Investigating Prevalence, Role of Psychological Distress, and Biopsychosocial Correlates of Subjective Cognitive Decline in Older Adults with a History of Mild Traumatic Brain Injury: A Secondary Analysis of the Canadian Longitudinal Study on Aging

by

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B.Sc. (Hons.), University of Toronto, 2022

Thesis Submitted in Partial Fulfillment of the
Requirements for the Degree of
Master of Arts

in the
Department of Psychology
Faculty of Arts and Social Sciences

SIMON FRASER UNIVERSITY
Summer 2024

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Abstract

Older adults with a history of mild traumatic brain injury (mTBI) might be uniquely vulnerable to subjective cognitive decline (SCD), which can adversely impact quality of life. Leveraging data from the Canadian Longitudinal Study on Aging, the present study aimed to determine the prevalence and correlates of SCD in adults and older adults with a history of mTBI. Adults with a single, remote mTBI (n = 861; 59.13%) endorsed SCD at a similar rate to those with no head injury (n = 11,417; 56.78%). Higher psychological distress predicted SCD, whereas change in global cognitive performance from baseline to 3-year follow-up did not. Using a biopsychosocial model, being female, greater depressive symptomatology, lower levels of conscientiousness and openness to experience, and worse self-reported hearing increased the likelihood of SCD. Results provide support for the use of interventions that alleviate psychological distress and target modifiable risk factors to promote cognitive health post-mTBI.

Keywords: Subjective cognitive decline; mild traumatic brain injury; psychological distress; biopsychosocial; aging; CLSA

Acknowledgements

Thank you to my supervisor, Dr. Molly Cairncross, for your support and enthusiasm through this research process and your mentorship through graduate school as a whole over these two years. Thank you to Dr. Allen Thornton, for the insightful discussions we had and your thoughtful ideas, and Dr. Rachel Fouladi, for your encouragement and helpful feedback – I enjoyed all our research conversations. Thank you to my examiner, Dr. Theodore Cosco, for your positive feedback and interesting discussion that helped improve the thesis. I am very grateful to have received CGS-M funding from Canadian Institutes of Health Research to support my master's degree.

Thank you to my mentors and role models in the field – Dr. Morgan Barense, for your continued support and guidance through academia since my early undergraduate years, and for all the conversations we had through this degree, thank you for always inspiring me. Thank you to my friends here and back in Toronto for the laughter, memories, and advice we have shared through the ups and downs of this journey. A very special thank you to my family – to my mom, dad, and brother for all our Zoom calls, for always being there for me, believing in me, teaching me so much, and motivating me to be the best version of myself.

This research was made possible using the data/biospecimens collected by the Canadian Longitudinal Study on Aging (CLSA). Funding for the Canadian Longitudinal Study on Aging (CLSA) is provided by the Government of Canada through the Canadian Institutes of Health Research (CIHR) under grant reference: LSA 94473 and the Canada Foundation for Innovation, as well as the following provinces, Newfoundland, Nova Scotia, Quebec, Ontario, Manitoba, Alberta, and British Columbia. This research has been conducted using the CLSA Baseline Comprehensive Dataset - Version 7.0 and Follow-up 1 Comprehensive Dataset - Version 4.0, under Application Number 2301010. The CLSA is led by Drs. Parminder Raina, Christina Wolfson and Susan Kirkland.

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List of Acronyms

SCD	Subjective cognitive decline
MCI	Mild cognitive impairment
AD	Alzheimer's disease
SCD-I	Subjective Cognitive Decline Initiative
mTBI	Mild traumatic brain injury
LOC	Loss of consciousness
GCS	Glasgow Coma Scale
SMC	Subjective memory complaints
SCC	Subjective cognitive complaints
PTSD	Post-traumatic stress disorder
CLSA	Canadian Longitudinal Study on Aging
BTBIS	Brief Traumatic Brain Injury Screen
RAVLT	Rey Auditory Verbal Learning Test
AFT	Animal Fluency Test
COWAT	Controlled Oral Word Association Test
MAT	Mental Alternation Test
CESD-10	Center for Epidemiologic Studies Short Depression Scale
K10	Kessler Psychological Distress Scale
PASE	Physical Activity Scale for the Elderly
TIPI	Ten-Item Personality Inventory
MOS-SSS	Medical Outcomes Study Social Support Survey
VIF	Variance Inflation Factor
CCHS-HA	Canadian Community Health Survey-Healthy Aging

Chapter 1. Introduction

1.1. Overview of Subjective Cognitive Decline

Subjective cognitive decline (SCD) is the self-reported experience of worsening memory in the absence of objective memory impairment on standardized neuropsychological testing (Jessen et al., 2020). SCD is estimated to affect 1 in 9 individuals aged 45 years and older (Taylor et al., 2018) and studies have shown that between 27% to 43% of community-dwelling older adults report subjective memory complaints (Zuniga et al., 2015). SCD is associated with functional and emotional difficulties, including reduced psychosocial well-being, mental health, and quality of life, which may further exacerbate memory problems (Mol et al., 2007; Sohrabi et al., 2009; Zuniga et al., 2016). Recently, SCD has been proposed as a sensitive, first symptomatic expression of age-related neurodegenerative changes such as mild cognitive impairment (MCI) and Alzheimer's disease (AD) (Jessen, 2014; Reisberg & Gauthier, 2008; Slot et al., 2018). In support of this, SCD can occur on average around 10 years before a dementia diagnosis (Verlinden et al., 2016), and the risk of developing dementia is doubled in older adults with SCD relative to those without SCD (Mitchell et al., 2014). A meta-analysis of longitudinal, epidemiological studies showed that approximately 11% and 25% of cognitively unimpaired individuals who endorsed SCD transitioned to dementia and MCI, respectively, whereas 4.6% of healthy older adults without SCD developed dementia over a five-year period (Jessen et al., 2020; Mitchell et al., 2014). Given the emerging link between SCD and future cognitive decline, it is critical to identify and evaluate modifiable risk and protective factors underlying SCD.

An operationalized definition of SCD in the context of preclinical AD was developed in response to the variability in SCD terminology and categorization across research and clinical settings (Abdulrab & Heun, 2008). Previous work has conflated SCD and related constructs (e.g., cognitive complaints, memory complaints, subjective cognitive impairment, subjective memory impairment) (Mendonça et al., 2015; Rabin et al., 2015), yet one distinction between complaints pertaining to *cognition* and *memory* is that the former encompasses declines in general cognitive abilities and non-memory related functioning (e.g., difficulty concentrating), whereas the latter refers to memory specifically (Burmester et al., 2016). When describing previous work, the terms "SCD" and "subjective cognitive complaints" will be used interchangeably. To refine the

construct of SCD, an international working group, the Subjective Cognitive Decline Initiative (SCD-I), defined SCD as a self-experienced persistent decline in cognitive capacity in comparison to a previously normal status that is not related to an acute event (Molinuevo et al., 2017). That is, SCD is often present when there is normal performance on standardized cognitive tests used to classify MCI, such that exceeding the threshold of a cognitive deficit on a neuropsychological test (e.g., often a deficit of at least 1 or 1.5 SD) would indicate the presence of MCI. Moreover, the SCD-I proposed a set of features that increases the likelihood of preclinical AD (i.e., SCD-plus), one of which is a particular concern or worry about SCD (Jessen et al., 2014; Molinuevo et al., 2017).

In contrast to standardized objective measures of cognition, the approaches used to quantify SCD are more diverse. A systematic review from 19 international cognitive aging studies affiliated with the SCD-I Working Group identified 34 different self-report measures of cognition (Rabin et al., 2015). Consistent with the idea that memory concerns are a harbinger of future neurodegenerative disease, items pertaining to the memory domain were most frequently surveyed in SCD measures (Rabin et al., 2015). Importantly, SCD measures differ in terms of their content, length, format, response options, and psychometric properties (Rabin et al., 2015). Examples of response options include the use of a single question with a dichotomous yes/no response, a single question with a graded response, scales composed of a set of questions with yes/no responses, and longer questionnaires in which a threshold must be met to indicate SCD (Abdulrab & Heun, 2008; Jessen et al., 2014; Molinuevo et al., 2017). Notably, single-question measures of SCD have been utilized in many population-based and clinical studies to determine whether self-perceived memory problems are meaningful indicators of increased risk of MCI and dementia (Abdulrab & Heun, 2020; Mitchell et al., 2014; Reid & MacLullich, 2006). As such, single dichotomous measures of SCD (e.g., “do you feel like your memory is becoming worse?”) are effective ways of classifying individuals who endorse SCD (Molinuevo et al., 2017; van Oijen et al., 2007).

1.2. Mild Traumatic Brain Injury

One important population in whom to investigate SCD is older adults with a history of mild traumatic brain injury (mTBI), who may be uniquely vulnerable to such decline. The most widely-conceived definition of mTBI comes from the American Congress of Rehabilitation Medicine mTBI Committee, which states that mTBI results in

one or more of the following: no more than thirty minutes loss of consciousness (LOC), less than 24 hours of post-traumatic amnesia, an initial Glasgow Coma Scale (GCS) score of 13-15 after thirty minutes post-injury, and/or other focal neurological deficits due to head trauma (Albrecht et al., 2016; Gardner & Yaffe, 2015). mTBI accounts for 70% to 90% of all TBI cases (Kristman et al., 2016) and >75% of older adult TBI cases are mild (Albrecht et al., 2016; Azouvi et al., 2017; Papa et al., 2012). After sustaining an mTBI, one might feel dazed, confused, and temporarily disoriented, and show memory gaps for the injury and some period afterwards (Vanderploeg et al., 2005). During acute and subacute phases of mTBI recovery, individuals commonly report somatic (e.g., headache, sleep disruptions, dizziness, nausea), cognitive (e.g., problems with attention, memory, processing speed, concentration), and affective (e.g., depression, anxiety, irritability) symptoms (Prince & Bruhns, 2017), which are often short-lived and resolve without complication within three months (Carroll et al., 2004). Individuals who sustained an mTBI at an older age, and older adults with a history of remote mTBI, may present with greater vulnerability to the development or persistence of cognitive decline after injury.

1.2.1. Effects of Mild Traumatic Brain Injury Sustained in Older Age

Older mTBI patients often present with pre-existing chronic conditions, medication usage, morphological brain changes, and normative cognitive decline that may influence their long-term prognosis (Markovic et al., 2021; Papa et al., 2012; Thompson et al., 2006). As a result, older adults have an increased risk of delayed recovery trajectories and mortality following mTBI (LeBlanc et al., 2006; Mosenthal et al., 2002; Peters & Gardner, 2018). Following mTBI in older adults, intracranial changes that occur with aging (e.g., dura adherence to skull, cerebrovascular atherosclerosis, bridging vein fragility), in addition to anticoagulant medication use for the management of chronic conditions, may increase the risk of intracranial bleeding visible on head computerized tomography despite normal neurological examination on the GCS following mTBI (Peters & Gardner, 2018; Thompson et al., 2006). Other mechanisms such as cerebral compensation, plasticity, and brain re-organization, in addition to psychological adjustments, might also be compromised in older adults following mTBI, increasing their vulnerability to cognitive decline and poor functional outcomes (LeBlanc et al., 2006).

1.2.2. Long-Term Cognitive Effects of a Remote Mild Traumatic Brain Injury in Older Adults

Emerging research has shown that normal aging processes may interact with long-term effects of a history of a single remote mTBI to exacerbate age-related cognitive decline in older adults. For example, older adults with either MCI or AD with a history of mTBI showed an earlier age at onset of cognitive impairment than those without a history of mTBI (Li et al., 2016). It has also been posited that the aging brain is unable to compensate for previously sustained brain damage, such that a remote mTBI precipitates age-related structural and functional decline (Henry et al., 2017). In support of this, neuroimaging work has shown that changes in brain structure integrity such as white matter anomalies in the frontal cortex and corpus callosum were present in older adults who sustained an mTBI in young adulthood to a degree like that of repetitive head trauma or normal aging (Tremblay et al., 2019). Specifically, older adults who sustained an mTBI in young adulthood more than three decades ago presented with more severe structural brain anomalies than age-matched individuals who sustained an mTBI in late adulthood around two years ago (Tremblay et al., 2019). Of note, both mTBI groups were compared to age-, sex-, and education-matched no head injury control groups to assess differences on neuroimaging measures of interest, to control for confounding variables between remote versus recent mTBI groups and to selectively characterize the effect of age at trauma onset on neurodegeneration. These findings support the notion that long-term effects of mTBI interact with normative age processes, which heightens vulnerability to neurodegeneration and accelerates cognitive decline.

To disentangle the long-term effect of a remote mTBI on cognitive function, one recent study in a nationally-representative sample of adults and older adults found that a small proportion of individuals experienced persistent mild executive and declarative memory impairment following a single mTBI with LOC compared to individuals who did not have a brain injury. Additionally, those who had mTBI with a greater duration of LOC were more likely to experience long-term cognitive impairment (Bedard et al., 2020). Bedard et al. (2018) also reported that adults and older adults with a history of a single remote mTBI showed a disproportionate deficit on time-based prospective memory performance compared to individuals who did not experience brain injury. These studies suggest that cognitive dysfunction may persist in the long-term (e.g., more than 12 months after mTBI) especially for tasks that involve greater frontal and executive

processes. However, a large proportion of individuals with a history of mTBI did not develop persistent cognitive dysfunction (Bedard et al., 2018, 2020). More recently, Bedard & Taler (2021) demonstrated that adults and older adults who reported a remote history of mTBI with LOC of 1–20 min were 60% more likely to experience global cognitive decline over a three-year period compared to individuals without a history of brain injury. Although adults and older adults with a single remote history of mTBI showed selective impairment and decline in certain cognitive domains, an open question is whether this population endorses subjective memory problems (i.e., SCD) to a greater degree than no brain injury controls. We know relatively little about the effects of a remote history of mTBI on the prevalence of SCD. It can be argued that prospective SCD is of considerable importance in this population, given that subjective cognitive symptoms following mTBI may be reported in the absence of cognitive impairment (Jamora et al., 2012; Stillman et al., 2020; Stulemeijer et al., 2007). Moreover, subjective experiences of cognitive change are related to feelings of anxiety and SCD-related worry, which may not only decrease quality of life but also heighten vulnerability to future cognitive decline (Buckley et al., 2016; Desai et al., 2021; Montejo et al., 2011).

1.3. Association Between Subjective and Objective Cognitive Functioning

1.3.1. Healthy Aging

Evidence for the link between SCD and objective memory performance is mixed. A meta-analysis of prospective longitudinal studies reported that rates of conversion to dementia were approximately twice as high for participants with subjective memory complaints (SMC) at baseline compared to those without memory complaints (Mitchell et al., 2014). In accordance with these findings, a systematic review revealed that the risk of developing dementia was 1.5 to 3 times greater for participants with subjective cognitive complaints (SCCs) at baseline than those without SCCs (Mendonça et al., 2016). Of note, the influence of depressive symptoms as well as other confounds such as measurement, severity, frequency, and functional impact of SCCs limited the explanatory value of SCCs in predicting the development of dementia (Mendonça et al., 2016).

One cross-sectional meta-analysis found that subjective memory measures explained less than 1% of the variance in objective memory performance, and that the

relationship between subjective and objective memory was better characterized by demographic (e.g., age, gender, education) and subjective measurement-related variables (e.g., questionnaire vs. interview format; longer vs. shorter questionnaires) (Crumley et al., 2014). Another systematic review with 50 cross-sectional studies also reported a small but significant correlation between subjective and objective cognition (Burmester et al., 2016). Many studies demonstrated evidence of a link between SCCs and rates of cognitive impairment and poorer objective cognitive performance (Calabria et al., 2011; de Jager et al., 2009; Jacinto et al., 2014; Montejo et al., 2011; Ossher et al., 2013; Rijs et al., 2013; Waldorff et al., 2012). However, these studies did not include depressive symptoms as a confound despite the role of depression in the association between SCCs and objective cognitive impairment (Reid & MacLullich, 2006). In contrast, some studies reported the absence of a link between SCCs and objective performance on neuropsychological tests (Mendes et al., 2008; Minett et al., 2008). Notably, a considerable number of studies reported that the association between subjective and objective cognition was diminished or eliminated after controlling for depressive symptoms (Genziani et al., 2013; Montejo et al., 2014; Zlatar et al., 2014). Importantly, research on the link between subjective and objective memory has primarily focused on depression and has not included other psychological symptoms such as anxiety and distress, which may also underlie the relationship.

