

Functional Social Support as a Mediator of the Association Between Anxiety and Executive Function: A
Moderated Mediation Analysis of the Canadian Longitudinal Study on Aging

by

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Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

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Abstract

Background: Anxiety in older adulthood may adversely affect executive function, a cognitive domain essential for adaptability and independence. Functional social support (FSS), the perception that others will provide help, care, or comfort when needed, may partially explain the link between anxiety and executive function. This link may differ by age or sex.

Objective: To examine whether FSS mediates the association between anxiety (self-reported clinical diagnosis of anxiety or anxiety symptoms) and executive function in middle-aged and older adults, stratified by age and sex.

Methods: Analyses included 6,719 community-dwelling adults aged 45 to 85 years at baseline, drawn from the Comprehensive cohort of the Canadian Longitudinal Study on Aging. Data were collected over three waves spanning six years. Clinical history of an anxiety disorder (yes/no) and anxiety symptoms (four items from the Kessler Psychological Distress Scale) were self-reported at baseline (T0). Three-year (T1) FSS was self-reported using the Medical Outcomes Study-Social Support Survey. Six-year (T2) executive function was obtained by standardizing and combining scores from five neuropsychological tests. Conditional process analysis with percentile bootstrapping was used to estimate mediation across levels of age and sex, adjusted for relevant covariates and antecedent measures of FSS (T0) and executive function (T0, T1).

Results: FSS did not significantly mediate the association between either anxiety measure (clinical anxiety or anxiety symptoms) and executive function for any age or sex subgroup ($bs = -0.0043$ to 0.0103 , $p > .05$).

Discussion: While social support has known benefits for cognition, the results suggest that the provision of FSS as a strategy to mitigate the impact of anxiety on executive function may not be needed in healthy middle-aged and older men and women. To promote the cognitive health of aging Canadians, interventions may be better directed to targeting other pathways linking anxiety to cognitive decline.

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List of Abbreviations

| | |
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| AD | Alzheimer's disease |
| AFT | Animal Fluency Test |
| CART | Classification and regression trees |
| CES-D-10 | Center for Epidemiological Studies Short Depression Scale |
| CLSA | Canadian Longitudinal Study on Aging |
| COWAT | Controlled Oral Word Association Test |
| FSS | Functional social support |
| GAD | Generalised anxiety disorder |
| GAD-7 | Generalized anxiety disorder 7-item scale |
| K10 | Kessler Psychological Distress Scale |
| MAT | Mental Alternation Test |
| MCI | Mild cognitive impairment |
| MI | Multiple imputation |
| MOS-SSS | Medical Outcomes Study-Social Support Survey |
| Stroop-VV | Stroop Neuropsychological Screening Test-Victoria Version |
| SSS | Structural social support |
| T0 | Baseline |
| T1 | Follow-up 1 |
| T2 | Follow-up 2 |
| TMT | Time-based Prospective Memory Test |

Chapter 1

Introduction

Declines in fertility and improved longevity are considered the primary drivers behind a globally ageing population (United Nations, Department of Economic and Social Affairs, Population Division, 2019a). By 2050, one in six people worldwide will be an older adult aged 65 and over (United Nations, Department of Economic and Social Affairs, Population Division, 2019b). Population ageing, in turn, is expected to increase the number of people living with dementia, a syndrome of significant cognitive and functional deterioration that is estimated to afflict about 7% of individuals aged 65 and over (Gale et al., 2018). Recent estimates of global prevalence suggest that approximately 60 million people are living with dementia worldwide, a figure that is projected to cross 150 million by 2050 (Nichols et al., 2022). In comparison, approximately 730,000 Canadians are living with dementia in 2024, a number that is forecasted to grow to 1.7 million cases by 2050 (Alzheimer Society of Canada, 2024). The percentage of those living with dementia aged 65 and over is expected to increase from 8.4% in 2020 to 13.2% by 2050, the majority of whom will comprise women and an aged “baby boomer” cohort (Armstrong, 2022).

Age-related cognitive decline affects the independence and quality of life of older adults and their care partners (Armstrong, 2022). The field of public health has generated a wealth of research related to the development, prevention, and treatment of dementia following the recognition that cognition is essential to a person’s everyday functioning and their ability to adapt to change. The cognitive domain of executive function, the basis of the ability to plan, problem-solve, self-regulate, and make decisions, is intrinsically linked to successful ageing (Fiocco & Yaffe, 2010). Deficits in executive function can make everyday activities of living, such as financial management, shopping for necessities, and managing a household, more challenging (Vaughan & Giovanello, 2010), disrupting an individual’s capacity to age in place. These deficits may also contribute to personality and behavioural changes that put a strain on one’s relationships with others (Adolphs, 2009; Cruz et al., 2020). Cognitive impairment is associated with great costs to the individual, the family, the healthcare system, and society (Public Health Agency of Canada, 2019). To promote healthy ageing and minimise the burden of dementia, there is a need to understand modifiable factors that impair or protect cognitive function, such as mental health and social support. Older adults are at an elevated risk of depression and social isolation. Of the 40% of dementia cases that are potentially modifiable, about 8% is attributable to these two risk factors (Livingston et al., 2020).

While less extensively studied compared to depression in the context of ageing, anxiety is commonly experienced by older adults and has been linked to poorer quality of life and increased disability (Bassil et al., 2011), as well as cognitive decline (Gulpers et al., 2016). Despite its prevalence, anxiety in late life is poorly understood and challenging to assess. In older adults, anxiety tends to be experienced at a subsyndromal level, failing to meeting diagnostic criteria for a disorder, and, as a result, is likely to go undetected and untreated (Chand et al., 2014). The questions that remain about anxiety, weighed against its ramifications for health and ageing, have elicited repeated calls to study late-life anxiety more in depth (Bryant et al., 2013; Byrne & Pachana, 2010; Ribeiro et al., 2020). More recently, the COVID-19 pandemic has put a spotlight on mental health and spurred a renewed interest in studying anxiety across different ethnic (West et al., 2021), gender (Heffner et al., 2021; Levy, 2022), and age groups (Cunningham et al., 2021; J. M. Wilson et al., 2021), prompting several large-scale studies (Cozen et al., 2024; Raina et al., 2021) and reviews focused on anxiety during the pandemic (Delpino et al., 2022; Kindred & Bates, 2023; Saeed et al., 2022).

Anxiety may impact executive function through direct and indirect pathways. While much research has focused on the cognitive and physiological mechanisms linking anxiety to executive function, little attention has been paid to psychosocial mechanisms, many of which are potentially modifiable and bidirectionally linked to anxiety, whereby improvements in one area of functioning may lead to improvements in the other, and vice versa. Social support, often impaired in those with anxiety (Stewart et al., 2022), has emerged as a protective factor for cognition (Mogic et al., 2023) and a promising area of intervention (Uchino et al., 2018). Social support encompasses both structural (e.g., network size) and functional (e.g., perceived availability of someone to confide in) elements. Functional social support, in particular, has demonstrated more direct associations with health than either structural or received support (Gable & Bedrov, 2022). The potential for late life anxiety to disrupt functional social support—and remove its potential benefits for executive function—should not be ignored. Research in this area constitutes a vital opportunity to improve on existing social support interventions and target subpopulations of aging adults whose cognition would profit from social-oriented programming.

The purpose of the current study was to determine whether functional social support mediated the association between anxiety and executive function, within specific age groups and across males and females. Better understanding of the relationship between anxiety, functional social support, and

executive function can inform development of interventions to improve cognitive health and preserve the abilities that make ageing independently in the community possible.

Chapter 2

Literature Review

2.1 Anxiety

Anxiety is a heightened state of arousal and vigilance induced by actual or perceived threats to well-being or survival and may manifest as symptoms of worry, restlessness, tension, irritability, or fatigue. Anxiety evolved as a protective response against natural and social threats and, at moderate levels, is adaptive, facilitating motivation, learning, problem-solving, and preparation for future challenges. Related to anxiety is the emotion of fear, which is a reaction to a known, external, and immediate threat that diminishes once the threat is removed (Steimer, 2002). In contrast, anxiety is marked by uncertainty and uncontrollability surrounding a threat that tends to be unknown, internal, and more distant in time (Steimer, 2002). Anxiety, fear, and stress share common neuroendocrine and arousal mechanisms and overlapping neurocircuitry that interact to increase “wear and tear” on physiological systems over time, elevating the risk for neuropsychiatric disorders such as depression and dementia (Mah et al., 2016). Chronic stress contributes to the development and maintenance of pathological anxiety by impairing neurocircuitry that downregulates fear and anxiety responses (Mah et al., 2016). Common coping strategies used to allay anxiety, such as avoidance, may also play a role in the maintenance of long-term anxiety (Hofmann & Hay, 2018). For example, maladaptive coping strategies that successfully reduce anxiety are more likely to be reinforced and used in the future but may decrease the likelihood of confronting and overcoming source(s) of anxiety (Hofmann & Hay, 2018).

When symptoms persist for long periods of time and undermine the ability to cope with life’s challenges, causing significant impairments in social, occupational, or other important areas of functioning, anxiety may evolve into a debilitating mental disorder. Most anxiety disorders emerge in adolescence and young adulthood and tend to be chronic and unremitting (Bassil et al., 2011; Sami & Nilforooshan, 2015). However, one-third of cases of generalised anxiety disorder (GAD), the most common type of anxiety disorder, is estimated to develop after the age of 50 (Bassil et al., 2011; Bryant et al., 2008; Wolitzky-Taylor et al., 2010) with symptoms enduring, on average, 20 years or longer (Lenze & Wetherell, 2011). Late-onset anxiety disorders are theorised to develop because of age-related neurobiological changes in the amygdala and frontal brain regions interacting with psychosocial, genetic, and early-life influences (Lenze & Wetherell, 2011). Among the anxiety

disorders listed in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychological Association, 2013), agoraphobia has been found to occur most frequently in late life, followed by panic disorder, GAD, specific phobias, and social phobia (Hellwig & Domschke, 2019). Although not a diagnosable disorder, fear of falling is also highly prevalent in older adults and, in severe forms, may be akin to agoraphobia in terms of curtailing one's activities (Lenze & Wetherell, 2011).

Older adults with anxiety tend to be female, unmarried, and childless; have lower education, lower income, and three or more chronic conditions; and report a history of traumatic experiences (Bassil et al., 2011; Lenze & Wetherell, 2011). Individuals scoring high on the personality trait of neuroticism are also more prone to feeling anxious. Both neuroticism and anxiety disorders are considered examples of trait anxiety, which is the individual tendency to perceive situations as threatening and to respond with more frequent and more intense elevations in state anxiety (Harrison et al., 2015; Wetherell et al., 2002). Neuroticism is known to play a role in the maintenance of anxiety disorders (Sami & Nilforooshan, 2015). Additionally, anxiety is present in the experience of psychological distress, referring to non-specific—usually transient and subthreshold—feelings of anxiety and depression that arise from exposure to stressful events and which has been linked to an increased risk of developing dementia (Simard et al., 2009). Neuroticism and psychological distress are related (Drapeau et al., 2011; Joshanloo, 2024; Panayiotou et al., 2014), and both are risk factors for future anxiety disorders (Keough et al., 2010; Wolitzky-Taylor et al., 2010), for example, by increasing vulnerability through negative thinking, increased reactivity, or ineffective coping (Dragan et al., 2012; Ormel et al., 2013).

Anxiety is highly prevalent in older adults, with estimates in community-dwelling samples ranging from 1.2% to 15% for anxiety disorders and 15% to 52% for anxiety symptoms (Bryant et al., 2008). This variation in estimates may partly be attributed to differences in how anxiety is defined and measured, with anxiety being more prevalent if subthreshold cases (i.e., symptoms not meeting the criteria for a diagnosis) are considered (Bryant et al., 2008). Unfortunately, anxiety has proven challenging to assess in older adults. Most instruments have been developed for use in younger adult samples and may be unsuitable for distinguishing between “normal” and “disordered” anxiety in older adults (Balsamo et al., 2018; Witlox et al., 2021; Wolitzky-Taylor et al., 2010). For instance, compared to younger adults, older adults may report fewer psychological symptoms and more somatic complaints and be less likely to report negative affect; however, they are more likely to

experience anxiety symptoms directly, such as fearfulness without the feelings of shame or guilt that characterise anxiety in younger adults, or express fear of situations generally not included on surveys, such as fear of being a burden on their families (Balsamo et al., 2018; Witlox et al., 2019; Wolitzky-Taylor et al., 2010). The failure to identify clinically relevant anxiety in older adults may be the reason that older adults reporting subthreshold anxiety appear to be highly similar to those diagnosed with anxiety in terms of physical and social activity restriction, decreased well-being, and health services utilisation (Witlox et al., 2018).

Other reasons for underdiagnosis and undertreatment of anxiety in older adults include the normalisation of anxiety in ageing, difficulty differentiating anxiety from symptoms of other medical conditions or pharmacological side-effects, and difficulty communicating one's symptoms due to cognitive impairment (Bryant et al., 2008; Wolitzky-Taylor et al., 2010). Older adults may also have more success coping with their anxiety and not meeting functional impairment criteria for a disorder as, compared to younger adults, they may be less active in society and less likely to confront anxiety-inducing situations (Bryant et al., 2008; Witlox et al., 2021; Wolitzky-Taylor et al., 2010). Help-seeking is low in older adults with anxiety, perhaps even lower than that of individuals who are depressed (Scott et al., 2010). Low help-seeking may be related to stigma, lack of mental health literacy, and socioeconomic barriers limiting access to health services (Elshaikh et al., 2023). For all these reasons, the population of older adults who report an anxiety disorder may not necessarily be representative of all older adults experiencing clinically relevant levels of anxiety. This is where examining anxiety symptoms may prove useful in understanding the experiences of anxious older adults, as self-administered screening tools for anxiety may capture a continuum of functioning and can easily be administered in large-scale studies where it is possible to recruit population-based samples. Research comparing self-reported diagnoses and self-reported symptoms of anxiety in the general population finds that both measures show high agreement when classifying individuals as not having a disorder, but self-reported symptoms are more likely to indicate a possible disorder where a diagnosis is not reported (Davies et al., 2022). Examining both self-reported symptoms and diagnoses may tap into different experiences of anxiety and help ascertain whether such experiences are related to differential health outcomes.

While anxiety is related to increased disability, lower quality of life, and a higher risk of mortality in older adults (Bassil et al., 2011; van Hout et al., 2004), standard treatments for anxiety disorders—pharmacotherapy and cognitive-behavioural therapy (CBT)—tend to be less effective in older adults

than they are in younger adults, indicating that current approaches to treating anxiety in late life are not optimally adapted to the needs of the older population (Wolitzky-Taylor et al., 2010). CBT may be complicated by impaired cognition and learning, and medications for anxiety, while effective at least in the short term (Balasubramaniam et al., 2019), are associated with adverse side-effects at lower doses in older adults, increasing the risk for falls, disability, and cognitive decline (Lenze & Wetherell, 2011). Undesirable side-effects, low treatment-seeking, and lower compliance exhibited by those with anxiety (Bassil et al., 2011) suggest the need for alternate strategies of long-term management of anxiety. Non-specific, non-pharmacological therapies, typically implemented in randomised controlled trials as active control conditions (e.g., group therapy), appear to be effective in treating anxiety in older adults, suggesting a possible direction forward in tailoring interventions to older individuals (Gonçalves & Byrne, 2012). Compared to more traditional mental health care, these naturalistic, more social approaches to treatment may be able to reduce stigma associated with treatment-seeking and consequently increase adherence (Gonçalves & Byrne, 2012). Further research is needed to elucidate the extent to which social interventions are effectual for mitigating the negative health impacts of anxiety.

To study anxiety in the older population, accounting for comorbid depression is important (Bryant et al., 2008) as anxiety frequently co-occurs with depression (Bassil et al., 2011) and this co-occurrence is associated with poorer health outcomes including greater chronicity, greater likelihood of relapse, greater symptom severity, greater functional impairment, and poorer response to treatment (Bassil et al., 2011; Sami & Nilforooshan, 2015; Wolitzky-Taylor et al., 2010). Anxiety and depression share numerous risk factors such as having chronic health conditions, poor self-rated health, functional limitations, and neuroticism; however, longitudinally, anxiety is more likely to precede depression than the reverse and may constitute a risk factor for late-life depression (Lenze & Wetherell, 2011). Because of the high comorbidity between anxiety and depression, many self-report assessments screen for both anxiety and depression, combining symptoms within the same instrument that may be further divided into anxiety and depression subscales for individual analysis. The Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983) and the Kessler Psychological Distress Scale (K10) (Brooks et al., 2006; Kessler et al., 2003) are some examples of composite instruments frequently implemented in clinical and research settings. On one hand, combining anxiety and depression in the same measure may provide a better sense of an individual's overall psychosocial functioning, but there is also an advantage to being able to distinguish symptoms using

anxiety and depression subscales in order to understand an individual's unique symptomatology and tailor treatments appropriately (Mordeno et al., 2024).

2.2 Executive Function

Executive function is one of six cognitive domains listed in the DSM-5, the others being language, learning and memory, social cognition, complex attention, and perceptual-motor function (APA, 2013). Executive function refers to a collection of cognitive abilities that govern and enable adaptive, goal-oriented behaviour (Rabinovici et al., 2015). These are the mental processes responsible for the ability to pay attention, organise, plan, make decisions, exert self-control, problem-solve, and multitask. Broadly, executive function can be divided into three subdomains: *inhibitory control*, the ability to control one's attention, behaviour, thoughts, or emotions to override a competing response; *working memory*, the ability to retain and manipulate information in the mind; and *cognitive flexibility*, the ability to change perspectives, spatially or from the point of view of others (Diamond, 2013). These subdomains may be assessed with standardised neuropsychological instruments that are usually part of a larger battery of tests, or with experimental paradigms that are non-standardised, computerised procedures that measure behavioural responses to a manipulation of a stimulus (Reuter-Lorenz et al., 2016). While such measures rarely capture a "pure" representation of a particular executive process by themselves, multiple tests may be employed in conjunction to derive an "overall" measure of executive function.

Executive processes are understood to originate from distributed neural networks that mainly encompass the prefrontal cortex but also engage the parietal cortex, basal ganglia, thalamus, and cerebellum (Rabinovici et al., 2015). The prefrontal cortex is one of the first areas to show age-related losses (Reuter-Lorenz et al., 2016), with declines in executive function manifesting as early as the third decade of life (Ferguson et al., 2021). Furthermore, these age-related changes occur at a greater magnitude than in other regions of the brain (Reuter-Lorenz et al., 2016). In healthy older adults, there is evidence of dedifferentiation of executive processes where additional brain networks are recruited to handle tasks (Spreng et al., 2017). For example, inhibitory control becomes less separable from working memory (Friedman & Miyake, 2017) such that age-related declines in working memory appear to be primarily driven by declining inhibitory control and general slowing of processing (Diamond, 2013). At the same time, working memory is increasingly relied on to compensate for degraded performance in other domains (Spreng et al., 2017). Consequently, to offset

the effort of increased working memory usage, older adults may draw on accumulated knowledge and experiences to inform their decisions (Spreng et al., 2017). Increased reliance on frontal brain regions in healthy older adults underscores the need to investigate ways to preserve these functions in later life and prevent the onset of more serious cognitive impairment.

Declines in cognition that deviate from the norms expected of one's age and education (Petersen et al., 1999) may signal abnormal changes that progress to dementia (Denver & McClean, 2018). Mild cognitive impairment (MCI) represents an intermediate stage between healthy cognitive ageing and the severe cognitive impairment seen in dementia (Petersen et al., 2014). Those classified as having MCI retain their functional independence but may exhibit deficits in episodic memory (amnestic subtype) or other cognitive domains such as executive function (non-amnestic subtype), as well as experience deficits in single or multiple domains (Petersen et al., 2014). MCI increases the risk of developing dementia, although this is not inevitable, and in some cases, MCI may revert and resume the normal trajectory of cognitive ageing (Iraniparast et al., 2022). MCI shares many risk factors with dementia, including older age, lower education, genetics, vascular diseases, and inadequate physical activity (Petersen et al., 2014), although recent research has shed light on executive function as a possible predictor of the risk of progression from MCI to dementia. In particular, deficits in executive function may be apparent even in those with pure amnestic MCI (Brandt et al., 2009), and executive dysfunction in amnestic MCI is associated with a greater risk of conversion to dementia (Junquera et al., 2020) over and above the rates seen in pure amnestic MCI or amnestic MCI with visuospatial or language dysfunction (Jung et al., 2020). Given the central role that executive function plays in enabling and sustaining daily life activities (Guarino et al., 2019), and its aforementioned ties to predicting progression to worse cognitive outcomes, understanding and targeting this domain of cognition is necessary to preserve the quality of life and independence of an ageing population.

Key to prevention is the observation that individuals show different rates of age-related cognitive decline. Some variation may be attributed to genetic susceptibility and some to environment/lifestyle, and it is the latter that is potentially modifiable. Factors such as educational attainment, occupation, leisure activity, and social connection contribute to cognitive reserve (Livingston et al., 2020), a term describing the brain's capacity to cope with age-related changes and pathology via cognitive-processing approaches or compensatory mechanisms that are conferred by experiences throughout the lifespan (Stern, 2012). Cognitive reserve allows the brain to withstand more severe disease without compromising functioning until a point is reached for which deterioration can no longer be tolerated,

whereupon decline proceeds rapidly and irreversibly. Cognitive reserve has been used to explain the seemingly paradoxical observation that individuals with brain pathology may not display overt clinical symptoms until much later in life, if ever (Stern, 2012). Executive function is sensitive to changes in lifestyle and is among the first cognitive domains to suffer from the effects of stress, loneliness, poor mental health, and inadequate physical fitness (Diamond, 2013). However, this vulnerability to lifestyle and behavioural changes may render executive function more amenable to intervention. Investigating modifiable risk and protective factors of executive function is a key step to achieving that aim.

2.3 Anxiety and Executive Function

Anxiety and executive function are intertwined. As previously discussed, normal feelings of anxiety facilitate adaptation to adverse or unexpected situations, while pathological anxiety interferes with the ability to cope successfully with life's challenges—an ability supported and made possible by the higher-order processes of executive function. The prefrontal cortex is known to play a key role in the downregulation, both voluntary and involuntary, of fear and anxiety responses, and in pathological anxiety, these “top-down” processes become dysregulated, leading to impairments in emotional regulation (Mah et al., 2016). Early work on anxiety and executive function has focused on the impact of anxiety through an experimental lens, typically involving the comparison of cognitive performance between anxious and non-anxious individuals (Bar-Haim et al., 2007). Subsequent research has upheld the finding that anxiety—at least anxiety experienced during testing conditions—impairs the components of executive function through threat-related attentional biases (Majeed et al., 2023), which is consistent with the *attentional control theory* of anxiety (Eysenck et al., 2007).

According to the attentional control theory, anxiety may worsen executive function by reducing working memory capacity. Specifically, anxiety impairs the ability to inhibit anxiety-related worry, subsequently leaving fewer cognitive resources to attend to the task at hand (Eysenck et al., 2007). This attentional bias toward negative, task-irrelevant stimuli (external or internal) over task-relevant stimuli results in decreased performance if the effort expended to compensate for this distraction exceeds an individual's working memory capacity (Eysenck et al., 2007; Owens et al., 2014). That is, the more difficult a task is for a given person's ability, the likelier that higher anxiety will be detrimental to that person's cognitive performance (C. MacLeod & Clarke, 2013). For those able to accommodate higher cognitive load, worry is less deleterious (Mella et al., 2020). The *processing*

efficiency theory of anxiety (Eysenck & Calvo, 1992) further qualifies that anxiety does not necessarily worsen accuracy, but rather the efficiency, or speed, of responses (Eysenck et al., 2007). That is, anxious individuals may take longer to accomplish a task, but the results of their efforts do not necessarily suffer in quality. Supporting this view is a meta-analysis by Majeed et al. (2023) that found slower reaction time but comparable accuracy on tasks of executive function in those with GAD compared to control groups, suggesting the use of effortful compensatory strategies to supplement reduced cognitive resources and enhance performance at the expense of speed. The gap between accuracy and efficiency may be more apparent in older adults who, in addition to being more susceptible to the effects of anxiety compared to younger adults, demonstrate age-related declines in speed of processing (Harrison et al., 2015) and show a greater top-down tendency to engage in slower and more deliberate decision-making in order to maintain accuracy in task performance (Reuter-Lorenz et al., 2016).

