missing, with only four studies explicitly stating their assumption of the missingness mechanism. While the most common method to handle missing data was exclusion of participants without complete data, suggesting reliance on the default method in statistical programs, there was substantial variability in the standard of reporting and handling of missing data.

Recognizing this variability in methodology, Richter and colleagues (2019) have outlined a framework for handling missing data in observational TBI research. Following the decision flow-chart, the approach includes comparison of participants with and without missing data, balancing the priorities of retaining power versus simplifying analyses, considering whether auxiliary data is available to help in predicting missing values, and determination of whether additional analysis are appropriate, such as multiple imputation, expectation maximization, and/or sensitivity analysis. Following this framework, our study conducted an in depth analysis of missing data patterns and reported the missingness mechanism (Rubin, 1976). The data was determined to be

missing at random and sufficient auxiliary data was available, allowing for the estimation of missing values using multiple imputation and the generation of unbiased and inclusive estimates of incident TBI. In contrast, previous research examining incident TBI in homeless and vulnerably housed adults (Nikoo et al., 2017) did not explore or identify the missingness mechanism of the data. Missing data was treated as *missing completely at random* and excluded from analyses, which may have resulted in a lack of appreciation of missing data patterns, over/underestimation of the true incidence proportion, and weakened generalizability of the findings to the complete sample. Adequate handling of missing data is an underappreciated methodology in TBI research, particularly in precariously housed populations. In order to best understand and handle missing data in TBI research, and to improve estimates of incident TBI in prospective studies, future research in this area may follow the framework outlined by Richter and colleagues (2019) for handling missing data.

5.2.3. Risk Factors for Traumatic Brain Injury

As an important contribution, this study identified several key risk factors for TBI occurrence, which has implications for prevention. Among broad predictor categories, psychological risk factors were found to predict brain injury occurrence. As a novel finding, TBI was more likely in those with worse role functioning, supporting a bidirectional relationship between role functioning and TBI. This highlights a group of individuals at risk for exacerbation of an already worsened capacity for independent living, social relationships, and working productivity. Ultimately, this would have negative implications at the level of the individual, community, and society as a whole. Fortunately, under bioecological systems theory (Bronfenbrenner 1979, 2005), this captures a set of under-recognized yet modifiable risk factors at which prevention strategies and treatment can be aimed. Under this model, there is a reciprocal connection between an individual's personal characteristics and the precariously housed environment at this period of personal and historical time. As an illustration, individuals experiencing physical, psychiatric, or cognitive limitations may be more vulnerable to victimization and assault in the SRO environment, resulting in increased likelihood for TBI and further reduced overall role functioning. Under Anatchkova and Bjorner's (2010) conceptual model of role functioning, managing health conditions, recognizing one's life stage, and providing choice and opportunity can improve an individual's role functioning.

Prevention and treatment targets including improving social connection, aiding with community involvement, and supporting everyday living activities in precariously housed persons will be important targets. These strategies are focused not only on the individual, but also address structural biopsychosocial factors that may reciprocally improve both person and contextual factors. This may result in improved individual role functioning, reduced TBI occurrence, and enhanced social economy. Further research into the bidirectional role of psychological risk factors for TBI is warranted.

While others have reported problematic alcohol and drug use to predict TBI in homeless and vulnerably housed persons (Nikoo et al., 2017), this study reveals that there is variability in this association between drug types. Alcohol dependence predicted TBI with LOC as well as TBI severity and falling appears to be the underlying mechanism accounting for this relationship. Opioid dependence predicted TBI, particularly due to assault, while other drug dependencies were not predictive. While TBI has been associated with an increased likelihood for opioid and other substance use disorders following the injury (Bjork & Grant, 2009), no known research has linked opioid dependence as a causal mechanism for TBI, although this link has been hypothesized (Corrigan & Adams, 2019). Replication of opioid dependence as a risk factor for incident TBI is needed. While psychiatric illness-related treatment is very important, those with a primary psychotic disorder may require less focused TBI prevention and screening strategies, but more networking resources and social supports.