Taken together, these findings suggest that standard neuropsychological tests might not have sufficient sensitivity to detect subtle changes in objective cognition in individuals who endorse SCD without objective cognitive impairment. As a result, inclusion of SCD measures might refine the detection of individuals who are at early prodromal stages of cognitive impairment despite showing normal cognitive performance. This may be especially useful for individuals with higher cognitive reserve or premorbid functioning, who might be aware of their decline, but their cognitive reserve might serve as a buffer and decrease the likelihood that their objective cognitive impairment is captured by neuropsychological tests (Jia et al., 2021). On the other hand, previous work showing that psychological or affective factors may heighten vulnerability to SCD suggests that cognitive assessments should not be limited to subjective ratings (e.g., check lists of symptoms), but should also include neuropsychological screenings. By further clarifying the nature and extent of associations between SCD, cognitive, and affective aetiologies, we can address potentially inaccurate self-representations as well

as provide treatment recommendations that can help mitigate subsequent cognitive decline.

1.3.2. Mild Traumatic Brain Injury

A substantial proportion of mTBI patients continue to report memory complaints months to years after the injury in the absence of compromised cognitive abilities on neuropsychological tests, termed the “subjective-objective discrepancy” (Stulemeijer et al., 2007). For instance, self-reported cognitive problems of adults with mTBI were endorsed and rated as being equally or more impairing than those of adults with moderate-to-severe TBI, although the moderate-to-severe TBI group showed greater objective memory impairment than the mTBI group (Jamora et al., 2012). Similarly, a population-based study of community-dwelling older adults without dementia found that prior TBI with LOC, but not prior TBI without LOC, was associated with a 38% increased risk for subjective cognitive impairment (i.e., “how would you rate your memory at the present time?”), despite lack of objective cognitive impairment in these individuals. Moreover, this increased risk for subjective cognitive impairment was partially mediated by comorbid depression, suggesting that depression is a modifiable variable of interest that might mitigate SCD (Gardner et al., 2017). This work stratified prior TBI according to LOC (i.e., with versus without LOC), yet was not specific to mTBI.

Prior work has examined explanatory factors for subjective changes in cognition in adult, athlete, and combat-exposed TBI populations. For instance, premorbid characteristics, post-injury emotional and physical status, and fatigue, rather than indices of clinical injury severity and neuropsychological performance, were strongly associated with subjective cognitive complaints six months following mTBI in an adult sample (Stulemeijer et al., 2007). This demonstrates that the dynamic relationship between perceived cognitive difficulties and emotional distress can perpetuate post-concussion cognitive sequalae (e.g., subjective-objective discrepancy). Likewise, emotional distress, namely depression, accounted for self-reported cognitive deficits following mild to moderate TBI (Chamelian & Feinstein, 2006). Consistent with these findings, affective distress (i.e., self-reported anxious and depressive symptoms) explained higher self-reported cognitive symptoms in the context of intact neuropsychological test performance after mTBI in adults (Hromas et al., 2021). Given that emotional difficulties might increase vulnerability to SCD following mTBI or exacerbate premorbid depression

and anxiety levels that further elevate cognitive decline, it is critical to assess how affective distress influences SCD in individuals with a history of mTBI.

In contact sport athlete populations where there is greater exposure to impacts of mTBI, a similar weak association between subjective and objective cognitive performance has been characterized. In a sample of former collegiate football players with a history of mTBI, lower subjectively rated cognitive abilities, rather than lower objective memory performance, were more likely among individuals with a greater history of self-reported mTBI (Bryant et al., 2023). In addition, ratings of subjective cognitive functioning were more strongly associated with psychological distress than objective cognitive performance (Bryant et al., 2023), which has also been reported in middle-aged adults with a history of mTBI (Stillman et al., 2020). Furthermore, studies with veterans who sustained military-related TBI suggest that psychological distress arising from comorbid conditions (e.g., PTSD, generalized anxiety, depression) might be the source of discordant subjective and objective reports of cognitive functioning (Donnelly et al., 2017; Drag et al., 2012; French et al., 2014).

Together, these studies suggest that psychological distress can explain subjective cognitive complaints in the context of normal neuropsychological functioning. However, these findings have been primarily focused on adult, athlete, and combat-exposed TBI populations. In addition, the work illustrated above measured subjective complaints in relation to mTBI symptomatology (e.g., feeling in a fog, difficulty remembering, confusion), rather than subjective complaints in relation to age-relevant memory changes (e.g., “Do you feel like your memory is becoming worse?”). Thus, it remains unclear whether SCD exists in community-dwelling adults and older adults with a history of a single remote mTBI, and the extent to which psychological distress more strongly predicts SCD in these individuals relative to those without a previous brain injury. In the case that SCD is predicted by psychologically relevant, modifiable risk factors, these findings will inform the development of mental health interventions that alleviate psychological distress in older individuals with a history of a single remote mTBI.

1.4. Biopsychosocial Correlates of Subjective Cognitive Decline

1.4.1. Risk and Protective Factors in Healthy Aging

Biopsychosocial models have been proposed in the study of dementia, which indicate that psychological and social factors, in addition to the impact of biomedical processes, influence cognitive changes (Spector & Orrell, 2010). Specifically, the biopsychosocial model proposes that fixed (e.g., biological factors that are not amenable to change) and tractable factors (e.g., psychosocial factors that may be amenable to change) influence dementia symptomatology and intervention effectiveness over the course of the disease. Critically, tractable factors can be modified by domain-relevant interventions (e.g., social support and cognitive stimulation for individuals who show deteriorations in mood and engagement) (Spector & Orrell, 2010). The biopsychosocial model can also be applied to SCD (Rabin et al., 2017). Etiologically-relevant factors can be identified to inform the development of targeted treatment and prevention approaches that promote cognitive and psychological functioning in individuals with SCD (Rabin et al., 2017). For instance, early interventions targeting psychological health can be beneficial for individuals in whom SCD is driven by mood, personality, and physical health concerns. In contrast, specialized interventions that enhance cognitive function might be useful to preclude the onset of cognitive decline for individuals with SCD that is associated with biomedical risk factors (Smart et al., 2017).

In healthy aging, psychological factors play an integral role in the development of SCD. There is a well-established link between SCD and affective symptoms (e.g., depression and anxiety) as well as personality traits (e.g., high neuroticism and low conscientiousness) (Hill et al., 2016; Reid & MacLullich, 2006; Snitz et al., 2015). Converging evidence from longitudinal community-based studies have found that depressive and anxiety symptoms and personality traits (e.g., less mastery, less perceived self-efficacy, high neuroticism) explained the endorsement of subjective memory complaints after controlling for objective memory performance (Comijs et al., 2002), and to a greater degree than cognitive factors (Slavin et al., 2010). Furthermore, cognitively unimpaired older adults with higher negative affect (e.g., depressive symptoms, anxiety complexity, worry) displayed greater memory complaints despite lack of objective cognitive impairment (Dux et al., 2008).

SCD and cognitive decline have also been associated with biological and health-related factors. Increased risk of cognitive decline is related to biological factors such as genetic susceptibility, sensory loss, hypertension, diabetes, cardiovascular disease, late-life high cholesterol, and use of cholesterol-lowering drugs (Baumgart et al., 2015; Qiu et al., 2009). Moreover, adults with multiple chronic diseases show greater prevalence of SCD, and the presence of common chronic diseases such as stroke, heart disease, and chronic obstructive pulmonary disease increase the likelihood of SCD as well as objective cognitive impairment (Taylor et al., 2020). Although many biological factors are non-modifiable, there are modifiable health-related behaviors that can aggravate or mitigate the course of cognitive decline. For example, heavy alcohol consumption and tobacco use may be risk factors for cognitive decline, whereas higher adherence to a nutritious diet may be protective against cognitive decline (Livingston et al., 2020; Qiu et al., 2009). Furthermore, clinical trials have suggested that physical activity interventions are one of the strongest protective lifestyle factors, with reported cognitive benefits in individuals with SCD and MCI (Lautenschlager et al., 2019).

There has been less attention on psychosocial factors associated with SCD. Social support, defined as (1) the perception that one is cared for and has supports available, (2) the actual assistance that is received, and/or (3) the level of integration within a social network, plays an important role in cognitive function in adults and older adults (Pillemer & Holtzer, 2016). For example, the presence and degree of social support has been shown to reduce the rate of cognitive decline and dementia (Barnes et al., 2004; Bassuk et al., 1999; Ertel et al., 2008; Fratiglioni et al., 2004). Distinct dimensions of perceived social support and cognitive function have also been identified, in which tangible (i.e., concrete and direct way of providing support, such as financial assistance, services, material items) and affectionate support (i.e., feeling of being loved and receiving attention) were not associated with cognitive function, whereas perceived emotional/informational support (i.e., nurturance and sense of being looked after) and positive social interaction (i.e., degree to which one has another to have a good time with and enjoy things with) exerted positive effects on cognition (Ellwardt et al., 2013; Pillmer & Holtzer, 2016). One explanation is that social support that promotes meaningful interpersonal interactions and cognitive engagement may exert the most protective effects on cognition (Cohen & Wills, 1985). Indeed, it has been proposed that the influence of biological and lifestyle-related factors (e.g., physical inactivity, tobacco use, alcohol use, cardiovascular and metabolic conditions) on cognitive impairment can

be modulated by psychosocial factors such as high level of social engagement, social support, and larger social networks in older age (Ellwardt et al., 2013; Marioni et al., 2015; Piolatto et al., 2022).

Given that SCD can be a harbinger for dementia, identifying biopsychosocial correlates of prospective SCD can bolster early prevention and intervention strategies for individuals at-risk for transition to dementia. To our knowledge, one study applied a biopsychosocial framework to SCD and found that well-established risk factors for cognitive aging and dementia, such as physical activity and health-related risk factors (e.g., hypertension, alcohol intake, smoking) did not predict SCD (Hopper et al., 2023). Notably, depression, perceived social status, and personality traits (e.g., conscientiousness, neuroticism), were associated with SCD, suggesting that psychosocial factors may have protective effects on SCD.

1.4.2. Risk and Protective Factors in Mild Traumatic Brain Injury

There is a paucity of research investigating biopsychosocial risk and protective correlates of prospective SCD in adults and older adults with a remote history of mTBI. Considerable research has revealed that pre-injury psychological factors and current symptomatology, rather than injury-related indicators (e.g., loss of consciousness, post-traumatic amnesia, microstructural MRI abnormalities) are associated with persistent post-concussive symptoms following mTBI (Ponsford et al., 2019; van der Naalt et al., 2017; Wäljas et al., 2015), yet biopsychosocial correlates of prospective SCD have not been translated to adults and older adults with a remote history of mTBI. One observational study examined subjective cognitive complaints in older adults with TBI. In the study, increased age, time-since-injury, communication difficulties, somatic symptoms, and chronic stress were associated with subjective memory complaints in older adults who were recovering from mild-to-moderate TBI (Bay et al., 2012), which suggests that SCD in older adults following mTBI might be explained by both medical and psychological factors. One longitudinal study examined the association between subjective and objective cognitive functioning at 2 weeks and 3 months after mTBI and reported that change in cognitive symptom severity was not related to cognitive test performance but was strongly associated with change in depression and anxiety symptoms (Stenberg et al., 2020). In other words, there was a discrepancy in recovery trajectory such that objective cognitive outcomes showed average improvement over

time, yet this objective improvement was not associated with self-reported cognitive symptoms (Stenberg et al., 2020). These findings suggest that emotional symptom severity might also be a relevant risk factor for cognitive complaints or SCD in adults and older adults with a history of single remote mTBI. The identification of psychological factors that can serve as modifiable intervention targets can help minimize SCD following mTBI.

However, it is important to note that biological risk factors of SCD may emerge as prominent risk factors in the adult and older adult mTBI population, in addition to the importance of psychological distress as established in other populations. Given that older adults are susceptible to poorer outcomes following mTBI, and the fact that mechanisms associated with a remote TBI sustained in early age or midlife might exacerbate normative age-related decline, recent work has underscored the importance of including age-relevant explanatory variables when studying cognitive decline in these mTBI populations. Studies have shown that pre-existing medical conditions such as hypertension is associated with the onset of cognitive impairment following mTBI in older adults (Karr et al., 2021; Ozono et al., 2022). It is reasonable that comorbid medical and behavioral health conditions might interact with long-term residue of cognitive effects in older adults with a history of mTBI. This is an important area of investigation to enhance prognosis and treatment of SCD in individuals with a remote history of mTBI, yet these etiologically-relevant prognostic variables have not been translated to the domain of prospective SCD. Moreover, key psychosocial factors that have been shown to mitigate cognitive decline in healthy and pathological aging, such as social engagement and social support (James et al., 2011; Ellwardt et al., 2013; Marioni et al., 2015) have not been included in research on long-term cognitive symptoms in older adults post-mTBI. A clearer understanding of the relative significance of modifiable (e.g., psychosocial factors; health-related behaviors) and non-modifiable correlates (e.g., biomedical) of prospective SCD in older adults with a history of single remote mTBI is needed. By leveraging a biopsychosocial framework to reveal prognostic factors of prospective SCD in this population, more effective treatment and rehabilitation tools can be developed.

1.5. Current Study

SCD is a common experience that increases with age and older adults with a remote history of mTBI might be more susceptible to SCD. Research in adult, athlete,

and veteran mTBI populations has shown that psychological or affective factors, rather than objective cognitive functioning, were responsible for subjective cognitive complaints, yet these relationships have not been studied in older adults with a single, remote history of mTBI. Understanding the biopsychosocial correlates of SCD in this population could also inform the design of targeted, etiologically-relevant interventions following mTBI. This thesis aimed to address the following objectives in a large-scale, nationally-representative sample of adults and older adults from the Canadian Longitudinal Study of Aging (CLSA; Raina et al., 2019). The hypotheses for each of these aims are described in turn, below:

Aim 1:

A) Determine the prevalence of SCD in participants with a self-reported history of a single remote mTBI relative to no brain injury controls.

Hypothesis: The prevalence of SCD will be higher in participants who self-report a history of mTBI compared to no brain injury controls.

B) Identify whether the prevalence of SCD differs based on mTBI-related factors, such as duration of loss of consciousness and number of currently experienced symptoms related to a possible head injury/concussion.

Hypothesis: Consistent with previous findings described above, the prevalence of SCD will be comparable across participants who reported longer versus shorter durations of LOC (i.e., 1-20 minutes unconscious versus less than one minute unconscious). The prevalence of SCD will be higher for participants who reported more currently experienced symptoms related to a possible head injury/concussion than those who currently endorsed fewer symptoms.

Aim 2:

A) Examine whether SCD is associated with objective measures of cognitive performance (i.e., global cognitive performance at follow-up 1), controlling for global cognitive performance at baseline in both groups.

Hypothesis: With respect to the well-established weak association between subjective and objective measures of cognition reported in healthy aging and various TBI

populations, endorsement of SCD at follow-up 1 will not be associated with objective memory performance for both the mTBI group and no brain injury control group.

B) Investigate the role of psychological distress and objective measures of cognitive performance (i.e., change in objective cognitive functioning and baseline objective cognitive performance) in explaining SCD, and whether the relationship between psychological distress and SCD differs in the mTBI versus no brain injury control group.

Hypothesis: Extending the findings described above in various mTBI populations, psychological distress will be associated with SCD after controlling for sociodemographic and health conditions that affect brain health, above and beyond change in objective cognitive performance and baseline objective cognitive performance, and that this relationship will be stronger in participants with a history of mTBI relative to no brain injury controls.

Aim 3:

Converge the complex interplay between biological, psychological, and social factors in age-related cognitive processes by exploring biopsychosocial risk and protective correlates of prospective SCD (e.g., demographics, injury-related factors, medical factors, health-related behaviors, psychological factors, social factors) in this mTBI sample.

Hypothesis: Although there are no specific hypotheses on the predictive power of individual biopsychosocial correlates on SCD given the explanatory nature of this aim, it is predicted that medical and health-related behaviors, in addition to psychosocial variables, will emerge as explanatory correlates of SCD in participants with a history of a single remote mTBI.

Chapter 2. Method

2.1 Data Source

The Canadian Longitudinal Study on Aging (CLSA) is a longitudinal research platform that investigates transitions and trajectories of healthy aging (Raina et al., 2019). CLSA consists of a nationally-representative random sample of over 50,000 Canadians aged 45-85 years at the time of recruitment (Raina et al., 2009, 2019). Data collection began in 2011 (baseline) and takes place at 3-year intervals for at least 20 years (follow-ups). The CLSA population was restricted to those between the ages of 45 to 85 years at baseline and who can read and speak either English or French. The CLSA exclusionary criteria consisted of individuals who: (1) were living in long-term care institutions, (2) presented with cognitive impairment at the time of recruitment, (3) lived in Canadian territories or remote areas, and (4) were full-time members of the Canadian Armed Forces.