In discussing experimental research, a further distinction should be made between trait anxiety (disposition) and state anxiety (emotional reaction), which differ in their chronicity and potential to influence long-term health outcomes (Harrison et al., 2015). It may very well be that outside of these testing conditions, executive function may be left intact when anxiety is absent. Indeed, cross-sectional examinations of anxiety and executive function may only be capturing transient states of anxiety, as individuals with social phobia or specific phobias do not exhibit the same degraded performance on tasks of executive function as those with GAD, perhaps owing to the fact that, in contrast to GAD, phobic disorders are characterised by worry of *specific* events or stimuli that are not at all activated under testing conditions, leaving performance relatively intact compared to control (Majeed et al., 2023). Given the temporal ambiguity of cross-sectional studies, it is also difficult to discount the possibility of preexisting executive dysfunction limiting the capacity to regulate emotions and inhibit intrusive thoughts, which may give rise to anxiety (Moran, 2016). However, there is growing longitudinal evidence that anxiety may leave gradual, more lasting impacts on cognitive function and increase the risk of cognitive decline (Jain et al., 2023; Santabárbara, Lipnicki, Bueno-Notivol, et al., 2020; Santabárbara, Lipnicki, Olaya, et al., 2020), including in the domain of executive function (Zainal & Newman, 2023).

2.3.1 Anxiety as a Risk Factor for Cognitive Decline

In more recent years, several systematic reviews and meta-analyses have been undertaken to summarise the literature on the longitudinal associations between anxiety and cognitive ageing, the majority of which have focused on anxiety as a potential risk factor for cognitive decline. One meta-analysis estimated that 6.5% and 7.9% of the overall risk for incident cognitive impairment and dementia respectively in the community was attributable to anxiety, placing the population attributable risk (PAR) of anxiety between other recognised risk factors such as midlife hypertension (global PAR 5.1%) and depression (global PAR 7.9%) (Gulpers et al., 2016). Meta-analyses of cohort studies focused on the incidence of all-cause dementia (Santabárbara, Lipnicki, Olaya, et al., 2020) and Alzheimer’s disease (AD)-specific dementia (Santabárbara, Lipnicki, Bueno-Notivol, et al., 2020) have also identified significant associations between baseline anxiety and cognitive decline. In contrast, other reviews (John et al., 2019; Stafford et al., 2022) were unable to draw a definite link between anxiety and cognitive decline—with included studies showing either a negative relationship or no relationship—and cite the need for further, high-quality longitudinal studies. The heterogeneity in findings has been attributed to differences in the severity of anxiety measured (subthreshold symptoms versus clinically significant anxiety); the duration of follow-up; and adjustment for potentially significant covariates, such as depression (Kootar et al., 2021; Santabárbara, Lipnicki, Olaya, et al., 2020). Few reviews have consolidated evidence on anxiety influencing the risk of decline in specific cognitive domains. The long-term impact of anxiety on executive function, in particular, remains understudied (Franks et al., 2022).

Several mechanisms have been forwarded to explain how anxiety may impair cognitive function over time, and these can be classified as being biological or behavioural in perspective. The most prominent biological explanation relates to the stress response, which may become dysregulated in chronic anxiety and accelerate ageing processes, including cognitive decline (Beaudreau & O’Hara, 2008; Perna et al., 2015). The chronic release of the stress hormone cortisol by the hypothalamic-pituitary-adrenal (HPA) axis may damage the hippocampus, resulting in atrophy, decreased neurogenesis, and an impaired ability of the hippocampus to inhibit HPA axis activity once the stressor has subsided, leading to prolonged HPA activation and further neuronal damage (Mah et al., 2016). Chronic stress may also damage the prefrontal cortex and weaken prefrontal regulation of the amygdala, which could further exacerbate anxiety and extend the stress response (Mah et al., 2016). Although the effect of psychological stress on executive function is less well studied than its effect on

memory (Franks et al., 2022), longitudinal studies have found associations between anxiety and executive dysfunction that are partially mediated by stress and inflammation (Da Silva Coelho et al., 2022; Zainal & Newman, 2022). A population-based study of women aged 38 to 54 years at baseline with a particularly long follow-up of 38 years found that distress mediated the association between neuroticism and later risk of dementia (Johansson et al., 2014).

More recent research that leverages multi-omics analysis, an analytic approach that integrates sources of biological “big data”, found that, across three large cohorts of non-demented older adults, anxiety impaired cognition over time via axon-synapse injury in the context of mitochondrial dysfunction (L. Sun et al., 2023). Specifically, mitochondrial biogenesis and energy production were suppressed in anxiety, potentially disrupting axon-synapse energy homeostasis and accelerating cognitive decline (L. Sun et al., 2023). Other proposed mechanisms linking anxiety to cognitive decline include cardiovascular disease and BDNF (brain-derived neurotrophic factor) suppression (Gulpers et al., 2016). Anxiety triggers physiological reactions such as increased heart rate, increased blood pressure, and vasoconstriction, which may increase the risk for cardiovascular disease and vascular dementia (Gulpers et al., 2016). BDNF, a brain chemical essential for synaptic plasticity, learning, and neuronal repair, has also been found in lower quantities in those with anxiety disorders (Vismara et al., 2020).

While anxiety may exert direct effects on the brain, it may also have indirect effects on cognitive function through lifestyle and behavioural choices. Maladaptive coping strategies such as avoidance (Aupperle et al., 2023) may decrease cognitive reserve over time, resulting in a reduced capacity to withstand pathological changes in brain ageing (Farina et al., 2021; Stern, 2012; Vance et al., 2010). As an illustration, fear of falling, a common source of anxiety in later life, has been associated with physical and social activity restriction (Bryant et al., 2008). While fear of falling may constitute a protective response to a situation in which there is a risk of harm to the self, avoiding healthy activities may, in the long term, result in decreased cognitive function and loss of independence (Bassil et al., 2011). Furthermore, anxiety has been associated with compromised self-care behaviours that may non-specifically increase the risk for cognitive decline alongside other adverse health outcomes (Duberstein et al., 2011). Those high in neuroticism, for instance, are less likely to eat a healthy diet (Diop et al., 2021), less likely to exercise, more likely to smoke (Graham et al., 2020), and less likely to adhere to medical treatment (Bassil et al., 2011), although the impact of these

lifestyle behaviours may be attenuated by high conscientiousness, a personality trait that reflects self-regulatory, careful, and diligent behaviour (Graham et al., 2020).

2.3.2 Anxiety: Causal or Prodromal Risk Factor for Cognitive Decline?

While thus far this review has focused on anxiety increasing the risk for cognitive decline through various biological and behavioural pathways, it should be acknowledged that debate surrounds the question of whether anxiety is a causal risk factor of cognitive decline or merely an early sign of worsening cognitive function, also known as a prodrome (Kuring et al., 2020; Santabárbara et al., 2019). Clarifying whether anxiety is a prodromal condition has relevance for treatment, given that primary and secondary interventions are most effective when targeting risk factors rather than symptoms or sequelae of the disease. Evidence both for and against anxiety being a prodrome of cognitive decline will be discussed in this section; research suggesting that anxiety and cognitive dysfunction reinforce each other in a bidirectional manner will also be presented.

Prodromal anxiety may manifest as a psychological reaction to subjective cognitive decline or from underlying disease processes that overlap with dementia (Gulpers et al., 2016). Age-related brain changes driving both anxiety and cognitive decline might explain why anxiety is commonly observed in those with cognitive impairment (Gallagher et al., 2017) and why the association between anxiety and cognitive decline appears to be strongest in the oldest old (Gulpers et al., 2016). Furthermore, some forms of anxiety do appear to be influenced by early cognitive impairment. Fear of falling may develop in the context of actual falls or in how individuals rate their physical ability and fall risk (Peeters, Bennett, et al., 2020). Research assessing balance and walking, particularly in dual-task conditions, have demonstrated that gait is a cognitively demanding task that can be predicted by executive function performance (Peeters et al., 2019). Accordingly, cognitive interventions that aim to improve balance confidence have observed reductions in fear of falling (Peeters, Bennett, et al., 2020). A *lack* of fear of falling may also be indicative of cognitive decline, as impairments in both memory and executive function have been associated with an absence of fear of falling in community-dwelling older adults (Shirooka et al., 2017; Uemura et al., 2012). There may come a point at which a person is no longer able to appraise their physical function and risk of falling, thus becoming insensitive to fear of falling.

However, other research argues against anxiety being a prodrome of cognitive decline. Evidence that assists in disentangling the prodrome or causal risk factor debate includes longitudinally

examining the impact of anxiety at earlier ages (e.g., midlife anxiety) on cognitive function and assessing the impact of anxiety severity on cognitive function (Kuring et al., 2020). To address the former question, a systematic review restricted their inclusion criteria to studies in which anxiety diagnosed during midlife preceded a diagnosis of dementia by at least ten years, thus minimizing the presence of prodromal anxiety, and found anxiety to be an independent risk factor for dementia (Gimson et al., 2018). In MCI populations, anxiety has been found to predict conversion of amnesic MCI to AD independent of depression, memory decline, or the extent of atrophy within AD-related brain regions over time (Mah et al., 2015) and may play a moderating role in preclinical AD by modifying the effect of beta amyloid and promoting more rapid cognitive decline (Pietrzak et al., 2015). In the context of MCI, anxiety appears to accelerate cognitive decline and increase the risk of progression to AD in a dose-dependent manner, suggesting that increasing levels of anxiety result in more deleterious cognitive outcomes (Mah et al., 2015; Pietrzak et al., 2015). Further evidence against anxiety's role as a prodrome comes from a recent systematic review and meta-analysis that tested but ultimately did not find evidence for a cross-sectional relationship between anxiety and the AD biomarkers tau and beta amyloid (Demnitz-King et al., 2023). While the association between AD pathology and presentation of anxiety symptoms appears unlikely based on this metanalytic study, it should be noted that biomarkers other than tau and beta amyloid were not explored and would require further research to clarify the extent of their relationship with anxiety (Demnitz-King et al., 2023).

The research presented thus far positions anxiety as an independent risk factor or prodrome of cognitive decline. As a risk factor, anxiety may exert its influence on cognitive function through biological or behavioural mechanisms; as a prodrome, anxiety may arise from distress to perceived cognitive impairment or be an early symptom of cognitive impairment that manifests before more profound cognitive and functional losses occur (Kuring et al., 2020). However, an alternative perspective is that anxiety and cognitive dysfunction co-occur and are mutually reinforcing, such that cognitive deficits (perceived or actual) increase distress and risk of developing anxiety, which may, in turn, precipitate further cognitive decline (An et al., 2024; Perna et al., 2015). Executive dysfunction, in particular, has been theorised to contribute to the risk of psychopathology by fostering maladaptive thinking and decreasing adaptive coping, which may trigger and maintain psychiatric conditions in late life (Moran, 2016; Zainal & Newman, 2018). More recent cohort studies spanning 3 to 26 years of observation have found evidence of a bidirectional relationship between anxiety and executive function within the same samples (Petkus et al., 2017; Zainal & Newman, 2021) that is partially

mediated by perceived stress (Jain et al., 2023). Considering how the hippocampus is impacted by stress and its involvement in memory formation and fear extinction (Mah et al., 2016), it is not surprising that cognitive deficits and anxiety may be able to exist simultaneously and affect each other via stress pathways such as the HPA axis.

While debate continues to surround the question of whether anxiety precedes or follows cognitive decline (Kassem et al., 2017; Santabábara et al., 2019), the two perspectives may be reconciled with the view that anxiety that manifests in the early stages of cognitive decline may accelerate pathological changes—for example, through perceived stress and elevated cortisol—and reduce one’s chances of recovering from mild forms of cognitive impairment.

2.4 Functional Social Support

Social support as a resource is fundamental to health and well-being, forged through intimate connections and interactions with others. The literature has distinguished two broad types of social support that, while related and interdependent, are distinct and may result in different outcomes for health. Social support is commonly divided into *structural* social support (the lack of which is sometimes termed social isolation) and *functional* social support (also called perceived social support or social support availability). While structural social support (SSS) refers to objective measures of one’s social network such as network size and frequency of contact (i.e., the quantity of social relationships), functional social support (FSS) refers to the extent to which one perceives that members of their social network will be available to help, care, or provide comfort when needed (i.e., the perceived quality of social relationships) (Sherbourne & Stewart, 1991). FSS may be further classified into subtypes such as emotional/informational support (e.g., provision of understanding, empathy, or advice), affectionate support (e.g., provision of love and physical affection), tangible support (e.g., provision of material aid or behavioural assistance), or positive social interaction (e.g., the availability of someone to have fun with) (Sherbourne & Stewart, 1991).

While there is a tendency to have greater FSS the larger one’s social network is (Harasemiw et al., 2019), this is not always the case. Some individuals may perceive themselves as having adequate FSS from one or two close individuals, whereas others may feel dissatisfied with the availability of social support from a larger social network with whom they may not be close or tend to have negative interactions (Wister et al., 2019). Furthermore, dissatisfaction with the discrepancy between desired and actual social relationships may increase feelings of loneliness (Peplau & Perlman, 1982). While

FSS and loneliness are both subjective evaluations of one's social relationships, loneliness is more emotional, reflecting a desire to have greater social connection (Newall & Menec, 2020; Wister et al., 2019). Low FSS may contribute to feelings of loneliness if one judges that they do not have a sufficient number of people on whom to rely for support (Ellwardt et al., 2013; Yeh & Liu, 2003). Despite the close associations between the types of social support, research suggests FSS may be more important for health outcomes than structural (T.-Y. Chen & Chang, 2016; Costa-Cordella et al., 2021; Melchior et al., 2003) or received social support (Gable & Bedrov, 2022). Indeed, FSS has been proposed as a mechanism by which social isolation impacts health and well-being, where FSS takes on a stress-buffering role (Gable & Bedrov, 2022). Specifically, the belief that others will respond to one's needs when life is difficult helps to lower stress by facilitating reappraisal of negative situations and increasing adaptive coping. An emerging body of research also suggests that having supportive others who respond enthusiastically and encouragingly can help an individual capitalise on positive events, increasing positive affect, subjective well-being, self-esteem, and even physical health beyond the original event. To put it another way, FSS helps to "maximise the ups *and* minimize the downs of life" (Gable & Bedrov, 2022, p. 89).

Social connection is a universal human experience and necessary for survival, yet research suggests that Western societies are growing increasingly disconnected (Holt-Lunstad et al., 2017). Older adults may be particularly vulnerable to the negative health impacts of social disconnection as they attempt to cope with changes in social roles and relationships common in late life, prompted by widowhood, retirement, and the development of health conditions, which can see their frequency of contact with sources of support reduced (Nicholson, 2012). Vulnerability to low social support, even amongst ageing adults, however, is not a universal experience. Compared to older women, older men are less likely to report adequate social support and more likely to rely on their spouse or partner for social support, increasing their risk for lowered social support when their spouse or partner passes (Menec et al., 2019; Newall & Menec, 2020). In contrast, older women are better able to cope with loss of a partner by drawing on connections in other social circles (Newall & Menec, 2020). Additionally, as one ages, the discrepancy between structural and functional social support may also increase such that satisfaction with one's relationships remains high even in the presence of a diminished social network (Menec et al., 2019). This is consistent with the socioemotional selectivity theory of ageing which posits that, as individuals approach the end of their lives, they may choose to prune their social

networks to focus on close, emotionally meaningful ties rather than continue to maintain relationships that are less emotionally satisfying (Lang & Carstensen, 2002).

Given the positive impact that FSS has on health, it would be useful to understand the situations in which social support may be enhanced or disrupted, so that efforts can be made to increase resilience against barriers to social support when they arise. The COVID-19 pandemic is an example of an event that disproportionately impacted older adults—a group already at heightened risk of social isolation and loneliness pre-pandemic—limiting their participation in social activities, disrupting their access to social support, and exacerbating feelings of loneliness (Frank, 2020; Ooi et al., 2023). Measures designed to curb the spread of the virus (e.g., physical distancing, stay-at-home orders) changed how people moved within and interacted with their social network (Finlay, Meltzer, et al., 2024). While there is evidence of resilience and return to pre-pandemic levels of loneliness following initial lockdown (Kadowaki & Wister, 2023), some older adults continued to experience altered socialisation and leisure routines post-pandemic, including spending more time at home, reducing engagement in cultural and recreational amenities, and limiting social interaction when in public (Finlay, Guzman, et al., 2024; Finlay, Meltzer, et al., 2024). In what has been termed the “COVID-19 paradox”, the pandemic may have encouraged individuals to self-isolate to mitigate the risk of immediate infection at the expense of long-term mental and physical well-being (S. MacLeod et al., 2021). The same can be said about common mental health conditions like anxiety, where coping strategies such as avoidance may be used to decrease symptoms in the short term but at the risk of decreased psychological, social, or physical functioning in the long term. In particular, anxiety may interfere with efforts to acquire or preserve social support, contributing to lowered levels of social support in late life and indirectly affecting health in other ways, such as reducing cognitive function.

2.5 Social Support as a Mediator of Anxiety and Executive Function

As discussed previously, anxiety may adversely impact cognitive function through stress pathways or via maladaptive behaviours that reduce psychological, social, or physical functioning. Of these two major perspectives, the latter has received less attention in research, particularly the social mechanisms linking anxiety to cognitive decline. It is unclear whether anxiety impairs executive function by reducing FSS. Anxiety’s potential to lower FSS is of concern from a therapeutic point of view because it may be minimizing the benefits that FSS might otherwise have for health, including cognitive function.

Whether FSS serves as a mechanism through which anxiety impacts executive function, and to what extent, can be clarified by examining FSS as a mediator of the association between anxiety and executive function. Mediation through FSS can be conceptualised as two paths: 1) the association between anxiety (exposure) and FSS (mediator), and 2) the association between FSS (mediator) and executive function (outcome). Evidence of an association on *both* paths indicates that FSS might be a mediator linking anxiety to executive function. Given the overlap in definitions between FSS, SSS, and loneliness, as well as research where social support is treated as purely structural (Menec et al., 2019) or a mixture of structural and functional support (Evans, Llewellyn, et al., 2019; Newall & Menec, 2020; Wister et al., 2019), the following review of FSS and its associations with anxiety and executive function includes studies that have investigated these broader definitions of social support.

2.5.1 Anxiety and Functional Social Support

The impact of anxiety on social relationships has mostly been studied in the context of social anxiety, which involves a chronic and impairing fear of social evaluation and avoidance of, or withdrawal from, social interaction (B. Chen et al., 2024). However, anxiety in general has been associated with social dysfunction (Choi et al., 2017; Del Carlo et al., 2013; Faes et al., 2010; Hansson, 2002; Hui et al., 2012; Schultz et al., 2004; Stopa & Clark, 2000), social isolation (Merchant et al., 2020; Norton et al., 2012; S. Sun et al., 2024), loneliness (Domènech-Abella et al., 2019; Hwang et al., 2022; Lim et al., 2016; Margrett et al., 2011; Nuyen et al., 2020; R. Smith et al., 2021; Van Bogart et al., 2023), and lower FSS (Hahn et al., 2021; Panayiotou & Karekla, 2013; Shurgot & Knight, 2005; Stewart et al., 2022; Vaingankar et al., 2020). There is some debate as to whether general anxiety may be related to social dysfunction or if this association is entirely accounted for by depression (Schultz et al., 2004; Seivewright et al., 2004; Thomas et al., 2022), although social anxiety appears to be a unique predictor of loneliness independent of depression (Wolters et al., 2023).

How might anxiety lower FSS? As mentioned previously, anxiety is associated with a cognitive bias for negative or threat-related stimuli, and this bias can extend to social interactions (Gray et al., 2020). Those with social anxiety, compared to those without, are less likely to see others in a positive light when forming first impressions (Hepp et al., 2021), more likely to classify neutral expressions as threatening (Bell et al., 2011), more likely to discount positive interpersonal events (Vassilopoulos & Banerjee, 2010), and more likely to interpret ambiguous social events as negative while catastrophising unambiguous, slightly negative events (Stopa & Clark, 2000). Social anxiety has been

associated with lower perceived social support through communication styles, which may differ between men and women (Barnett et al., 2021). Furthermore, not only are those with social anxiety more dissatisfied with their interpersonal relationships, but others are also less likely to perceive socially anxious individuals in a positive manner. Anticipation of social evaluation or rejection may result in self-protective behaviour that comes across as less warm, less friendly, and less likeable, halting further relationship development (Alden & Taylor, 2004). Socially anxious behaviour coupled with others' unfavourable reactions could result in a self-perpetuating cycle where socially anxious individuals think and then behave in ways that provoke negative reactions from others, leading to a discrepancy in desired relationships, which may strengthen preexisting negative cognitive schemas of the self in relation to others and lead to social anxiety in future interactions (Alden & Taylor, 2004).

The literature has documented that lower perceived support can result from anxious individuals adopting avoidance strategies to escape anxiety-inducing problems and situations (Whitworth et al., 2013). However, anxiety is also characterised by interpersonal dependency, or an overreliance on others, that can increase interpersonal stress and lead to lower FSS (Darcy et al., 2005; Davila & Beck, 2002). To the extent that close relationships are marked by mutual perceived support, others in the relationship may experience diminished support and availability from the individual with anxiety (Zaider et al., 2010). If the response of others is to withdraw from anxious individuals who cannot provide them with adequate support, this may, in turn, lower the anxious person's FSS. Thus, anxiety-related behaviours in social relationships can lead to withdrawal in those with anxiety or withdrawal of others from them. A high degree of communication and accommodation of anxiety may protect against interpersonal strain exacerbated by an individual's anxiety (Zaider et al., 2010), but research on anxiety in romantic relationships suggests that anxious individuals may rely on partners to meet social demands and avoid social exposure themselves, which contradicts treatment efficacy research that recommends long-term reductions in anxiety symptoms be facilitated by exposure (Gordon et al., 2012). There is also evidence to suggest that these over-reliant interpersonal styles may place anxious individuals at risk of developing depressive symptoms (Grant et al., 2007), which may provoke further withdrawal from others and further decrease FSS (Iacono et al., 2024).

Most research on anxiety and interpersonal functioning has been conducted in younger populations (e.g., undergraduate university samples) and examined cross-sectionally, so data on how anxiety may impact FSS over time in older populations is lacking. Of the research that has been conducted, the situation appears similar to younger populations: older adults with anxiety report lower intimacy with

others (Drageset et al., 2013), higher loneliness (Domènech-Abella et al., 2019; Fees et al., 1999; Long & Martin, 2000; Margrett et al., 2011), and lower FSS (Couture et al., 2005; O’Conor et al., 2019). However, there may be some social experiences that are unique to anxious older adults. For example, older adults with social anxiety report greater loneliness than their younger counterparts, challenging the late-life motivation to focus on pruning less intimate relationships in favour of investing in more meaningful, if fewer, social relationships in line with the socioemotional selectivity theory of aging (Hoffman et al., 2021). FSS may protect against loneliness in socially anxious older adults by offsetting the impact of a reduced social network; however, this protection weakens with increasing social anxiety, as FSS is also ultimately impacted by a decreased social network (S. Sun et al., 2024). Compared to younger adults, older adults with anxiety may also be more reluctant to discuss the subject of their mental health with friends and family—members of their immediate social circle—and instead prefer to seek social support from others who share their experiences but with whom they may be less familiar (Alkholy et al., 2022). Stigma, shame, and embarrassment about one’s mental health can contribute to lowered FSS if the individual with anxiety fears that their problems will not be taken seriously or be viewed as a burden.

Like the association between anxiety and executive function, the relationship between anxiety and FSS is likely bidirectional (B. Chen et al., 2024; Domènech-Abella et al., 2019; Lim et al., 2016; Nuyen et al., 2020; Van Bogart et al., 2023). A perceived lack of support may increase the risk of poor mental health, including anxiety, while initial anxiety may lower subsequent perceived support (Santini et al., 2020). Furthermore, the discussion of anxiety and social support thus far has focused on their negative association, but anxiety and social support may also be *positively* related. For instance, anxiety may increase social support because individuals are seeking more support for their mental health (Robitaille et al., 2012), or social support may increase anxiety because individuals are made more aware of their needs and dependence on others (DiNicola et al., 2013). While the literature tends to agree that FSS is protective of mental health (Wickramaratne et al., 2022), this protection appears to weaken in the presence of higher levels of anxiety (Panayiotou & Karekla, 2013; Park et al., 2013; S. Sun et al., 2024), suggesting the potential for decreased social support over time and increased or sustained anxiety due to weakened perceived support.