In line with predictions and previous research, persons with a lifetime history of TBI and increased number of lifetime TBIs were more likely to sustain a TBI. While this association is unsurprising, the amount of increased likelihood for TBI highlights the vulnerability of this population for subsequent, and sometimes repeated, brain injuries. Considering that 33% (this study) to more than half (53.1%; Stubbs et al., 2020) of homeless and precariously housed individuals report a lifetime history of TBI, with a median of three lifetime TBI, a large number of individuals are at increased risk for TBI as an ongoing neurological insult. Further, given that repetitive mild TBI events have been found to result in chronic neuropathological and neurobehavioural changes, including continuing white matter degradation, progressive neuroinflammation, and persistent cognitive deficits (Mouzon et al., 2013), the impact of recurrent TBI appears to be an underappreciated factor in accounting for the current functioning of the large proportion of individuals experiencing repeated TBI events. Given that this population

faces high rates of traumatic brain injury, identifying individual characteristics associated with more severe and/or repetitive injuries, may help to identify those at increased risk of worsened outcomes.

Lastly, in a sub-sample of individuals with incident brain injury, several risk factors for more severe TBI were identified. In a population facing the aggregate impact of multimorbidity, harm reduction strategies targeting those most vulnerable are imperative to improve functioning. Persons with alcohol dependence, worse sustained attention, and worse physical health symptoms would likely benefit most from a comprehensive educational and screening approach in a harm reduction strategy to prevent more severe and debilitating brain injuries.

5.2.4. Recommendations

The pervasiveness of incident traumatic brain injury in precariously housed persons is currently an unrecognized endemic. Under bioecological systems framework (Bronfenbrenner, 2005) and the ecology of homelessness (Nooe & Patterson, 2010), addressing both individual and structural factors will have the most impact on individual outcomes, including TBI occurrence. Addressing factors inherent to the homeless and precarious housing risk environment (Rhodes, 2002), including physical, social, economic, and policy-related factors, can produce intersecting influence to help mitigate risk for brain injury and associated outcomes. First, knowledge translation efforts are needed at all levels. On an individual level, persons living in homelessness and precarious housing would benefit from education on the sequelae and pervasiveness of TBI in this population. Increased research, clinical, and media attention is warranted and needed in this area to increase awareness. Education should also be provided to health care providers on the rate of TBI in this vulnerable population, along with factors making an individual more apt to acquire a TBI, in order to help with risk stratification efforts. Structurally, supportive housing models, increased housing stability, and treatment for psychiatric and substance use disorders may mitigate many of the risks for TBI inherent in the current environment. This will also provide a social environment conducive to improved role functioning and social connection. Appropriate funding and public policy is needed to outline and implement TBI screening procedures that are deployed frequently. Planning will be best done by involving a wide range of stakeholders, including all levels of government, service providers, health professionals, researchers, community groups,

and individuals who are homeless of living in precarious housing (Frankish, Hwang, & Quantz, 2005).

5.3. Limitations

Despite the novel contributions of this study, limitations need to be acknowledged. First, this study relied on self-report for the identification of incident traumatic brain injuries and associated symptoms. This method may have been susceptible to variance in response styles, lack of insight, and recall errors due to cognitive limitations. Although these problems are inherent to self-report measures, the impact of these factors was likely reduced given that participants were screened monthly for TBI, resulting in high frequency and consistency of TBI education and screening with relatively short inter-screening intervals. In addition, reliability analyses found that participants of this study reliably reported injury details across two assessment time points.

Second, this population acquired some TBI events in the context of drug overdose (9.7 percent of all events) or acute alcohol intoxication (at least 26.4 percent of all events), which may have somewhat confounded the definition of TBI for some events in this study. In defining TBI, loss of consciousness and post-traumatic amnesia are symptoms that indicate TBI occurrence (standard definition) when occurring along with a clear impact to the head. Drug overdoses can result in a LOC and alcohol intoxication can result in a blackout and/or amnestic period, all of which can be indistinguishable from a LOC due to TBI specifically. Within this context, while the clinical symptoms may present as the same, the specific mechanism through which they occurred (e.g., TBI versus drug overdose versus alcohol intoxication) is unclear, and could have resulted from a combination of these factors (e.g., drug overdose resulting in LOC that is prolonged with additional TBI upon impact to ground). Thus, some TBI events occurring in the context of a drug overdose or acute alcohol intoxication may have been wrongly classified as a TBI occurrence (i.e., false positive error). While efforts were made to avoid this, by requiring an observer report or observable sign of head trauma when reporting a TBI in these contexts, this may still have resulted in a somewhat higher proportion and rate of TBI. On the other hand, experiencing periods of amnesia and LOC more frequently in the context of drug overdose and/or alcohol intoxication may make a person less apt to report these symptoms as associated with TBI events, which may

have resulted in more frequent reports of drug and alcohol related negative experiences but fewer self-reported TBI occurrences (i.e., false negative errors). Thus, this potential confound for a subset of TBI events may have resulted in an over- or under-estimation of the pervasiveness of TBI in precariously housed persons.