The initial CLSA sample consisted of two cohorts: (1) Comprehensive Cohort (n = 30,097), in which participants were first evaluated through 90-minute at-home interview (computer-assisted personal interview) and then completed in-person comprehensive physical, biological, and cognitive assessments and (2) Tracking Cohort (n = 21,241), in which participants were evaluated through telephone interviews. For recruitment, the CLSA team collaborated with Statistics Canada and utilized the Canadian Community Health Survey 4.2 (CCHS 4.2) as a recruitment vehicle, followed by additional recruitment from provincial health registration databases for an estimated sample of 50,000 Canadians. Participants in the Comprehensive Cohort were randomly selected from within 25-50 kilometers of 11 data collection sites in seven provinces (Victoria, Vancouver, Surrey, Calgary, Winnipeg, Ottawa, Hamilton, Montreal, Sherbrooke, Halifax, and St. John's). Participants in the Tracking Cohort were randomly selected Canadian Community Health Survey participants from within all ten provinces who consented to future contact from CLSA principal investigators. Full details of CLSA sampling and recruitment are described in Raina et al. (2009, 2019).

All participants provided informed consent prior to completing the questionnaires and neuropsychological assessments described below. The CLSA study (project # 2301010) was reviewed and approved by the Ethical, Legal, and Social Issues Committee of the Canadian Institutes of Health Research. The current study was

reviewed and approved by the Research Ethics Board at Simon Fraser University (# 30001712).

2.2 Participants

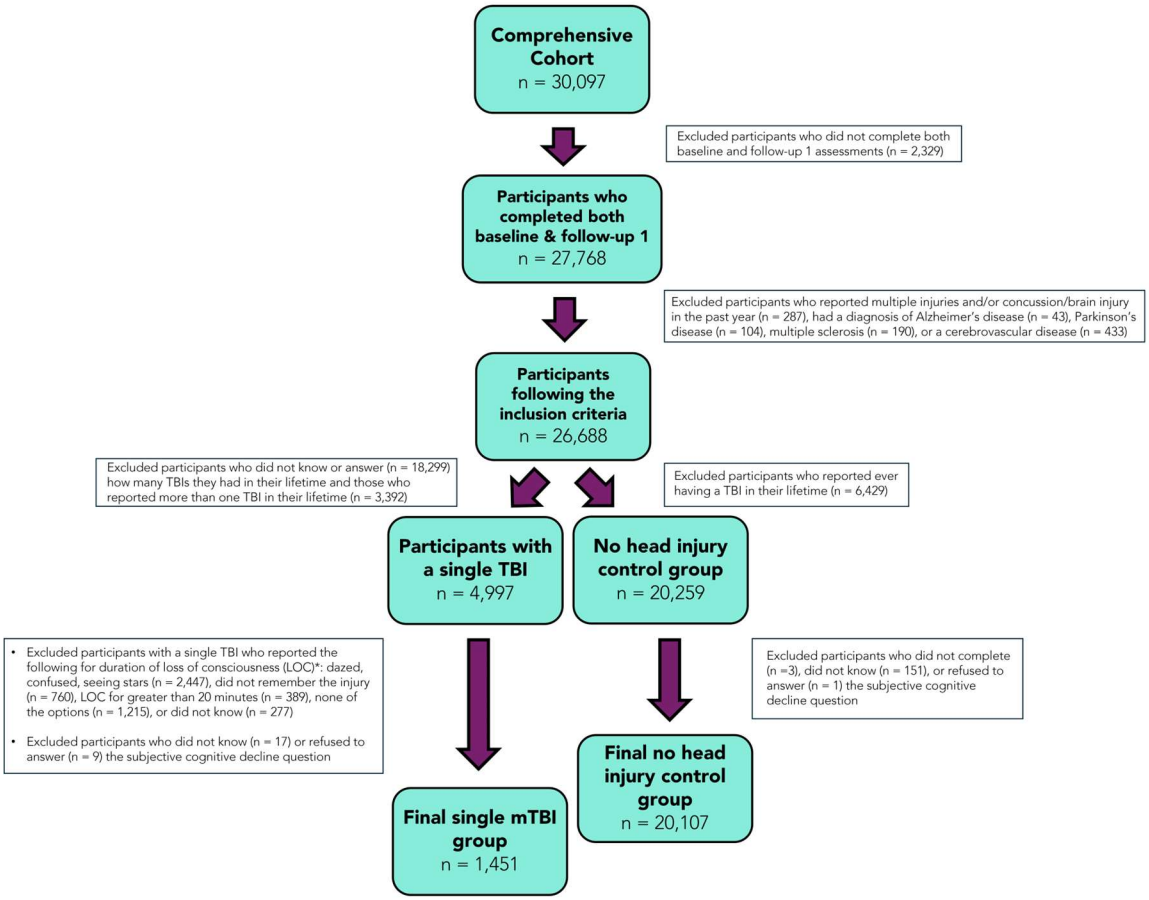
Data from participants who completed baseline (2011-2015) and follow-up 1 (2015-2018) data collection waves were analyzed. Only participants from the Comprehensive Cohort were included, since participants in the Tracking Cohort were not asked to report prior TBI. In line with established definitions of mTBI as described in Silverberg et al. (2023), an mTBI involves a duration of LOC of no more than thirty minutes; in the current work, participants who reported a TBI with LOC greater than 20 minutes were excluded, as this was the highest duration of LOC collected in the CLSA (see Measures). Note that CLSA does not provide information on post-traumatic amnesia or Glasgow Coma Scale score.

Participants who reported a concussion or extracranial injury (e.g., broken bone, burn, poisoning, etc.) in the past 12 months were excluded. The rationale for doing so was that CLSA does not provide information on when the injury occurred, such that it is unclear whether individuals in that category were in the acute phase of recovery at the time of data collection (e.g., within one-month post-injury). Moreover, participants who reported multiple head injuries were excluded. Given that our outcome variable is SCD, participants who reported at baseline or follow-up 1 a diagnosis of a neurodegenerative disease, neurological disorder, and/or medical condition that could impact cognition (i.e., dementia or AD, multiple sclerosis, Parkinson's disease, stroke/cerebrovascular event) were also excluded.

In sum, participants were included in our mTBI sample if they completed both baseline and follow-up studies, endorsed mTBI with LOC less than 20 minutes more than 12 months prior to study recruitment at baseline, did not report a brain injury or other injury in the past 12 months, did not experience multiple head injuries, and were not diagnosed with a neurodegenerative disease, neurological disorder, and/or medical condition that could impact cognition. Participants who never experienced a brain injury were included in the no head injury control group; the same set of exclusion criteria as noted above was applied to the control group. Figure 2.1 provides a CONSORT flow diagram representing the two groups.

Figure 2.1

CONSORT Flow Diagram of mTBI and No Head Injury Control Groups



Note: *The CLSA allowed for multiple selections in their classification of duration of loss of consciousness, such that participants were able to select more than one of the following: (1) dazed, confused, seeing stars; did not remember the injury; LOC for <1 minute; LOC for 1-20 minutes; LOC for >20 minutes.

2.3 Measures

The CLSA collected data on biological, clinical, psychological, lifestyle and behavior, and social measures that influence disease, health, and aging (Raina et al., 2009). The measures relevant to our questions of interest are described below. Demographic variables such as age, sex, education level, marital status, self-rated general health (5-point rating scale: “In general, would you say that your health is

excellent, very good, good, fair, or poor?"), and perceived social status (self-reported social standing in their community on a scale of 1-10 on a ladder) were inquired at baseline. Age, self-rated general health, and perceived social status were treated as continuous variables; sex, education level, and marital status were treated as categorical variables.

2.3.1 Mild Traumatic Brain Injury

TBI history was assessed using the Brief Traumatic Brain Injury Screen (BTBIS), a quick self-report screening tool of TBI. The BTBIS collects data on the mechanism of head injury (e.g., vehicular crash; fall; sports-related activity; etc.), number of lifetime head injuries; duration of LOC (i.e., <1 minute; 1-20 minutes; >20 minutes), currently experienced problems that they believe might be related to a possible head injury or concussion (i.e., headaches, dizziness, memory problems, balance problems, ringing in the ears, irritability, sleep problems), and medical care received for the head injury (i.e., physician assessment; emergency department visit, hospitalization, none).

2.3.2 Cognition

Subjective Cognitive Decline (SCD). At follow-up 1, participants were asked whether they experienced SCD with the question "Do you feel like your memory is becoming worse?". Participants responded yes or no. This measure allowed the assessment of prospective SCD, which was defined as an endorsement of SCD up to three years following baseline study participation. A single measure of SCD is effective for the classification of SCD endorsement and has been used in community-based studies to predict the incidence of cognitive decline (Geerlings et al., 1999; Molinuevo et al., 2017; van Oijen et al., 2007).

Objective Cognitive Performance. At baseline and follow-up, participants completed a standardized neuropsychological test battery. The cognitive measure in the current work included tests of episodic memory, language, and executive function. For an overview of the implementation of cognitive measures in the CLSA, see Tuokko et al. (2016). Memory was assessed using an abbreviated version of the Rey Auditory Verbal Learning Test (RAVLT; Rey, 1964), consisting of two trials in which participants were presented with 15 words and asked to recall as many words as possible (immediate

recall) and again, after a five-minute delay (delayed recall). The total RAVLT score ranged from 0 to 30, with higher scores indicating better verbal memory.

Executive function tasks included the Animal Fluency Test (AFT; Rosen, 1980), Controlled Oral Word Association Test (COWAT; Lezak et al., 2004), Mental Alternation Test (MAT; Teng, 1995), and the Victoria Stroop Test (Stroop; Strauss et al., 2006). In the AFT, participants were asked to list as many animals as possible within one minute. One point was awarded for each animal listed; the overall AFT score reflected the number of valid animals that were listed. In the COWAT, participants were asked to generate as many words as possible beginning with a given letter within one minute. Three trials were administered for the letters F, A, and S. One point was awarded for each unique word named per trial; the overall COWAT score consisted of the summed points from all three trials. In the MAT, participants were asked to alternate between the numbers 1-26 and letters of the alphabet in ascending order (i.e., 1-A, 2-B, 3-C, etc.) as quickly as possible within 30 seconds. The overall MAT score ranged from 0 to 51, which was defined as the number of correct number and letter alterations made in 30 seconds, discounting any errors. The Stroop task included three trials, during which participants were instructed to (1) name the ink colors of the dots presented on a card (Dot condition), (2) name the ink color of non-color words presented on a card (Word condition), and (3) name the color of the ink that color words are printed in and not read the color name (Interference condition) (i.e., say ‘yellow’ for the word ‘blue’ written in yellow ink). An interference Stroop score was derived by dividing task time of the interference condition by the completion time of the Dot condition. For all measures except the Stroop task, higher scores indicated better cognitive performance.

Standardized z-scores for individual neuropsychological tests at both timepoints were created using sample means and standard deviations at baseline. To quantify global cognitive performance at both timepoints, a composite score was created by standardizing the sum of all individual z-scores for each cognitive test (i.e., RAVLT total score, AFT score, COWAT score, MAT score, reversed Stroop interference score). To assess the change in global cognitive performance across baseline and follow-up 1 (Aim 2B), the composite score at baseline was subtracted from the composite score at follow-up 1. Given the exploratory nature of Aim 3, the cognitive domain was quantified by memory performance on the RAVLT (total raw score).

2.3.3 Psychological Factors

Depression: The Center for Epidemiologic Studies Short Depression Scale (CESD-10) measured depressive symptoms at baseline and follow-up 1 (Andresen et al., 1994). The CESD-10 includes ten questions regarding depressive symptoms over the past week rated on a 4-point Likert rating scale: all of the time (5-7 days), occasionally (3-4 days), some of the time (1-2 days), or rarely or never (<1 day). Total CESD-10 scores range from 0-30, with scores 10 or more indicating a positive screen for depression. The CESD-10 has shown strong construct validity and good internal consistency in community samples (González et al., 2017; Mohebbi et al., 2018). In the current sample, internal consistency on the CESD-10 was acceptable to good (Taber, 2018), with a Cronbach's alpha of 0.76 at baseline and 0.80 at follow-up 1.

Psychological Distress: The Kessler Psychological Distress Scale (K10) assessed levels of psychological distress with 10 questions on anxiety and depressive symptoms (e.g., "how often did you feel hopeless?", "how often did you feel depressed?", "how often did you feel nervous?", "how often did you feel restless or fidgety?") in the previous 30 days at follow-up 1 (Kessler et al., 2003). Participants responded to the questions with a 5-item rating scale: none of the time (1), a little of the time (2), some of the time (3), most of the time (4), and all of the time (5). Scores across items were summed to create a total score ranging from 10 to 50. The K10 has demonstrated high levels of internal consistency and convergent validity in many populations (Bougie et al., 2016; Jong Won et al., 2015; Oakley Browne et al., 2010; Sampasa-Kanyinga et al., 2018). Although the CESD and K-10 may have overlapping items, this was of minimal effect, given the number of items in these scales and their unique underlying constructs (Wister et al., 2018). In the current sample, internal consistency on the K10 was good to high (Taber, 2018), with a Cronbach's alpha of 0.82 at baseline and 0.97 at follow-up 1.

Psychological Distress Composite Measure: To assess the influence of psychological distress on SCD (Aim 2B), scores from the CESD-10 and K10 at follow-up 1 were standardized into z-score metrics and averaged together to establish the psychological distress composite measure.

2.3.4 Medical Factors

Chronic Conditions: In the CLSA, chronic conditions were defined as "long-term conditions which are expected to last or have already lasted 6 months or more and that have been diagnosed by a health professional." There were six groups of chronic conditions: (1) Cardiac/Cardiovascular (i.e., heart disease, peripheral vascular disease/poor circulation in your limbs), (2) Gastrointestinal (i.e., stomach ulcers, bowel disorder, bowel and urinary incontinence), (3) Cancer (e.g., multiple types), (4) Mental Health (e.g., anxiety disorders, mood disorders), (5) Other Conditions (i.e., allergies, back problems, kidney disease/failure, etc.), and (6) Infections (i.e., pneumonia, flu, urinary tract infection). Additionally, participants were asked about the presence of other diseases, including the presence of Diabetes, Stroke/Cerebrovascular Event, Hypo and Hyperthyroidism, and Hypertension. For each health condition, participants were asked "Has a doctor ever told you that you have [condition]?" (yes/no). Participants reported chronic conditions and diseases at baseline and follow-up 1.

For the chronic conditions measure, the number of conditions that participants reported that fell under a Cardiac/Cardiovascular condition, Diabetes, and/or Hypertension were summed, and the number of reported conditions were categorized into 0, 1, or 2 or more. The rationale for this decision was that adults with multiple of these chronic conditions show greater prevalence of SCD (Taylor et al., 2020), and that these specific chronic conditions are established risk factors of cognitive decline (Picano et al., 2014; Song et al., 2020). Moreover, age-relevant comorbid conditions (e.g., hypertension) are prevalent in mTBI populations and are associated with subsequent cognitive impairment following mTBI (Gardner et al., 2017; Karr et al., 2021; Ozono et al., 2022), underscoring the importance of determining the role of these chronic conditions in prospective SCD in individuals with a history of a single remote mTBI. Of note, the chronic conditions measure served as an important covariate for subsequent analyses, given that these factors are linked to increased risk of SCD and can account for cognitive aging across our three-year window of observation (i.e., baseline to follow-up 1) (Hopper et al., 2023; Murman, 2015).

Hearing and Vision: Hearing and vision were both self-reported on a 5-point rating scale: excellent (1), very good (2), good (3), fair (4), and poor (5) at baseline and follow-up 1.

2.3.5 Health-Related Behaviors

Alcohol Use: Alcohol use was measured using a series of questions about how often the respondent drinks alcohol and the frequency of heavy drinking (e.g., 5 drinks or more) in the past 12 months at baseline and follow-up 1. Alcohol use was based on participants' self-reported alcohol use history (i.e., "Have you ever drank alcohol?") and frequency (i.e., "About how often in the past 12 months did you drink alcohol?"). Alcohol use was categorized into the following four categories: (1) non-drinker (never drank alcohol and did not drink in the last 12 months), (2) former/12-month abstainer (drank alcohol in the past but not in the last 12 months, or drank alcohol in the past but had less than one drink per month in the last 12 months), (3) occasional drinker (drank alcohol in the past and had at least one drink per month in the last 12 months), and (4) regular drinker (drank alcohol in the past and had at least one drink per week in the last 12 months).

Smoking Habits: Smoking status was assessed using a series of questions about current smoking habits (e.g., frequency of smoking, number of cigarettes smoked in a day, use of other tobacco products, exposure to other people's tobacco smoke, use of electronic cigarettes) at baseline and follow-up 1. Smoking status was based on the participant's self-reported smoking history (i.e., "Have you smoked at least 100 cigarettes in your life?") and frequency (i.e., "At the present time, do you smoke cigarettes daily, occasionally, or not at all?"). Smoking status was classified as non-smokers (never smoked ≥100 cigarettes), former smokers (smoked ≥100 cigarettes in their lifetime but have not smoked in the past month), and current smokers (smoked ≥100 cigarettes in their lifetime and smoked in the past month).