2.5.2 Functional Social Support and Executive Function

Much of the literature on social support and its potential benefits for cognition has focused on older populations, an interest that has been fuelled by concerns of population ageing, a rise in the prevalence of dementia, and the ensuing need to investigate strategies to promote healthy ageing (Costa-Cordella et al., 2021). In light of evolutionary theories proposing that human brains have evolved to depend on social relationships as a key means of survival (Adolphs, 2009), it is not surprising that having low social support (structural or functional) is associated with a range of negative health outcomes, including poorer cardiovascular health, poorer mental health, cognitive decline, and a heightened risk of mortality (Leigh-Hunt et al., 2017). In contrast, higher FSS protects overall cognitive function, including executive function, in middle-aged and older adults (Mogic et al., 2023). While much of the research on social support and cognition has been undertaken to examine the influence of social support on subsequent cognitive function, there may be bidirectional effects in which those with declining cognition may face challenges engaging in social activities and gradually withdraw from social life; increased social isolation, in turn, may place them at risk of further cognitive decline (Porcelli et al., 2019).

Social connection and executive function are intertwined. Evolutionary perspectives contend that higher-order cognitive processes, such as executive function, evolved to enable cooperation and living in large groups (Adolphs, 2009; Ardila, 2008). Modern human children are born with markedly underdeveloped brains relative to other members of the hominid family and, to survive, must rely on social support from relatives over the course of development (Adolphs, 2009). Early life socialisation and positive social interaction have a cumulative impact on the brain, shaping vulnerability and resilience to future stress as well as conferring the personality, secure attachment style, and social skills needed for the formation of future supportive social networks (Uchino et al., 2018). In older age, social support continues to play an important role in protecting cognitive function by providing a source of mental stimulation, an idea closely related to the cognitive reserve hypothesis (Stern, 2012). Social interaction relies on a complex set of skills that includes knowing others' minds, knowing one's own mind, memory recall of past interactions and evaluations of others, and the ability to interpret and use social cues to act in a manner that is appropriate for a given situation—the coordination of which is facilitated by processes in the prefrontal cortex (Adolphs, 2009). Through the act of establishing, navigating, and maintaining social relationships, individuals continue to hone these competencies, encouraging the growth of neurons in preexisting pathways or in alternate

compensatory pathways which may be used to preserve current levels of functioning amid structural brain losses (Stern, 2012).

Besides the cognitive reserve hypothesis, another theory posits that social support enhances cognitive function more indirectly by facilitating and motivating healthy behaviours such as increased physical activity, less smoking/alcohol consumption, and adherence to treatment regimens, which in turn may improve mood, enhance cardiovascular, neuroendocrine, and immune function, and lower the risk of disease (Uchino, 2006; Uchino et al., 2018). However, social support may not always be optimal for making healthy lifestyle changes if individuals resent being controlled (Uchino et al., 2018). There is also evidence suggesting that this pathway to well-being through health-promoting behaviours differs by age (Mo et al., 2022). Adolescents and young adults may benefit more from social support while they are in the process of learning the skills to cultivate healthy lifestyles and maintain those skills through adulthood, but older adults may choose to adhere to an established and preferred lifestyle that may or may not support their health and thus may not experience improvements in health despite encouragement from one's social circle (Mo et al., 2022).

By far the most influential theory linking social support to cognitive health is the stress-buffering hypothesis, in which social support mitigates the negative impacts of stress on health. Having high social support may aid in fostering more positive appraisals of stressors, lowering exposure to stressors, improving problem-solving and coping strategies, or increasing feelings of self-control, self-efficacy, and self-esteem, which, in turn, lower cardiovascular reactivity, blood pressure, cortisol, and inflammation (Uchino et al., 2018), known risk factors for cognitive decline (Gulpers et al., 2016). Some work indicates that social support is more beneficial in the presence of higher levels of stress (Moskowitz et al., 2013) whereas studies on social support outside of stressful contexts suggest social support may have benefits regardless of the level of stress, such as by enhancing positive affective experiences (Gable & Bedrov, 2022). Having shown consistent benefits to health, FSS may have a greater stress-buffering effect than received social support due to perceptions of a "safety net" that fosters feelings of personal control and precludes received support that may be unhelpful, intrusive, or insensitive (Uchino et al., 2018). Unless social support is desired and perceived as responsive to one's needs, the actual receipt of it may increase, rather than decrease, stress (Uchino et al., 2018).

2.5.3 Mediation Studies on Anxiety, Social Support, and Cognition

While the summary of literature on the individual paths outlined in Sections 2.5.1 and 2.5.2 offers the preliminary suggestion that FSS may link anxiety to cognitive function, stronger evidence of a mediated effect can be attained by testing hypotheses with all three variables in a single model. This section provides an overview of the more limited mediation research that has been undertaken with anxiety, social support, and cognitive function in the same analytic model. A mediated effect can be suggested by models that account for the exposure (i.e., anxiety)—through adjustment, stratification, matching, standardization, or restriction—when estimating the effect of the mediator (i.e., FSS) on the outcome (i.e., executive function). If the effect of the mediator on the outcome changes, but remains significant after accounting for the exposure, the mediator is independently associated with the outcome, albeit impacted by the exposure. Similarly, adding the mediator as a covariate into the model can suggest a mediated effect if the addition of the mediator eliminates or partially eliminates the direct effect of the exposure on the outcome. In either case, it may not be clear whether the exposure impacts the mediator or vice versa unless temporality can be ascertained (e.g., the exposure was measured before the mediator). The strongest evidence of mediation, therefore, would come from studies that explicitly treat social support as a mediator of anxiety and cognitive function and, more advantageous still, utilise longitudinal data to preserve the temporal ordering of the exposure, mediator, and outcome.

A systematic search was conducted to retrieve literature of relevant mediation and related studies (see Appendix A for the search strategies and PRISMA flowchart, as well as Appendix B for a summary of relevant literature). Six studies assessed social support (structural or functional) as a mediator of anxiety and cognitive function, and of those six studies, four utilised longitudinal data. Two were cross-sectional (Bethell et al., 2024; Li et al., 2021), two were longitudinal with three waves of available data (Best et al., 2021; Stephan et al., 2024), and two had only two waves of available data, so the mediator was modelled at time 1 (Peeters et al., 2018) or the mediator was modelled at time 2 in the first mediation path and at time 1 in the second mediation path (McHugh Power et al., 2017). Overall, the six studies were generally mixed in terms of finding significant mediation. To begin with studies that did not contain evidence of significant mediation, Best et al. (2021) found that, after controlling for age, gender, race, and years of education, social engagement (wave 2) did not mediate the association between neuroticism (wave 1) and cognitive function (wave 3) in adults aged 60 and over. Similarly, Stephan et al. (2024) found that physical and cognitive

activities, but not social activity (wave 2), mediated the association between neuroticism (wave 1) and cognition (wave 3). Utilising two waves of data, Peeters et al. (2018) found an association between fear of falling at baseline and cognitive function at follow-up that was also not mediated by baseline social activity.

In contrast, Bethell et al. (2024), in their cross-sectional study, examined the mediation of neuroticism and cognitive function (memory and executive function) through both loneliness and social isolation and found evidence of significant mediation that did not differ by sex. Li et al.'s (2021) research question was unique compared to the other five studies in that they examined *serial* mediation of leisure activity and cognitive function through anxiety symptoms and loneliness, in that order. However, following the logic of mediation, as anxiety symptoms preceded loneliness in their model, anxiety symptoms served as an exposure for loneliness once leisure activity was controlled for. When accounting for leisure activity (a measure of participation in physical, cognitive, and/or social activities), loneliness mediated the association between anxiety symptoms and cognitive function in adults aged 65 and over. Specifically, anxiety symptoms were positively related to loneliness, and loneliness was negatively related to cognitive function (Li et al., 2022). In their two-wave study, McHugh-Power et al. (2017) found that social support (structural and functional) mediated the effect of neuroticism on cognitive function after two years of follow-up in adults aged 60 and over. Their findings differed from Li et al.'s (2021) in that neuroticism was negatively associated with social support, but social support still exerted a net positive effect on cognitive function. Considering the six mediation studies altogether, the studies that found evidence of significant mediation tended to utilise fewer waves of data and incorporated more subjective measures of social support (e.g., loneliness, FSS).

Due to the sparsity of literature on the mediation of anxiety and executive function through FSS, another 23 studies were reviewed as potential evidence to support further investigation of FSS as a mediator and to generate predictions for expected findings. These 23 related studies did not explicitly aim to determine FSS's role as a mediator but rather adjusted for anxiety or social support (structural or functional) to ascertain independent associations between either predictor and the outcome of cognitive function. Although these studies did not provide direct evidence of mediation, they offered grounds for further examination and discrimination of FSS as a mediator if, in their analyses, accounting for either anxiety or social support changed the other variable's relationship with cognitive function. The majority of these studies (Alper et al., 2020; Foong et al., 2018a; Fung &

Lam, 2017; Hsieh et al., 2021; James et al., 2011; Kassem et al., 2018; Kobayashi et al., 2022; Nakanishi et al., 1998; Nayak et al., 2019; Olaru et al., 2023; Patel et al., 2020; Peeters, Romero-Ortuno, et al., 2020; Sutin et al., 2019; Tomaszewski Farias et al., 2024; Verma et al., 2020; Watfa et al., 2011) included anxiety and social support simultaneously in models where the outcome was cognitive function. Three of the twenty-three studies assessed anxiety as a mediator of social support and cognitive function (Foong et al., 2018b; Q. Wang et al., 2022; Y. Wang et al., 2022), one study examined the association between social support and cognitive function restricted to a population with anxiety (Evans, Llewellyn, et al., 2019), and three studies examined the interaction of anxiety and social support on cognitive function (D'Amico et al., 2024; Liu et al., 2024; Segel-Karpas & Lachman, 2018).

Of the 23 studies mentioned, the majority reported associations between anxiety and cognitive function or between social support and cognitive function that were in the direction consistent with other literature, with a minority finding nonsignificant associations. Specifically, 10 reported negative associations between anxiety and cognitive function after adjusting for social and other covariates (Alper et al., 2020; Foong et al., 2018a; Fung & Lam, 2017; Kassem et al., 2018; Kobayashi et al., 2022; Liu et al., 2024, p. 202; Nakanishi et al., 1998; Olaru et al., 2023; Segel-Karpas & Lachman, 2018; Sutin et al., 2019) versus four that reported no association between anxiety and cognitive function (Hsieh et al., 2021; Nayak et al., 2019; Patel et al., 2020; Verma et al., 2020). Eleven studies reported positive associations between social support and cognitive function after adjusting for anxiety and other covariates (Foong et al., 2018a; James et al., 2011; Kobayashi et al., 2022; Liu et al., 2024; Nakanishi et al., 1998; Olaru et al., 2023; Patel et al., 2020; Segel-Karpas & Lachman, 2018; Verma et al., 2020; Q. Wang et al., 2022; Y. Wang et al., 2022) versus three that reported no association between social support and cognitive function (Foong et al., 2018b; Hsieh et al., 2021; Nayak et al., 2019). The remaining studies found partial support for the associations between anxiety and cognitive function or between social support and cognitive function. In a population restricted to individuals with anxiety, social support was positively associated with cognitive function cross-sectionally but not longitudinally (Evans, Llewellyn, et al., 2019). In two other studies, anxiety was negatively associated with cognitive function cross-sectionally but not longitudinally while social support was positively associated with cognitive function longitudinally but not cross-sectionally (Peeters, Romero-Ortuno, et al., 2020; Watfa et al., 2011). Another study found that both neuroticism and loneliness were independently and negatively associated with cognition cross-sectionally but not

longitudinally (Tomaszewski Farias et al., 2024). In contrast, in a study that stratified by sex, anxiety was negatively associated with cognitive function longitudinally but not cross-sectionally and in males only, while overall, social support was positively associated with cognitive function longitudinally but not cross-sectionally (D'Amico et al., 2024). In summary, findings from these studies appear to support the direct effect of anxiety on cognitive function (after accounting for potential mediators, including social support) and the effect of social support on cognitive function independent of anxiety, both of which help inform the results that might be expected from the mediated pathway.

In general, evidence from mediation studies appears equivocal in terms of whether social support mediates the association between anxiety and cognitive function, with slightly more supportive evidence from studies that utilised functional measures of social support or fewer follow-ups. Studies that adjusted for anxiety or social support in models with cognitive function as the outcome tended to support the independent effects of anxiety and social support on cognitive function. However, because in most studies anxiety or social support was not entered as a separate covariate, it remains unclear how their addition changes the main effect, over and above the other covariates. A finding of no effect of anxiety on cognitive function could result from a possible mediator or mediators accounting for the association, but whether social support is one such mediator cannot be determined at this time. Likewise, a finding of a significant effect of anxiety on cognitive function could signal possible mediation if the addition of social support weakened the association; however, among studies in which estimates initially unadjusted for social factors were also reported (Hsieh et al., 2021; Kassem et al., 2018; Kobayashi et al., 2022; Nayak et al., 2019; Verma et al., 2020), in no case was social support later assessed separately from other predictors in the model. On the other hand, an enduring association between social support and cognitive function after accounting for anxiety may signal the potential for a Path II association between social support and cognitive function (that controls for anxiety), although whether a significant path association translates to significant mediation must be determined by treating anxiety explicitly as an exposure in mediation analyses.

The inconsistency across studies, including the mediation studies, could be attributed to differences in research methodology (e.g., cross-sectional versus longitudinal data), differences in variables or measurement tools (e.g., anxiety symptoms versus trait anxiety, structural versus functional social support, global cognition versus specific subdomains), or underpowered analyses. The studies were about evenly divided between cross-sectional and longitudinal designs, although in the majority of

longitudinal studies, the predictors—including anxiety and social support as exposures or covariates—were measured at baseline. Most studies included neuroticism as a measure of anxiety, and trait anxiety tends to be more stable than symptoms measured at a given period and may exert cumulative effects on cognitive function over time. Seven studies adjusted for both functional and structural measures of social support (Alper et al., 2020; Bethell et al., 2024; Evans, Llewellyn, et al., 2019; Fung & Lam, 2017; Hsieh et al., 2021; McHugh Power et al., 2017; Segel-Karpas & Lachman, 2018), fourteen studies adjusted for structural only (Best et al., 2021; D’Amico et al., 2024; Foong et al., 2018a; James et al., 2011; Kassem et al., 2018; Liu et al., 2024; Nakanishi et al., 1998; Oлару et al., 2023; Patel et al., 2020; Peeters et al., 2018; Peeters, Romero-Ortuno, et al., 2020; Stephan et al., 2024; Sutin et al., 2019; Verma et al., 2020), five examined functional measures of social support while accounting for structural measures of social support (Foong et al., 2018b; Li et al., 2021; Nayak et al., 2019; Q. Wang et al., 2022; Y. Wang et al., 2022), and three studies adjusted for purely functional measures (Kobayashi et al., 2022; Tomaszewski Farias et al., 2024; Watfa et al., 2011). While definite conclusions cannot be drawn regarding which measure of social support (structural or functional) is more consistently related to cognitive function, the current literature suggests that social support may influence cognitive function independent of anxiety. Thus, it may be reasonable to hypothesise that social support in general is beneficial to cognitive function.

2.6 Potential Moderators of the Indirect Effect of Anxiety on Executive Function Through Functional Social Support

While the mediation studies cited in 2.5.3 did not set out to examine whether the indirect effect of anxiety on executive function through FSS was moderated by subgroup status (with the exception of one cross-sectional study by Bethell et al. [2024] that examined the indirect effect of neuroticism on executive function by sex only), it is plausible, based on research focused on the individual paths, that mediation of the association of anxiety and executive function by social support may differ based on age group and sex. More specifically, we might expect age group or sex to moderate the relationship between anxiety and social support and/or the relationship between social support and executive function.

2.6.1 Age

2.6.1.1 Anxiety and Functional Social Support

The interaction of age and anxiety can result in different outcomes for FSS depending on the relative strengths of the negative attentional bias associated with anxiety and the positive emotional bias of older age. While the socioemotional selectivity theory of ageing predicts that adults become more motivated to prioritise their emotional well-being in late life and will use emotional regulation strategies to help maintain their positivity (e.g., seeking more rewarding relationships and pruning less meaningful ties) (Carstensen et al., 2003), this tendency to focus on positive information may be attenuated by a bias for negative information in anxiety (Cabrera et al., 2020; Knight & Durbin, 2015), leading to poorer perceived relationships and greater feelings of loneliness (Hoffman et al., 2021). If anxiety is disrupting the positivity bias of older age, the social networks of anxious older adults may not necessarily be perceived as being more positive and emotionally meaningful as the socioemotional selectivity theory of ageing would suggest. Furthermore, if *other* older adults are motivated to maintain positive emotionality, they may distance themselves from a friend experiencing negative emotions and rescind their support (Matt & Dean, 1993). Thus, by experiencing a weaker positivity bias, anxious older adults may be at greater risk of poorer FSS and be more similar to their middle-aged counterparts who are less driven by emotional positivity (Carstensen & Gottman, 1995). Alternatively, the positivity bias of older age may endure and counteract anxiety by directing one's focus to maintaining relationships that are personally meaningful and downplaying interactions that are threatening or anxiety-inducing (Weisman et al., 2015), potentially protecting mental health and FSS in older adults. The sparse evidence on the buffering role of emotional positivity (Gray et al., 2020) underscores the need to examine the relationship between anxiety and FSS by age.

Although the discussion thus far has focused on more extreme feelings of anxiety, it may be that a different pattern of age effects would emerge for moderate levels of anxiety. Mental health literacy, stigma, and cost have been cited as barriers to reaching out for help regarding one's anxiety, and use of health services for anxiety has been found to be lowest in older adults while being the highest in middle-aged adults (Hohn & Maricutoiu, 2024). Willingness to access social support in middle-aged adults may increase, rather than decrease, perceptions of social support, particularly if levels of anxiety are sufficient to drive individuals to seek help instead of withdrawing from others. Non-pathological anxiety, as noted previously, serves an adaptive role by anticipating threats and

encouraging problem-solving, and may lead to higher FSS through an increase in support-related coping (Robitaille et al., 2012). Screening instruments that assess anxiety symptoms may be more likely than diagnostic criteria for an anxiety disorder to capture non-pathological levels of anxiety and the trend of higher levels of help-seeking in middle-aged adults. In summary, the impact of age on the association between anxiety and FSS may depend on the measure of anxiety, shaping not only the strength, but also the direction of the association.

2.6.1.2 Functional Social Support and Executive Function

The strength and direction of the effect of FSS on executive function may also differ by age. The stress-buffering model of social support (Cohen, 2004) suggests that FSS may play a larger role in mitigating the negative health impacts of stress in older versus middle age, as a lifetime of experiencing and adapting to stress increases allostatic load—an accumulation of wear-and-tear on neuroendocrine, autonomic, and immune systems—and decreases the body’s ability to respond effectively and efficiently to stress (Lavretsky & Newhouse, 2012). This might indicate that an absence of FSS during stressful times would have more severe ramifications for cognitive function in the oldest old compared to other older adults. In support of this hypothesis, studies that have examined the link between indicators of low social support and cognitive impairment have identified stronger associations in older adults compared to younger adults (Håkansson et al., 2009; Hatch et al., 2015; Olaru et al., 2023; R. S. Wilson et al., 2015). Recent research utilising data from the Canadian Longitudinal Study on Aging (CLSA) features more consistent evidence of moderation of age in the cognitive domain of executive function. One cross-sectional study suggested that measures of FSS and SSS were more important for adults aged 65 to 85 (versus those aged 45 to 64) in mediating the association between sensory impairment and executive function (Hämäläinen et al., 2019). A two-wave longitudinal study that examined FSS as a mediator of depression and executive function similarly found stronger associations in individuals aged 75 and over (Iacono et al., 2024). In contrast, a cross-sectional study of the association between FSS and memory found no evidence of moderation by age (Oremus et al., 2020), a finding that persisted in a two-wave longitudinal examination of FSS subtypes and memory (Yoo et al., 2023).

However, not all evidence is consistent that FSS is beneficial to cognitive function in older adults. Some studies have found that social support was more important for middle-aged adults (Atti et al., 2010; Cohrdes & Bretschneider, 2018). Other research suggests a specific window of time, between

the ages of 71 and 80 years, during which FSS would be most beneficial for cognition: those younger may not require a strong support network as a result of being mostly self-sufficient whereas social support after 80 years may be insufficient to offset the losses associated with cognitive impairment (Zamora-Macorra et al., 2017). A “needs-based” theory of social support contends that those who report higher FSS demonstrate a greater need and use of social support due to declining health, and that this increased use of social support may accompany more measurable cognitive decline in the ensuing years (Pillemer et al., 2019). This hypothesis may be applicable to the case of older adults who, due to advancing age, are more likely to experience declines in skills and abilities that would necessitate drawing on social support to compensate for such losses. The reciprocity theory of social support (Gleason et al., 2008; Uehara, 1995) further posits that the receipt of support that cannot be returned due to physical or cognitive limitations may come to be viewed as a burden or stressor, and this increase in stress and negative affect would then have detrimental effects on cognitive function (Du et al., 2023; Pillemer et al., 2019; Sims et al., 2014). Thus, FSS may exhibit positive or negative associations with executive function depending on an individual’s age.

2.6.2 Sex

2.6.2.1 Anxiety and Functional Social Support

The negative impact of anxiety on FSS may vary by sex. Women report higher levels of anxiety (Tetzner & Schuth, 2016) and are almost twice as likely to develop anxiety disorders compared to men (Canuto et al., 2018). These gender differences in anxiety are believed to be largely driven by socialisation factors related to expressing and coping with anxiety in a manner consistent with gender expectations (i.e., gender effects) rather than an enduring biological predisposition to anxiety (i.e., sex effects) (McLean & Anderson, 2009). Girls are nurtured in ways that promote anxious traits and lower self-efficacy in controlling their anxiety, whereas boys are encouraged to confront the source(s) of their anxiety directly as expressing fear and avoidance is viewed as less acceptable (McLean & Anderson, 2009). However, while anxiety appears to be more common in women, more recent reviews suggest that men may not be as protected from developing anxiety as originally thought (B. Chen et al., 2024; Fisher et al., 2021). Specifically, traditional masculine norms may act as a double-edged sword that, on one hand, promote proactive, problem-focused coping and self-reliance, but on the other, act as an additional source of distress when anxiety can no longer be controlled through the usual problem-solving strategies (Fisher et al., 2021). Beliefs that worry and fear are incompatible

with masculine gender norms may encourage concealment of anxiety and discourage the seeking of formal help, which may not only exacerbate symptoms but also contribute to lower reporting, diagnosis, and prevalence rates (Fisher et al., 2021, 2022). However, men who seek help may be more likely than women to actually receive mental health services (McLean et al., 2011). In terms of the impacts of anxiety on social support, while anxiety tends to be more disabling in women, characterised by higher comorbidity with other anxiety and mood disorders (McLean et al., 2011), research on anxiety in older adults indicates that the social networks and relationships of anxious women tend to be more resilient than those of anxious men (Curran et al., 2020). Given the unique risk and protective factors associated with each sex, and limited understanding on the impact of anxiety on FSS in the aging population, moderation of this association by sex should not be discounted when assessing mediation of anxiety on executive function through FSS.

Anxiety may also contribute to *higher* FSS depending on how each sex utilises social support. Anxiety in women may trigger coping behaviour that maintains social networks, known as the *tend-and-befriend* response, which increases the seeking of support from others and may protect women against the worst of their anxiety (Farhane-Medina et al., 2022; McLean & Anderson, 2009). In contrast, men are more likely to emphasise self-reliance when dealing with anxiety and may resort to denial, avoidance, or self-medication when more familiar strategies no longer prove effective (Fisher et al., 2022). When men do seek help, they prefer to access more informal sources of support (Fisher et al., 2022), though this may prove more challenging in late life due to older men's networks being overall less diverse than older women's (Menec et al., 2019; Newall & Menec, 2020). Gender differences in willingness to report anxiety, ability to cope with feelings of anxiety, and openness to accepting support from others may contribute to higher risk of suicidality (Fisher et al., 2022) and all-cause mortality (van Hout et al., 2004) in anxious men compared to anxious women. Thus, the traits that initially protect men against anxiety may benefit them until they develop anxiety, at which point these traits become detrimental to their health; an opposite pattern emerges in women, whose socialisation may increase their risk of developing anxiety but may also motivate them to maintain and draw on their social ties when they are anxious. Because women demonstrate more regular involvement in their social relationships and are more likely to reach out for social support when needed (Belle, 1991; Kawachi & Berkman, 2001), their perceptions of social support may not change after seeking help from others. On the other hand, men may be less likely to perceive having adequate levels of FSS until they are given the opportunity to utilise their social support. Thus, while anxious

men may be more reluctant to seek help, which may contribute to adverse outcomes such as increased mortality, those who do may consequently report higher FSS after receiving social support.