Third, TBI events occurring in the context of drug overdose or acute alcohol intoxication may also confound the severity classification of a subset of TBI events. The severity of TBI events were classified according to the occurrence and length of loss of consciousness (i.e., mild 0-30 mins LOC; moderate/severe > 30 mins LOC). As drug overdose or alcohol intoxication can result in a LOC or blackout period, TBI sustained in these contexts may have been more likely to be classified as moderate to severe TBI than mild TBI. For TBI events occurring in the context of drug overdose, only 3 TBI events (15.8% of all events in this context) were classified as moderate to severe. This is a similar proportion of moderate to severe events than that found for all TBI events (18.3%), suggesting TBI events in the context of drug overdose were not more likely to be classified as more severe. For TBI events occurring in the context of alcohol intoxication, 12 TBI events (25% of all events in this context) were classified as moderate to severe. This is an apparently slightly higher proportion of moderate to severe events, suggesting TBI events in the context of alcohol intoxication may be somewhat more likely to be classified as more severe. For some of these TBI events, it is possible that they were incorrectly classified as moderate to severe versus mild. This would occur with a LOC due to TBI of 30 minutes or less that is prolonged due to a LOC arising from drug overdose or alcohol intoxication of more than 30 minutes. On the other hand, it is also possible that events were appropriately classified as moderate to severe TBI versus mild TBI. This would occur with a LOC due to TBI of longer than 30 minutes, which may or may not have been prolonged by drug overdose or alcohol intoxication. Considering both of these possibilities, including TBI events occurring in the context of drug overdose or alcohol intoxication could result in an overestimation of the proportion of moderate to severe TBI (likely more so for events in the context of alcohol intoxication), while excluding these events could result in false negative errors and an underestimation of the severity of TBI events occurring in precariously housed persons. As the proportion of influence from TBI versus overdose/intoxication on loss of consciousness cannot be determined in this study, further research is needed on the impact of drug overdose and/or alcohol intoxication on length of loss of consciousness in

the context of TBI. This research may help to determine whether severity classification based on the occurrence and length of loss of consciousness is appropriate in this population.

Fourth, this study relied on the attendance of each participant at monthly TBI screening assessments over a one person-year period. While the amount of missing screening data was considered an acceptable amount, there was a considerable amount of missing data that may have resulted in a reduced observed proportion of TBI and/or under- or over-estimate of the observed rate of TBI. With this, the observed incidence proportion should be viewed as the minimum proportion of TBI. Compared to the observed estimate, the rate of TBI is best estimated with imputed methods, since estimated rate using approach C is likely unaffected since the data was found to be missing at random (MAR) and imputation allowed for a complete dataset.

Fifth, in an effort to engage study participants and reduce the amount of missing data, participants were given small monetary honoraria after each completed screening assessment. Additionally, as part of the larger study, a subset of participants was enrolled in additional neuropsychological testing after reporting a TBI occurrence. This raises concern of participants falsely reporting TBI events in order to receive additional testing opportunities and associated compensation. In order to examine this possibility, longitudinal analyses were performed to determine whether relevant variables were associated with whether participants came in to report a TBI or not (see Procedures section). Fortunately, no observed relevant variable was found to predict whether participants were more or less likely to come in and report a TBI, suggesting that motivation for compensation did not significantly alter the proportions or rates of TBI occurrence. Analysis did, however, reveal that recruitment date and visit month predicted whether participants were more likely to report a head injury (without associated symptoms of TBI). This suggests that requiring associated symptoms of TBI in screening for TBI occurrence is essential in mitigating the risk of false reports. This also necessitates the need to examine missing data patterns in order to determine whether motivation for compensation has reduced the validity of screening. Alternative compensation strategies may be warranted to further mitigate this risk in future studies (e.g., equal opportunity for additional testing and compensation for both those who self-report TBI and those who do not).

Lastly, this study examined traumatic brain injury in individuals living in precarious housing. Considering this environment can provide considerable risk for TBI, and that this population experiences multiple comorbid risk factors that further increase risk for TBI occurrence, this population is uniquely apt to experience a high rate of TBI. With this, the generalizability of the findings of this study is limited to similar marginalized samples with multiple comorbid risk factors.

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Appendix A.