Physical Activity: The Physical Activity Scale for the Elderly (PASE) assessed frequency and duration of activities (i.e., walking, housework, yard work, and caring for others) over the last week at baseline and follow-up 1 (Washburn et al., 1993). Physical activity was defined as the summed score of the frequency of activity (hours/day) multiplied by intensity level of the activity for each activity. All scores were summed, ranging from 0 to 739, with higher scores indicating greater levels of physical activity. PASE has demonstrated good validity and test-retest reliability (Loland, 2002; Washburn et al., 1993).

2.3.6 Social Factors

Personality Traits: The Ten-Item Personality Inventory (TIPI) assessed the Big-Five personality traits, which included extraversion, agreeableness, conscientiousness, emotional stability (i.e., opposite of neuroticism), and openness to experience dimensions at baseline and follow-up 1 (Gosling et al., 2003). Considering personality traits that are most relevant to SCD, the current work focused on conscientiousness, emotional stability, and openness to experience dimensions. Each dimension was represented by two items, with responses ranging from strongly disagree to strongly agree (1-7). Items were reverse scored as appropriate and higher scores indicated more of the trait. TIPI has shown adequate convergent validity and test-retest reliability and is a useful tool for personality measurement (Gosling et al., 2003). Given that each dimension only comprised two brief items, internal consistency for each dimension on the TIPI in the current sample was weak (Taber, 2018), with Cronbach's alpha of 0.39 (conscientiousness), 0.58 (emotional stability), and 0.36 (openness to experience) at baseline, which is consistent with previously reported low internal consistency across TIPI versions and subscales (Thørrisen & Sadeghi, 2023).

Social Support Availability: The Medical Outcomes Study Social Support Survey (MOS-SSS) assessed levels of perceived social support at baseline and follow-up 1 (Sherbourne & Stewart, 1991). The MOS-SSS includes 19 items rated on a 5-point Likert scale to denote how often each item of support was available to the individual if they needed it: none of the time (1), a little of the time (2), some of the time (3), most of the time (4), and all of the time (5). The MOS-SSS includes four subscales: (1) emotional or informational support (e.g., positive affect expressions, empathic understanding, encouragement of expressions of feelings, offering of advice, guidance, or feedback); (2) tangible support (e.g., instrumental aid or behavioral assistance); (3) positive social interaction (e.g., availability of others to positively engage with); and (5) affectionate support (e.g., expression of love and admiration). Total MOS-SSS scores ranged from 0-100, with higher scores indicating a greater perceived level of social support. The MOS-SSS has demonstrated high convergent validity, discriminant validity, and internal-consistency reliability (Sherbourne & Stewart, 1991). In our current sample, internal consistency for each subscale on the MOS-SSS was high to excellent (Taber, 2018), with Cronbach's alpha of 0.93 (emotional or informational support), 0.86 (tangible support), 0.88 (positive social interaction), and 0.86 (affectionate support).

Social Participation: Social participation was measured by self-reported frequency of participation for 8 types of community activities during the past year: (1) family- or friendship-based activities outside the household, (2) church or religious activities, (3) sports or physical activities, (4) educational and cultural activities, (5) service club or fraternal organization activities, (6) neighborhood, community, or professional association activities, (7) volunteer or charity work, and (8) any other recreational activities involving other people (e.g., hobbies, gardening, bridge, cards) at baseline and follow-up 1. Participants responded “at least once a day”, “at least once a week”, “at least once a month”, “at least once a year”, and “never” to these questions on community engagement. CLSA has a derived variable, termed frequency of community-related activity participation, which categorized respondents by the frequency of their participation in any type of community-related activity during the past 12 months on a scale of “did not participate”, “daily”, “weekly”, “monthly” and “yearly”. These categories were collapsed to create a three-level frequency of the social participation variable: (1) infrequent/no participation (i.e., never, at least once/year, or at least once/month), (2) moderate participation (i.e., at least once/week), and (3) frequent participation (i.e., at least once/day).

2.4 Statistical Analysis

Demographics and Clinical Characteristics

Baseline demographic information (e.g., age, sex, educational level, marital status, self-rated general health, level of depressive symptomatology) were reported for the mTBI group and no brain injury control group (Table 3.1). Clinical characteristics of the mTBI group (e.g., mechanism of head injury, duration of LOC, number of symptoms currently endorsed, type of medical care received) are presented in Table 3.2. Number of participants and percentages were used to depict categorical data, whereas means and standard deviations were used to depict continuous data. Continuous variables were tested for normality with Q-Q plots and skew statistics; all continuous variables were normally distributed, with the exception of Stroop Interference scores, which were positively skewed. To compare differences in baseline demographic and clinical data across groups, chi-squared tests were used to analyze categorical data, and t-tests were used for normally distributed continuous data. Significance levels were set to $p < .05$ for all statistical analyses unless otherwise specified.

Raw Neuropsychological Test Performance

Although beyond the scope of the current thesis, for completeness, means and standard deviations for raw neuropsychological data at baseline and follow-up 1 timepoints across group membership (i.e., mTBI vs. no head injury controls) and endorsement of SCD are illustrated in Table 3. Separate linear-mixed models were conducted for each neuropsychological test, with group (mTBI vs. no head injury controls), endorsement of SCD (yes vs. no), timepoint (baseline vs. follow-up 1), and their interactions as fixed-effects. Participants were included as a random intercept to account for within-subject variability. The Benjamini and Hochberg method was applied to generate adjusted p-values, to account for the false discovery rate given the number of statistical tests conducted on the same data.

Analysis 1A: Prevalence of Subjective Cognitive Decline

A chi-squared test was used to compare the prevalence of prospective SCD (i.e., the proportion of participants who reported *yes* to “do you feel like your memory is becoming worse?”) at follow-up 1 in the mTBI group and no brain injury controls.

Analysis 1B: Influence of Injury-Related Factors on Prevalence of Subjective Cognitive Decline

A chi-squared test was conducted to compare differences in the proportion of participants who endorsed SCD at follow-up 1 across categorical injury-related measures, such as duration of LOC and number of currently experienced symptoms related to a possible head injury or concussion in the mTBI group. LOC was categorized into less than one minute and between 1-20 minutes. Currently experienced symptoms were categorized into zero and more than one.

Analysis 2A: Association Between Subjective Cognitive Decline and Objective Measures of Cognitive Performance

To assess the association between SCD and objective cognitive performance, linear models were conducted to predict global objective cognitive performance at follow-up 1 by group (mTBI vs. no head injury controls) and SCD (yes vs. no), controlling for global objective cognitive performance at baseline. Age, sex, education, perceived social status, and health conditions that affect brain health (i.e., hypertension, diabetes,

cardiovascular diseases) were included as covariates. Hypothesis 2A would be supported if SCD is not significantly associated with global objective cognitive performance at follow-up 1 after controlling for global objective cognitive performance at baseline and relevant covariates. Given that the SCD in CLSA specifically referenced *memory* (i.e., Do you feel like your memory is becoming worse?), a post-hoc linear model was conducted to predict delayed RAVLT performance at follow-up 1 by group and SCD, controlling for delayed RAVLT performance at baseline.

Analysis 2B: Role of Psychological Distress Versus Objective Cognitive Performance in Explaining Subjective Cognitive Decline

A binomial logistic regression was conducted to investigate (1) whether psychological distress more strongly explains SCD compared to change in objective cognitive performance, and (2) whether this relationship is stronger in participants with a history of mTBI compared to no brain injury controls. SCD was the outcome variable, with a psychological distress composite measure, change in global cognitive performance (i.e., baseline – follow-up 1), group membership (i.e., mTBI vs. no head injury controls), and their interactions, as predictors. Group membership was effect-coded, with participants with a history of mTBI coded as “1” and no brain injury controls coded as “0”. Endorsement of SCD was coded as “1” and no endorsement of SCD was coded as “0”. Scores from the CESD-10 and K10 at follow-up 1 were standardized into z-score metrics and averaged together to establish the psychological distress composite measure. Age, sex, education, perceived social status, and health conditions that affect brain health (i.e., hypertension, diabetes, cardiovascular diseases) were included as covariates. Specifically, the number of health conditions that affect brain health were categorized into zero, one, or two or more. Hypothesis 2B(1) would be supported if odd ratios and corresponding 95% confidence intervals show that increasing psychological distress is significantly associated with a higher risk of SCD after controlling for covariates. Hypothesis 2B(2) would be supported if there was a significant two-way interaction between psychological distress and group membership, and if simple slope analyses indicate that the magnitude of increased risk for the SCD associated with psychological distress is greater in the mTBI group compared to no brain injury controls. Multicollinearity was assessed using variance inflation factor (VIF) statistics; the presence of multicollinearity is detected by VIF values above five. Odd ratios and corresponding 95% confidence intervals were reported. To assess the amount of

variance explained by the predictors in the model, Cox and Snell R^2 and Nagelkerke R^2 values were generated.

Analysis 3: Biopsychosocial Correlates of Subjective Cognitive Decline

To explore biopsychosocial correlates of SCD in adults and older adults with a history of single remote mTBI, univariate and multivariate binomial generalized linear models were conducted. Six biopsychosocial domains were constructed, each of which contained relevant variables from baseline: (1) demographics: age, sex, education, perceived social status; (2) injury-related factors: loss of consciousness, currently experienced symptoms that might be related to a head injury or concussion; (3) medical factors: comorbid chronic conditions, vision, hearing; health-related behaviors: alcohol use, smoking habits, physical activity; (4) cognitive: performance on the Rey Auditory Verbal Learning Test (RAVLT); (5) psychological: depression; and (6) social factors: personality traits, social support availability, social participation predicting SCD at follow-up 1. Endorsement of SCD was coded as “1” and no endorsement of SCD was coded as “0”. Categorical variables such as sex (reference level = “female”), education (reference level = “College level”), loss of consciousness (reference level = “<1 minute”), alcohol use (reference level = “regularly”), smoking habits (reference level = “non-smoker”), and social participation (reference level = “moderate”) were indicator-coded. Continuous variables such as age, perceived social status, comorbid chronic conditions, vision, hearing, RAVLT performance, depression, personality traits, and social support availability were mean-centered.

Based on the exploratory nature of this work, an omnibus multivariate regression model was conducted, to assess the relationships between predictors and SCD while accounting for the other predictors. Variables were entered into the multivariate model simultaneously. Multicollinearity was assessed using variance inflation factor (VIF) statistics; the presence of multicollinearity is detected by VIF values above five. Odd ratios and corresponding 95% confidence intervals were reported. To assess the amount of variance explained by the predictors in the model, Cox and Snell R^2 and Nagelkerke R^2 values were generated.

Chapter 3. Results

3.1 Demographics and Clinical Characteristics

Table 3.1 presents participant characteristics of the mTBI group and no head injury controls. Of the 30,097 participants in the Comprehensive Cohort, 20,107 participants were included in the no head injury control group (i.e., no positive screen for TBI) and 1,451 participants were included in the single mTBI group. Comparing both groups, the mTBI group was statistically younger [$t(1679.9) = -3.23$, $p = .001$], had a larger proportion of males relative to females [$\chi 2\ (1) = 56.16$, $p < .001$], had higher self-rated general health [$t(1653.8) = 3.22$, $p = .001$], and endorsed greater levels of depressive symptomatology [$t(1639) = 2.19$, $p = .028$], relative to the no head injury control group. However, these group differences of statistical significance might be due to large sample sizes, given that the effect size for these group differences was small (Cohen's d = 0.06-0.09). Both groups reported similar levels of perceived social status [$t(1628.2) = 1.30$, $p = .194$] and levels of education [$\chi 2\ (4) = 6.42$, $p = .170$].

Table 3.1

Participant Characteristics

	mTBI Group n = 1451	**No Head Injury Controls n = 20107**
Age (M, SD)	62.11 (9.88)	62.98 (10.18)
Sex (n, %)		
Female	642 (44.25)	10946 (54.44)
Male	809 (55.75)	9161 (45.56)
Education (n, %)		
<High School	45 (3.56)	733 (4.17)
High School	125 (9.88)	1900 (10.81)
College Degree	407 (32.17)	5695 (32.40)
University Degree	333 (26.32)	4822 (27.43)
Graduate Degree	355 (28.06)	4427 (25.19)
Marital status (n, %)		
Single	119 (8.20)	1706 (8.48)
Married	1057 (72.85)	13979 (69.52)
Widowed	103 (7.10)	1873 (9.32)
Divorced	137 (9.44)	2039 (10.14)
Separated	35 (2.41)	506 (2.52)
Perceived social status (M, SD)	6.30 (1.80)	6.23 (1.85)
Self-rated general health (M, SD)	2.30 (0.93)	2.22 (0.90)
Depression rating (M, SD)	5.23 (4.67)	4.95 (4.46)

In characterizing the mTBI group, 55.41% (n = 804) reported experiencing loss of consciousness (LOC) for less than one minute at the time of the injury and 44.59% (n = 647) participants experienced LOC between 1-20 minutes. It is important to note that CLSA allowed participants to indicate multiple selections for the duration of LOC. Ten participants selected both LOC of less than one minute and between 1-20 minutes. 84.01% (n = 1,219) of the mTBI group reported no currently experienced symptoms. Of those (n = 230) who reported current symptoms, 4.55% reported memory issues (n =

66), 4.62% reported headaches (n = 67), 2.14% reported dizziness (n = 31), 2.14% reported balance issues (n = 31), 3.03% reported ear issues (n = 44), 1.45% reported irritability (n = 21), 2.69% reported sleep issues (n = 39), and 4.82% reported other issues (n = 70). Full details on clinical characteristics of the mTBI group, including cause of head injury and type of medical care received for the head injury, are depicted in Table 3.2.

Table 3.2

Clinical Characteristics of mTBI Group

	mTBI Group n = 1451
Mechanism of head injury (n, %)	
Vehicular crash	299 (20.61)
Fall	512 (35.29)
Sports-related activity	488 (33.63)
Other	171 (11.78)
Duration of loss of consciousness (n, %)	
<1 minute	804 (55.41)
1-20 minutes	647 (44.59)
Total number of symptoms currently endorsed that can be attributed to the remote mTBI (n, %)	
Zero	1323 (91.18)
Greater than or equal to one	128 (8.82)
Type of medical care received for head injury (n, %)	
Physician assessment/visit	469 (32.32)
Emergency department visit	700 (48.24)
Hospitalization	330 (22.74)
None	457 (31.50)
Do not know	14 (0.96)

3.2 Neuropsychological Test Performance

Although beyond the scope of the thesis, for completeness, Table 3.3 provides raw neuropsychological data across group membership (i.e., mTBI vs. no head injury controls), endorsement of SCD, and timepoint (i.e., baseline vs. follow-up 1). In relation to group differences, linear-mixed models showed that both groups performed similarly on the following neuropsychological tests: Controlled Oral Word Association Test [β = 1.03, *SE* = 0.67, *t*(16013) = 1.55, *p* = .243], Mental Alternation Test [β = 0.41, *SE* = 0.42, *t*(18203) = 0.97, *p* = .533], Victoria Stroop Test [β = 0.008, *SE* = 0.02, *t*(21251) = 0.56, *p* = .769], and Rey Auditory Verbal Learning Test [β = 0.33, *SE* = 0.22, *t*(18719) = 1.51, *p* = .262]. Performance on the Animal Fluency Test was better in the mTBI group relative to no head injury controls [β = 1.56, *SE* = 0.62, *t*(18329) = 2.52, *p* = .031].

In respect to time point differences, performance across groups was better at follow-up 1 on the Controlled Oral Word Association Test [β = -0.82, *SE* = 0.11, *t*(12993) = -7.36, *p* < .001] and the Rey Auditory Verbal Learning Test [β = -1.40, *SE* = 0.05, *t*(12993) = -28.95, *p* < .001]. In contrast, performance was better at baseline on the Mental Alternation Test [β = 0.38, *SE* = 0.09, *t*(12993) = 4.17, *p* < .001]. There was no change in performance on the Animal Fluency Test [β = 0.26, *SE* = 0.13, *t*(12993) = 1.94, *p* = .105] and Victoria Stroop Test [β = 0.02, *SE* = 0.01, *t*(12993) = 2.10, *p* = .145] at both timepoints.

In terms of SCD, participants who did not endorse SCD performed better at follow-up 1 on the Animal Fluency Test [β = -0.64, *SE* = 0.21, *t*(18329) = -3.05, *p* = .009], Mental Alternation Test [β = -0.41, *SE* = 0.14, *t*(18203) = -2.89, *p* = .008], and Rey Auditory Verbal Learning Test [β = -0.26, *SE* = 0.07, *t*(18719) = -3.56, *p* = .001]. In contrast, participants who endorsed SCD performed better at follow-up 1 on the Controlled Oral Word Association Test [β = 0.95, *SE* = 0.23, *t*(16013) = 4.19, *p* < .001]. There was no difference in performance across endorsement of SCD on the Victoria Stroop Test at follow-up 1 [β = 0.01, *SE* = 0.01, *t*(21251) = 1.01, *p* = .624]. Full results with unadjusted and adjusted p-values are provided in Table A.1.