2.6.2.2 Functional Social Support and Executive Function

Sex or gender may also moderate the association between FSS and cognitive function. On one hand, having the protection of FSS may be particularly important in women due to dementia risk factors being more prevalent in this group. On the whole, women live longer, which increases their chances of developing dementia, but they are also likelier than men to develop depression, be less educated historically, exhibit higher levels of cortisol in response to stress, be more adversely impacted by the apolipoprotein E- ϵ 4 allele, show poorer outcomes for lifestyle behaviours such as smoking and alcohol consumption, and lose the protective cognitive benefits of oestrogen and testosterone after menopause (Sindi et al., 2021). On the other hand, men and women may demonstrate differential risk to losing the protection of FSS itself. The finding that men are more likely than women to rely on the support of their spouses solely versus other network ties (Newall & Menec, 2020) has prompted suggestions that FSS may be more important for men because it is more easily lost due to their relative lack of supportive social ties (Gurung et al., 2003). Women, on the other hand, report wider, more diverse networks from whom they draw their support (Newall & Menec, 2020) and would therefore derive the most benefit from more, rather than fewer, social ties, a state that also renders their FSS more resilient to changes in network structure (Gurung et al., 2003). Indeed, findings from a recent systematic review suggest that both structural and functional social support, but not marital status, may be relevant to cognitive function in women, as being married may be associated with caregiving stress at older ages (Costa-Cordella et al., 2021). CLSA studies that have examined moderation of social support and cognitive function by sex appear to reflect the pattern that social support (functional or structural) is more important for women (Hämäläinen et al., 2019; Iacono et al., 2024; Oremus et al., 2019; Rutter et al., 2024), although associations in men may be underestimated due to selective survival, in which men are likelier to die of cardiovascular causes before they reach the age to develop cognitive decline, leaving those who do survive to appear healthier overall (Sindi et al., 2021).

2.6.3 Age and Sex

It is also possible that age and sex interact to produce different patterns of mediation. While women generally report higher levels of anxiety compared to men, this is more the case in older women

(Tetzner & Schuth, 2016), which may translate to reduced social support and subsequently lower executive function in the oldest group of women. Amongst the oldest men, the ability to cope with feelings of anxiety may pose the greatest challenge after the loss of a spouse or partner (Sialino et al., 2021), exacerbated by a frequently reported lack of diverse support networks outside of that spouse or partner (Menec et al., 2019; Newall & Menec, 2020). At this stage, anxiety may not be able to further decrease social support that is already low (i.e., a floor effect), which may explain the nonsignificant indirect effect of mental health on executive function that has been observed in some groups of older men (Iacono et al., 2024). Furthermore, FSS has been found to mediate the association between depression and executive function, but in women aged 75 years and over only (Iacono et al., 2024). Such findings may reflect the higher burden of depression in older women over the life course, resulting in lower FSS and lower executive function at the oldest ages, whereas men may have been less likely to survive with the same burden and be captured in analyses (Iacono et al., 2024). A similar pattern may emerge with anxiety as a baseline predictor. Given that anxiety tends to develop early in life and persist without remitting, the cumulative burden of living with anxiety and chronic levels of stress may be most evident later in life, manifesting most markedly in cardiovascular disease and accelerated cognitive decline (Lenze & Wetherell, 2011).

2.7 Summary

Anxiety, FSS, and executive function exhibit complex and reciprocal associations with one another. Anxiety symptoms and anxiety disorders differ in symptom severity and health service utilisation, which may result in different consequences for FSS and executive function. Scholarship, both theoretical and empirical, is generally in agreement that social support predicts cognition independently of anxiety predicting levels of social support, although mediation studies explicitly evaluating social support as a link between anxiety and cognitive function have produced conflicting results. Similarly, evidence of age or sex moderating the mediated association of anxiety and cognitive function through social support is based on analyses of the discrete paths rather than the indirect effect itself. It is not clear which moderator is more important at which path, and no study to the best of the author's knowledge has explicitly investigated moderated mediation by FSS where anxiety is the exposure. The impact of age and sex requires further investigation due to the potential for disparate health outcomes in these subgroups.

Chapter 3

Rationale and Objective

3.1 Study Rationale

Current evidence on FSS as a mediator of anxiety and executive function is limited to studies that assess global cognition, leaving the long-term impacts of anxiety on cognitive domains such as executive function unclear. Most investigations relating anxiety to impaired executive function have been undertaken within cross-sectional psychological experiments, where deficits may be temporary and context-driven (i.e., attention or working memory is impaired only if a threatening stimulus is present and the cognitive task is difficult). Longitudinal research is needed to establish temporality and determine whether and to what extent anxiety (clinical diagnosis or subclinical symptoms) affects the trajectory of executive function over time. To reduce the potential for confounding and aid interpretation, controlling for multiple covariates is important. Thus, a dataset that includes at least three waves of observations to establish temporality of the mediated effect, a large selection of variables with which to address possible confounding, and a sample size to ensure sufficient statistical power to detect an effect is ideal for mediation analyses. Two mediation studies utilised data at three timepoints (Best et al., 2021; Stephan et al., 2024), but both examined neuroticism, employed a structural measure of social support, and did not consider moderated mediation, which may limit the generalisability of findings given the potential for the effect of anxiety on FSS, and FSS on executive function, to differ by age and sex. In particular, moderated mediation has not been explored in younger cohorts of aging adults. To summarise, few studies have examined the role of FSS in linking anxiety to changes in executive function. While some evidence is suggestive of social support mediating the association between anxiety and cognitive function (Bethell et al., 2024; Li et al., 2021; McHugh Power et al., 2017), prior mediation work has tended to focus on neuroticism as an exposure, SSS or a mix of SSS and FSS as the mediator, and global cognition as the outcome; none have explicitly sought to examine mediation of anxiety and executive function through FSS while integrating longer follow-up periods and moderation by age and sex.

This thesis addressed these gaps in the literature by drawing on data from the CLSA. The CLSA is a population-based, panel study designed to explore health over the life course from middle to old age (Raina et al., 2019). At the time of this thesis, the dataset includes three waves of data and the availability of a wide range of sociodemographic, health, and lifestyle covariates to analyse the

potential for confounding when exploring complex temporal relationships. Furthermore, the scope of cognitive tests administered in the CLSA offers an opportunity to examine the impact of anxiety and FSS on the specific cognitive domain of executive function. This thesis was able to leverage the middle-aged and older adult sample to examine diverse age ranges and determine whether mediated effects vary by age as well as by sex. Moderated mediation was conducted using conditional process analysis, an analytic technique that combines moderation and mediation into one statistical model (Hayes, 2022). To the author's knowledge, this is the first study to examine whether FSS mediates the association between anxiety and executive function in a large sample of community dwelling, middle-aged and older adults. A greater understanding of FSS as a link between anxiety and executive function will inform the development of programs that support the cognitive health of aging adults by clarifying whether interventions for anxiety should include programming that strengthens FSS (e.g., social prescribing). Knowledge of whether this mediation is moderated by age and sex will help target social interventions to the most vulnerable subgroups, ensuring that public health initiatives have the greatest impact for the individuals most in need of support.

3.2 Objectives

Using baseline (T0), first follow-up (T1), and second follow-up (T2) data from the CLSA Comprehensive cohort, this study assessed whether FSS (T1) mediated the association between anxiety (T0) and executive function (T2) within sex and age subgroups, controlling for baseline sociodemographic, social, health, and lifestyle covariates.

Chapter 4

Methods

4.1 Sample

4.1.1 Data Source

The CLSA was established to prospectively explore the needs of an aging population (Raina et al., 2019). The total CLSA cohort of 51,388 participants is composed of two complementary cohorts: Tracking and Comprehensive. Recruitment was completed in 2015 and participants were to be followed for at least 20 years, with data being collected every three years (Raina et al., 2019). At the time of this thesis, three timepoints of data were available for analysis: baseline (T0), follow-up 1 (T1), and follow-up 2 (T2).

The Comprehensive cohort consists of 30,097 community-dwelling Canadians aged 45 to 85 years at baseline (CLSA, n.d.). Participants were recruited through provincial healthcare registration databases, random digit dialling, and the Québec Longitudinal Study on Nutrition and Aging (CLSA, 2017a; Gaudreau et al., 2007). All lived within a 25 to 50 km radius of 11 data collection sites located in the provinces of Alberta, British Columbia, Manitoba, Newfoundland and Labrador, Nova Scotia, Ontario, and Québec (Raina et al., 2008). Sampling was stratified by sex, age group, province, and data collection site to ensure adequate statistical power in the estimation of parameters (CLSA, 2023). Later, during baseline recruitment, participants with lower levels of education were also oversampled (CLSA, 2023). The CLSA excluded participants who resided in the three territories or on federal First Nations reserves or other First Nations settlements, who were full-time members of the Canadian Armed Forces, or who lived in long-term care institutions (Raina et al., 2008). Furthermore, the CLSA excluded individuals who showed obvious signs of cognitive impairment at baseline and could not provide consent or reliable information, as well as those who could not speak English or French (Raina et al., 2008). Compared to the Tracking cohort, which undergoes interviews via telephone, the Comprehensive cohort completes assessments through a combination of in-home interviews and on-site data collection (questionnaires, physical examinations, and biological samples) (Raina et al., 2008). As a result, more measures are available for analysis in the Comprehensive cohort, including tests of executive function and measures of anxiety.

4.1.2 Analytic Sample

Participants were included in the analytic sample if they were in the Comprehensive cohort at T0, T1, and T2 and had complete data on all variables described in Section 4.2. On March 16, 2020, the CLSA imposed COVID-19 restrictions that altered data collection procedures for the remainder of the Comprehensive cohort at T2 (CLSA, 2024). Specifically, assessments which were previously administered in person were then completed by telephone. Three of the five tests of executive function could not be administered due to these changes. To ensure executive function scores reflected consistent testing conditions and the same number of cognitive tests were included at all three timepoints, only participants who completed an in-person DCS visit (i.e., assessments completed before these COVID-19 protocol changes) were included in the analytic sample. Additionally, participants who self-reported a physician’s diagnosis of Alzheimer’s disease or memory problems at baseline or had a missing response to these questions were excluded. Those who completed their executive function tests bilingually were also excluded, as the process of deriving the executive function measure involved standardising scores separately for those who tested in English or in French (see Section 4.2.2 for more information). After applying these exclusions, the final analytic sample comprised 6,719 individuals. Appendix C provides additional information on how this sample was derived.

4.2 Measures

4.2.1 Exposure: Anxiety

Anxiety at baseline (T0) was assessed by a self-reported clinical diagnosis of anxiety or self-reported anxiety symptoms. Appendix D includes additional details on each of these measures. In brief, clinical anxiety (yes/no) was self-reported in response to the question: “Has a doctor ever told you that you have an anxiety disorder such as a phobia, obsessive-compulsive disorder or a panic disorder?” Anxiety symptoms were a sum of four items related to anxiety (Brooks et al., 2006; Buchanan, 2020; Lace et al., 2019) from the Kessler Psychological Distress Scale (K10) (Kessler et al., 2003). Participants were asked to rate their symptoms on a 5-point Likert scale from 1 (all of the time) to 5 (none of the time). To sum the items so that a higher score corresponded to higher symptoms, the categories were reverse coded starting at 0 for “none of the time” and ending with 4 for “all of the time”. In the CLSA, two items on the K10 were skipped if answers to a related question were “none of the time” (CLSA, 2017b). Specifically, if participants answered that they felt nervous

“none of the time”, the question related to “feeling so nervous that nothing could calm you down” was skipped and labelled as missing. Similarly, if participants answered they felt restless “none of the time”, the question related to “feeling so restless that you could not sit still” was skipped and labelled as missing. To include these responses in the data, skipped items were recoded as 0 (none of the time) if answers to the preceding related questions were also “none of the time”. While the K10 can be analysed continuously either as a sum of responses or with a cut-off score (Andrews & Slade, 2001), cut-off scores for subscales within the K10 have not been well validated in older or non-U.S.-based populations (Lace et al., 2019). Therefore, to minimise the uncertainty of an optimal cut-off score, the K10 anxiety subscale was analysed as a continuous variable.

4.2.2 Outcome: Executive Function

Executive function was a composite measure of five cognitive tests administered in the CLSA. These measures were selected for use in the CLSA based on literature reviews and their psychometric properties (e.g., sensitivity, specificity, responsiveness), sensitivity to cognitive change, and relevance to the study population (Tuokko et al., 2017). The five executive function tests are the Mental Alternation Test (MAT) (Billick et al., 2001; Teng, 1995), the Animal Fluency Test (AFT) (Crossley et al., 1997), the Stroop Neurological Screening Test-Victoria Version (Stroop-VV) (Bayard et al., 2009, 2011; Moroni & Bayard, 2009; Spreen & Strauss, 1998; Strauss et al., 2006; Troyer et al., 2006), the Controlled Oral Word Association Test (COWAT) (Benton & Hamsher, 1978), and the Time-based Prospective Memory Test (TMT) (Lowenstein & Acevedo, 2001). The Stroop-VV was modified for use in the CLSA whereby errors made during the test were recorded but not reflected in the interference score due to extreme skewness in this variable (i.e., most participants did not make errors) (O’Connell et al., 2023). Descriptions for each of these tests can be found in Appendix D. A composite score of executive function was derived for each of the three timepoints by standardising individual test scores based on the mean and standard deviation of the sample at the relevant timepoint, with standardisation taking the form of a z-score (mean = 0; standard deviation = 1). Then, the z-scores of each cognitive test were summed together to obtain the composite score, as detailed in previous work (Iacono et al., 2024; Oremus et al., 2019). For executive function at baseline (T0) and follow-up 1 (T1), standardisation of scores was based on the entire available sample at that timepoint. Before standardising scores for executive function at follow-up 2 (T2), the sample was restricted to participants who completed their DCS visit pre-COVID-19. This was done so that the z-scores of the MAT and AFT were not based on the entire sample at T2, which would have reflected a mix of in-

person and telephone assessments, but on the subset of participants who were able to complete all their executive function tests in person, consistent with previous timepoints. Raw cognitive test scores were converted into z-scores separately for French and English speakers to account for differences in cognitive performance due to language (Tuokko et al., 2017, 2020). The executive function composite score was analysed as a continuous variable to retain detailed variation in performance across the sample.

4.2.3 Mediator: Functional Social Support

Functional social support (FSS) was assessed using the 19-item Medical Outcomes Study-Social Support Survey (MOS-SSS) (Sherbourne & Stewart, 1991). The original MOS-SSS included an additional item that assessed SSS; to only reflect FSS, this item has been omitted consistent with the usual derivation of overall FSS. Eighteen items on the scale belong to one of four subtypes of FSS and an additional item did not belong to any subscale. Details on each of the 19 items can be found in Appendix D. Items were rated on a 5-point Likert scale from 1 (none of the time) to 5 (all of the time). To reduce missing data and avoid exacerbating bias associated with a healthier sample at follow-up, up to one item on the MOS-SSS was imputed using the mean of a subscale (if the missing item belonged to said subscale) or using the mean of the other 18 items on the MOS-SSS (if the missing item did not belong to any subscale). A more detailed description of the imputation can be found in previous work (Lupoi, 2024). Once imputation was conducted, overall FSS was calculated by taking the mean of all items (including the imputed item, if up to one item was imputed). Deriving overall FSS by averaging all 19 items on the MOS-SSS is consistent with the approach taken by the CLSA, except that the CLSA transforms overall FSS scores on a scale that ranges from 0 to 100 (CLSA, 2018). Since this transformation is not necessary for the purposes of the current analyses and interpretation is more intuitive with the original response categories, the FSS scores in this thesis retain the MOS-SSS's ordinal response categories, which range from 1 to 5. FSS was analysed continuously to retain variation in FSS reported across the sample.

4.2.4 Moderators: Age Group and Sex

Age group and sex at baseline were tested as moderators of the indirect effect of anxiety on executive function through FSS. The age categories were 45–54, 55–64, 65–74 and 75+ years old. Sex was reported based on the question: “Are you male or female?” Gender was not assessed at baseline in the CLSA.

4.2.5 Covariates

There are several variables related to anxiety and executive function that may confound their mediated association. These covariates were measured at baseline and categorised as sociodemographic, social, health, or lifestyle factors. Covariate selection was informed by the CLSA's complex sampling strategy (CLSA, 2017a), literature on anxiety in older adults (e.g., Bryant et al., 2008; Wolitzky-Taylor et al., 2010), and previous work on social support and cognitive function using CLSA data (Endresz et al., 2025; Iacono et al., 2024; Ohman et al., 2022; Oremus et al., 2019, 2020; Rutter et al., 2024; Yoo et al., 2023). Covariates may be associated with the exposure, mediator, or outcome. Categories with a cell count of <3% in the analytic sample were collapsed to ensure adequate statistical power in analyses.

4.2.5.1 Sociodemographic Covariates

In addition to being moderators, age and sex were assessed as covariates as they are associated with anxiety (Bethell et al., 2024; Olaru et al., 2023), social support (Menec et al., 2019; Newall & Menec, 2020), and cognition (Tuokko et al., 2020).

Indicators of socioeconomic status such as education and income influence mental health outcomes (Barakat & Konstantinidis, 2023) and cognitive function (A.-Y. Wang et al., 2023). Education was assessed in terms of highest educational level attained: less than high school, high school graduate, some post-secondary education, or post-secondary degree/diploma. Total annual household income was divided into five levels: < \$20,000; ≥ \$20,000 and < \$50,000; ≥ \$50,000 and < \$100,000; ≥ \$100,000 and < \$150,000; and ≥ \$150,000. Participants rated their income adequacy (i.e., whether their income met their needs) as totally inadequately, not very well, with some difficulty, adequately, or very well.

Province was controlled for as there are regional differences in social support and cognition in Canada (Oremus et al., 2019). Participants in the Comprehensive cohort were recruited from the provinces of Alberta, British Columbia, Manitoba, Newfoundland and Labrador, Nova Scotia, Ontario, and Quebec. Additionally, because participants were recruited based on age, sex, education, and province, the CLSA (2023) also recommends adjusting for these variables in analyses.

4.2.5.2 Social Covariates

Structural social support indicators such as marital status and living arrangement were controlled for due to their associations with mental (Hakulinen et al., 2016; Stewart et al., 2022), social (Holt-Lunstad, 2017), and cognitive health (Evans, Martyr, et al., 2019). Marital status was classified as partnered, single/never married, widowed, or divorced/separated. Living arrangements were dichotomised as either living alone or living with others.

Social support may also be derived from sources other than people: animal companionship is associated with anxiety (Bolstad et al., 2021) and cognitive function (Friedmann et al., 2023). Thus, pet ownership was also included as a covariate and was dichotomised as yes or no.

4.2.5.3 Health Covariates

Self-rated health, functional impairment, chronic conditions, and depression are closely associated with anxiety (Lenze, 2003; Van Der Weele et al., 2009; Wolitzky-Taylor et al., 2010) and cognitive function in older adults (Bennett & Thomas, 2014; Bourassa et al., 2017; Wittenberg et al., 2022). Self-rated health, functional impairment, and chronic conditions have also been linked to other measures of social support, such as loneliness and social isolation (Menec et al., 2019). Participants rated their general health as poor, fair, good, very good, or excellent. Their number of chronic conditions was categorised as 0, 1, 2, and 3+. Functional impairment, a derived variable based on a modified version of the Older Americans' Resources and Services Multidimensional Functional Assessment Questionnaire (Fillenbaum, 2005; Fillenbaum & Smyer, 1981), was dichotomised as yes (mild to total impairment) or no (absence of impairment). Self-reported clinical depression was assessed by the question: "Has a doctor ever told you that you suffer from clinical depression?" and was dichotomised as yes or no. Depressive symptoms were assessed in the last 30 days, summed from six items on the K10, which were rated as "none of the time", "a little of the time", "some of the time", "most of the time", or "all of the time" (refer to Appendix D for the specific depression items).

4.2.5.4 Lifestyle Covariates

Anxiety is often associated with substance use, such as smoking and drinking (Mathew et al., 2011). Smoking and alcohol use are also associated with cognition (Peters, 2012). Alcohol use was defined as nonuser (no alcohol consumed in the last 12 months), occasional user (alcohol consumed less than once per month), or regular user (alcohol consumed at least once per month for the last year).

Smoking status was defined as current smoker, former smoker (do not smoke currently but have in the past), and never smoker (do not smoke and never have).

4.3 Descriptive Analysis: Univariate and Bivariate

All descriptive analyses were performed on unweighted data due to the lack of available weights for all timepoints and to maintain consistency with unweighted multivariable analyses (Section 4.4). Frequencies were computed for categorical variables. Means and standard deviations were calculated for continuous variables; since most data were skewed, medians and interquartile ranges were also computed. For bivariate associations between continuous variables, Spearman's rank correlation coefficient, a nonparametric alternative to Pearson's correlation coefficient, was reported. For bivariate associations between categorical variables, a chi-square test was used. For bivariate associations between a categorical variable and a continuous variable, a t-test or ANOVA was used when the continuous variable was normally distributed, and a nonparametric Mann-Whitney U or Kruskal-Wallis test was used when the continuous variable was highly skewed. Post-hoc analyses were then conducted to assess significant pairwise differences between levels of a multi-categorical variable: Tukey's test and Dunn's test were employed for normally distributed data and skewed data, respectively.

4.4 Multivariable Analysis: Moderated Mediation

Conditional process analysis, developed by Andrew Hayes (2022), is an ordinary least squares (OLS)-based regression analysis capable of assessing moderation, mediation, or a combination thereof. For this thesis, the PROCESS macro version 4.3.1 for SAS was used to address moderated mediation of the indirect effect of anxiety on executive function. The exposure (anxiety), mediator (FSS), and outcome (executive function) were modelled at T0, T1, and T2, respectively, and age group and sex were assessed as moderators of the mediated effect. Separate analyses were conducted on the two measures of anxiety, self-reported clinical anxiety and anxiety symptoms, where each measure was the exposure variable of their respective models. Unweighted data were used in all moderated mediation models.

4.4.1 Estimating the Indirect Effect

When an effect is partially mediated, some of the total effect of an exposure X on outcome Y is explained by a mediator M. The mediated path through which X affects Y is the *indirect effect* of X

on Y, while the remaining effect not explained by M is termed the *direct effect* of X on Y. The total effect of X on Y (c) can therefore be modelled with the following equation: $c = c' + ab$, where c' is the direct effect of X on Y controlling for M, and ab is the indirect effect of X on Y through M. The regression coefficient ab is the product of the effect of X on M (a , also called Path I) and the effect of M on Y when controlling for X (b , also called Path II). Evidence of moderated mediation will generate multiple ab coefficients for the specified moderator, for up to two moderators with PROCESS: W and Z (Hayes, 2022). The indirect effect conditioned on a moderator quantifies the amount by which two cases with the same value of the moderator would differ on Y through M if they differed by one unit on X. Moderation of the indirect effect by W or Z may occur on paths I or II in various combinations. Refer to Figure 1 for a conceptual diagram of a moderated mediation model based on the variables in this study.

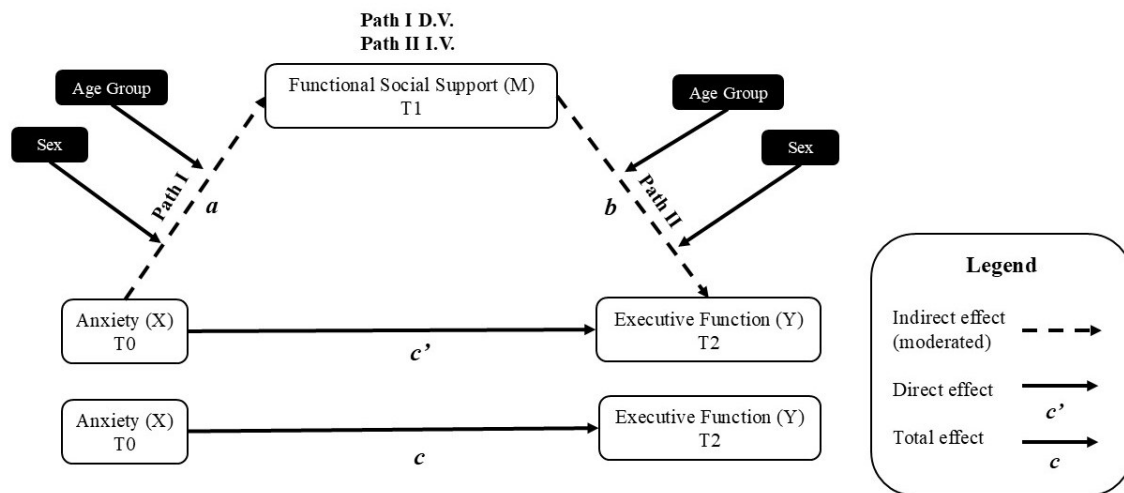


Figure 1. Conceptual Moderated Mediation Model

Note. Age group and sex are represented as potential moderators of the indirect effect of anxiety on executive function through functional social support.