Traumatic Brain Injury Educational Handout

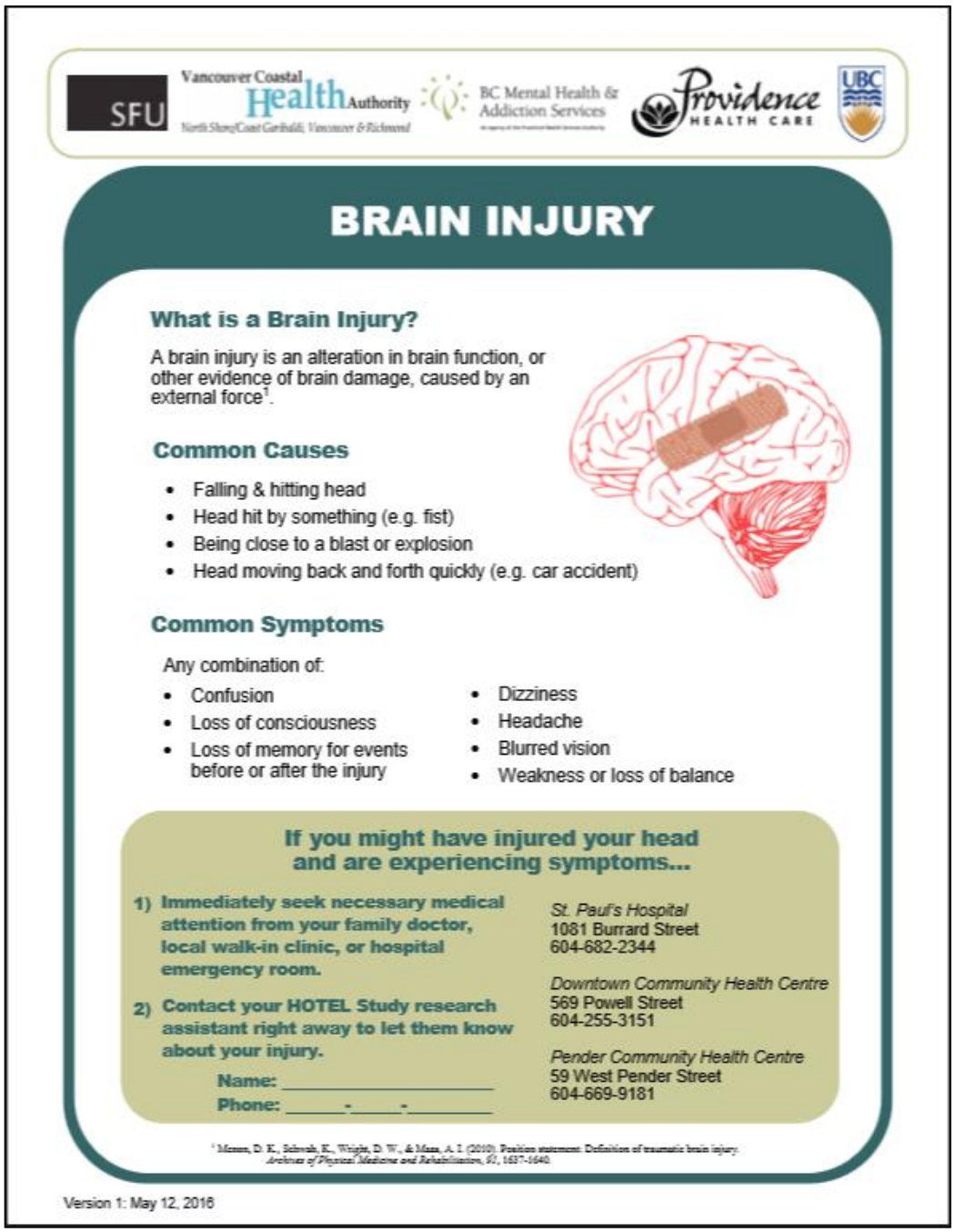

Figure A1. Traumatic brain injury educational handout

Appendix B.

Traumatic Brain Injury Screening Questionnaire

Hotel-TBI Screening Questionnaire

Interviewer initials: _____ Participant number: _____

Date: _____ (☐ BL ☐ Monthly ☐ Reliability) Participant age: _____

Any injuries to head/neck within past year? Yes / No *(fill below for all injuries)*
If none within past year, any injuries to head/neck ever? Yes / No *(fill below for most recent)*