Table 3.3

Neuropsychological Test Performance Across Group, Subjective Cognitive Decline, and Timepoint

	mTBI Group				No Head Injury Controls			
	Baseline		Follow-Up 1		Baseline		Follow-Up 1	
	Yes SCD	No SCD	Yes SCD	No SCD	Yes SCD	No SCD	Yes SCD	No SCD
Animal Fluency Test	44.17 (11.65)	44.14 (11.06)	43.41 (10.97)	44.00 (10.23)	42.38 (11.67)	42.70 (11.87)	41.81 (11.17)	42.45 (10.92)
Controlled Oral Word Association Test	41.54 (12.24)	41.00 (12.08)	42.05 (11.80)	41.98 (11.92)	41.11 (12.49)	40.13 (12.49)	41.89 (12.17)	40.94 (12.32)
Mental Alternation Test (/51)	28.22 (8.03)	28.17 (7.52)	27.24 (7.44)	27.82 (6.83)	27.73 (8.19)	27.78 (8.18)	27.00 (7.45)	27.41 (7.28)
Stroop Task	2.15 (0.86)	2.10 (0.48)	2.11 (0.59)	2.09 (0.52)	2.11 (0.62)	2.11 (0.65)	2.11 (0.79)	2.09 (0.63)
Rey Auditory Verbal Learning Test Total (/30)	10.28 (3.81)	10.70 (3.78)	11.60 (4.58)	12.25 (4.41)	10.33 (3.73)	10.53 (3.59)	11.66 (4.37)	11.93 (4.13)

Note. Means (SD) are denoted for raw neuropsychological test data.

3.3 Analysis 1A: Prevalence of Subjective Cognitive Decline

59.13% of participants with a self-reported history of single remote mTBI endorsed prospective SCD, and 56.78% of participants with no head injury endorsed prospective SCD. A Pearson's Chi-Squared test showed that the prevalence of SCD was comparable across groups [$\chi 2$ (1) = 3.52, p = .061]. Given that subjective changes in cognition have been found to be greater in individuals with multiple chronic conditions, and the fact that cardiovascular conditions and diabetes are established risk factors for cognitive decline, an additional control analysis of the prevalence of SCD in participants

with these chronic conditions was conducted. In the current sample, out of 13,262 participants with a history of a chronic condition (i.e., Cardiac/Cardiovascular condition, Diabetes, and/or Hypertension), 58.28% endorsed prospective SCD. A Pearson's Chi-Squared test showed that the prevalence of SCD was equivalent across the mTBI and chronic conditions groups [$\chi 2$ (1) = 0.56, p = .454].

3.4 Analysis 1B: Influence of Injury-Related Measures on Prevalence of Subjective Cognitive Decline

To evaluate whether SCD varied as a function of injury-related factors in the mTBI group, a Pearson's Chi-Squared test showed that the prevalence of SCD did not differ as a function of LOC [$\chi 2$ (1) = 2.40, p = .121] or number of currently experienced symptoms that might be related to a possible head injury/concussion [$\chi 2$ (1) = 0.73, p = .392]. Full details on injury-related measures across endorsement of SCD are presented in Table 3.4.

Table 3.4

Injury-Related Measures Across Subjective Cognitive Decline in mTBI Group

	Presence of Subjective Cognitive Decline	
	Yes SCD n = 861	**No SCD n = 590**
Duration of loss of consciousness (n, %)		
<1 minute	492 (33.91)	312 (21.50)
1-20 minutes	369 (25.43)	278 (19.16)
Total number of symptoms currently endorsed that are related to the head injury (n, %)		
Zero	780 (53.76)	543 (37.42)
Greater than or equal to one	81 (5.58)	47 (3.24)

3.5 Analysis 2A: Association Between Subjective Cognitive Decline and Objective Measures of Cognitive Performance

In assessing the association between subjective and objective measures of cognition, a linear model showed that endorsement of SCD at follow-up 1 was

significantly weakly related to global objective cognitive performance at follow-up 1, after controlling for global objective cognitive performance at baseline (β = -0.02, *SE* = 0.01, *t*(12496) = -2.09, *p* = .037, squared semi-partial correlation = 0.02%). Moreover, global objective cognitive performance at follow-up 1 did not differ across mTBI and no head injury control (β = 0.04, *SE* = 0.03, *t*(12496) = 1.12, *p* = .264). The two-way interaction between group and SCD was not significant (β = -0.02, *SE* = 0.04, *t*(12496) = -0.57, *p* = .567). Full results are presented in Table A.2.

Given that the SCD question in CLSA specifically referenced *memory* (i.e., Do you feel like your memory is becoming worse?) and the delayed RAVLT assesses episodic memory retrieval, a post-hoc exploratory analysis was conducted to examine whether endorsement of SCD was related to performance on delayed RAVLT trial at follow-up 1, controlling for performance on the delayed RAVLT trial at baseline. Interestingly, there was a statistically significant difference on the delayed RAVLT for participants who did not endorse SCD compared to those who endorsed SCD (β = -0.04, *SE* = 0.02, *t*(12496) = -2.44, *p* = .015, squared semi-partial correlation = 0.04%), with those endorsing SCD remembering a fewer number of words ($M_{\text{yes SCD}}$ = 4.87, $SD_{\text{yes SCD}}$ = 2.44; $M_{\text{no SCD}}$ = 5.07, $SD_{\text{no SCD}}$ = 2.32). However, the effect size was 0.04%, which is small (Selya et al., 2012). Full results are presented in Table A.3.

3.6 Analysis 2B: Role of Psychological Distress and Objective Cognitive Performance in Explaining Subjective Cognitive Decline

Results from the binomial logistic regression with SCD as the outcome variable are presented in Table A.4 and A.5. In support of our hypothesis, higher levels of psychological distress at follow-up 1 significantly predicted SCD by 52% (β = 0.42, *SE* = 0.02, z = 17.40, *p* < .001; OR = 1.52; CI: 0.37-0.47), whereas change in global objective cognitive performance across timepoints did not predict SCD (β = 0.06, *SE* = 0.03, z = 1.82, *p* = .069), after controlling for age, sex, education, perceived social status, and health conditions that affect brain health (i.e., hypertension, diabetes, cardiovascular diseases). Contrary to our prediction, the relationship between psychological distress and SCD was not stronger in the mTBI group, relative to no head injury controls (group X psychological distress interaction: β = 0.13, *SE* = 0.10, z = 1.36, *p* = .174) (Table A.4). To further explore the role of psychological distress in SCD above and beyond objective

measures of cognition, an identical analysis using objective cognitive performance at baseline as the cognitive predictor was conducted. This model similarly revealed that higher levels of psychological distress at follow-up 1 significantly predicted SCD by 53% (β = 0.42, *SE* = 0.02, z = 17.48, *p* < .001, OR = 1.53, CI: 0.38-0.47), but that higher baseline objective cognitive performance was also associated with SCD by 2% (β = 0.05, *SE* = 0.02, z = 2.12, OR = 1.02, CI: -0.02-0.06, *p* = .034) (Table A.5). Both models accounted for 12% to 16% of the variance (Cox and Snell R^2 = 0.12; Nagelkerke R^2 = 0.16). All VIF values for predictors in both models were below five, suggesting that there are no issues of multicollinearity.

3.7 Analysis 3: Biopsychosocial Correlates of Subjective Cognitive Decline in Older Adults With Single Remote History of mTBI

Table 3.5 presents descriptive statistics on biopsychosocial variables for the mTBI group across endorsement of SCD.

Table 3.5

mTBI Participant Characteristics as a Function of Subjective Cognitive Decline

	mTBI group (n = 1451)	
Variables	**Yes SCD (n = 861)**	**No SCD (n = 590)**
Age (M, SD)	62.56 (9.98)	61.46 (9.72)
Sex (n, %)		
Female	389 (26.81)	253 (17.44)
Male	472 (32.53)	337 (23.23)
Education (n, %)		
<High school	23 (1.59)	22 (1.52)
High school	70 (4.82)	55 (3.79)
College degree	241 (16.61)	166 (11.44)
University degree	192 (13.23)	142 (9.72)
Graduate degree	219 (15.09)	136 (9.37)
Perceived social status (M, SD)	6.25 (1.76)	6.36 (1.85)
Duration of loss of consciousness (n, %)		
<1 min	492 (33.91)	312 (21.50)
1-20 mins	369 (25.43)	278 (19.16)

Total number of symptoms currently endorsed related to the head injury (n, %)		
Zero	780 (53.76)	543 (37.42)
One	45 (3.10)	29 (2.00)
Two	16 (1.10)	14 (0.96)
Greater than two	20 (1.38)	4 (0.28)
Depression rating (M, SD)	5.67 (4.80)	4.58 (4.41)
Baseline RAVLT performance (M, SD)	10.09 (3.80)	10.21 (3.68)
Hearing rating (M, SD)	2.49 (0.96)	2.25 (0.99)
Vision rating (M, SD)	2.21 (0.91)	2.11 (0.87)
Number of chronic conditions (n, %)		
Zero	436 (30.05)	318 (21.92)
One	272 (18.75)	172 (11.85)
Two	124 (8.55)	85 (5.86)
Three	29 (2.00)	15 (1.03)
Personality traits (M, SD)		
Conscientiousness	6.07 (1.16)	6.35 (0.94)
Emotional stability	5.74 (1.41)	6.02 (1.25)
Openness to experience	5.43 (1.32)	5.57 (1.33)
Social support availability (M, SD)		
Affection	85.93 (19.89)	87.13 (19.48)
Emotional & informational	79.98 (18.68)	81.48 (18.79)
Positive social interaction	81.72 (19.37)	83.56 (18.60)
Tangible	82.00 (19.57)	82.37 (20.46)
Social participation (n, %)		
Infrequent/No participation	133 (9.17)	95 (6.55)
Moderate	589 (41.21)	404 (27.77)
Frequently	133 (9.17)	95 (6.55)
Alcohol frequency (n, %)		
Non-drinker	15 (1.03)	10 (0.69)
12-month abstainer	155 (10.68)	133 (9.17)
Occasionally	142 (9.79)	99 (6.82)
Regularly	549 (37.84)	347 (23.98)
Smoking status (n, %)		
Non-smoker	368 (25.36)	295 (20.33)
Former	419 (28.88)	250 (17.23)
Occasionally	15 (1.03)	12 (0.83)
Regularly	55 (3.79)	32 (2.21)
Physical activity PASE (M, SD)	148.38 (72.39)	147.56 (76.62)

Note. Social participation was indicator-coded, with “moderate participation” as the reference level (“infrequent/no participation” = never, at least once/year, or at least once/month, “moderate participation” = at least once/week, and “frequent participation” = at least once/day). Smoking status was indicator-coded, with “non-smokers” (i.e., never

smoked) as the reference level ("never smoked" = ≥100 cigarettes), "former smokers" = smoked ≥100 cigarettes in their lifetime but have not smoked in the past month, and "current smokers" = smoked ≥100 cigarettes in their lifetime and smoked in the past month. Alcohol frequency was indicator-coded, with "regular drinker" as the reference level ("non-drinker" = never drank alcohol and did not drink in the last 12 months, "former/12-month abstainer" = drank alcohol in the past but not in the last 12 months, or drank alcohol in the past but had less than one drink per month in the last 12 months, "occasional drinker" = drank alcohol in the past and had at least one drink per month in the last 12 months, and "regular drinker" = drank alcohol in the past and had at least one drink per week in the last 12 months. Physical activity was quantified as the summed score of the frequency of activity (hours/day) multiplied by intensity level of the activity.

3.7.1 Univariate Analyses

Demographic Factors. There was an association between age (β = 0.11, *SE* = 0.05, z = 2.06, OR = 1.12, CI: 1.01-1.24, *p* = .039) and SCD, such that older age resulted in greater likelihood of SCD by 12%. There were no associations between sex (β = -0.09, *SE* = 0.11, z = -0.87, *p* = .387), level of education obtained (< High School vs. College Degree: β = -0.33, *SE* = 0.31, z = -1.04, *p* = .297; High School vs. College Degree: β = -0.13, *SE* = 0.21, z = -0.64, *p* = .524; University Degree vs. College Degree: β = 0.10, *SE* = 0.15, z = 0.70, *p* = .486; Graduate Degree vs. College Degree: β = -0.06, *SE* = 0.15, z = -0.43, *p* = .669), or perceived level of social status (β = -0.06, *SE* = 0.05, z = -1.08, *p* = .278) at baseline and prospective SCD at follow-up 1.

Injury-Related Factors. Endorsing zero or more than one symptom attributed to an mTBI did not change the likelihood of SCD (β = -0.18, *SE* = 0.19, z = -0.95, *p* = .342). There was no association between duration of loss of consciousness and prospective SCD (β = 0.17, *SE* = 0.11, z = 1.60, *p* = .109).

Medical Conditions. Worse self-reported hearing problems increased the likelihood of SCD by 28% (β = 0.25, *SE* = 0.05, z = 4.52, *p* < .001; OR = 1.28, CI: 1.15-1.43). Moreover, worse self-reported vision problems increased the likelihood of SCD by 13% (β = 0.12, *SE* = 0.05, z = 2.20, OR = 1.13, CI: 1.01-1.25, *p* = .028). There was no association between the number of chronic conditions (β = 0.07, *SE* = 0.06, z = 1.11, *p* = .268) and prospective SCD.

Health-Related Behaviors. Higher levels of smoking frequency (i.e., being a former smoker vs. non-smoker) increased the likelihood of SCD by 38% (β = 0.32, *SE* = 0.24, z = 1.36, *p* = .008; OR = 1.38, CI: 1.08-1.67), although results were not significant for occasional (β = 0.002, *SE* = 0.40, z = 0.01, *p* = .996) or regular smokers (β = 0.32, *SE* = 0.24, z = 1.36, *p* = .174). Moreover, reduced alcohol use (i.e., 12-month abstainer

from alcohol vs. regular user of alcohol) decreased the likelihood of SCD by 26% (β = -0.30, *SE* = 0.14, z = -2.22, *p* = .027, OR = 0.74, CI: 0.57-0.97), although results were not significant for non-drinkers (B = -0.05, *SE* = 0.41, z = -0.12, *p* = .903) or occasional drinkers (B = -0.10, *SE* = 0.15, z = -0.64, *p* = .519).

Cognitive Factors. Cognitive performance on the RAVLT at baseline was not associated with prospective SCD (β = -0.03, *SE* = 0.05, z = -0.60, *p* = .549).

Psychological Factors. Depression and SCD were significantly associated, such that greater levels of depressive symptomatology increased the likelihood of SCD by 28% (β = 0.25, *SE* = 0.06, z = 4.34, *p* < .001; OR = 1.28, CI: 1.15-1.44).

Psychosocial Factors. With respect to personality traits, higher levels of conscientiousness (β = -0.27, *SE* = 0.06, z = -4.68, *p* < .001; OR = 0.76, CI: 0.68-0.85), emotional stability (β = -0.22, *SE* = 0.06, z = -3.81, *p* <.001; OR = 0.81, CI: 0.72-0.90), and openness to experience (β = -0.11, *SE* = 0.06, z = -2.04, OR = 0.89, CI: 0.80-0.99, *p* = .041) were associated with a decreased likelihood of SCD by 24%, 19%, and 11%, respectively. There was no association between other psychosocial measures, such as level of perceived social support across multiple domains (Affection: β = -0.06, *SE* = 0.05, z = -1.13, *p* = .258; Emotional and Informational: β = -0.08, *SE* = 0.05, z = -1.49, *p* = .135; Positive: β = -0.10, *SE* = 0.05, z = -1.81, *p* = .071; Tangible: β = -0.02, *SE* = 0.05, z = -0.35, *p* = .728), and level of social participation (Infrequent/No Participation vs. Moderate Participation: β = -0.05, *SE* = 0.15, z = -0.32, *p* = .746; Frequent Participation vs. Moderate Participation: β = -0.06, *SE* = 0.15, z = -0.39, *p* = .696) and SCD.

Given the large number of univariate statistical tests performed, it is important to control for Type I error using the Bonferroni correction. In this way, the original statistical alpha level of 0.05 was divided by 22 (number of predictors), therefore establishing *p* = .002 as the threshold for statistical significance for this set of univariate analyses. With this approach, only greater depressive symptomatology, lower levels of conscientiousness, lower levels of emotional stability, and worse self-reported hearing problems remained significant univariate predictors of SCD. Of note, rather than solely relying on the statistical significance, odds ratios (i.e., the relative odds of the outcome of interest in the presence of the explanatory variable) were used to contextualize the significance of the results above. The odd ratios for the significant predictors ranged from 0.76 to 1.34, suggesting that the influence of these variables in altering the odds of endorsing SCD were relatively small or weak (Chen et al., 2010). Full results for univariate analyses are presented in Table A.6.