DV = dependent variable; IV = independent variable; M = mediator; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; X = exposure; Y = outcome.

Conditional process analysis uses an index test of mediation to quantify and test the indirect effect, namely, whether the product, ab , differs from zero. Evidence of significant mediation is informed by whether zero falls outside a 95% bootstrap confidence interval (CI). The 95% bootstrap CI is constructed from an empirically derived representation of the sampling distribution of ab . To derive this sampling distribution, observations are randomly sampled with replacement from the original dataset, creating a bootstrap sample. Using this bootstrap sample, ab is computed. This procedure to create a bootstrap sample and then estimate ab within that bootstrap sample is repeated 10,000 times, saving the value of ab each time. After 10,000 resamples and estimations, a distribution of ab is created, sorted from low to high. The 95% bootstrap CI captures the values of ab that fall within the upper and lower bounds of a 95th percentile range. If the 95% bootstrap CI does not contain 0, one can conclude that the indirect effect is statistically significant. The index of *moderated* mediation observes a similar approach to hypothesis testing. To generate 95% bootstrap CIs for conditional indirect effects, 10,000 bootstrap samples are taken and ab is estimated for a given value of a moderator. A 95% CI is then calculated for this bootstrapped sampling distribution. This process is repeated for various values of the moderator and then PROCESS outputs all ab estimates for the levels of the moderator(s) at the end. Thus, separate mediation analyses do not have to be run for each stratum to obtain their indirect effect.

The index approach using percentile bootstrapping makes no assumption of the shape of the sampling distribution of ab , which is unlikely to be normal, nor does it require the components a or b to be statistically significant, since evidence of mediation is based on a single statistic, the ab product. The index approach purports to overcome limitations associated with component approaches to mediation analyses, such as the causal steps approach proposed by Baron and Kenny (1986) and the more recent joint-significance test proposed by Yzerbyt et al. (2018). Advocates of the index approach contend that component approaches may be more prone to reduced power and Type II error because multiple statistical tests must be conducted to infer the presence of mediation (Hayes, 2022). Furthermore, testing individual paths, by themselves, does not quantify the mediated relationship of interest nor speak to the confidence of this estimate (Hayes, 2022). More recently, proponents of the component approach have demonstrated that requiring statistical significance of *both* component paths as criteria to establish mediation does not inflate Type II error and is more inherently conservative than the index approach, which, reliant on one inferential test, is more susceptible to Type I error (Yzerbyt et al., 2018). These findings have guided a set of recommendations that support

the use of the joint-significance test *in conjunction with* the index test, acting as a gatekeeper for further analysis involving quantifying the magnitude of the significant mediated effect using the index test (Yzerbyt et al., 2018).

The moderated mediation analysis in this thesis employed the index test over the joint-significance test based on several factors. First, the joint-significance test relies on assumptions of normality and homoscedasticity of residual errors in the estimation of M and Y (Hayes, 2022; Yzerbyt et al., 2018). The nonparametric index test with percentile bootstrapping does not require these assumptions and may produce more reliable inferences when the assumptions of regression analysis are not met (Hayes, 2022). Second, conducting the joint-significance test on a moderated mediation model decreases power due to lower sample sizes in each stratum of the moderator. Alternatively, the component paths may be estimated without moderators to preserve power, but this strategy was less compatible with the current analysis, which involved mediation that was moderated. The index test, which exploits information contained in the entire dataset to estimate conditional indirect effects, was the preferred approach due to its potential to overcome issues of power associated with subgroup analysis (Hayes, 2022). Third and last, a criticism of the index test is that focussing on the ab product alone cannot adequately lead to an understanding of the mediated effect, and that the plausibility of the mediation model depends on a critical examination of the component paths (Yzerbyt et al., 2018). While proponents of the index test maintain that significance of the component paths is not required to establish mediation, they advocate for examination of the size and sign of the component paths when interpreting the indirect effect (Jollineau & Bowen, 2023). For example, Type I error may result where one component path is small and far from significant and the other is large and highly significant (often attributable to a poorly chosen mediator) (Jollineau & Bowen, 2023; Yzerbyt et al., 2018). PROCESS facilitates the identification of such cases by including path effects when estimating the mediated effect (Hayes, 2022). Thus, the index test was the preferred analytical technique of this thesis because it does not rely on normal distribution assumptions, can preserve statistical power when estimating conditional indirect effects, and can simultaneously output (moderated) path effects to aid in interpreting (moderated) indirect effects.

4.4.2 Building the Moderated Mediation Model

Before assessing moderated mediation, the moderated mediation model had to be specified. The first step in constructing the model was to create a conceptual diagram, which included age group and sex as potential moderators of the indirect effect of anxiety on executive function through FSS (Figure 1). From there, interactions with age group and sex were tested in fully adjusted linear regression models at Paths I and II, starting with three-way interactions (i.e., $X*W*Z$ or exposure*age group*sex) and proceeding to lower order interactions if higher order interactions were nonsignificant. Since the current analysis included two moderators, age group and sex, the two-way interaction terms containing age group and sex were tested simultaneously before each interaction term was tested individually. PROCESS provides a joint test of interaction when XW and XZ are both added to the model; this is a test of the difference in R^2 that results when the two products are added to the model already containing X , Y , W , and Z as individual predictors (Hayes, 2022). This difference in R^2 follows the F-distribution under the assumption that neither W nor Z moderate X 's effect on Y ; a statistically significant increase in R^2 would indicate that X has explained additional variance in Y when allowed to simultaneously vary as a function of W and Z (Hayes, 2022). If the joint test of interaction was significant, the individual contributions of W and Z were then examined to determine which moderator was significant.

Following this hierarchical testing of interactions, a moderated mediation model was created based on the significant interactions at each path, and indirect effects were estimated for different levels of the moderator(s); if no interactions were significant, an overall indirect effect was estimated on a simple (i.e., unmoderated) mediation model. Separate moderated mediation models were constructed for clinical anxiety and anxiety symptoms. The final step in model building was to conduct sensitivity analyses to address data limitations (see Section 4.4.3).

For each moderated mediation model, two versions of the model were created to test the effect of adding covariates to the model (Table 1). In the *base model*, only the exposure, mediator, outcome, significant interactions with the moderator(s), and prior measures of the mediator and outcome (as recommended by Hayes [2022]), were included. In the *final model*, sociodemographic, social, health, and lifestyle covariates were added to the base moderated mediation model. Models were specified with the most appropriate measure of depression: the model with anxiety symptoms as the exposure added depressive symptoms as a covariate, while the model that included clinical anxiety added clinical depression instead, to emulate the severity and chronicity of the exposure.

Table 1. Analysis Plan Based on Conceptual Moderated Mediation Model

| | Path I <i>Anxiety (X) → FSS (M)</i> | Path II <i>FSS (M) → Executive Function (Y)</i> |
|---|--|--|
| Model 1 <i>Base</i> | <p>Exposure (X)</p> <ul style="list-style-type: none"> • Clinical anxiety (T0) or anxiety symptoms (T0) <p>Outcome (M)</p> <ul style="list-style-type: none"> • FSS (T1) <p>Moderator(s)</p> <ul style="list-style-type: none"> • Age group (T0) • Sex (T0) <p>Antecedent measures¹</p> <ul style="list-style-type: none"> • FSS (T0) • Executive function (T0) | <p>Exposure (M)</p> <ul style="list-style-type: none"> • FSS (T1) <p>Outcome (Y)</p> <ul style="list-style-type: none"> • Executive function (T2) <p>Moderator(s)</p> <ul style="list-style-type: none"> • Age group (T0) • Sex (T0) <p>Antecedent measures¹</p> <ul style="list-style-type: none"> • Clinical anxiety (T0)² or anxiety symptoms (T0)² • FSS (T0) • Executive function (T0, T1) |
| Model 2 <i>Fully Adjusted</i> | <p>Base Model +</p> <p>Sociodemographic covariates (T0)</p> <ul style="list-style-type: none"> • Age group, sex, province, education, annual household income, income adequacy <p>Social covariates (T0)</p> <ul style="list-style-type: none"> • Marital status, living arrangements, pet ownership <p>Health covariates (T0)</p> <ul style="list-style-type: none"> • Self-rated health, number of chronic conditions, functional impairment, clinical depression (when X = clinical anxiety) or depressive symptoms (when X = anxiety symptoms) <p>Lifestyle covariates (T0)</p> <ul style="list-style-type: none"> • Smoking status, alcohol use | |

Note. FSS = functional social support; M = mediator; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; X = exposure; Y = outcome.

¹Prior measurements of the mediator and outcome are controlled for as recommended by Hayes (2022).

²PROCESS also controls for X at Path II.

4.4.3 Sensitivity Analyses

4.4.3.1 Omitting T1 Executive Function

While the literature recommends adjusting for baseline measures of the mediator(s) and outcome(s) to reduce bias when estimating mediational effects (Loh & Ren, 2023; Selig & Preacher, 2009), this thesis followed the recommendation of Hayes (2022) and adjusted for *all* prior measures of the mediator and outcome. Because three timepoints of data were available for analysis, prior measures of executive function at second follow-up included executive function at first follow-up in addition to baseline. However, given the possibility of minimal change in executive function over three years of follow-up (versus six years), a second set of moderated mediation analyses were conducted on models that omitted executive function at T1 as a covariate. This sensitivity analysis enabled a comparison between indirect effects that accounted for executive function at T1 versus indirect effects that did not, providing insight into the role that executive function at T1 plays in informing the mediated pathway.

4.4.3.2 Multiple Imputation of Missing Covariate Data

Multiple imputation (MI) is a commonly used strategy in epidemiological research to retain participants with missing data and thereby increase statistical power (Harel et al., 2018). The process of MI involves replacing each missing value with a set of $m > 1$ plausible values, generating m complete datasets that can be analysed individually, with the results then being pooled using Rubin's rules (Rubin, 1987). MI was undertaken as a sensitivity analysis in this thesis to assess the impact of missing data, informed by previous work by our research group (Endresz et al., 2025; Golberg, 2023b, 2023c; Kang et al., 2024) and adapted for the purposes of estimating conditional indirect effects using the imputed data. For this thesis, only MI of missing covariates was undertaken to balance the advantage of retaining more participants, and therefore increasing statistical power, against the potential disadvantage of biasing main associations by imputing and analysing key variables that were not originally observed. Thus, the analytic sample for this sensitivity analysis was restricted to complete data on clinical anxiety at T0, anxiety symptoms at T0, FSS at T0 and T1, and executive function at T0, T1, and T2, while data on T0 sociodemographic, social, health, and lifestyle covariates were allowed to be missing. Covariates whose levels were collapsed due to low cell counts retained those collapsed categories for imputation. The dataset was imported into R v4.4.2 (The R Center for Statistical Computing, Vienna, Austria) and imputed using the *mice* package (Buuren &

Groothuis-Oudshoorn, 2011). All analytic variables except participant ID were selected as predictors. Additionally, the predictor matrix included interaction terms that were significant in the main analysis (i.e., *FSS*age group*, *anxiety symptoms*sex*age group*). Including interaction terms that are present in the analytic model is recommended when using *mice* (Tilling et al., 2016).

Classification and Regression Trees (CART) was the chosen method to impute missing data. CART does not require specification of an imputation model and is suitable for modelling large, complex health data (Strobl et al., 2009) as well as for capturing complex relationships such as interactions and nonlinearities (Doove et al., 2014). CART imputes data by finding partitions in the predictor space that have relatively homogeneous response values; these partitions are found by recursive binary splits of the predictors (Burgette & Reiter, 2010). When the splitting stops, the terminal node results in a prediction for the target variable, chosen randomly from among the members in the node (Doove et al., 2012). A total of nine imputation cycles was conducted, generating nine imputed datasets. This number was selected based on prior recommendations that $m = 5$ is sufficient for calculating point estimates and that m should not exceed 10 to minimise computational expenses associated with larger datasets (Golberg, 2023a; Rubin, 1987). As a compromise between precision and efficiency, $m = 9$ falls within the recommended range of imputation cycles that increases precision of point estimates and standard error (Bodner, 2008; White et al., 2011) while maintaining computational efficiency. Conditional process analysis, specified using the same models as in the main analysis, was then run on each of the nine datasets. Once conditional indirect effects were obtained for the nine datasets, and after verifying that there were at least 10 participants per predictor variable to indicate that violations of normality for regression coefficients would not impact inferences (Schmidt & Finan, 2018), the results were pooled using Rubin's rules (Rubin, 1987), whose equations were programmed into an Excel spreadsheet.

4.5 Ethics

This research received ethics approval from the University of Waterloo's Office of Research Ethics (#45693). Data access was approved by the CLSA (#2307011), and this thesis utilised Comprehensive dataset version 7.0, which was released on December 7, 2023.

Chapter 5

Results

5.1 Descriptive Analyses

5.1.1 Univariate Analyses

To describe the key variables involved in the mediated pathway, histograms of anxiety symptoms at T0, FSS at T1, and executive function at T2 are depicted in Figures 2–4, which include information on central tendency (mean, median) and variability (standard deviation, interquartile range). Anxiety symptoms at T0 (Figure 2) and FSS at T1 (Figure 3) both had skewed distributions, where most participants reported lower levels of anxiety and higher levels of FSS. Executive function at T2 (Figure 4) had little skewness as it is a standardised score.

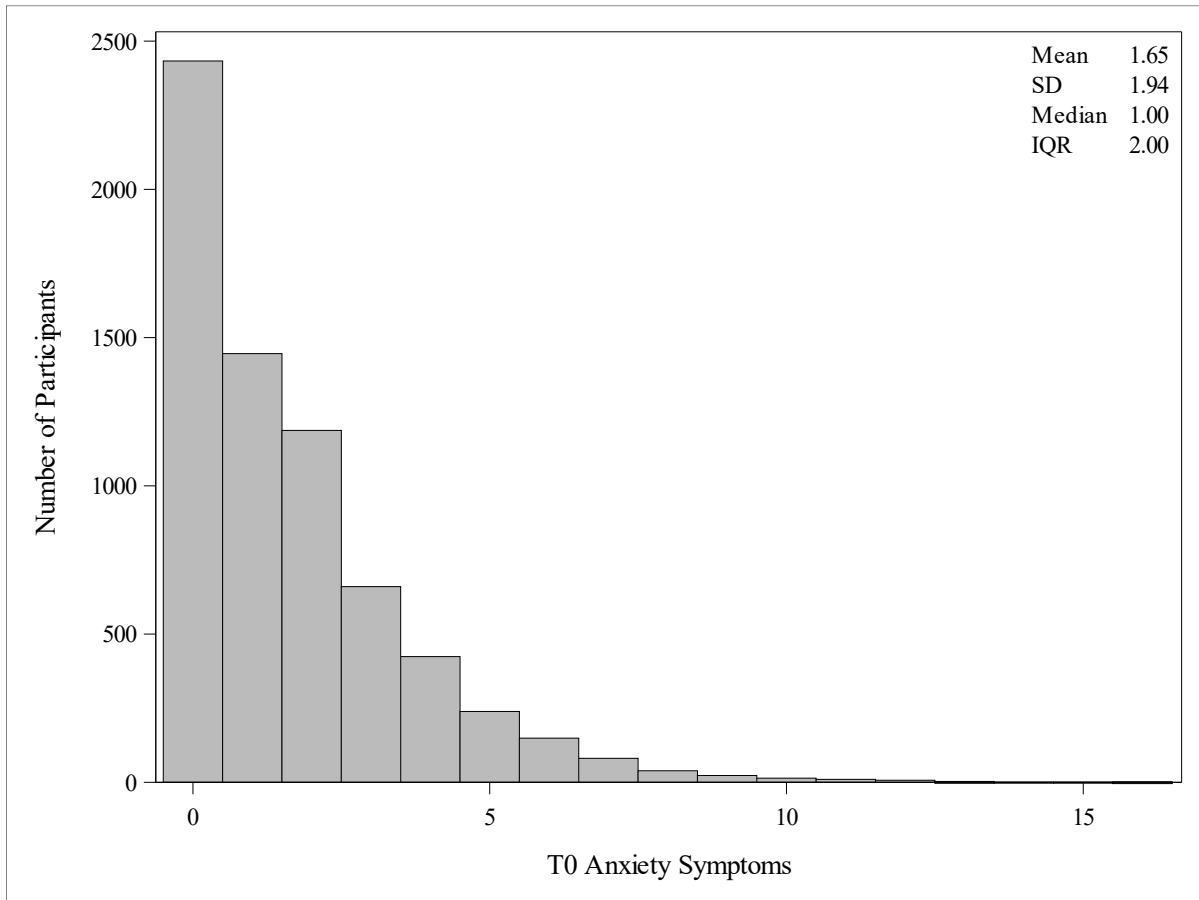


Figure 2. Distribution of Anxiety Symptoms at Baseline (T0), Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

Note. Anxiety symptoms were summed across four items from the Kessler Psychological Distress Scale (K10). Scores range from 0 to 16. A higher value on the scale indicates higher levels of anxiety.

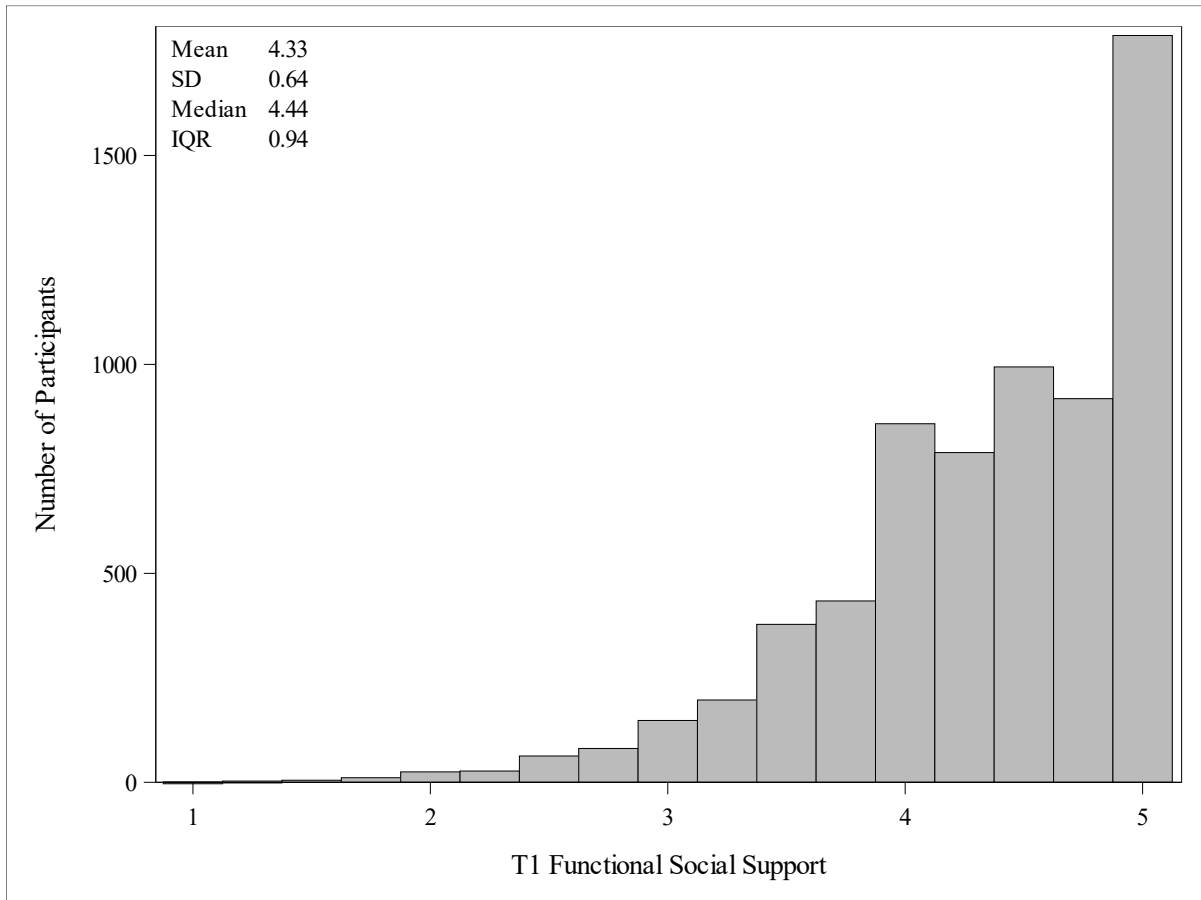


Figure 3. Distribution of Functional Social Support at Follow-up 1 (T1), Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

Note. Functional social support was measured using the Medical Outcomes Survey-Social Support Survey (MOS-SSS). Scores range from 1 to 5, with higher values indicating higher levels of functional social support.

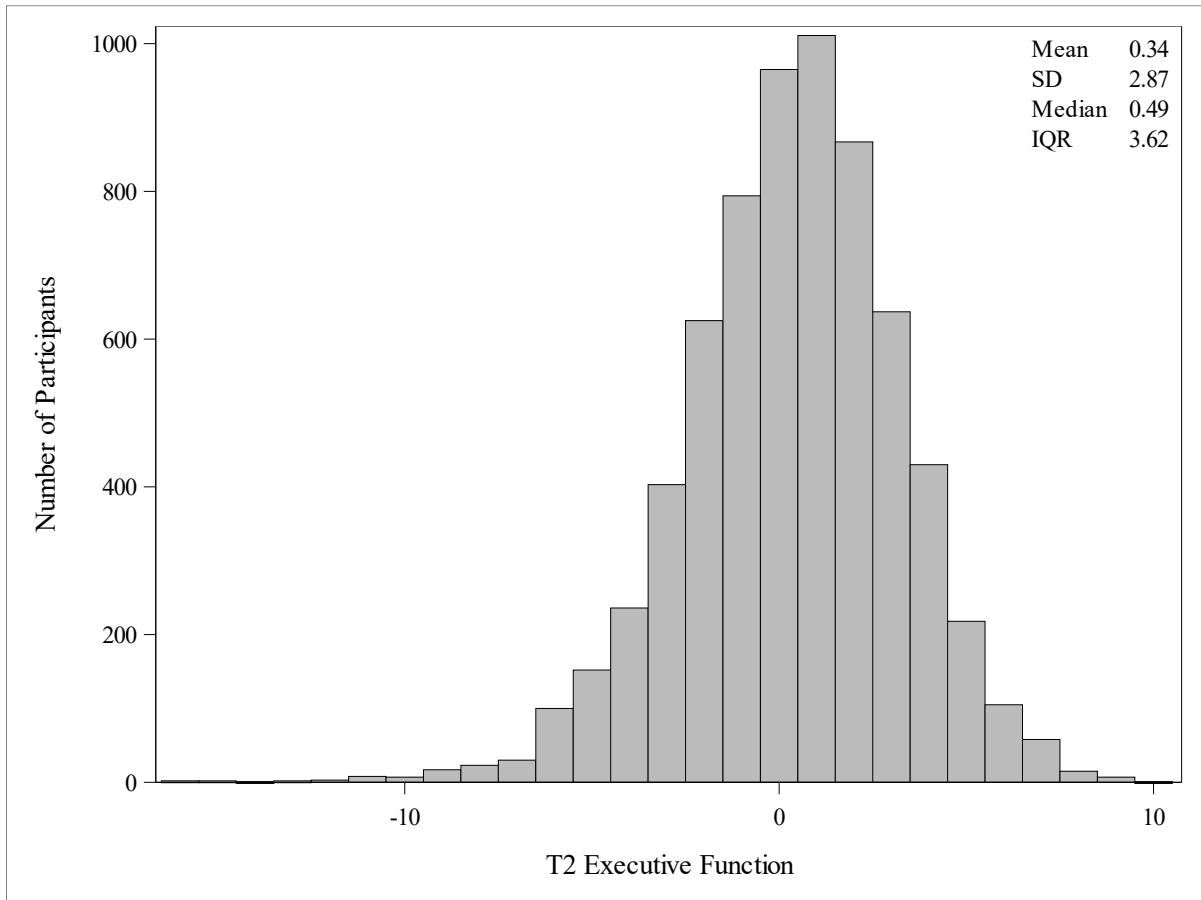


Figure 4. Distribution of Executive Function at Follow-Up 2 (T2), Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

Note. An overall score of executive function was created by standardising and then summing the scores of five neurocognitive tests of executive function.