Signs/Symptoms	**Injury #1**	**Injury #2**
Cause of Injury *(If assault, clearly indicate by who [e.g., stranger, friend, partner])*		
Time Since Injury		
If known, Injury Date		
Treatment at Hospital/Clinic		
Yes / No		
LOSS OF CONSCIOUSNESS *("how do you know?")*		
Yes / No		
If yes, Length		
MEMORY GAP		
Yes / No		
If yes, Length		
Anterograde *(after injury; "1st memory?")*		
Yes / No		
If yes, Length		
Retrograde *(before injury; "last memory?")*		
Yes / No		
If yes, Length		
DAZED OR CONFUSION		
Yes / No		
If yes, Length		
Dazed *(foggy, out of it, processing problems)*		
Yes / No		
If yes, Length		
Confusion		
Yes / No		
If yes, Length		
If present, indicate √ and length		
Headaches		

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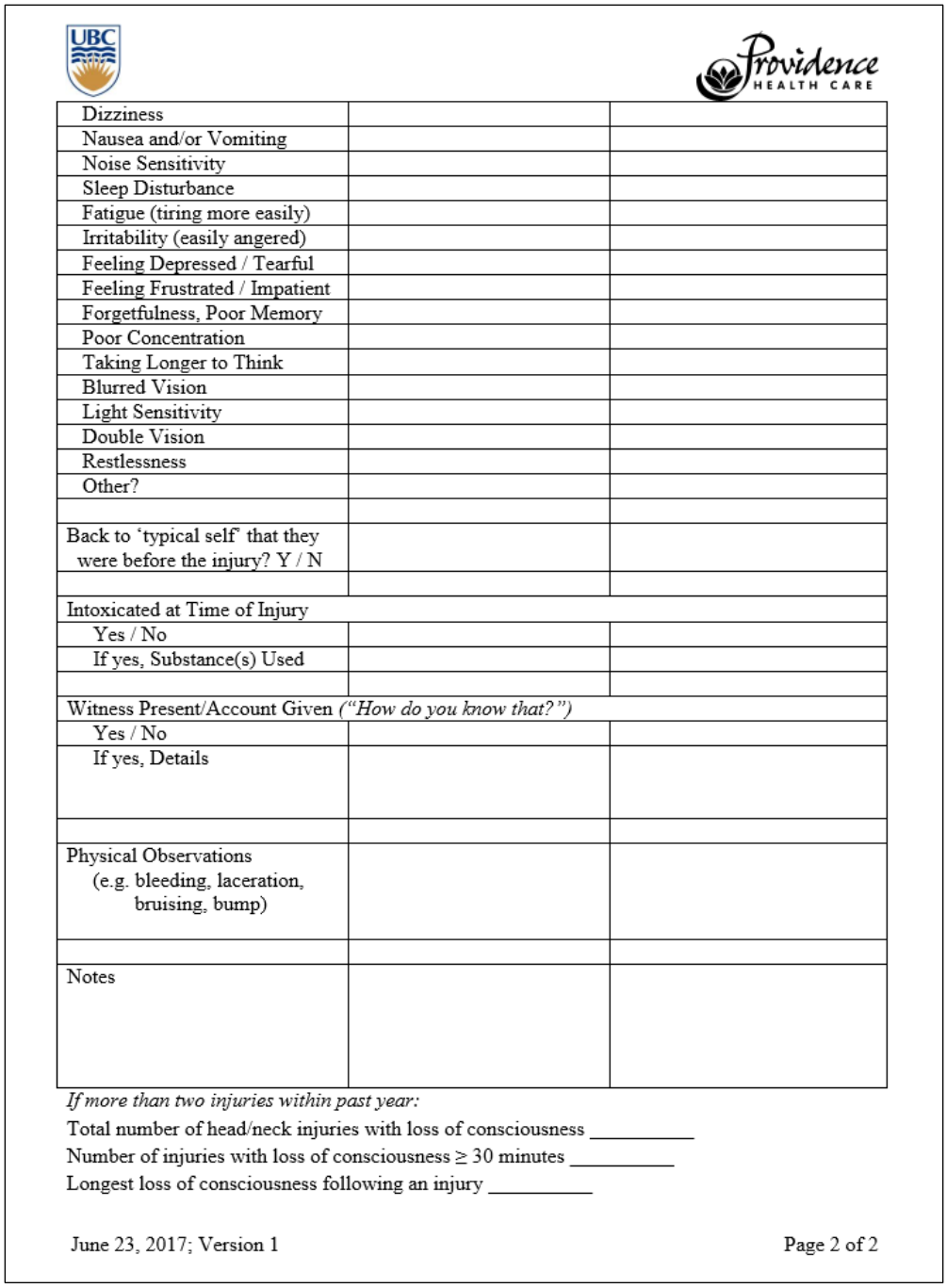
Figure B1. Traumatic brain injury screening questionnaire page 1