3.7.2 Multivariate Analysis

A multivariate binomial logistic regression model including all variables and relevant covariates that impact cognition showed that only sex, level of depressive symptomatology, level of openness to experience, level of conscientiousness, and self-reported hearing problems predicted prospective SCD. More specifically, relative to males, females showed a greater likelihood of SCD by 26% (β = -0.30, *SE* = 0.14, z = -2.11, *p* = .035, OR = 0.74, CI: 0.56-0.98). Depressive symptomatology was associated with SCD (β = 0.19, *SE* = 0.08, z = 2.34, *p* = .019, OR = 1.21, CI: 1.03-1.42), with an increase in depressive symptoms associated with 21% greater odds of SCD. In terms of personality traits, lower levels of openness to experience (β = -0.17, *SE* = 0.07, z = -2.42, *p* = .015, OR = 0.85, CI: 0.74-0.97) increased the likelihood of SCD by 15%, and lower levels of conscientiousness (β = -0.22, *SE* = 0.07, z = -3.05, *p* = .002, OR = 0.80, CI: 0.69-0.92) increased the likelihood of SCD by 20%. Worse self-reported hearing problems were associated with an increased likelihood of SCD by 26% (β = 0.23, *SE* = 0.07, z = 3.48, *p* = .001; OR = 1.26, CI: 1.11-1.44). The model accounted for 36% to 44% of the variance (Cox and Snell R^2 = 0.36; Nagelkerke R^2 = 0.44). All VIF values for predictors in the model were below five, suggesting that there are no issues of multicollinearity. Full results are presented in Table A.7.

Chapter 4. Discussion

This thesis aimed to (1A) determine the prevalence of SCD in adults and older adults with a self-reported single, remote history of mTBI, (1B) evaluate whether injury-related factors, such as duration of LOC and number of current concussion symptoms endorsed, influence the prevalence of SCD, (2A) examine whether SCD is related to objective cognitive functioning, (2B) determine the role of psychological distress in explaining SCD, relative to objective measures of change in cognitive performance, as well as whether this relationship is stronger in mTBI participants compared to no head injury controls, and (3) explore the biopsychosocial correlates of SCD in adults and older adults with a single, remote history of mTBI.

4.1 High Prevalence of Subjective Cognitive Decline in Both mTBI and No Head Injury Controls

The prevalence of SCD was high in the mTBI group, with 59.13% of participants endorsing SCD at follow-up 1. Prior work has primarily focused on subjective cognitive complaints in the context of a *recent* mTBI, with studies indicating that 39-68% of mTBI patients who presented to a trauma center or concussion specialty clinic reported high levels of cognitive complaints six months after their injury (Ngwenya et al., 2018; Stulemeijer et al., 2007). To our knowledge, this is the first study investigating the prevalence of SCD in a nationally-representative sample of adults and older adults with a single *remote* mTBI at least 12 months prior to recruitment. As such, the high prevalence of SCD in the mTBI group in the CLSA dataset might reflect an age-related increase in SCD rather than an observation that is specific to mTBI, especially given that a comparable percentage of no head injury control participants (56.78%) also endorsed SCD. Our findings are in accordance with the prevalence of SCD in the entire CLSA sample, which was reported to be 58% (Hopper et al., 2023). Rates of SCD in this sample appear to be within the range of those reported in other studies with community-dwelling older adults, which is between 50-80% (Balash et al., 2013; Holmen et al., 2013). Although direct comparisons are difficult because of differing SCD measures, our prevalence rates are consistent with another large-scale study showing that 65% of older participants endorsed memory complaints to a similar SCD question (i.e., “have you noticed difficulties with your memory?”) (Slavin et al., 2010) and other work from

cognitively healthy samples enrolled in studies of cognitive decline (Ahmed et al., 2008; Lam et al., 2005). Our findings support previous evidence that SCD is a common experience in aging individuals and that adults with a single, remote history of mTBI are not at greater risk for SCD. Of note, certain mTBI groups might show an increased risk of SCD compared to individuals with no history of brain injury, such as veterans with a blast-related mTBI or those who were exposed to psychological trauma (e.g., PTSD). Previous studies in veteran populations showed that the TBI+ group (i.e., reported an mTBI on average 41 months ago) experienced more subjective cognitive problems over time (i.e., concentration, memory, decision-making, slowed thinking) compared to the TBI- group, although objective cognitive performance was not considerably impaired in the TBI+ group (Donnelly et al., 2017; Spencer et al., 2010). Similarly, a vast majority of veterans with a history of blast-related mTBI (82%) reported greater difficulty with executive functioning (e.g., planning, cognitive flexibility, working memory) post-injury, compared to their abilities pre-injury (Karr et al., 2019), which suggests that veteran populations with a remote history of mTBI might be a qualitatively distinct group where SCD is a greater area of concern.

Interestingly, the self-reported duration of LOC at the time of the injury, as well as the number of symptoms currently experienced that participants attributed to their mTBI, did not influence the endorsement of SCD. Our findings support prior evidence showing that measures of mTBI severity, such as duration of LOC, do not predict subjective changes in cognition (Stillman et al., 2019; Stulemeijer et al., 2007) nor alter objective cognitive impairment or long-term neurocognitive outcomes post-TBI (Lovell et al., 1999; Tripodis et al., 2017). With respect to the number of symptoms currently experienced by mTBI participants, it is important to note that the majority of participants (84.01%) declined experiencing any current symptoms related to their remote head injury. Those who attributed symptoms to their remote mTBI most frequently endorsed headache (4.62%) and memory (4.55%) symptoms (4.62%). The low prevalence of current concussion symptoms endorsed could suggest that the mTBI group was mostly recovered from their remote injury. Moreover, these symptoms are non-specific and are commonly seen in healthy and clinical populations (Donnell et al., 2012; Iverson & Lange, 2010; Smith-Seemiller et al., 2003). If participants were experiencing these symptoms, they did not attribute them to their remote mTBI.

Taken together, the results indicate that a remote mild concussive injury does not increase endorsement of SCD. The rate of SCD was consistent with that of an aging

population who did not sustain a remote head injury. This suggests that a single remote history of mTBI may not be the most sensitive and critical factor when conceptualizing prospective SCD. Importantly, the majority of individuals with a single, remote mTBI in this study reported no currently endorsed symptoms that they would attribute to the mTBI, suggesting that they were recovered. This could also imply that the remote mild head injury is not as central to older adults' conceptualization of identity and physical and cognitive change. Due to methodological variability across studies (e.g., diagnostic criteria; heterogeneity of clinical samples; sensitive and specific biomarkers of neurodegeneration; injury characterization), research on the impact of a single mTBI on subsequent neurodegeneration is mixed (Brett et al., 2022). Some studies have shown that a single remote history of mTBI increases the likelihood of a subsequent dementia diagnosis, which can be driven by long-term neurostructural changes and compromised brain integrity (Rajesh et al., 2017; Snowden et al., 2020). In contrast, research has also suggested that moderate/severe TBI exposure and/or repetitive TBI are risk factors for neurodegenerative disease, rather than a single, uncomplicated mTBI (Brett et al., 2022; LoBue et al., 2019).

4.2 Association Between Psychological Distress and Subjective Cognitive Decline Above and Beyond Objective Cognitive Performance in Older Adults with mTBI

Results showed that the endorsement of SCD at follow-up 1 was weakly associated with global cognitive performance at follow-up 1, after controlling for demographic factors and global cognitive performance at baseline, in both the mTBI group and no head injury group. More specifically, participants who endorsed SCD showed better global objective cognitive performance at follow-up 1. Our findings are in accordance with previous literature, which demonstrate a weak relationship between subjective and objective memory performance. Cross-sectional studies have demonstrated that SCD and concurrent objective memory performance are not closely linked, although there is a small but significant correlation between the two measures (Burmester et al., 2016; Zlatar et al., 2018). Rather than reflecting actual memory performance, the endorsement and severity of SCD have been more tightly associated with concurrent depressive and anxious symptomatology (Buckley et al., 2013; Slavin et al., 2010; Zlatar et al., 2018).

Of note, when evaluating the association between SCD and concurrent delayed recall performance on the RAVLT, participants who did not endorse SCD remembered a slightly greater number of words on the delayed RAVLT trial at follow-up 1, after controlling for delayed RAVLT performance at baseline. We note that the slight increase in words recalled in participants without SCD (i.e., consistency between subjective and objective measures of cognitive functioning) compared to those with SCD might be due to time-saving-modifications for the RAVLT in the CLSA administration (i.e., 1-trial vs. 5-trials; 5-minute delay vs. 30-minute delay), and it is likely that this benefit will be stronger with more standard RAVLT administrations. List learning tests are sensitive to changes in episodic memory in mild cognitive impairment and Alzheimer's disease (Rabin et al., 2009). Specifically, delayed recall tests capture episodic retrieval, which is highly-dependent on hippocampal systems that are known to be affected in cognitive decline (Esteves-Gonzalez et al., 2003; Shankle et al., 2005). Given that the SCD measure in CLSA specified "do you feel like your *memory* is becoming worse?", our finding suggests that list learning episodic memory tests might be an ecologically-valid predictor of real-world memory changes and forgetfulness that are experienced in older age (Buckley et al., 2015; Corner & Bond, 2004), and that endorsement of SCD may indeed indicate age-related episodic memory changes that are pronounced in neurodegeneration.

Furthermore, currently experienced psychological distress was associated with SCD, whereas change in objective cognitive performance was not. Previous studies have shown that SCD was more closely related to current levels of depression and anxiety than levels of objective memory decline (Gustavson et al., 2021; Markova et al., 2017). One study found that change in objective memory retention scores did not predict subjective memory ratings, and that psychological distress was predictive of subjective memory complaint above and beyond longitudinal change on cognitive measures or current objective performance (Smith et al., 1996). In the present work, we had access to an extensive, nationally-representative dataset with clinically-relevant, performance-based measures of cognitive functioning measured at baseline and follow-up 1 timepoints (Tuokko et al., 2017). These results suggest that the endorsement of SCD may not always be consistent with measurable cognitive change between original and follow-up evaluations, but that the reporting of perceived memory problems may instead be driven by negative emotional states, such as depression and anxiety. There is an established bi-directional relationship between psychological distress and SCD, such that emotional problems can increase one's worry of changes in cognition; alternatively,

perceived changes in cognition can be worrisome and increase psychological distress (Liew, 2020; Podlesek et al., 2021). More broadly, emotion and cognition are interconnected in that psychological distress (e.g., anxiety) can alter attentional processing, which increases one's hyper fixation on threat and biases the perception of stimuli (e.g., memory changes) (Okon-Singer et al., 2015). Concurrently, cognition (e.g., real-world memory difficulties) can also influence the reappraisal and regulation of difficult emotions, resulting in elevated distress (Paradise et al., 2011; Podlesek et al., 2021). It is important to note that when examining the association between psychological distress and baseline objective cognitive performance on SCD, results showed that higher baseline objective cognitive performance increased the likelihood of SCD by 2%. This reflects the fact that SCD may occur without objective evidence of worse cognitive performance, although interpretation should be cautioned given the small effect size of 2%, in comparison to the fact that psychological distress was positively associated with SCD by 53%. Moreover, as expected, we found that sociodemographic factors such as older age, being female, and having a lower perceived social status significantly predicted SCD (Cedres et al., 2018; Giacomucci et al., 2022; Gupta, 2021).

These results are consistent with research showing that psychological distress was associated with the subjective experience of cognitive decline in adults, athletes, and combat-exposed mTBI groups (Chamelian et al. 2006; French et al., 2014; Gass et al. 1997; Karr et al. 2019; Satz et al., 1998; Stillman et al., 2019; Stulemeijer et al., 2007). To our knowledge, this is the first investigation of these relationships in older adults with a history of mTBI. Several studies in mTBI veteran populations have demonstrated small associations between subjective cognition and objective cognitive performance. In examining the incongruence, and more specifically, the factors that explain SCD post-mTBI, one study found that psychological distress, captured by depression, anxiety, and PTSD measures, was elevated and strongly related to subjective cognitive problems (Donnelly et al., 2018). Similarly, self-reported cognitive complaints in past military members with mild-severe TBI were associated with psychological distress, as opposed to overall neurocognitive functioning (French et al., 2014). Furthermore, another study reported that change in subjective cognitive symptom severity was not related to change in cognitive test performance, but rather, was associated with change in self-reported depressive and anxiety-related symptoms over a 3-month period after mTBI (Stenberg et al., 2020).

The relationship between psychological distress and SCD was comparable in both the mTBI and no head injury control groups. Interestingly, a recent study found that higher levels of psychological distress were more strongly associated with greater levels of cognitive complaint in an adult mTBI group in the post-acute period of recovery (i.e., average two months since injury), when compared to a no brain injury control group (Anderson, 2021). It is likely that psychological distress may explain SCD to a stronger extent in individuals who are recovering from a more recent injury (i.e., post-acute period), due to time-limited alterations in the influence of affective state on subjective cognitive factors, which may fade as the time-from-injury increases.

4.3 Biopsychosocial Correlates of Subjective Cognitive Decline in Older Adults with mTBI

Here, we explored biopsychosocial correlates of prospective SCD in a sample of participants who self-reported a single, remote mTBI more than 12 months ago, by assessing the relationship between *baseline* demographic, injury-related factors, medical factors, health-related behaviors, cognitive performance, psychological factors, and social factors and SCD at *follow-up* three years later. At the univariate level, after controlling for multiple comparisons, greater depressive symptomatology, lower levels of conscientiousness, lower levels of emotional stability, and worse self-reported hearing problems increased the likelihood of SCD. In the multivariate logistic regression model with all predictors entered simultaneously, only being female**,** greater depressive symptomatology, lower levels of conscientiousness, lower levels of openness to experience, and worse self-reported hearing problems emerged as significant predictors of SCD.

Broadly, this exploratory work provides further evidence that psychological and social factors are closely linked to SCD. An extensive body of literature has demonstrated that lower conscientiousness, higher neuroticism (i.e., low emotional stability), and lower openness to experience are associated with subjective memory complaints (Koller et al., 2019; Slavin et al., 2010; Smit et al., 2021; Studer et al., 2013). It has been posited that high levels of neuroticism may predispose an individual to negative affect and difficulty responding to distress, which in turn may elevate the endorsement of subjective memory complaints (Koller et al., 2019). The degree of organization, dependability, and discipline (i.e., conscientiousness) and creativity and

curiosity (i.e., openness to experience) are negatively related to subjective memory complaints, and stronger alignment with these personality traits can support cognitive function (Luchetti et al., 2016). Furthermore, we found that psychological factors, namely depressive symptomatology, showed the second highest association with SCD. This extends previous cross-sectional findings showing that depressive symptoms are associated with SCD (Balash et al., 2013; Markova et al., 2017; Zlatar et al., 2018), because we showed that higher levels of depression at *baseline* increased the likelihood of SCD *three years later*. One explanation is that SCD can be conceptualized as a psycho-affective problem, such that the inaccurate perception of cognitive problems is driven by worry and depressive tendencies, as opposed to measurable objective cognitive impairment (Hill et al., 2016). Alternatively, SCD can also be conceptualized as an early sign of MCI, in which case a depression diagnosis and/or depressive symptomatology, accompanied by SCD, has been shown to increase risk of cognitive decline and dementia (Mourao et al., 2016; Wang et al., 2021). A recent paper on biopsychosocial predictors of SCD in a nationally-representative general adult population (n = 21,920) reported similar findings, in which individuals with a positive screen for depression, low conscientiousness, high neuroticism, and low openness to experience had an increased risk of SCD (Hopper et al., 2023). Our findings show that these relationships can be extended to adults who have experienced a remote head injury.

We found that select medical factors, specifically worse self-reported hearing problems, increased vulnerability to SCD in the current older mTBI sample. Longitudinal studies have shown that hearing loss is associated with a higher risk of SCD (Curhan et al., 2019; Curhan et al., 2020) and cognitive decline (Lin et al., 2013), with greater severity of hearing loss showing higher incidence of risk for these effects. Age-related changes in hearing have also been shown to increase risk of incident dementia (Deal et al., 2017; Loughrey et al., 2018). The mechanisms underlying the relationship between hearing impairment and cognitive decline is the fact that there is increased cognitive load, progressive damage to cochlear structures which precipitates neural reorganization and atrophy of the temporal brain regions, and hampered levels of social engagement resulting in social isolation (Lin & Albert, 2014). Critically, our results demonstrate the value of treating hearing loss as a modifiable risk factor for SCD (Fortunato et al., 2016; Loughrey et al., 2018), which might help mitigate progression to cognitive decline. These results highlight the need for cognitive interventions to implement screening and delivery of hearing services and assistive services.