Table 2 provides additional univariate data on the continuous variables used in the analyses, which include anxiety symptoms, depressive symptoms, FSS, and executive function at T0, as well as FSS and executive function at T1. Median FSS remained high from T0 to T1. Mean executive function decreased from T0 to T1 (Table 2), with a further decrease at T2 (Figure 4).

Table 2. Univariate Descriptives of Continuous Predictors and Bivariate Correlations of Continuous Predictors with Follow-up 1 Functional Social Support and Follow-up 2 Executive Function, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Continuous Predictors | \bar{x} (SD) | MD (IQR) | Mediator (T1) | Outcome (T2) |
|----------------------------------|----------------|-------------|------------------|---------------------------------|
| | | | FSS ¹ | Executive Function ² |
| | | | r_s | r_s |
| Exposure (T0) | | | | |
| Anxiety Symptoms ³ | 1.65 (1.94) | 1.00 (2.00) | -0.17**** | 0.06**** |
| Covariates (T0) | | | | |
| FSS ¹ | 4.32 (0.64) | 4.47 (0.89) | 0.71**** | 0.09**** |
| Executive Function ² | 0.64 (2.66) | 0.76 (3.34) | 0.09**** | 0.73**** |
| Depressive Symptoms ⁴ | 2.22 (2.78) | 1.00 (3.00) | -0.26**** | -0.05**** |
| Mediator (T1) | | | | |
| FSS ¹ | 4.33 (0.64) | 4.44 (0.94) | — | 0.10**** |
| Covariates (T1) | | | | |
| Executive Function ² | 0.54 (2.70) | 0.62 (3.47) | 0.09**** | 0.77**** |

Note. Spearman's rank correlation coefficient was used given the data were skewed.

FSS = functional social support; IQR = interquartile range; MD = median; r_s = Spearman correlation coefficient; SD = standard deviation; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; \bar{x} = mean.

¹Functional social support ranges from 1 to 5, with higher scores indicating higher levels of functional social support.

²Executive function is a standardised and summed score of five neurocognitive tests of executive function.

³Anxiety symptoms were summed across four items from the Kessler Psychological Distress Scale (K10). Scores range from 0 to 16, with higher values indicating higher levels of anxiety.

⁴Depressive symptoms were summed across six items from the Kessler Psychological Distress Scale (K10). Scores range from 0 to 24, with higher values indicating higher levels of depression.

*** $p < .001$, **** $p < .0001$

Proportions for categorical predictors (T0), including the exposure self-reported clinical history of anxiety and various sociodemographic, social, health, and lifestyle covariates, are provided in Table 3. The prevalence of anxiety disorders (T0) was approximately 7.6%. Across the moderators age group and sex, slightly more than half of the sample was female (51%) and slightly more than one-third (34%) of participants were 65 years or older. The sample consisted of participants who were highly educated (82% of participants had a postsecondary degree/diploma), had a mid-to-high income (41% of individuals had a household income \geq \$100,000), and were healthy (94% rated their health as good, very good, or excellent).

Table 3. Frequencies of Categorical Predictors and Associations of Categorical Predictors with Follow-up 1 Functional Social Support and Follow-up 2 Executive Function, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Characteristics (T0) | Total % | Mediator (T1) | Outcome (T2) |
|---|---------|--|---|
| | | Functional social support ¹ MD (IQR) | Executive function ² \bar{x} (SD) |
| Exposure | | | |
| <i>Clinical anxiety (self-reported)</i> | | | |
| Presence | 7.56 | 4.28 (1.03) ^a | 0.28 (2.93) |
| Absence | 92.44 | 4.50 (0.89) ^b | 0.34 (2.86) |
| Sociodemographic factors | | | |
| <i>Age group (years)</i> | | | |
| 45–54 | 28.93 | 4.50 (0.89) ^a | 1.51 (2.38) ^a |
| 55–64 | 37.21 | 4.50 (0.89) ^a | 0.74 (2.52) ^b |
| 65–74 | 23.01 | 4.44 (0.89) ^a | -0.54 (2.75) ^c |
| 75+ | 10.85 | 4.28 (1.00) ^b | -2.34 (3.15) ^d |
| <i>Sex</i> | | | |
| Female | 51.48 | 4.44 (0.89) ^a | 0.36 (2.84) |
| Male | 48.52 | 4.50 (0.89) ^b | 0.31 (2.90) |
| <i>Province</i> | | | |
| Ontario | 20.36 | 4.50 (0.89) ^{a,b} | 0.35 (2.90) ^{a,b} |
| Alberta | 8.84 | 4.39 (0.94) ^b | 0.35 (2.84) ^{a,b,c} |
| British Columbia | 21.12 | 4.50 (0.89) ^{a,b} | 0.62 (2.87) ^b |
| Manitoba | 11.42 | 4.39 (0.94) ^{b,d} | 0.68 (2.78) ^b |
| Newfoundland and Labrador | 9.97 | 4.53 (0.89) ^c | -0.10 (2.73) ^c |
| Nova Scotia | 11.52 | 4.56 (0.83) ^{a,c} | -0.04 (2.90) ^c |
| Quebec | 16.77 | 4.39 (0.83) ^d | 0.23 (2.89) ^{a,c} |
| <i>Education</i> | | | |
| Less than high school | 3.33 | 4.28 (1.03) ^a | -2.28 (3.17) ^a |
| High school graduate | 8.36 | 4.39 (1.00) ^{a,b} | -0.56 (2.84) ^b |
| Some post-secondary | 6.47 | 4.33 (0.94) ^a | -0.13 (2.68) ^b |
| Post-secondary degree/diploma | 81.83 | 4.50 (0.89) ^b | 0.57 (2.80) ^c |
| <i>Total household income</i> | | | |
| < \$20,000 | 3.26 | 3.89 (1.28) ^a | -1.15 (3.00) ^a |
| ≥ \$20,000 to < \$50,000 | 18.47 | 4.17 (1.00) ^b | -0.81 (3.09) ^a |
| ≥ \$50,000 to < \$100,000 | 36.94 | 4.44 (0.94) ^c | 0.12 (2.75) ^b |
| ≥ \$100,000 to < \$150,000 | 22.29 | 4.56 (0.83) ^d | 0.95 (2.57) ^c |
| ≥ \$150,000 | 19.04 | 4.72 (0.72) ^c | 1.41 (2.59) ^d |
| <i>Income meets needs</i> | | | |
| Totally inadequately | 0.49 | 3.94 (1.22) ^{a,b} | 0.89 (2.30) ^{a,b} |
| Not very well | 1.22 | 3.82 (1.17) ^a | 0.39 (2.89) ^{a,b} |
| With some difficulty | 5.46 | 4.11 (1.11) ^a | 0.05 (2.91) ^a |
| Adequately | 34.81 | 4.33 (0.94) ^b | 0.03 (2.90) ^a |
| Very well | 58.01 | 4.56 (0.83) ^c | 0.54 (2.83) ^b |
| Social factors | | | |
| <i>Marital status</i> | | | |

| | | | |
|--|-------|----------------------------|-------------------------------|
| Married/common-law | 75.04 | 4.61 (0.83) ^a | 0.48 (2.82) ^a |
| Single/never married | 7.08 | 3.94 (1.11) ^b | 0.40 (2.76) ^a |
| Widowed | 6.53 | 4.11 (0.89) ^b | -1.29 (3.32) ^b |
| Divorced | 9.09 | 4.06 (0.97) ^b | 0.16 (2.72) ^a |
| Separated | 2.25 | 4.00 (1.22) ^b | 0.57 (2.64) ^a |
| <i>Living arrangements</i> | | | |
| Lives alone | 17.84 | 4.00 (1.00) ^a | -0.33 (3.02) ^a |
| Lives with others | 82.16 | 4.56 (0.83) ^b | 0.48 (2.82) ^b |
| <i>Pet ownership</i> | | | |
| Yes | 44.92 | 4.50 (0.89) | 0.69 (2.79) ^a |
| No | 55.08 | 4.44 (0.94) | 0.05 (2.90) ^b |
| Health factors | | | |
| <i>Self-rated general health</i> | | | |
| Poor | 0.82 | 4.17 (1.44) ^a | -0.37 (2.69) ^{a,b,c} |
| Fair | 5.22 | 4.22 (1.17) ^a | -0.47 (3.00) ^a |
| Good | 26.57 | 4.33 (0.94) ^a | 0.01 (2.91) ^b |
| Very good | 43.89 | 4.50 (0.89) ^b | 0.44 (2.84) ^c |
| Excellent | 23.50 | 4.61 (0.78) ^c | 0.70 (2.77) ^d |
| <i>Number of chronic conditions</i> | | | |
| 0 | 33.92 | 4.50 (0.89) ^a | 0.97 (2.67) ^a |
| 1 | 35.66 | 4.44 (0.89) ^{a,b} | 0.38 (2.75) ^b |
| 2 | 19.24 | 4.44 (0.89) ^b | -0.17 (2.94) ^c |
| 3+ | 11.18 | 4.28 (0.94) ^c | -0.87 (3.15) ^d |
| <i>Functional impairment</i> | | | |
| Yes | 6.01 | 4.22 (1.00) ^a | -1.14 (3.25) ^a |
| No | 93.99 | 4.50 (0.89) ^b | 0.43 (2.82) ^b |
| <i>Clinical depression (self-reported)</i> | | | |
| Presence | 15.30 | 4.28 (1.00) ^a | 0.40 (2.87) |
| Absence | 84.70 | 4.50 (0.89) ^b | 0.32 (2.87) |
| Lifestyle factors | | | |
| <i>Smoking status</i> | | | |
| Never smoked | 49.31 | 4.50 (0.89) ^a | 0.58 (2.85) ^a |
| Former smoker | 43.80 | 4.44 (0.94) ^b | 0.10 (2.90) ^b |
| Current smoker | 6.89 | 4.22 (1.00) ^c | 0.11 (2.65) ^b |
| <i>Alcohol use</i> | | | |
| No | 9.36 | 4.33 (1.00) ^a | -0.08 (3.03) ^a |
| Occasional | 10.86 | 4.33 (1.06) ^a | -0.33 (2.96) ^a |
| Regular | 79.77 | 4.50 (0.89) ^b | 0.47 (2.82) ^b |

Note. Medians and interquartile ranges were calculated for functional social support because the data were skewed; Mann-Whitney U-tests and Kruskal-Wallis with post-hoc Dunn's tests were used to detect significant pairwise differences. Means and standard deviations were calculated for executive function, which approximated a normal distribution; t-tests and ANOVA with post-hoc Tukey tests were used to detect significant pairwise differences. Different superscript letters denote values with significant pairwise differences at the $p < .05$ level.

IQR = interquartile range; MD = median; SD = standard deviation; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; \bar{x} = mean.

¹Functional social support ranges from 1 to 5, with higher scores indicating higher levels of functional social support.

²Executive function is a standardised and summed score of five neurocognitive tests of executive function.

5.1.2 Bivariate Analyses

5.1.2.1 Associations with Functional Social Support

Self-reported clinical diagnosis of anxiety and self-reported anxiety symptoms at T0 were both significantly associated with FSS at T1. As a continuous measure of anxiety, anxiety symptoms at T0 were negatively correlated with FSS at T1 ($r_s = -0.17, p < .0001$) (Table 2). As a dichotomous measure of anxiety, individuals reporting a physician-diagnosed anxiety disorder at T0 had a lower median FSS score at T1 compared to individuals without a physician-diagnosed anxiety disorder (4.28 versus 4.50, $p < .0001$) (Table 3).

Regarding the key moderator age group (Table 3), median FSS decreased with advancing age, although the results of Dunn's tests showed that only the 75+ age group experienced significantly lower FSS compared to the other age groups. In terms of the moderator sex, females reported significantly lower median FSS compared to males. Stratifying sex by age group (Table 4) showed that only females aged 65–74 and 75+ reported significantly lower median FSS compared to males in the same group.

Table 4. Association of Baseline Sex with Follow-up 1 Functional Social Support and Follow-up 2 Executive Function, by Age Group, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Age Group (Years) by Sex | Total % | Mediator (T1) Functional social support¹ MD (IQR) | Outcome (T2) Executive function² \bar{x} (SD) |
|---------------------------------|----------------|---|--|
| <i>45–54</i> | | | |
| Males | 44.60 | 4.56 (0.94) | 1.58 (2.42) |
| Females | 55.40 | 4.50 (0.83) | 1.46 (2.36) |
| <i>55–64</i> | | | |
| Males | 48.48 | 4.40 (1.00) | 0.70 (2.57) |
| Females | 51.52 | 4.44 (0.89) | 0.78 (2.48) |
| <i>65–74</i> | | | |
| Males | 52.26 | 4.56 (0.83) ^a | -0.49 (2.80) |
| Females | 47.74 | 4.39 (0.83) ^b | -0.61 (2.69) |
| <i>75+</i> | | | |
| Males | 51.17 | 4.33 (0.94) ^a | -2.17 (3.13) |
| Females | 48.83 | 4.14 (0.97) ^b | -2.51 (3.15) |

Note. T-tests for normally distributed variables and Mann-Whitney U tests for skewed variables were used to detect significant group differences. Medians and interquartile ranges were given for functional social support because the data were skewed. Means and medians with superscript letters denote significant pairwise differences at the $p < .05$ level.

IQR = interquartile range; MD = median; SD = standard deviation; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; \bar{x} = mean.

¹Functional social support ranges from 1 to 5, with higher scores indicating higher levels of functional social support.

²Executive function is a standardised and summed score of five neurocognitive tests of executive function.

All covariates at T0, except pet ownership, were significantly associated with FSS at T1 (Table 3). Individuals with higher levels of FSS were more likely to be more highly educated, have higher household incomes, report greater income adequacy, be married, live with others, report better health, have fewer chronic conditions, be functionally unimpaired, smoke less frequently, and consume alcohol regularly. Depression, whether as self-reported symptoms (Table 2) or a self-reported clinical diagnosis (Table 3), was associated with decreased levels of FSS.

5.1.2.2 Associations with Executive Function

Anxiety symptoms at T0 (Table 2) were significantly associated with higher executive function at T2 ($r_s = 0.06$, $p < .0001$). Individuals with an anxiety disorder (Table 3) tended to have lower average executive function at T2 compared to individuals without an anxiety disorder, but this difference was not statistically significant (0.28 versus 0.34, $p = .64$). Additionally, FSS at T1 was significantly associated with higher executive function at T2 ($r_s = 0.10$, $p < .0001$) (Table 2).

Regarding the association between age and executive function (Table 3), the four age groups significantly differed from one another, with increasingly older age groups associated with decreasing levels of executive function. Female participants on average had slightly higher executive function at T2, but this difference was not significant (0.36 versus 0.31, $p = .53$). When stratified by age group (Table 4), females tended to have lower executive function than males in every age group but 55–64; however, none of these differences were statistically significant.

All T0 covariates but sex and clinical depression were significantly associated with T2 executive function (Table 3), including depressive symptoms (Table 2). Participants who had higher executive function at T2 tended to be more educated, report higher household incomes, not be widowed, live with others, own a pet for companionship, report better health, have fewer chronic conditions, be functionally unimpaired, never smoke, and consume alcohol regularly.

5.2 Multivariable Analyses

5.2.1 Building the Moderated Mediation Model

5.2.1.1 Testing Interactions at Path I: The Effect of Anxiety on Functional Social Support

To test interactions at Path I, multiple linear regression models were run with T0 anxiety as the exposure and T1 FSS as the outcome. Models with clinical anxiety and anxiety symptoms were tested separately. All models were fully adjusted for sociodemographic, social, health, and lifestyle factors, as well as T0 measures for FSS and executive function. The three-way interaction term (*anxiety*age group*sex*) was tested for significance first. In a fully adjusted model, the three-way interaction term was only significant for *anxiety symptoms*age group*sex* (R^2 change = 0.0006, $p = .036$) and not *clinical anxiety*age group*sex* (R^2 change = 0.0003, $p = .18$). Since a three-way interaction was significant for anxiety symptoms at Path I, the three-way interaction was included and no further interactions with lower-order terms were tested. For the model with clinical anxiety, because the three-way interaction was not significant, two-way interaction terms were subsequently tested.

The two-way interaction terms tested at Path I for clinical anxiety were *clinical anxiety*age group* and *clinical anxiety*sex*. In a model where both two-way interaction terms were included, the joint test of interaction between *clinical anxiety*age group* and *clinical anxiety*sex* was not statistically significant (R^2 change = 0.0001, $p = .74$). Thus, two-way interactions were tested separately. In these models, the moderator that was not included in an interaction term was controlled for as a covariate. Both the *clinical anxiety*age group* interaction term (R^2 change = 0.0001, $p = .64$) and the *clinical anxiety*sex* interaction term (R^2 change = 0.0000, $p = .56$) were nonsignificant. Thus, the final Path I model for clinical anxiety did not include any interaction terms.

5.2.1.2 Testing Interactions at Path II: The Effect of Functional Social Support on Executive Function

Interaction testing at Path II proceeded similarly to interaction testing at Path I, with several key differences: T1 FSS was the exposure, T2 executive function was the outcome, and fully adjusted models additionally controlled for T0 anxiety and T1 executive function. Again, models for clinical anxiety and anxiety symptoms were tested separately. Starting with three-way interactions, the three-way interaction term between FSS, age group, and sex (*FSS*age group*sex*) was not significant for

either clinical anxiety (R^2 change = 0.0003, $p = .11$) or anxiety symptoms (R^2 change = 0.0003, $p = .12$).

Two-way interaction terms for both models of anxiety were then tested. When the two-way interaction terms of *FSS*age group* and *FSS*sex* were simultaneously included in models, their joint test of interaction was significant for clinical anxiety (R^2 change = 0.0007, $p = .0097$) and anxiety symptoms (R^2 change = 0.0007, $p = .0098$), indicating that at least one of the interaction terms significantly increased the proportion of variance explained in the model.

Examining the individual contributions of *FSS*age group* and *FSS*sex* when the other interaction term was controlled for, *FSS*age group* was a larger contributor to the explained variance (R^2 change = 0.0007, $p = .0043$) than *FSS*sex* (R^2 change = 0.0000, $p = .53$) in the model with clinical anxiety; in the model with anxiety symptoms, *FSS*age group* also uniquely explained more variance (R^2 change = 0.0007, $p = .0043$) than *FSS*sex* (R^2 change = 0.0000, $p = .54$). Since the unique contribution of *FSS*sex* to the R^2 change was smaller, the two-way interaction terms were tested separately to determine whether these contributions to the explained variance held when the other interaction term was *not* controlled for. When tested individually in models, *FSS*age group* was significant for both clinical anxiety (R^2 change = 0.0007, $p = .0048$) and anxiety symptoms (0.0007, $p = .0048$). *FSS*sex* was not significant for either clinical anxiety (R^2 change = 0.0000, $p = .68$) or anxiety symptoms (R^2 change = 0.0000, $p = .69$). Thus, only the two-way interaction of *FSS*age group* was included in the final Path II models of clinical anxiety and anxiety symptoms.

5.2.1.3 Finalised Moderated Mediation Model

Based on the results of testing interactions at Path I and Path II, a final moderated mediation model was created for clinical anxiety (Figure 5) and anxiety symptoms (Figure 6). In the model where clinical anxiety was the exposure, Path I was unmoderated, as the three-way interaction term, the joint two-way interaction terms, and the single two-way interaction term were all nonsignificant. In contrast, the model where anxiety symptoms was the exposure included a three-way interaction of *anxiety symptoms*age group*sex* at Path I. At Path II, for both models of anxiety, moderation by age group was included, as the *FSS*age group* interaction term was significant while *FSS*sex* was not.

In the PROCESS macro, Model 14 (Hayes, 2022) allows for the estimation of moderated mediation with one moderator at Path II, such as that depicted in the moderated mediation model for clinical anxiety (Figure 5). For the moderated mediation model for anxiety symptoms (Figure 6), Model 68

(Hayes, 2022) specifies moderation of Path I by moderators $W*Z$ (i.e., three-way interaction term between the exposure and two moderators) and moderation of Path II by moderator W (i.e., two-way interaction term with the moderator that is also present in Path I). The final analysis plan, which reflects the results of testing interactions at Paths I and II, can be found in Table 5.

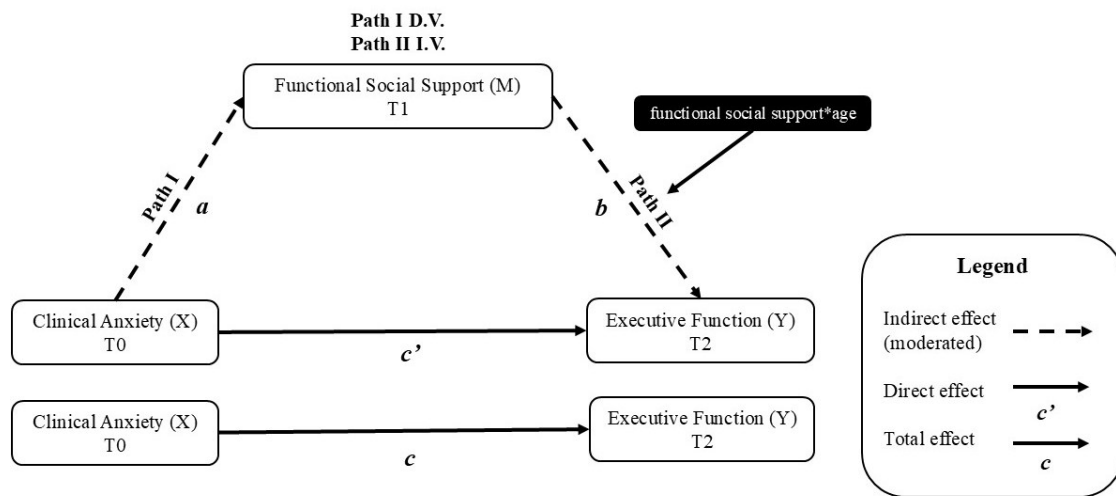


Figure 5. Finalised Moderation Mediation Model (Clinical Anxiety)

Note. Clinical anxiety was a self-reported physician diagnosis of an anxiety disorder. Functional social support was measured using the 19-item Medical Outcomes Survey-Social Support Survey. Executive function was a standardised and summed score of five neurocognitive tests of executive function. Age group was a moderator at Path II.

DV = dependent variable; IV = independent variable; M = mediator; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; X = exposure; Y = outcome.

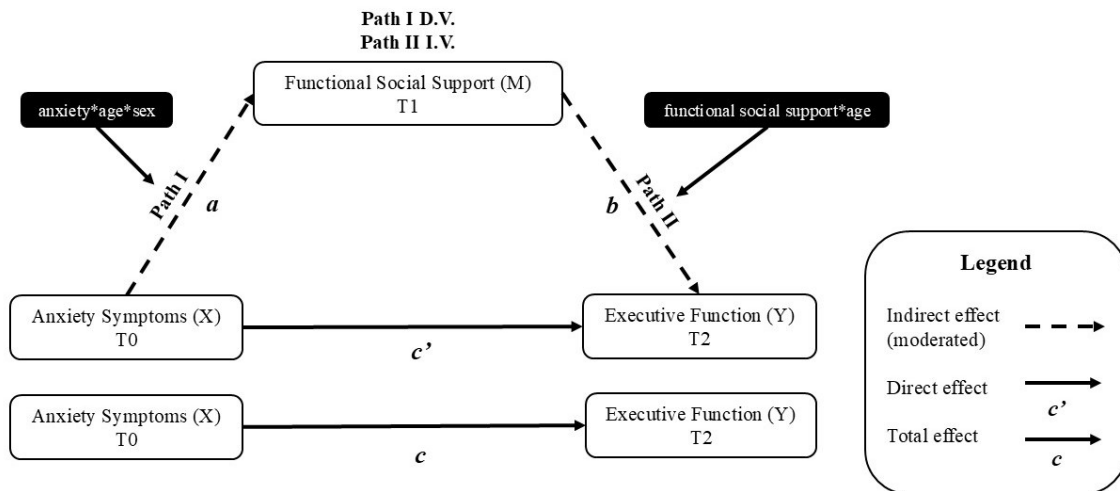


Figure 6. Finalised Moderated Mediation Model (Anxiety Symptoms)

Note. Anxiety symptoms were measured using four items from the Kessler Psychological Distress Scale. Functional social support was measured using the 19-item Medical Outcomes Survey-Social Support Survey. Executive function was a standardised and summed score of five neurocognitive tests of executive function. Age group*sex was a moderator at Path I. Age group was a moderator at Path II.

DV = dependent variable; IV = independent variable; M = mediator; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; X = exposure; Y = outcome.