Dizziness		
Nausea and/or Vomiting		
Noise Sensitivity		
Sleep Disturbance		
Fatigue (tiring more easily)		
Irritability (easily angered)		
Feeling Depressed / Tearful		
Feeling Frustrated / Impatient		
Forgetfulness, Poor Memory		
Poor Concentration		
Taking Longer to Think		
Blurred Vision		
Light Sensitivity		
Double Vision		
Restlessness		
Other?		
Back to 'typical self' that they were before the injury? Y / N		
Intoxicated at Time of Injury		
Yes / No		
If yes, Substance(s) Used		
Witness Present/Account Given *("How do you know that?")*		
Yes / No		
If yes, Details		
Physical Observations (e.g. bleeding, laceration, bruising, bump)		
Notes		

If more than two injuries within past year:

Total number of head/neck injuries with loss of consciousness _____

Number of injuries with loss of consciousness ≥ 30 minutes _____

Longest loss of consciousness following an injury _____

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Figure B2. Traumatic brain injury screening questionnaire page 2

Appendix C.

Screened Risk Factors for Traumatic Brain Injury Severity

Includes all potential risk factors screened for association with TBI severity. Effect sizes marked with an asterisk (*) denotes those with a Cohen's *d* of at least 0.2 that were subsequently included in the final model. Those marked with a tilde (~) denotes a *d* of at least 0.2 that were not included into the model due to the choice of a conceptually similar predictor variable included in the model.

Table C1 Potential Risk Factors Screened for Inclusion in Predicting Traumatic Brain Injury Severity

Variable	Description	Coding	Correlation (*r*)	Effect Size (*d*)
Demographics				
Age	Age in years	Continuous variable	.100*	.200*
Education	Years of education attained	Continuous variable	.009	.018
Monthly income	Self-reported monthly income	Continuous variable	.010	.020
Sex	Male/Female	[0,1] where males = 0, females = 1	-.175*	-.350*
Ethnicity	Self-identified ethnicity (note: no individual dummy coded variables were associated with the dependent variable)	Categorical [1,2,3] grouped by frequency where Caucasian = 1, Indigenous = 2, all other = 3	.043	.086
Drug/Alcohol Dependence				
Alcohol dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.218*	.436*
Cocaine dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	-.018	-.036
Methamphetamine dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.027	.054
Stimulant dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where both diagnoses (Cocaine or methamphetamine dependence) absent = 0, either present = 1	-.035	-.070
Heroin dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.031	.062
Opioid dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where all diagnoses (heroin or other opioid dependence) absent = 0, any present = 1	-.018	-.036
Cannabis dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.118*	.236*
Other drug dependence	Diagnosed using the BECED-II	[0,1] where diagnosis	-.067	-.134

Variable	Description	Coding	Correlation (*r*)	Effect Size (*d*)
	according to DSM-IV criteria	absent = 0, present = 1		
Mental Illness				
Depression	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.041	.082
Bipolar spectrum disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where both diagnoses (bipolar I and II disorder) absent = 0, either present = 1	-.108~	-.216~
Schizophrenia	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	-.109~	-.218~
Schizoaffective disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	-.101~	-.202~
Schizophrenia spectrum disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where both diagnoses (schizophrenia and schizoaffective disorder) absent = 0, either present = 1	-.110~	-.220~
Primary psychotic disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where all diagnoses (schizophrenia, schizoaffective, bipolar I disorder) absent = 0, either present = 1	-.149*	-.298*
Substance induced psychotic disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	-.072	-.144
Other disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.043	.086
Medical/Cognitive				
Lifetime number of TBI	Total of number of self-reported TBI from any mechanism from Brain Injury Screening Questionnaire	Continuous variable with maximum 50 events	.099	.198
Lifetime history of TBI	Self-reported history of mild to moderate TBI from Medical Review Questionnaire	[0,1] where no history of self-reported TBI = 0, reported history of TBI = 1	.137*	.274*
MRI-defined TBI	Radiologically confirmed TBI on most proximal pre-enrollment MRI scan	[0,1] where no MRI defined TBI = 0, MRI defined TBI = 1	-.051	-.102
History of seizures/epilepsy	Self-reported history of seizure/epilepsy, verified by history of anticonvulsant use	[0,1] where no history = 0, history = 1	.066	.132
History of stroke	Self-reported history of stroke	[0,1] where no history = 0, history = 1	.024	.048
Psychological				
Attention deficit hyperactivity disorder	Self-reported history of diagnosis of ADHD	[0,1] where diagnosis absent = 0, present = 1	-.081	-.162
Crystallized reading ability	Standard reading score from the Wechsler Test of Adult Reading	Continuous variable	-.046	-.092
Selective attention	Assessed using the Stroop Color Word Test	Continuous variable	-.027	-.054
Sustained attention & working memory	Assessed using the RVP subtest of the CANTAB	Continuous variable	-.274*	-.548*
Verbal learning &	Assessed using the HVLT	Continuous variable	-.098	-.196