Health-related behaviors such as smoking history and frequency of alcohol use did not emerge as significant predictors after controlling for multiple comparisons at the univariate level and were also not associated with SCD at the multivariate level. This suggests that although lower frequency of smoking and reduced alcohol consumption are established protective factors against cognitive decline (Anstey et al., 2007; Kim et al., 2012; Lee et al., 2009), the extent to which these health-related behaviors are related to SCD might not be as critical in the context of other biopsychosocial factors. We also found that physical activity was not associated with SCD. Although physical activity has been shown to be protective against cognitive decline and neurodegeneration due to cardiovascular, neurogenesis, or anti-inflammatory mechanisms (Blondell et al., 2014; Sofi et al., 2010; Valenzuela et al., 2020), the relationship between physical activity and SCD has been understudied and warrants further investigation. Of note, in a large-scale general older adult sample, common risk factors for dementia, such as biological and health-related behaviors (e.g., alcohol use, smoking, hypertension, physical activity) were not predictive of SCD (Hopper et al., 2023). The consistency of our findings with those of the general older adult sample suggests that the impact of psychosocial factors, rather than preventative health measures and lifestyle modifications, on SCD may be extended to older adults with a history of mTBI.

Interestingly, social participation and perceived social support were not associated with SCD. Past literature has shown that poorer social relations, including infrequent or lack of social engagement, integration, and social support, is a risk factor for SCD (Weng et al., 2020; Zullo et al., 2021), as well as cognitive decline and/or dementia (Baumgart et al., 2015; Dickinsin et al., 2011; Holtzman et al., 2004; Kuiper et al., 2015; Pillemer et al., 2016; Zunzunegui et al., 2003). Although outside of the scope of the current thesis, it may be the case that the influence of social participation and perceived social support in mitigating SCD was reduced because it was already accounted for by other strong psychosocial factors included in the analysis, such as depression and personality. Indeed, one mechanism underlying the association between social factors and cognitive functioning is that engagement in social activities and greater social support can increase one’s psychological state and decrease loneliness, which may protect against cognitive decline (Lakey & Orehek, 2011). Within the CLSA dataset, cross-sectional analyses have shown that social support availability was associated with memory performance (Bedard & Taler 2021; Ohman et al., 2022;

Oremus et al., 2020). These results suggest that social factors can buffer against cognitive decline according to the slowing of neurodegeneration (Anatürk et al., 2018), but that these social factors may be less indicative of *subjective* memory changes that may occur prior to, or in the absence of, documented neurodegenerative changes.

The current results contextualize the relevance of multiple biopsychosocial factors on SCD following mTBI. For example, it could be the case that individuals who sustained a single, remote mTBI, and experience various medical and psychological concerns, such as depression and hearing problems, may be more susceptible to future cognitive deterioration. Affective symptoms might contribute to the perception of worsening memory without detectable cognitive impairment on standardized tests (Buckley et al., 2013; Yates et al., 2015). The perception of worsening memory can also be compounded by other age-related medical challenges, such as hearing loss, which can accelerate cognitive decline (Lin et al., 2013). The dynamic relationship between these factors can also be illustrated by the fact that psychological distress (e.g., depression, anxiety) and medical conditions (e.g., hearing loss) might result in withdrawal from positive health behaviors, such as physical and social activity engagement (Lin et al., 2013; Naismith et al., 2009). In sum, this exploratory work presents biopsychosocial correlates that may hold prognostic value for SCD in an older adult sample with a single, remote history of mTBI, which can be targeted in rehabilitative and intervention work to mitigate cognitive decline and promote psychological well-being.

4.4 Limitations & Future Directions

We acknowledge several limitations in the current study. For example, the CLSA utilizes a self-report measure of previous TBIs. As such, participants might have under- or over-estimated the number of previous TBIs they had, as well as the duration of LOC at the time of injury, especially considering the length of time that elapsed since the injury (i.e., one year or more). The inclusion of Glasgow Coma Scale scores, data regarding post-traumatic amnesia, and acute symptoms attributable to the injury as documented close to the time of injury would have allowed for a more comprehensive assessment of mTBI (Silverberg et al., 2023). Additionally, confirmation of a physician-diagnosed mTBI or clinical interview would increase reliability, yet this is not feasible for large-scale studies. Of note, it is unclear the exact date of the remote head injury.

Although we aimed to circumvent this limitation by excluding individuals who experienced a concussion/brain injury, or other injury in the past 12 months, we are unable to make claims regarding the time course of subjective changes in cognition. Future work can examine whether the prevalence of SCD differs across various timepoints of an mTBI recovery course (e.g., acute, post-acute), as well as the features of mTBI recovery that may influence the strength of the association between one's psychological state and subjective evaluation of cognition. For example, although outside the scope of the data collected by the CLSA, further examination of mTBI-relevant variables (e.g., coping styles, sleep disturbance, fatigue, illness perception) that have been shown to be sensitive to the development of persistent cognitive complaints following an mTBI can be informative (Le Sage et al., 2022). Additionally, given that age-related brain changes accompanied by TBI-related neuropathology might advance cognitive deterioration and result in poorer outcomes (Kristman et al., 2016; Thompson et al., 2006), an important line of future investigation would be to evaluate the prevalence and correlates of SCD in older adults who sustained an mTBI during older adulthood, as well as older adults with mTBI who are acutely injured. In these populations, it is likely that the prevalence of SCD would be greater than that of those with a remote injury and no brain injury controls. It would be interesting to examine the trajectory of the association between SCD and objective functioning as a function of time since injury in older adults, such as whether there is a tighter link between SCD and objective functioning in individuals showing evidence of neuropsychological impairment acutely post-mTBI, relative to the association between SCD and psychological distress, which may emerge as subjective complaints persist long after objective cognitive function is recovered.

Similarly, we recognize that CLSA data primarily utilized self-reported measures, such that the reporting of certain factors such as medical conditions (e.g., hearing, vision, chronic conditions) and health-related behaviors (e.g., level of physical activity, alcohol use, smoking frequency) may be biased and less definitive than objective measures. Given that the majority of the sample endorsed SCD, there may be incongruencies between self-report data and objective markers on the aforementioned measures. Importantly, issues of self-presentation behaviors may be evident in the older adult population, such that responses may be biased toward a more positive self-image to distance themselves from stigma and internalized negative views around aging while conveying a sense of physical and psychological health (Martin et al., 2000). This is an

important area of consideration since sociodemographic, psychosocial, lifestyle/behavior, and health measures from the Comprehensive Cohort were obtained via in-person computer-assisted personal interview software, in which questions were read to the participants and responses were recorded by the interviewer accordingly.

It is also likely that the large sample size may have increased the likelihood of statistically significant results that may not bear clinical significance. To mitigate concerns, corrections for Type I error and false discovery rate were implemented as appropriate. However, access to a large-scale, population-based dataset allowed for an exploration of multiple factors concurrently, which could inform the design of focused studies that elaborate upon the current findings. We also acknowledge the low internal consistency on the TIPI, which was used as a measure of personality traits in CLSA. Although the TIPI has psychometric shortcomings due to its brevity and the fact that each subscale is comprised of two items, it has acceptable convergence with other comprehensive and more prominent measures of personality (e.g., 240-item NEO Personality Inventory-Revised; 60-item NEO Five-Factor Inventory; 44-item Big Five Inventory), and the TIPI is encouraged for use in studies that explore personality among several other constructs (Thørrisen & Sadeghi, 2023).

Another limitation in the current study pertains to the CLSA measure of SCD. Brief, dichotomous (yes/no) SCD measures have been shown to be meaningful indicators of increased risk of MCI and dementia (Abdulrab & Heun, 2008; Mitchell et al., 2014; Reid & MacLullich, 2006). Considering the extensive questionnaire data and broader scope of the CLSA study, a single-question measure of SCD is an efficient way to classify individuals who endorse SCD. Nevertheless, more fine-grained measures of SCD, such as scales that assess subjective changes in relation to specific cognitive domains or assess the frequency and/or severity of subjective complaints, may be more sensitive to the weak but significant association between SCD and objective measures of cognition (Burmester et al., 2016; Rabin et al., 2015). Additionally, the SCD measure was non-time-specific (“do you feel like your memory is becoming worse?”), which suggests that participants were likely anchoring their perception of worsening memory to different points in time (e.g., ten years ago; past year). Indeed, atemporal SCD measures may be conflated by older adults’ global beliefs about cognitive aging, which can be mitigated by questions that ask one to recall specific episodes of cognitive decline over a narrow reference period. Given that subjective changes in certain areas of cognition (e.g., getting lost, processing speed) may be more sensitive to objective

cognitive functioning (Burmester et al., 2016), and the fact that a single-question measure of SCD can be confounded by affective factors (e.g., depressive symptomatology), a more comprehensive assessment of SCD targeting *specific* cognitive problems might help disentangle the relationship between SCD and change in objective cognitive performance. Future research can also consider a continuous measure of SCD to not only capture the endorsement of SCD, but also to examine how *the degree of* psychological distress is associated with *the degree of* self-perceived worsening memory. Furthermore, at the time of writing, cognitive data from the CLSA were only available for the baseline and the 3-year follow-up timepoints, which limited our investigation of cognitive change to a three-year window. Although SCD has a weak concordance with objective cognitive functioning, research suggests that SCD precedes and increases the risk of developing cognitive decline and dementia (Pike et al., 2022). Beyond the current analyses of the association between SCD and objective memory performance at a concurrent timepoint, future work could evaluate whether SCD at follow-up 1 predicts cognitive change over subsequent follow-up timepoints.

Lastly, it is important to recognize the limitations on generalizability of the current findings. Given the nature of participation in the Comprehensive Cohort which involves considerable time and effort (e.g., travel to data collection sites, physical assessments), participants were required to be physically and cognitively able to participate. As reported by the CLSA, the Comprehensive Cohort was generally more educated, had higher household income, had a greater percentage of Canadian born participants, and rated their general health more positively than the general Canadian population as referenced in Statistics Canada's Canadian Community Health Survey-Healthy Aging (CCHS-HA) (Raina et al., 2019). Moreover, individuals showing signs of cognitive impairment were screened out prior to data collection. As a result, the implications of our research may be less translatable to individuals who are more cognitively impaired and/or in whom health-related barriers constrain ability to participate in research (e.g., sensory or physical limitations). For example, the association between subjective and objective cognitive functioning may be stronger in a population experiencing greater cognitive impairment, relative to a sample that may resonate with the "worried well" (i.e., concerned about cognitive decline despite being neurologically intact upon examination) yet remain cognitively intact (Burmester et al., 2016; Sutherland et al., 2021). The Comprehensive Cohort comprised individuals who lived 25-50 kilometers from the 11 CLSA data collection sites across seven provinces, and followed a similar exclusion

criterion as CCHS-HA (e.g., excluded residents of the Canadian territories and some remote regions, persons on First Nations reserves and settlements, members of the Canadian Armed Forces, institutionalized persons), which suggests that the results may not be generalizable to the entire Canadian population. Importantly, further work is needed to explore intersectional effects of minority stress (e.g., race, gender, sexual orientation) and sociocultural influences (e.g., individuals with lower levels of literacy; level of acculturation; place of residence) on SCD, objective cognitive functioning, and the role of psychological factors underpinning SCD, which may not be fully captured by the CLSA.

4.5 Implications

SCD is a common experience that increases with age, with studies reporting prevalence rates of between 50-80% in community-dwelling older adults (Desai et al., 2021; Jessen et al., 2020). There is accumulating research suggesting that SCD is a harbinger for pathologic cognitive decline and dementia (Rabin et al., 2017). In many cases, endorsement of SCD may represent the initial symptomatic manifestation of Alzheimer's disease, prior to mild cognitive impairment. Additionally, SCD can adversely impact one's psychological well-being, quality of life, and self-perceived health (Hill et al., 2017; Jenkins et al., 2019). Although most of the research has focused on how moderate-to-severe TBI negatively affects cognition in aging, mTBI accounts for 60-95% of the million TBIs that occur annually. Research suggests that older adults with a remote history of mTBI may have heightened vulnerability to SCD and progressive cognitive impairment (Brown et al., 2011; McInnes et al., 2017; Whiteneck et al., 2016). Furthermore, anxiety and negative emotions could elevate one's perception of worsening cognition, but SCD could also in turn be exacerbated by psychological distress (Hill et al., 2016; Liew, 2020). As such, it is critical to determine the prevalence and determinants of prospective SCD, which can help inform the development of interventions that mitigate cognitive deterioration and alleviate emotional causes underlying SCD in this population.

This work underscores the value of implementing early psychological interventions in both healthy individuals and in various etiologies (e.g., post-mTBI) that target depressive and anxiety symptoms to improve quality of life, which could mitigate the onset of SCD. For instance, evidence-based psychological interventions (e.g.,

cognitive behavioral therapy) can provide a unique opportunity to understand how other issues might be influencing SCD, such as hypervigilance to cognitive changes, negative subjective appraisal of memory, symptom misattribution, illness perception, and coping styles (Byrne et al., 2018; Silver et al., 2009). Additionally, psychoeducation programs can encourage individuals to understand the discrepancy between subjective and objective cognitive changes, as well as their attitudes and perceptions toward healthy aging, which can restructure the fears and insecurities associated with aging to promote cognitive health and well-being. Of note, in cases where SCD may represent a preclinical form of neurodegeneration, cognitive interventions (e.g., cognitive training, cognitive rehabilitation, cognitive stimulation) are effective ways to support the maintenance of cognitive functions, especially when there is consistency between the treatment type and the area of cognitive concern (e.g., memory training to improve objective memory function) (Smart et al., 2017). The implementation of intervention efforts is important for individuals with SCD, given that these individuals may have largely preserved cognitive functioning and access to cognitive reserve (Smart et al., 2017), and the fact that effective interventions can positively influence quality of life.

Lastly, given the multifaceted nature of SCD, there is increasing application of biopsychosocial frameworks to elucidate the importance of biological and physical health and psychosocial factors in cognitive decline (Livingston et al., 2020), as well as multi-dimensional interventions that target several risk factors (Naismith et al., 2009; Plassman et al., 2010). In the case that modifiable risk factors are targeted early, such as prior to the onset of SCD, the progression of neurodegenerative changes might be preventable. Older adults with a remote history of mTBI may be more susceptible to diminished medical, behavioral, and cognitive outcomes due to the exacerbation of age-related memory changes (Anderson et al., 2005; Moretti et al., 2012). Thus, the current work highlights key factors that can be integrated into multi-dimensional rehabilitative and intervention programs to maximize cognitive health in older adults with a history of mTBI.

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Appendix A. Supplementary Tables

Table A.1.

Separate Linear-Mixed Models for Neuropsychological Test Data as a Function of Group Membership, Subjective Cognitive Decline, and Timepoint

Variable	Estimate	SE	DF	T Value	Lower CI	Upper CI	Unadjusted P Value	Adjusted P Value
Animal Fluency Test								
Group	1.56	0.62	18329.47	2.52	0.35	2.77	0.012*	0.031*
SCD	-0.64	0.21	18329.47	-3.05	-1.05	-0.23	0.002**	0.009**
Timepoint	0.26	0.13	12993.00	1.94	-0.003	0.52	0.052	0.105
Group * SCD	0.05	0.80	18329.47	0.06	-1.51	1.61	0.950	0.950
Group * Timepoint	-0.12	0.52	12993.00	-0.23	-1.14	0.90	0.816	0.933
SCD * Timepoint	0.31	0.18	12993.00	1.77	-0.03	0.66	0.077	0.123
Group * SCD * Timepoint	0.31	0.67	12993.00	0.46	-1.01	1.62	0.646	0.861
Controlled Oral Word Association Test								
Group	1.03	0.67	16013.02	1.55	-0.27	2.34	0.121	0.243
SCD	0.95	0.23	16013.02	4.19	0.50	1.39	<.001***	<.001***
Timepoint	-0.82	0.11	12993.00	-7.36	-1.03	-0.60	<.001***	<.001***
Group * SCD	-0.87	0.86	16013.02	-1.01	-2.56	0.81	0.311	0.498
Group * Timepoint	-0.16	0.43	12993.00	-0.37	-1.01	0.69	0.715	0.796
SCD * Timepoint	0.04	0.15	12993.00	0.26	-0.25	0.33	0.796	0.796
Group * SCD * Timepoint	0.42	0.56	12993.00	0.75	-0.67	1.52	0.451	0.601
Mental Alternation Test								
Group	0.41	0.42	18203.34	0.97	-0.42	1.24	0.333	0.533
SCD	-0.41	0.14	18203.34	-2.89	-0.69	-0.13	0.004**	0.008**
Timepoint	0.38	0.09	12993.00	4.17	0.20	0.55	<.001***	<.001***

Group * SCD	-0.16	0.54	18203.34	-0.30	-1.23	0.91	0.767	0.877
Group * Timepoint	-0.03	0.35	12993.00	-0.07	-0.71	0.66	0.942	0.942
SCD * Timepoint	0.36	0.12	12993.00	3.04	0.13	0.59	0.002**	0.006**
Group * SCD * Timepoint	0.26	0.45	12993.00	0.58	-0.62	1.15	0.560	0.747
Stroop Task								
Group	0.001	0.04	22668.22	0.03	-0.07	0.07	0.976	0.977
SCD	0.01	0.012	22668.22	1.10	-0.011	0.04	0.272	0.435
Timepoint	0.02	0.010	12993.00	2.10	0.001	0.04	0.036*	0.145
Group * SCD	-0.001	0.05	22668.22	-0.03	-0.09	0.09	0.977	0.977
Group * Timepoint	-0.02	0.04	12993.00	-0.47	-0.10	0.06	0.642	0.856
SCD * Timepoint	-0.02	0.01	12993.00	-1.42	-0.05	0.007	0.155	0.414
Group * SCD * Timepoint	0.06	0.05	12993.00	1.22	-0.04	0.17	0.221	0.435
Rey Auditory Verbal Learning Test								
Group	0.33	0.22	18719.94	1.51	-0.10	0.75	0.131	0.262
SCD	-0.26	0.07	18719.94	-3.56	-0.41	-0.12	<.001***	0.001**
Timepoint	-1.40	0.05	12993.00	-28.95	-1.49	-1.30	<.001***	<.001***
Group * SCD	-0.39	0.28	18719.94	-1.39	-0.94	0.16	0.164	0.263
Group * Timepoint	-0.15	0.19	12993.00	-0.82	-0.52	0.22	0.415	0.474
SCD * Timepoint	0.06	0.06	12993.00	0.98	-0.06	0.19	0.328	0.437
Group * SCD * Timepoint	0.17	0.24	12993.00	0.69	-0.31	0.64	0.491	0.491

Note. SCD = subjective cognitive decline, SE = standard error, DF = degrees of freedom, CI = confidence interval. * $p < .05$; ** $p < .01$; *** $p < .001$.