Table 5. Analysis Plan Based on Final Moderated Mediation Model

| | Path I <i>Anxiety (X) → FSS (M)</i> | Path II <i>FSS (M) → Executive Function (Y)</i> |
|--|--|--|
| Clinical Anxiety PROCESS Model 14 ¹ | | |
| Model 1 <i>Base</i> | Exposure (X) <ul style="list-style-type: none"> Clinical anxiety (T0) Outcome (M) <ul style="list-style-type: none"> FSS (T1) Interaction term(s)² <ul style="list-style-type: none"> None Antecedent measures³ <ul style="list-style-type: none"> FSS (T0) Executive function (T0) | Exposure (M) <ul style="list-style-type: none"> FSS (T1) Outcome (Y) <ul style="list-style-type: none"> Executive function (T2) Interaction term(s)² <ul style="list-style-type: none"> Two-way: <ul style="list-style-type: none"> FSS (T1)*Age group (T0) Antecedent measures³ <ul style="list-style-type: none"> Clinical anxiety (T0)⁴ FSS (T0) Executive function (T0, T1) |
| Model 2 <i>Fully Adjusted</i> | Base Model + Sociodemographic covariates (T0) <ul style="list-style-type: none"> Age group, sex, province, education, annual household income, income adequacy Social covariates (T0) <ul style="list-style-type: none"> Marital status, living arrangements, pet ownership Health covariates (T0) <ul style="list-style-type: none"> Self-rated health, number of chronic conditions, functional impairment, clinical depression Lifestyle covariates (T0) <ul style="list-style-type: none"> Smoking status, alcohol use | |
| Anxiety Symptoms PROCESS Model 68 ¹ | | |
| Model 1 <i>Base</i> | Exposure (X) <ul style="list-style-type: none"> Anxiety symptoms (T0) Outcome (M) <ul style="list-style-type: none"> FSS (T1) Interaction term(s)² <ul style="list-style-type: none"> Three-way: <ul style="list-style-type: none"> Anxiety symptoms*age group*sex (T0) Antecedent measures³ <ul style="list-style-type: none"> FSS (T0) Executive function (T0) | Exposure (M) <ul style="list-style-type: none"> FSS (T1) Outcome (Y) <ul style="list-style-type: none"> Executive function (T2) Interaction term(s)² <ul style="list-style-type: none"> Two-way: <ul style="list-style-type: none"> FSS (T1)*Age group (T0) Antecedent measures³ <ul style="list-style-type: none"> Anxiety symptoms (T0)⁴ FSS (T0) Executive function (T0, T1) |
| Model 2 <i>Fully Adjusted</i> | Base Model + Sociodemographic covariates (T0) <ul style="list-style-type: none"> Age group, sex, province, education, annual household income, income adequacy Social covariates (T0) <ul style="list-style-type: none"> Marital status, living arrangements, pet ownership | |

Health covariates (T0)

- Self-rated health, number of chronic conditions, functional impairment, depressive symptoms

Lifestyle covariates (T0)

Smoking status, alcohol use

Note. FSS = functional social support; M = mediator; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; X = exposure; Y = outcome.

¹Preprogrammed PROCESS models are denoted by numbers, as found in Hayes (2022).

²Only significant highest-order interactions were included. Lower-order terms were automatically controlled for.

³Prior measures of the mediator and outcome were controlled for as recommended by Hayes (2022).

⁴PROCESS automatically controls for X in Path II.

5.2.2 Estimating Moderated Mediation

The mediated (indirect) effect of FSS was not significant in any subgroup defined by age or sex (Table 6). This result was consistent for both anxiety models, as well as across their base (unadjusted) and final (fully adjusted) models.

Table 6. Indirect Effect of Anxiety on Executive Function Through Functional Social Support by Sex and Age Group, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Moderators | | Indirect Effect <i>b</i> (95% bootstrap CI) | |
|------------|-------------------|--|---------------------------|
| Sex | Age Group (years) | Clinical Anxiety | |
| | | Base Model ¹ | Final Model ² |
| Overall | 45–54 | 0.0057 (-0.0024, 0.0167) | 0.0045 (-0.0015, 0.0137) |
| | 55–64 | -0.0070 (-0.0196, 0.0017) | -0.0043 (-0.0149, 0.0020) |
| | 65–74 | -0.0037 (-0.0157, 0.0065) | -0.0026 (-0.0121, 0.0049) |
| | ≥ 75 | 0.0141 (-0.0013, 0.0367) | 0.0103 (-0.0014, 0.0301) |
| Sex | Age Group (years) | Anxiety Symptoms | |
| | | Base Model ¹ | Final Model ² |
| Male | 45–54 | -0.0000 (-0.0022, 0.0020) | -0.0019 (-0.0059, 0.0005) |
| | 55–64 | -0.0018 (-0.0058, 0.0005) | -0.0003 (-0.0028, 0.0014) |
| | 65–74 | -0.0014 (-0.0064, 0.0027) | -0.0006 (-0.0040, 0.0017) |
| | ≥ 75 | -0.0015 (-0.0096, 0.0059) | -0.0043 (-0.0145, 0.0027) |
| Female | 45–54 | 0.0019 (-0.0008, 0.0053) | 0.0002 (-0.0013, 0.0020) |
| | 55–64 | -0.0029 (-0.0080, 0.0007) | -0.0007 (-0.0037, 0.0007) |
| | 65–74 | 0.0001 (-0.0016, 0.0022) | 0.0009 (-0.0018, 0.0046) |
| | ≥ 75 | -0.0007 (-0.0085, 0.0076) | -0.0041 (-0.0142, 0.0030) |

Note. *b* = unstandardised regression coefficient; CI = confidence interval.

¹Included T0 functional social support, T0 executive function, and T1 executive function.

²Included the same measures as the base model, with the addition of sociodemographic, social, health, and lifestyle covariates at T0.

No significant indirect effects were detected at $p < .05$.

5.2.3 Path I and Path II Effects

While there were no significant mediated effects of anxiety on executive function through FSS, it may be useful to examine the independent path effects (moderated or unmoderated) to obtain a more thorough understanding of the relationships between exposure, mediator, and outcome. In Path I, clinical anxiety was unmoderated, and anxiety symptoms were moderated by the interaction of age group and sex. In Path II, FSS was moderated by age group for both models of anxiety.

5.2.3.1 Clinical Anxiety

As shown in Table 7, in Path I of the fully adjusted model, clinical anxiety was significantly associated with lower FSS ($b = -0.0433$, 95% CI: $-0.0843, -0.0024$). In Path II of the fully adjusted model, the effect of FSS on executive function differed significantly by age group ($F = 4.32$, $p = .0048$). FSS was negatively associated with executive function in age groups 45–54 and 75+, and positively associated in age groups 55–64 and 65–74, although the effect of FSS on executive function was significant only in those aged 75+ ($b = -0.2383$, 95% CI: $-0.4393, -0.0372$).

Table 7. Effect of Clinical Anxiety on Functional Social Support (Path I) and Effects of Functional Social Support on Executive Function (Path II) by Age, Canadian Longitudinal Study on Aging (n = 6,719)

| Path I: Clinical Anxiety (X) → FSS (M) | | |
|--|--|--|
| <i>b</i> (95% CI) | | |
| | Base Model¹ | Final Model² |
| All Participants | -0.0616 (-0.1010, -0.0223)* | -0.0433 (-0.0843, -0.0024)* |
| Path II: FSS (M) → Executive Function (Y)³ | | |
| <i>b</i> (95% CI) | | |
| | Base Model¹ | Final Model² |
| Age Group | FSS*Age Group ($\Delta R^2=0.0007$, $F=4.35$) | FSS*Age Group ($\Delta R^2=0.0007$, $F=4.32$) |
| 45–54 | -0.0926 (-0.2356, 0.0504) | -0.1027 (-0.2470, 0.0416) |
| 55–64 | 0.1137 (-0.0125, 0.2399) | 0.1000 (-0.0274, 0.2274) |
| 65–74 | 0.0604 (-0.0925, 0.2132) | 0.0603 (-0.0934, 0.2140) |
| 75+ | -0.2292 (-0.4296, -0.0287)* | -0.2383 (-0.4393, -0.0372)* |

Note. *b* = unstandardised regression coefficient; CI = confidence interval; FSS = functional social support; ΔR^2 = R-squared change.

¹Included T0 functional social support, T0 executive function, and T1 executive function.

²Included the same measures as the base model, with the addition of sociodemographic, social, health, and lifestyle covariates at T0.

³Path II models additionally controlled for anxiety symptoms and T1 executive function.

**p* < .05

5.2.3.2 Anxiety Symptoms

In Path I of the fully adjusted model for anxiety symptoms (Table 8), the association between anxiety symptoms and FSS was moderated by age group and sex in a three-way interaction ($F = 2.8530$, $p = .036$), where, more specifically, the association between anxiety symptoms and FSS varied by age in males ($F = 2.8620$, $p = .035$) but not females ($F = 2.0761$, $p = .10$). In the unadjusted model, higher anxiety symptoms in men were significantly related to lower FSS in those aged 55–64 ($b = -0.0163$, 95% CI: $-0.0298, -0.0027$) and 65–74 ($b = -0.0241$, 95% CI: $-0.0425, -0.0058$), while higher anxiety symptoms in women were significantly related to lower FSS in those aged 45–54 ($b = -0.0203$, 95% CI: $-0.0319, -0.0086$) and 55–64 ($b = -0.0253$, 95% CI: $-0.0372, -0.0135$). However, once all covariates were entered in analyses, the association between anxiety and FSS was significant and positive only in men aged 45–54 ($b = 0.0176$, 95% CI: $0.0034, 0.0318$).

In Path II of the fully adjusted model for anxiety symptoms (Table 8), the effect of FSS on executive function differed significantly by age group ($F = 4.32$, $p = .0048$). Here, the pattern across age groups was similar to the model for clinical anxiety: FSS was negatively associated with executive function in age groups 45–54 and 75+ and positively associated in age groups 55–64 and 65–74, but the effect of FSS on executive function was significant only in those aged 75+ ($b = -0.2417$, 95% CI: $-0.4428, -0.0407$).

Table 8. Effects of Anxiety Symptoms on Functional Social Support (Path I) and Effects of Functional Social Support on Executive Function (Path II) by Age and Sex, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Path I: Anxiety Symptoms¹ (X) → FSS (M) <i>b</i> (95% CI) | | |
|---|---|--|
| | Base Model² Anxiety Symptoms*Sex*Age Group ($\Delta R^2=0.0006$, $F=2.91$) | Final Model³ Anxiety Symptoms*Sex*Age Group ($\Delta R^2=0.0006$, $F=2.85$) |
| Age Group | | |
| Males | <i>Anxiety Symptoms*Age Group</i> ($F = 2.24$) | <i>Anxiety Symptoms*Age Group</i> ($F = 2.86$) |
| 45–54 | 0.0005 (-0.0135, 0.0145) | 0.0176 (0.0034, 0.0318)* |
| 55–64 | -0.0163 (-0.0298, -0.0027)* | -0.0034 (-0.0171, 0.0104) |
| 65–74 | -0.0241 (-0.0425, -0.0058)** | -0.0111 (-0.0295, 0.0073) |
| 75+ | 0.0065 (-0.0207, 0.0338) | 0.0180 (-0.0091, 0.0450) |
| Females | <i>Anxiety Symptoms*Age Group</i> ($F = 2.98$) | <i>Anxiety Symptoms*Age Group</i> ($F = 2.08$) |
| 45–54 | -0.0203 (-0.0319, -0.0086)*** | -0.0020 (-0.0141, 0.0101) |
| 55–64 | -0.0253 (-0.0372, -0.0135)*** | -0.0071 (-0.0194, 0.0052) |
| 65–74 | 0.0019 (-0.0159, 0.0197) | 0.0154 (-0.0025, 0.0333) |
| 75+ | 0.0030 (-0.0224, 0.0285) | 0.0171 (-0.0084, 0.0427) |
| Path II: FSS (M) → Executive Function (Y)⁴ <i>b</i> (95% CI) | | |
| | Base Model FSS*Age Group ($\Delta R^2=0.0007$, $F=4.33$) | Final Model FSS*Age Group ($\Delta R^2=0.0007$, $F=4.32$) |
| Age Group | | |
| 45–54 | -0.0927 (-0.2359, 0.0504) | -0.1079 (-0.2527, 0.0370) |
| 55–64 | 0.1137 (-0.0126, 0.2399) | 0.0955 (-0.0323, 0.2234) |
| 65–74 | 0.0589 (-0.0941, 0.2118) | 0.0564 (-0.0974, 0.2102) |
| 75+ | -0.2282 (-0.4287, -0.0277)* | -0.2417 (-0.4428, -0.0407)* |

Note. *b* = unstandardised regression coefficient; CI = confidence interval; FSS = functional social support; M = mediator; ΔR^2 = R-squared change; X = exposure; Y = outcome.

¹Anxiety symptom scores were a sum of four items from the Kessler Psychological Distress Scale (K10). Scores range from 0 to 16, with higher values indicating higher levels of anxiety.

²Included T0 functional social support and T0 executive function.

³Included the same measures as the base model, with the addition of sociodemographic, social, health, and lifestyle covariates at T0.

⁴Path II models additionally controlled for anxiety symptoms and T1 executive function.

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

5.2.4 Covariate Effects in the Moderated Mediation Model

5.2.4.1 Clinical Anxiety

In Path I of the model with clinical anxiety (Table E1), T0 FSS was significantly associated with T1 FSS. Age group, province, total household income, income adequacy, marital status, self-rated general health, smoking, and alcohol use were also associated with T1 FSS; sex, educational level, living arrangement, pet ownership, number of chronic conditions, functional impairment, and clinical depression were not associated with T1 FSS. The direction of significant effects was consistent with expectations: older age, being single/never married, and being a current smoker were associated with lower FSS, while higher income, greater income adequacy, better self-rated health, and more frequent alcohol use were associated with higher FSS.

In Path II of the model, T0 and T1 executive function were both significantly associated with T2 executive function. Province, educational level, and number of chronic conditions were also associated with T2 executive function; clinical anxiety, sex, total household income, income adequacy, marital status, living arrangement, pet ownership, self-rated health, functional impairment, clinical depression, smoking status, and alcohol use were not associated with T2 executive function. As expected, lower education and a higher number of chronic conditions were associated with lower executive function.

5.2.4.2 Anxiety Symptoms

In Path I of the model with anxiety symptoms (Table E2), T0 FSS was significantly associated with T1 FSS. Province, income adequacy, marital status, depressive symptoms, smoking status, and alcohol use were also associated with T1 FSS; educational level, total household income, living arrangement, pet ownership, self-rated health, number of chronic conditions, and functional impairment were not associated with T1 FSS. Again, the significant associations were in the expected direction: being single/never married, reporting a higher level of depressive symptoms, and current smoking were associated with lower FSS, whereas greater income adequacy and more frequent alcohol use were associated with higher FSS.

In Path II of the model with anxiety symptoms, associations with T2 executive function followed the same patterns as Path II of the clinical anxiety model, including the direction of effects (see Section 5.2.4.1 above).

5.2.5 Sensitivity Analysis 1: Estimating Indirect Effects Without T1 Executive Function

While adjusting for antecedent measures of the mediator and outcome in conditional process analysis is recommended by Hayes (2022), results may not encapsulate significant changes in executive function due to a lack of significant cognitive change occurring in this sample over three years (if controlling for T1 executive function, in addition to T0 executive function) versus six years (if only controlling for T0 executive function). Thus, a sensitivity analysis was undertaken to see if omitting T1 executive function from moderated mediation models would result in significant changes in one or more indirect effects. Base and fully adjusted models in the sensitivity analysis retained all key variables and covariates used in the main analysis, differing only by the exclusion of T1 executive function as a covariate at Path II. Because omitting a variable could alter the nature of effect modification at one or more paths, interactions were tested on fully adjusted models before indirect effects were estimated.

5.2.5.1 Testing Interactions

Since T1 executive function was only adjusted for in Path II models, retesting Path I was not necessary as the results would remain unaffected. As with the main analysis, three-way interactions followed by two-way interactions (if three-way interactions were nonsignificant) were tested at Path II in multiple linear regression models.

In the model that controlled for clinical anxiety, the *FSS*age group*sex* interaction was not significant (R^2 change = 0.0004, $p = .15$). In a model that included *FSS*age group* and *FSS*sex* as interaction terms, the joint test of interaction was not significant (R^2 change = 0.0006, $p = .081$). As individually entered interaction terms, neither *FSS*age group* (R^2 change = 0.0005, $p = .052$) nor *FSS*sex* (R^2 change = 0.0000, $p = .56$) were significant. The model that controlled for anxiety symptoms was similar: the *FSS*age group*sex* interaction was not significant (R^2 change = 0.0003, $p = .16$), the joint test of interaction for *FSS*age group* and *FSS*sex* was not significant (R^2 change = 0.0006, $p = .081$), and, individually, *FSS*age group* (R^2 change = 0.0005, $p = .051$) and *FSS*sex* (R^2 change = 0.0000, $p = .58$) were not significant. These results diverge from the interactions at Path II in the main analysis, where *FSS*age group* was significant for both models of anxiety. To summarise these differences, when T1 executive function was included as a covariate in the main analysis, age group was a significant moderator at Path II for both anxiety models; when T1 executive function was

excluded as a covariate in the sensitivity analysis, age group was no longer a significant moderator at Path II for either anxiety model.

For clinical anxiety, then, a simple mediation model was estimated using PROCESS's preprogrammed Model 4 (Hayes, 2022). For anxiety symptoms, PROCESS Model 11 (Hayes, 2022) specified a three-way interaction at Path I.

5.2.5.2 Estimating Indirect Effects

The results of estimating indirect effects in models without T1 executive function are given in Table 9. For clinical anxiety, the (unmoderated) indirect effect in the base model was significant but became nonsignificant once all covariates were added to the model. For anxiety symptoms, the indirect effects for females aged 45–54, females aged 55–64, and males aged 65–74 were significant in the base model but not in the final model. No indirect effects, as moderated by age group or sex, were significant in fully adjusted models.

In the main analysis, indirect effects within sex and age subgroups were nonsignificant across base and fully adjusted models, for both clinical anxiety and anxiety symptoms (Table 4). Compared to those results, this sensitivity analysis produced statistically significant indirect effects in the base models of clinical anxiety and anxiety symptoms but not in the fully adjusted model. Thus, estimates of indirect effects differed only in the base models across the main and sensitivity analyses. When excluding T1 executive function in base models, T1 FSS significantly mediated the association between T0 anxiety (clinical anxiety and anxiety symptoms) and T2 executive function. After adding T1 executive function to the base models, these mediated effects were no longer significant. In contrast, the absence or presence of T1 executive function did not influence the pattern of results in fully adjusted models. That is, when all covariates were controlled for, indirect effects were consistently nonsignificant in both main and sensitivity analyses.

Table 9. Sensitivity Analyses: Indirect Effect of Anxiety on Executive Function Through Functional Social Support by Sex and Age Group (Unadjusted for T1 Executive Function), Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Moderators | | Indirect Effect <i>b</i> (95% bootstrap CI) | |
|------------------|-------------------|--|---------------------------|
| Sex | Age Group (years) | Clinical Anxiety | |
| | | Base Model ¹ | Final Model ² |
| All Participants | | -0.0079 (-0.0183, -0.0004)* | -0.0012 (-0.0075, 0.0044) |
| Sex | Age Group (years) | Anxiety Symptoms | |
| | | Base Model ¹ | Final Model ² |
| Male | 45–54 | 0.0001 (-0.0025, 0.0024) | 0.0003 (-0.0020, 0.0026) |
| | 55–64 | -0.0022 (-0.0058, 0.0000) | -0.0001 (-0.0013, 0.0010) |
| | 65–74 | -0.0034 (-0.0082, -0.0001)* | -0.0002 (-0.0022, 0.0017) |
| | ≥ 75 | 0.0009 (-0.0029, 0.0055) | 0.0003 (-0.0023, 0.0034) |
| Female | 45–54 | -0.0027 (-0.0061, -0.0003)* | -0.0000 (-0.0010, 0.0007) |
| | 55–64 | -0.0034 (-0.0078, -0.0003)* | -0.0001 (-0.0017, 0.0010) |
| | 65–74 | 0.0003 (-0.0023, 0.0031) | 0.0003 (-0.0018, 0.0027) |
| | ≥ 75 | 0.0004 (-0.0040, 0.0050) | 0.0003 (-0.0023, 0.0034) |

Note. *b* = unstandardised regression coefficient; CI = confidence interval; T0 = baseline; T1 = follow-up 1.

¹Included T0 functional social support and T0 executive function.

²Included the same measures as the base model, with the addition of sociodemographic, social, health, and lifestyle covariates at T0.

**p* < .05

5.2.6 Sensitivity Analysis 2: Estimating Indirect Effects Using Imputed Data

Conditional process analysis was run on each of the nine imputed datasets, where only missing covariate data were imputed. Note that results for the base (unadjusted) models were not pooled, since none of the variables included in the base models were imputed. The PROCESS models (Hayes, 2022) specified for clinical anxiety (Model 14) and anxiety symptoms (Model 68) were the same models used in the main analysis. After obtaining the results for each dataset, the indirect effects (i.e., *ab* estimates) for each level of the moderator(s) were pooled using Rubin's rules (Rubin, 1987).

Overall, the results of the sensitivity analysis incorporating imputed covariate data (Table 10) did not differ from the results observed in the main analysis (Table 4). Indirect effects were nonsignificant across age and/or sex for both clinical anxiety and anxiety symptoms in both base and fully adjusted analyses.

Table 10. Sensitivity Analyses: Indirect Effect of Anxiety on Executive Function Through Functional Social Support by Sex and Age Group After Multiple Imputation of Covariates, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 7,479)

| Moderators | | Indirect Effect <i>b</i> (95% bootstrap CI) | |
|-----------------|-------------------|--|---------------------------|
| Sex | Age Group (years) | Clinical Anxiety | |
| | | Base Model ¹ | Final Model ² |
| Male and female | 45–54 | 0.0019 (-0.006, 0.0104) | 0.0020 (-0.0040, 0.0079) |
| | 55–64 | -0.0057 (-0.0165, 0.0018) | -0.0033 (-0.0104, 0.0038) |
| | 65–74 | -0.0046 (-0.0157, 0.0042) | -0.0031 (-0.0106, 0.0043) |
| | ≥ 75 | 0.0054 (-0.0079, 0.0221) | 0.0042 (-0.0069, 0.0154) |
| Sex | Age Group (years) | Anxiety Symptoms | |
| | | Base Model ¹ | Final Model ² |
| Male | 45–54 | 0.0000 (-0.0014, 0.0013) | -0.0009 (-0.0035, 0.0016) |
| | 55–64 | -0.0016 (-0.0049, 0.0006) | -0.0002 (-0.0018, 0.0014) |
| | 65–74 | -0.0018 (-0.0064, 0.0017) | -0.0007 (-0.0031, 0.0018) |
| | ≥ 75 | -0.0003 (-0.0036, 0.0057) | -0.0008 (-0.0055, 0.0039) |
| Female | 45–54 | 0.0007 (-0.0019, 0.0036) | 0.0001 (-0.0009, 0.0011) |
| | 55–64 | -0.0025 (-0.0072, 0.0009) | -0.0006 (-0.0025, 0.0014) |
| | 65–74 | -0.0001 (-0.0022, 0.0018) | 0.0009 (-0.0016, 0.0034) |
| | ≥ 75 | -0.0004 (-0.0052, 0.0037) | -0.0021 (-0.0079, 0.0038) |

Note. *b* = unstandardised regression coefficient; CI = confidence interval.

¹Included T0 functional social support, T0 executive function, and T1 executive function. Results were not pooled since the data were not imputed.

²Included the same measures as the base model, with the addition of sociodemographic, social, health, and lifestyle covariates at T0. Results were pooled since the covariate data were imputed.

No significant indirect effects were detected at $p < .05$.

To compare the characteristics of the sample pre- and post-imputation, frequencies for categorical variables, mean/SD for normally distributed variables, and median/IQR for skewed variables are provided in Table F1. Statistics were pooled if the variable was imputed. Imputed results for skewed variables were pooled by taking the averages of the median, Q1, and Q3, and deriving the IQR from the pooled Q1 and Q3 estimates. Imputed proportions for categorical variables were pooled using Rubin's rules, which have been coded into the R function `mi.n.p()` (Nahhas, 2025). The function `mi.n.p()` and other functions that pool descriptive data for continuous variables are available through `Functions_rmph.R`, which can be downloaded from https://github.com/rwnahhas/RMPH_Resources.