Variable	Description	Coding	Correlation (*r*)	Effect Size (*d*)
memory	immediate			
Composite fluid cognition	Composite cognition using Stroop Color Word, HVLT immediate, RVP A tasks.	Continuous variable	-.083	-.166
Decision Making	Mean net score of performance on Iowa Gambling Task	Continuous variable	-.044	-.088
Impulsiveness	Self-reported impulsiveness using Barratt Impulsiveness Scale.	Continuous variable	.005	.010
Health Risk	Rated using the Maudsley Addiction Profile i) Physical symptoms i) Mental symptoms	Continuous variable	 .215* .025	 .430* .050
Adaptive functioning	Rated using the Role Functioning Scale	Continuous variable	-.086	-.172
Social & occupational functioning	Rated using the Social and Occupational Functioning Assessment Scale	Continuous variable	-.028	-.056
Composite role functioning	Composite functioning using RFS & SOFAS	Continuous variable	-.028	-.056

Appendix D.

Screened Risk Factors for Traumatic Brain Injury Count

Includes all potential risk factors screened for association with TBI count. Effect sizes marked with an asterisk (*) denotes those with a Cohen's *d* of at least 0.2 that were subsequently included in the final model. Those marked with a tilde (~) denotes a *d* of at least 0.2 that were not included into the model due to the choice of a conceptually similar predictor variable included in the model.

Table D1 Potential Risk Factors Screened for Inclusion in Predicting Traumatic Brain Injury Count

Variable	Description	Coding	Correlation (*r*)	Effect Size (*d*)
Demographics				
Age	Age in years	Continuous variable	.089	.178
Education	Years of education attained	Continuous variable	-.092	-.184
Monthly income	Self-reported monthly income	Continuous variable	-.052	-.104
Sex	Male/Female	[0,1] where males = 0, females = 1	-.188*	-.376*
Ethnicity	Self-identified ethnicity (note: no individual dummy coded variables were associated with the dependent variable)	Categorical [1,2,3] grouped by frequency where Caucasian = 1, Indigenous = 2, all other = 3	-.030	-.060
Drug/Alcohol Dependence				
Alcohol dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.105*	.210*
Cocaine dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.069	.138
Methamphetamine dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.000	.000
Stimulant dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where both diagnoses (Cocaine or methamphetamine dependence) absent = 0, either present = 1	.020	.040
Heroin dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.009	.018
Opioid dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where all diagnoses (heroin or other opioid dependence) absent = 0, any present = 1	.034	.068
Cannabis dependence	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.037	.074
Other drug dependence	Diagnosed using the BECED-II	[0,1] where diagnosis	-.018	-.036

Variable	Description	Coding	Correlation (*r*)	Effect Size (*d*)
	according to DSM-IV criteria	absent = 0, present = 1		
Mental Illness				
Depression	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.083	.166
Bipolar spectrum disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where both diagnoses (bipolar I and II disorder) absent = 0, either present = 1	.032	.064
Schizophrenia	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	-.091	-.182
Schizoaffective disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	-.095	-.190
Schizophrenia spectrum disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where both diagnoses (schizophrenia and schizoaffective disorder) absent = 0, either present = 1	-.134*	-.268*
Primary psychotic disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where all diagnoses (schizophrenia, schizoaffective, bipolar I disorder) absent = 0, either present = 1	-.058	-.116
Substance induced psychotic disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	-.025	-.050
Other disorder	Diagnosed using the BECED-II according to DSM-IV criteria	[0,1] where diagnosis absent = 0, present = 1	.092	.184
Medical/Cognitive				
Lifetime number of TBI	Total of number of self-reported TBI from any mechanism from Brain Injury Screening Questionnaire	Continuous variable with maximum 50 events	.153*	.306*
Lifetime history of TBI	Self-reported history of mild to moderate TBI from Medical Review Questionnaire	[0,1] where no history of self-reported TBI = 0, reported history of TBI = 1	.094	.188
MRI-defined TBI	Radiologically confirmed TBI on most proximal pre-enrollment MRI scan	[0,1] where no MRI defined TBI = 0, MRI defined TBI = 1	-.048	-.096
History of seizures/epilepsy	Self-reported history of seizure/epilepsy, verified by history of anticonvulsant use	[0,1] where no history = 0, history = 1	.086	.172
History of stroke	Self-reported history of stroke	[0,1] where no history = 0, history = 1	.010	.020
Psychological				
Attention deficit hyperactivity disorder	Self-reported history of diagnosis of ADHD	[0,1] where diagnosis absent = 0, present = 1	.096	.192
Crystallized reading ability	Standard reading score from the Wechsler Test of Adult Reading	Continuous variable	-.082	-.164
Selective attention	Assessed using the Stroop Color Word Test	Continuous variable	-.116~	-.232~
Sustained attention & working memory	Assessed using the RVP subtest of the CANTAB	Continuous variable	-.259~	-.518~
Verbal learning &	Assessed using the HVLT	Continuous variable	-.079	-.158