Table A.2.

Linear Model for Subjective Cognitive Decline and Global Cognitive Performance at Follow-Up 1

Variables	Estimate	Standard Error	T-Value	Lower CI	Upper CI	P-Value
(Intercept)	1.06	0.04	25.46	0.84	1.02	<.001***
Group	0.04	0.03	1.12	-0.01	0.12	0.264
Subjective cognitive decline	-0.02	0.01	-2.08	-0.03	0.01	0.037*
Baseline cognitive performance	0.71	0.01	120.23	0.68	0.70	<.001***
Age	-0.02	0.001	-28.83	-0.02	-0.01	<.001***
Sex	-0.10	0.01	-9.26	-0.13	-0.08	<.001***
Education (<High School)	-0.03	0.03	-1.08	-0.04	0.08	0.280
Education (High School)	-0.05	0.02	-2.81	-0.08	0.01	0.005**
Education (University)	0.09	0.01	6.75	0.04	0.10	<.001***
Education (Graduate)	0.14	0.01	9.88	0.09	0.15	<.001***
Chronic conditions	-0.02	0.01	-3.72	-0.04	-0.01	<.001***
Perceived social status	0.0086	0.003	2.93	-0.01	0.01	0.003**
Group * Subjective cognitive decline	-0.02	0.04	-0.57	-0.15	0.03	0.567

Note. SE = standard error, CI = confidence interval. ** $p < .01$; *** $p < .001$.

Table A.3.

Linear Model for SCD and Delayed RAVLT Performance at Follow-Up 1

Variables	Estimate	SE	T Value	Lower CI	Upper CI	P Value
(Intercept)	2.11	0.06	34.66	1.99	2.23	<.001***
Group	0.06	0.05	1.27	-0.03	0.15	0.205
Subjective cognitive decline	-0.04	0.02	-2.44	-0.07	-0.01	0.015*
Baseline delayed RAVLT performance	0.52	0.01	62.36	0.50	0.53	<.001***
Age	-0.03	0.001	-32.57	-0.03	-0.03	<.001***
Sex	-0.37	0.02	-23.01	-0.40	-0.34	<.001***
Education (<High School)	-0.09	0.04	-2.12	-0.17	-0.01	0.034*
Education (High School)	-0.06	0.03	-2.18	-0.11	-0.01	0.029*
Education (University)	0.13	0.02	6.62	0.09	0.17	<.001***
Education (Graduate)	0.24	0.02	11.81	0.20	0.28	<.001***
Chronic conditions	-0.02	0.01	-2.50	-0.04	-0.01	0.012*
Perceived social status	0.01	0.004	3.13	0.01	0.02	0.002**
Group * Subjective cognitive decline	-0.05	0.06	-0.81	-0.17	0.07	0.418

Note. SE = standard error, CI = confidence interval. * $p < .05$; ** $p < .01$; *** $p < .001$.

Table A.4.

Logistic Regression of the Relationships Between Psychological Distress and Change in Objective Cognitive Performance and SCD

Variable	Estimate	SE	Z Value	Odds Ratio	Lower CI	Upper CI	P Value
(Intercept)	-0.21	0.15	-1.43	0.80	-0.52	0.06	0.153
Psychological distress	0.42	0.02	17.40	1.52	0.37	0.47	<.001***
Group	0.15	0.08	1.96	1.16	0.001	0.30	0.050
Change in composite cognitive performance	0.06	0.03	1.72	1.01	-0.04	0.07	0.086
Age	0.01	0.002	6.48	1.01	0.01	0.02	<.001***
Sex	-0.09	0.04	-2.42	0.91	-0.16	-0.01	0.015*
Education (<High School)	0.00	0.11	-0.03	1.00	-0.20	0.21	0.976
Education (High School)	-0.05	0.07	-0.72	0.95	-0.18	0.09	0.470
Education (University)	0.13	0.05	2.77	1.15	0.04	0.23	0.006**
Education (Graduate)	0.14	0.05	2.85	1.15	0.05	0.24	0.004**
Chronic conditions	-0.05	0.02	-2.05	0.95	-0.10	-0.002	0.040*
Perceived social status	-0.05	0.01	-4.33	0.95	-0.07	-0.03	<.001***
Psychological distress & Group	0.13	0.10	1.38	1.16	-0.04	0.35	0.167
Psychological distress * Baseline cognitive performance	-0.03	0.04	-0.83	1.01	-0.05	0.07	0.407
Group * Baseline cognitive performance	0.03	0.12	0.26	1.10	-0.11	0.31	0.795
Psychological distress * Group * Baseline cognitive performance	0.23	0.15	1.51	1.36	0.06	0.56	0.132

Note. SE = standard error, CI = confidence interval. * $p < .05$; ** $p < .01$; *** $p < .001$.

Table A.5.

Logistic Regression of the Relationships Between Psychological Distress and Baseline Cognitive Performance and SCD

Variable	Estimate	SE	Z Value	Odds Ratio	Lower CI	Upper CI	P Value
(Intercept)	-0.32	0.15	-2.12	0.78	-0.54	0.04	0.034*
Psychological distress	0.42	0.02	17.48	1.53	0.38	0.47	<.001***
Group	0.16	0.08	2.12	1.16	0.00	0.30	0.034*
Baseline cognitive performance	0.05	0.02	2.12	1.02	-0.02	0.06	0.034*
Age	0.02	0.002	6.95	1.01	0.01	0.02	<.001***
Sex	-0.08	0.04	-2.14	0.92	-0.16	-0.01	0.033*
Education (<High School)	0.002	0.11	0.02	1.00	-0.21	0.21	0.981
Education (High School)	-0.04	0.07	-0.61	0.96	-0.18	0.09	0.542
Education (University)	0.12	0.05	2.39	1.14	0.03	0.23	0.017*
Education (Graduate)	0.12	0.05	2.32	1.15	0.04	0.24	0.020*
Chronic conditions	-0.05	0.02	-1.89	0.95	-0.10	0.00	0.059
Perceived social status	-0.05	0.01	-4.34	0.95	-0.07	-0.03	<.001***
Psychological distress & Group	0.13	0.10	1.37	1.13	-0.06	0.31	0.170
Psychological distress * Baseline cognitive performance	0.00	0.02	0.17	1.03	-0.01	0.08	0.866
Group * Baseline cognitive performance	-0.07	0.08	-0.84	0.97	-0.18	0.13	0.400
Psychological distress * Group * Baseline cognitive performance	0.15	0.10	1.53	1.11	-0.09	0.29	0.127

Note. SE = standard error, CI = confidence interval. * $p < .05$; ** $p < .01$; *** $p < .001$.

Table A.6.

Univariate Logistic Regression for Biopsychosocial Correlates of Subjective Cognitive Decline

	Univariate (Unadjusted) Analysis						
Predictor	**Coefficient**	**SE**	**Z Value**	**Odds Ratio**	**Lower CI**	**Upper CI**	**P Value**
Age	0.11	0.05	2.06	1.12	1.01	1.24	0.039*
Sex	-0.09	0.11	-0.87	0.91	0.74	1.12	0.387
Education: <High school	-0.33	0.31	-1.04	0.72	0.39	1.34	0.297
Education: High school	-0.13	0.21	-0.64	0.88	0.59	1.32	0.524
Education: University	0.10	0.15	0.70	0.94	0.70	1.26	0.486
Education: Graduate	-0.06	0.15	-0.43	1.11	0.83	1.49	0.669
Perceived social status	-0.06	0.05	-1.08	0.94	0.85	1.05	0.278
Loss of consciousness	0.17	0.11	1.60	1.19	0.96	1.47	0.109
Total symptoms endorsed	-0.18	0.19	-0.95	0.83	0.57	1.21	0.342
Depressive symptoms	0.25	0.06	4.34	1.28	1.15	1.44	<.001***
Baseline cognitive performance	-0.03	0.05	-0.60	0.97	0.87	1.08	0.549
Level of conscientiousness	-0.27	0.06	-4.68	0.76	0.68	0.85	<.001***
Level of emotional stability	-0.22	0.06	-3.81	0.81	0.72	0.90	<.001***
Level of openness to experience	-0.11	0.06	-2.04	0.89	0.80	0.99	0.041*
Social support: Affection	-0.06	0.05	-1.13	0.94	0.84	1.05	0.258
Social support: Emotional & Informational	-0.08	0.05	-1.49	0.92	0.83	1.02	0.135
Social Support: Positive	-0.10	0.05	-1.81	0.91	0.81	1.01	0.071
Social Support: Tangible	-0.02	0.05	-0.35	0.98	0.88	1.09	0.728
Social Participation: Infrequent/No	-0.05	0.15	-0.32	0.95	0.71	1.28	0.746
Social Participation: Frequent	-0.06	0.15	-0.39	0.94	0.71	1.27	0.696
Self-rated hearing	0.25	0.05	4.52	1.28	1.15	1.43	<.001***
Self-rated vision	0.12	0.05	2.20	1.13	1.01	1.25	0.028*
Number of chronic conditions	0.07	0.06	1.11	1.07	0.95	1.22	0.268

Level of physical activity	0.01	0.05	0.20	1.01	0.91	1.12	0.838
Smoking status: Former	0.30	0.11	2.64	1.34	1.08	1.67	0.008**
Smoking status: Regular	0.32	0.24	1.36	1.38	0.87	2.21	0.174
Smoking status: Occasional	0.002	0.40	0.01	1.00	0.46	2.22	0.996
Alcohol frequency: Non-drinker	-0.05	0.41	-0.12	0.95	0.43	2.21	0.903
Alcohol frequency: 12-month abstainer	-0.30	0.14	-2.22	0.74	0.57	0.97	0.027*
Alcohol frequency: Occasional	-0.10	0.15	-0.64	0.91	0.68	1.22	0.519

Note. Social participation was indicator-coded, with “moderate participation” as the reference level (“infrequent/no participation” = never, at least once/year, or at least once/month, “moderate participation” = at least once/week, and “frequent participation” = at least once/day). Smoking status was indicator-coded, with “non-smokers” (i.e., never smoked) as the reference level (“never smoked” = ≥100 cigarettes), “former smokers” = smoked ≥100 cigarettes in their lifetime but have not smoked in the past month, and “current smokers” = smoked ≥100 cigarettes in their lifetime and smoked in the past month. Alcohol frequency was indicator-coded, with “regular drinker” as the reference level (“non-drinker” = never drank alcohol and did not drink in the last 12 months, “former/12-month abstainer” = drank alcohol in the past but not in the last 12 months, or drank alcohol in the past but had less than one drink per month in the last 12 months, “occasional drinker” = drank alcohol in the past and had at least one drink per month in the last 12 months, and “regular drinker” = drank alcohol in the past and had at least one drink per week in the last 12 months. Physical activity was quantified as the summed score of the frequency of activity (hours/day) multiplied by intensity level of the activity. CI = confidence interval. * $p < .05$; ** $p < .01$; *** $p < .001$.

Table A.7.

Multivariate Logistic Regression for Biopsychosocial Correlates of Subjective Cognitive Decline

	Multivariate (Adjusted) Analysis						
Predictor	**Coefficient**	**SE**	**Z Value**	**Odds Ratio**	**Lower CI**	**Upper CI**	**P Value**
Age	0.14	0.08	1.78	1.15	0.99	1.33	0.076
Sex	-0.30	0.14	-2.11	0.74	0.56	0.98	0.035*
Education: <High school	-0.52	0.38	-1.39	0.59	0.28	1.25	0.166
Education: High school	-0.27	0.23	-1.15	0.76	0.48	1.21	0.251
Education: University	0.03	0.17	0.15	1.03	0.74	1.43	0.878
Education: Graduate	0.24	0.17	1.40	1.27	0.91	1.79	0.162
Perceived social status	0.01	0.07	0.07	1.01	0.87	1.16	0.944
Loss of consciousness	0.17	0.13	1.36	1.19	0.93	1.53	0.173
Total symptoms endorsed	0.02	0.24	0.09	1.02	0.63	1.63	0.931
Depressive symptoms	0.19	0.08	2.34	1.21	1.03	1.42	0.019*
Baseline cognitive performance	-0.03	0.07	-0.38	0.97	0.85	1.12	0.702
Level of conscientiousness	-0.22	0.07	-3.05	0.80	0.69	0.92	0.002**
Level of emotional stability	-0.10	0.07	-1.36	0.90	0.78	1.04	0.174
Level of openness to experience	-0.17	0.07	-2.42	0.85	0.74	0.97	0.015*
Social support: Affection	0.09	0.11	0.80	1.09	0.88	1.35	0.425
Social support: Emotional & Informational	0.01	0.11	0.13	1.01	0.82	1.26	0.894
Social Support: Positive	-0.10	0.12	-0.84	0.90	0.71	1.15	0.401
Social Support: Tangible	0.05	0.09	0.50	1.05	0.87	1.26	0.617
Social Participation: Infrequent/No	-0.11	0.18	-0.60	0.90	0.62	1.29	0.546
Social Participation: Frequent	-0.07	0.18	-0.39	0.93	0.66	1.32	0.700
Self-rated hearing	0.23	0.07	3.48	1.26	1.11	1.44	0.001**
Self-rated vision	0.02	0.07	0.29	1.02	0.89	1.16	0.769
Number of chronic conditions	0.06	0.08	0.72	1.06	0.90	1.25	0.474
Level of physical activity	0.12	0.07	1.76	1.13	0.99	1.30	0.078
Smoking status: Former	0.24	0.14	1.79	1.28	0.98	1.67	0.074

Smoking status: Occasional	0.03	0.48	0.06	1.03	0.40	2.71	0.951
Smoking status: Regular	0.37	0.30	1.22	1.44	0.81	2.62	0.221
Alcohol frequency: Non-drinker	0.15	0.48	0.32	1.16	0.46	3.10	0.753
Alcohol frequency: 12-month abstainer	-0.31	0.18	-1.73	0.74	0.52	1.04	0.084
Alcohol frequency: Occasional	0.01	0.17	0.03	1.01	0.72	1.42	0.975

Note. Social participation was indicator-coded, with “moderate participation” as the reference level (“infrequent/no participation” = never, at least once/year, or at least once/month, “moderate participation” = at least once/week, and “frequent participation” = at least once/day). Smoking status was indicator-coded, with “non-smokers” (i.e., never smoked) as the reference level (“never smoked” = ≥100 cigarettes), “former smokers” = smoked ≥100 cigarettes in their lifetime but have not smoked in the past month, and “current smokers” = smoked ≥100 cigarettes in their lifetime and smoked in the past month. Alcohol frequency was indicator-coded, with “regular drinker” as the reference level (“non-drinker” = never drank alcohol and did not drink in the last 12 months, “former/12-month abstainer” = drank alcohol in the past but not in the last 12 months, or drank alcohol in the past but had less than one drink per month in the last 12 months, “occasional drinker” = drank alcohol in the past and had at least one drink per month in the last 12 months, and “regular drinker” = drank alcohol in the past and had at least one drink per week in the last 12 months. Physical activity was quantified as the summed score of the frequency of activity (hours/day) multiplied by intensity level of the activity. CI = confidence interval. * $p < .05$; ** $p < .01$.