Variable	Description	Coding	Correlation (*r*)	Effect Size (*d*)
memory	immediate			
Composite fluid cognition	Composite cognition using Stroop Color Word, HVLT immediate, RVP A tasks.	Continuous variable	-.212*	-.424*
Decision Making	Mean net score of performance on Iowa Gambling Task	Continuous variable	.090	.180
Impulsiveness	Self-reported impulsiveness using Barratt Impulsiveness Scale.	Continuous variable	-.043	-.086
Health Risk	Rated using the Maudsley Addiction Profile i) Physical symptoms i) Mental symptoms	Continuous variable	 .080 .026	 .160 .052
Adaptive functioning	Rated using the Role Functioning Scale	Continuous variable	-.140~	-.280~
Social & occupational functioning	Rated using the Social and Occupational Functioning Assessment Scale	Continuous variable	-.140~	-.280~
Composite role functioning	Composite functioning using RFS & SOFAS	Continuous variable	-.158*	-.316*

Appendix E.

Plot of Statistical Power to Sample Size

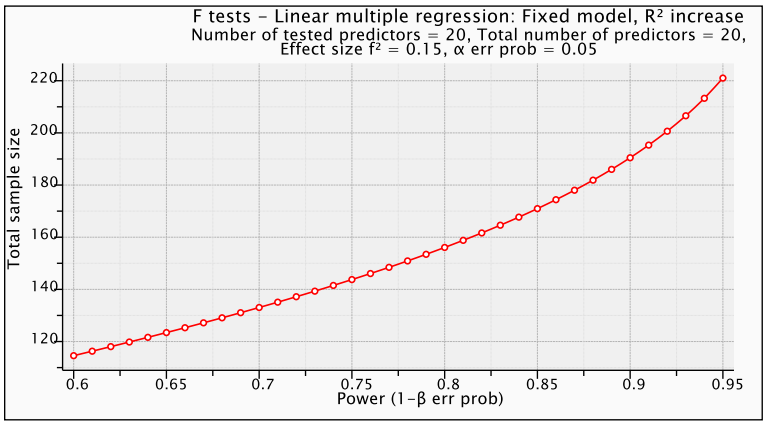

Figure E1. Plot of Statistical Power to Sample Size for Examination of Risk Factors for Incident Traumatic Brain Injury Occurrence

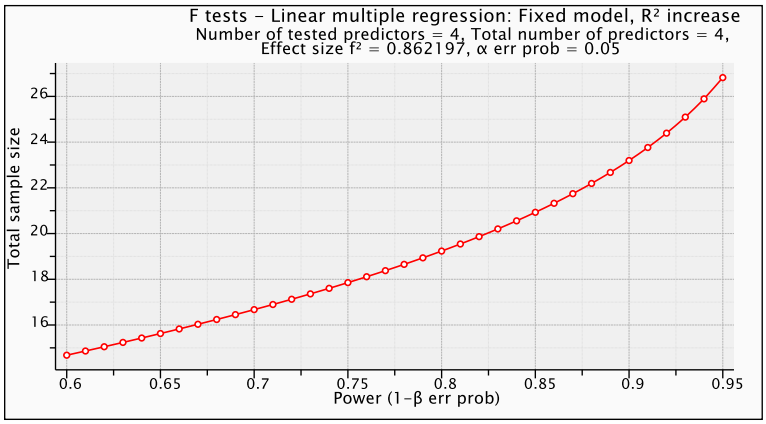

Figure E2. Plot of Statistical Power to Sample Size for Examination of Risk Factors for Incident Traumatic Brain Injury Severity