Comparing these univariate descriptives, mean executive function (at any timepoint) was lower in the pooled imputed data than in the complete-case data. Furthermore, the new participants included in the imputed sample shifted the sample toward being less healthy on most covariates compared to the more restrictive sample in the complete-case dataset. For example, the proportion of participants who had less than a post-secondary education, had lower levels of income, were widowed or divorced/separated, lived alone, had poorer self-rated health, reported more chronic conditions, or were functionally impaired was higher in the imputed sample than in the complete-case sample. In terms of the moderators sex and age group, the relative proportion of women and older individuals was higher in the imputed sample than in the complete-case sample.

Table F2 provides the path effects of the complete-case and pooled imputed data, the latter of which were pooled using Rubin's rules (Rubin, 1987) to further characterise the impact of retaining additional participants and imputing missing covariate data. Broadly speaking, regression coefficients obtained using the imputed data were smaller than the coefficients in the complete-case analysis. Path II effects, which were significant in the 75+ year age group in the complete-case analysis, were no longer significant following imputation for either model that included clinical anxiety or anxiety symptoms as the exposure. However, the Path I association between anxiety symptoms and FSS remained significant among men aged 45 to 54 years. The overall (unstratified) Path I association between clinical anxiety and FSS became nonsignificant after imputing and pooling the effects.

5.2.7 Model Diagnostics

The Path I and Path II models for clinical anxiety and anxiety symptoms did not appear to violate the assumptions of linear regression. Figures Figure G1–Figure G4 depict standard model diagnostic plots for linear regression that demonstrate minimal heteroscedasticity and approximate normal distribution of residuals. There were no influential outliers, since no observation surpassed the Cook’s D threshold of 1 (Kleinbaum et al., 2014). Furthermore, multicollinearity was assessed for Path I and Path II of both anxiety models and judged to be acceptable based on existing guidelines. Specifically, although the variance inflation factor exceeded 10 for several predictors at Path II (for both anxiety models), in no case where condition indices were greater than 30 did two or more variance proportions exceed 0.9 (Kleinbaum et al., 2014).

Chapter 6

Discussion

6.1 Summary of Study Findings

The aim of this study was to determine whether T1 FSS mediated the prospective association between T0 anxiety and T2 executive function across age and sex subgroups, after controlling for prior measures of the mediator and outcome in addition to baseline sociodemographic, social, health, and lifestyle covariates. The results of the main analysis indicated that FSS was not a significant mediator of anxiety (measured either as clinical anxiety or anxiety symptoms) and executive function, for any subgroup defined by age and sex. Furthermore, the sensitivity analyses demonstrated that omitting T1 executive function from adjusted models or imputing covariate data using multiple imputation did not change the overall pattern of effects. That is, the finding of no significant mediation moderated by age or sex remained robust even when greater cognitive change was introduced through the exclusion of T1 executive function or when additional participants were retained in the sample through multiple imputation of missing covariates.

6.2 Mediation Results

The results of this study were consistent with some, but not all, mediation studies that assessed social support (structural or functional) as a mediator of the association between anxiety and cognitive function. Due to the paucity of research on social support as a mediator of anxiety and executive function, the scope of the studies reviewed in this thesis was broadened to include other anxiety-related variables, structural measures of social support, and global cognition or other cognitive domains. However, because of this broadened scope, the inevitable increase in heterogeneity among relevant studies renders direct comparisons more challenging. Inconsistencies in study findings may result from differences in choice of exposure, mediator, outcome, study design, measurement instruments, or a combination thereof.

One notable example of heterogeneity between studies is that most mediation studies examined neuroticism as an exposure, rather than anxiety. As noted previously, neuroticism is an example of trait anxiety—an enduring disposition to perceive threats in the environment and react with anxiety in these situations (Harrison et al., 2015; Wetherell et al., 2002)—that may be similar to clinical anxiety in terms of its chronicity and long-term impacts on health. However, while related, neuroticism

differs from anxiety in that it increases the *tendency* to experience anxiety but, in and of itself, does not always or exclusively entail feelings of anxiety. In total, four mediation studies examined neuroticism as the exposure (Best et al., 2021; Bethell et al., 2024; McHugh Power et al., 2017; Stephan et al., 2024) while one examined fear of falling (Peeters et al., 2018) and one examined anxiety symptoms (Li et al., 2021). If considering only similar exposure variables, Li et al. (2021) matches the current thesis most closely as they also examined anxiety symptoms; however, while they found evidence of significant mediation, the current study did not. Different scales used to measure the same construct may also give rise to different patterns of association. Therefore, the current study's findings may have diverged from Li et al. (2021) because they implemented the Generalized Anxiety Disorder 7-item scale (GAD-7) (Spitzer et al., 2006) to measure anxiety symptoms, whereas this study utilised a 4-item K10 anxiety subscale. The findings of this study were more in line with the studies that assessed different exposures of anxiety (neuroticism and fear of falling), which shared the general conclusion that social support (structural or functional) was not a significant mediator of the association between anxiety and cognitive function (Best et al., 2021; Peeters et al., 2018; Stephan et al., 2024).

Another source of heterogeneity between studies is the variation in the type of social support examined (structural or functional) as the mediating variable. SSS and FSS are related concepts but differ in definition as well as outcomes for health, which may have resulted in divergent findings across this mediation study and others. SSS refers to the size and frequency of engagement with one's social networks, while FSS captures the quality of one's social relationships, regardless of network size or frequency of contact (Sherbourne & Stewart, 1991). In terms of the type of social support assessed as the mediator, three studies examined SSS, namely social activity (Peeters et al., 2018; Stephan et al., 2024) and social engagement (Best et al., 2021); one examined loneliness (Li et al., 2021); and the remaining two incorporated a combined measure of FSS and SSS (Bethell et al., 2024; McHugh Power et al., 2017). However, the findings of the current study were more in line with the studies that assessed SSS as a mediator than similar studies assessing loneliness or a combined FSS/SSS measure. The studies that solely examined SSS did not find significant mediation (Best et al., 2021; Peeters et al., 2018; Stephan et al., 2024) whereas the studies that examined more functional or qualitative aspects of social support did (Bethell et al., 2024; Li et al., 2021; McHugh Power et al., 2017). Because of the close conceptual link between SSS and FSS, investigating the independent effect of one social support variable would be best supported by accounting for the other social

support variable in analyses (as this study attempted to do by controlling for various SSS-related measures); however, most studies did not adjust for the other social support variable. Thus, results may conflict among mediation studies due to the choice of mediator, but also due to possible confounding from other types of social support.

In terms of measures of cognitive function, all but one study (Bethell et al., 2024) assessed global cognition (Best et al., 2021; Li et al., 2021; McHugh Power et al., 2017; Peeters et al., 2018; Stephan et al., 2024), and findings related to mediation were generally mixed. Notably, the study by Bethell et al. (2024) utilised baseline CLSA Comprehensive cohort data to assess mediation. Investigating the subdomains of memory and executive function, they found that social connection (a combined measure of social isolation and loneliness) significantly mediated the association between neuroticism and several tests of executive function (except the Stroop-VV test). They were also the only study to investigate moderation of the mediated effect, specifically by sex; however, like the current study, they did not find sex to be a significant modifier of the mediated effect. Given the similarities in study population, method of data collection, outcome, and moderator, the study by Bethell et al. (2024) may be the most comparable to the current investigation. As previously noted, the current study shares similar findings with other research that examined different exposures (e.g., neuroticism) and mediators (e.g., SSS). Thus, additional sources of heterogeneity in Bethell et al.'s (2024) study may provide a possible explanation for the divergent findings, such as differences in how executive function was analysed, the inclusion of different covariates, and the number of timepoints that were included. First, Bethell et al. (2024) assessed the executive tests individually rather than as a composite score, and doing so may provide an advantage by retaining variations in performance across the tests; however, these variations appeared to be minimal, as all but one of the tests were impacted by neuroticism through social connection. Covariate selection may have had a greater impact on findings, as Bethell et al. (2024) found evidence of significant mediation after adjusting for age, education, and hearing ability (unadjusted mediated effects were not reported). In the current study, significant mediated effects were only detected in the sensitivity analysis that excluded executive function at T1, specifically in the base models that adjusted for T0 FSS and executive function as the sole covariates. When other sociodemographic, social, health, and lifestyle covariates were added to the model, or when T1 executive function was added to the model, these results became nonsignificant, suggesting that mediated effects were fully accounted for by the addition of these variables. In particular, the fact that T1 executive function alone shifted the mediation results

from significant to nonsignificant highlights the minimal cognitive change present in the main analytic sample. This points to another important difference between the studies, which is that Bethell et al. (2024) undertook a baseline cross-sectional analysis while this study leveraged three timepoints of data to conduct a longitudinal investigation.

One may argue that differences in study design, which is of relevance to inferences of temporality in mediation, could have contributed most to the differences in findings observed across studies. Bethell et al. (2024) adopted a cross-sectional model of moderated mediation after determining that there was little cognitive change across baseline and first follow-up CLSA data. Although analysing baseline data may have improved the likelihood of detecting significant mediated effects, it was noted that bidirectional and prodromal effects could have also accounted for the findings due to the absence of temporal ordering in the exposure, mediator, and outcome (Bethell et al., 2024). Mediation studies are recommended to model at least three timepoints to meet the assumption of mediational relationships, which is that the exposure precedes the mediator which itself precedes the outcome. In other words, temporality is key to the interpretation of mediated effects. However, frequently trade-offs occur in longitudinal mediation studies between being able to detect a mediated effect and being able to preserve the temporal ordering of the main analytic variables. With a greater likelihood of losing participants to attrition in longitudinal studies, effects may be underestimated (and thus potentially nonsignificant) due to a lack of cognitive change across follow-up assessments. Among studies that utilised at least three timepoints of data to assess mediation, the current study paralleled their findings of no significant mediation of the association between neuroticism and cognitive function through social engagement (Best et al., 2021) or social activity (Stephan et al., 2024). Notably, with one exception (Peeters et al., 2018), the studies which found statistically significant mediation of anxiety/neuroticism and cognitive function employed cross-sectional or half-longitudinal designs (i.e., the mediator was modelled cross-sectionally with either the exposure or outcome) (Bethell et al., 2024; Li et al., 2021; McHugh Power et al., 2017). Although temporality is unclear when exposure, mediator, and outcome variables are analysed cross-sectionally or at two timepoints, there may have been sufficient variation in cognitive function in these samples to detect differences among those who varied by levels of anxiety or social support. The current study was better able to rule out bidirectional or prodromal explanations by analysing mediation of anxiety and executive function through FSS longitudinally, but attrition and selection biases could have limited detection of greater cognitive change across follow-ups.

While the current thesis attempted to mitigate selection biases associated with healthier individuals returning for follow-up assessments by: 1) excluding T1 executive function from analyses to allow for more cognitive change, and 2) imputing missing covariate data and retaining participants that would have otherwise been excluded for missing responses on these variables, the findings of these sensitivity analyses did not change the overall conclusion that FSS was not a significant mediator of the association between anxiety and executive function. It could be argued that modelling executive function even six years apart in the sensitivity analysis (versus three years apart in the main analysis) is still too short an interval to detect significant changes in cognitive function, particularly for middle-aged participants who may not experience noticeable cognitive decline until later in life, or even for cognitively healthy older participants who are atypical for their age and may not decline as rapidly as the average individual. The inclusion of a middle-aged sample and longer follow-ups are particularly important for informing the role of anxiety as a prodrome versus independent risk factor of cognitive decline. The meta-analysis by Stephan et al. (2024) was able to pool results across four prospective samples of middle-aged and older adults in which cognition was measured in a third wave, 8 to 20 years later. However, even with these longer follow-up periods, the authors found that social activity did not mediate the association between neuroticism and cognition. Weighing the findings of both cross-sectional and longitudinal mediation studies, then, there appears to be a greater likelihood of detecting significant mediated effects when utilising fewer waves of data, raising a possible prodromal interpretation of the associations between anxiety, social support, and cognitive function (e.g., those with greater anxiety may be more likely to report lower social support as well as exhibit poorer cognitive function when measured at the same timepoint). Although other mechanisms appear to link anxiety to poorer cognitive function over time (Stephan et al., 2024), past and current research suggest that the association between anxiety and cognitive function is more likely explained by social support when mediation is examined cross-sectionally rather than longitudinally. Despite the strengths of Stephan et al.'s (2024) meta-analysis, comparisons should still be undertaken with caution, as the current thesis assessed not neuroticism, but anxiety; not social activity, but FSS; and not global cognition, but the specific domain of executive function.

6.3 Path Effects

When assessing the plausibility of the mediated effect, one should pay attention to the magnitude and direction of the path effects (Hayes, 2022). Regarding the inverse association between clinical anxiety

and FSS (Path I), most of the literature is in agreement with the negative impact that anxiety has on social and interpersonal functioning in the general population, including in older adults (Couture et al., 2005; Domènech-Abella et al., 2019; Drageset et al., 2013; Fees et al., 1999; Hoffman et al., 2021; Long & Martin, 2000; Margrett et al., 2011; O’Conor et al., 2019; S. Sun et al., 2024). An attentional bias for threat-related stimuli in those with anxiety (Gray et al., 2020) may lead to negative appraisals of social interactions and relationships (Bell et al., 2011; Stopa & Clark, 2000; Vassilopoulos & Banerjee, 2010), which may come to be held as enduring beliefs by the anxious person as well as affect others’ perception of them (Alden & Taylor, 2004; Hepp et al., 2021). These interpersonal impairments, endorsed by anxious individuals and perceived as a less warm and friendly demeanour by others, may then create a self-perpetuating cycle that lowers FSS over time (Alden & Taylor, 2004). That age was not a significant modifier of the association between clinical anxiety and FSS is consistent with the hypothesis that an attentional bias for negative information may disrupt the late-life motivation to focus on maintaining close, meaningful ties (Carstensen et al., 2003; Hoffman et al., 2021). Because anxious older adults might perceive their relationships less positively compared to others of their age, they may find themselves more similar to their middle-aged counterparts who are less driven by the motivation to maintain emotional positivity (Carstensen & Gottman, 1995). Sex also did not modify the relationship between clinical anxiety and FSS. While anxiety has been found to be less deleterious for women’s social networks than men’s and may serve as motivation in women to seek social support (Curran et al., 2020; McLean & Anderson, 2009), *pathological* anxiety, characterised by higher levels of comorbidity with other anxiety disorders and major depressive disorder, may be more disabling in women compared to men (McLean et al., 2011). Thus, while women bear a higher burden of mental illness, their social networks are more robust and diverse compared to men (Menec et al., 2019; Newall & Menec, 2020), so the impact of clinical anxiety for either sex may ultimately have similar outcomes in terms of the level of social impairment.

In contrast to the negative association between clinical anxiety and FSS, the association between anxiety symptoms and FSS in males aged 45 to 54 years was positive (i.e., higher anxiety symptoms at baseline were associated with higher FSS three years later). This result was contrary to predictions given the impact of clinical anxiety in the literature, which is that higher levels of anxiety would be detrimental to social relationships and lower levels would be more beneficial. However, the measure of anxiety symptoms analysed in this study could have reflected non-pathological levels of anxiety, which may serve more of an adaptive function that, instead of provoking withdrawal, elicits higher

FSS as a form of coping (Robitaille et al., 2012). Men aged 45 to 54 may be better placed than other age groups to seek social support for their anxiety. They may have more social resources to fall back on in times of need (e.g., from children, their spouse, parents, and co-workers) and may be more willing to draw on those resources when faced with familial and professional pressures to address their mental health. That is, at higher levels of anxiety, middle-aged men may feel more motivated to reach out to others for help rather than cope with poor mental health on their own, as is commonly observed among men who endorse less severe symptoms or who have a less robust social network (Murayama et al., 2022; D. T. Smith et al., 2018). Middle-aged women may face similar drivers and facilitators of help-seeking, but they may not necessarily report higher FSS after utilising their social support. While research has shown that actually using social support when it is needed tends to increase perceived social support (Melrose et al., 2015), women demonstrate more regular involvement in their social relationships and are more likely to reach out for social support when needed (Belle, 1991; Kawachi & Berkman, 2001); thus, their perceptions of social support may not change to a great extent after drawing on it to cope with their stress and anxiety. Rather, it may be that middle-aged men are *less* likely to appreciate the support they have available to them until the opportunity arises when they must use it.

Regarding the Path II effects, this study found that FSS at first follow-up, independent of either measure of anxiety, was associated with lower executive function three years later. Two theories have been proposed to explain the negative association between FSS and executive function, one of which links FSS to the increased need and use of social support and the second which asserts that worsening cognitive function may be a negative reaction to increasingly relying on others, also called the reciprocity theory. The first explanation follows a similar logic to the discussion of anxiety symptoms and FSS in men aged 45 to 54, where participants aged 75 years and over may be rating their FSS more highly based on an increased need and use of social support. In particular, at the oldest ages, older adults may be experiencing greater health needs and may find themselves drawing on more social support to compensate for the loss of skills and other abilities. Thus, in cases where FSS is a recent evaluation of the receipt of social support, FSS may be acting as a marker that signals incipient cognitive decline that assessments at a later time may detect following this initial endorsement of high social support (Pillemer et al., 2019). Another explanation for the negative relationship between FSS and executive function is the reciprocity theory of social support (Gleason et al., 2008; Uehara, 1995). According to this theory, receiving support that cannot be returned, whether because of physical or

cognitive limitations, may come to be viewed as a burden or stressor as the individual notices their dependence and begins to doubt their usefulness in the relationship (Uehara, 1995). Consequently, the burden of social support may have deleterious effects on cognitive function through an increase in stress and negative affect (Du et al., 2023; Pillemer et al., 2019; Sims et al., 2014). Younger individuals who are healthier may not need to rely as much on social support, and because they may be more capable of reciprocating social support, they are less likely to experience the negative emotions associated with a lack of reciprocity. In this study, the *positive* effects of social support may have also been attenuated in the younger age groups given the healthier nature of participants at follow-up: some theories, such as the stress-buffering hypothesis, suggest fewer health benefits of social support in the absence of significant stressors (Moskowitz et al., 2013). In contrast, stronger associations between FSS and cognitive function have been found in cross-sectional baseline CLSA studies (Oremus et al., 2020; Rutter et al., 2024) where samples are more likely to reflect a wider continuum of psychosocial functioning. For these reasons, the impact of FSS on executive function may have been stronger and related to a decline in executive function in those aged 75 and over, but not in other age groups.

While the discussion so far suggests that the *direction* of the significant path effects is plausible, a nonsignificant mediated effect may result if these path effects are small or if unique sample characteristics are contributing to an underestimation of the mediated effect. Regarding the path effects, because the indirect effect is defined as the product of Paths I and II, a small effect at either path would result, accordingly, in a small (and potentially nonsignificant) mediated effect. Thus, while there appeared to be an impact of clinical anxiety on FSS (Path I) that could have resulted in changes in executive function in those aged 75 years and over (Path II), the Path I effect was small and would have contributed to a very weak, nonsignificant mediated effect. Similarly, while the association between anxiety symptoms and FSS in men aged 45 to 54 (Path I) was probable given the literature, the magnitude of the effect was small, and mediation may be less likely in this subgroup due to their younger age and the possibility that changes in FSS at these ages would not yet translate to noticeable changes in executive function. To summarise, while clinical anxiety and anxiety symptoms appeared to affect levels of FSS at three-year follow-up, these changes were not substantial enough to influence FSS's impact on executive function at six-year follow-up. The negative association between FSS and executive function (Path II) in individuals aged 75 and over was stronger relative to the impact of clinical anxiety or anxiety symptoms on FSS, but in this age group,

this Path II association may be influenced to a greater extent by exposures other than anxiety, such as a greater need and use of social support.

In addition to small path effects, unique sample characteristics could have also limited the ability to detect a mediated effect. Anxiety, social support, and cognitive function may have stronger associations cross-sectionally than longitudinally because, as noted in other literature, anxiety symptoms may improve between measurements (Evans, Llewellyn, et al., 2019), alleviating their impact and potentially allowing FSS and/or executive function to recover to baseline levels. As was also mentioned earlier, a lack of cognitive change could have also reduced the ability to identify mediated effects, as the current study was only able to model executive function across three years (if T1 executive function was controlled for) or six years (if T1 executive function was *not* controlled for). Factors contributing to this lack of cognitive change include volunteer bias and attrition bias. Since CLSA participants as a whole tend to be healthier, wealthier, and more educated than the general Canadian population (Raina et al., 2019), these characteristics may become overrepresented with subsequent waves of data collection, particularly when complete data are needed for longitudinal analyses and some variables may be linked to reasons for dropout (e.g., poor mental health, low FSS, and low executive function). Consequently, the participants included in the analytic sample—even those who reported an anxiety disorder or higher levels of anxiety symptoms at baseline—may be healthier and possibly more resilient to factors that lower FSS and cognitive function over time than the participants who were excluded, including those in the general population. A highly selective sample, therefore, could have led to an underestimation of mediated effects and biased measures of association towards the null.

Appendix Tables F1 and F2, which summarize results from the second sensitivity analysis on multiple imputation of covariates, aid in characterising some of the selection bias that may be present in the analytic sample. The decrease in mean executive function observed after retaining more participants in the imputed sample (Table F1) suggests that those with missing covariate data tended to have lower executive function, which would have increased the likelihood of nonresponse and noncompletion on assessments (hence the missingness on covariates). While the inclusion of these participants did not change the overall findings, the problem of selection bias may be more substantial than what this thesis has been able to identify, as many more participants were missing data on the key variables of anxiety, FSS, and executive function (see Table C1 for derivation of the analytic sample). Selection bias could have influenced Path II effects in particular, if those with poorer

executive function—most likely those belonging to the oldest age group—had participated in the study *only if* they had adequate FSS. Thus, it might appear that high FSS was associated with decreases in executive function in individuals aged 75 years and over, but the association may be nonsignificant or possibly even reversed if more older individuals with lower FSS had been retained in the sample. The pooled path effects provided in Table F2 offer some support for this hypothesis, as after including more participants in the imputed dataset, the association between FSS and executive function (Path II) was no longer significant in the 75 and older age group. In contrast, the association between anxiety symptoms and FSS (Path I) remained significant for men aged 45 to 54 years, possibly because men and younger individuals were less likely to be excluded from the complete-case sample than were women and older individuals (Table F1).

Despite these differences in sample characteristics and associations at the individual paths, it should be noted that multiple imputation of covariates did not produce moderated mediation results that differed from the results of the main analysis. In either analysis, FSS was not a significant mediator, in any age and sex subgroup, of the relationship between anxiety and executive function. Because this pattern remained relatively consistent across main and sensitivity analyses, as well as across base and fully adjusted models (only the base models without T1 executive function produced significant mediated effects), this study suggests that FSS is unlikely to mediate the association between anxiety and executive function in middle-aged and older men and women.

6.4 Strengths of the Study

Key strengths of the study include quantification of indirect effects moderated by age and sex, the use of a population-based data source that stratified sampling by age and sex, the incorporation of three timepoints of data to preserve the temporal ordering of key variables in the mediation model, examination of two measures of anxiety, the inclusion of numerous covariates to address confounding, and sensitivity analyses that explored the impact of attrition and selection biases on the sample.

Conditional process analysis using PROCESS (Hayes, 2022) allowed for the simultaneous estimation and quantification of indirect effects while requiring fewer statistical assumptions compared to component approaches of estimating mediation, such as the causal steps (Baron & Kenny, 1986) or joint-significance (Yzerbyt et al., 2018) approaches. Moreover, PROCESS estimates conditional indirect effects simultaneously rather than independently, minimising the number of

inferential tests needed to assess moderated mediation and thus improving statistical power and reducing Type II error. The examination of moderated mediation was aided by the use of population-based data that stratified sampling by age, sex, and province, and later oversampled from geographic areas with lower levels of education (CLSA, 2017a, 2023; Raina et al., 2019), ensuring that groups who tend to be underrepresented in population-based studies had adequate sample sizes to support investigating mediated effects across age and sex subgroups.

Also aiding the investigation of mediation was the ability to analyse up to three timepoints of data, which allowed modelling of the exposure at T0, the mediator at T1, and the outcome at T2, as well as adjustment of the antecedent measures of the mediator and outcome. Modelling the key variables in the mediation model at different timepoints is important as an assumption of temporality is intrinsic to the logic of causal mediation, where for an exposure to affect a mediator which in turn affects an outcome, the exposure must precede the mediator which itself precedes the outcome. The availability of three timepoints of data thus allowed the current analyses to satisfy this assumption of temporality.

The breadth of the CLSA allowed the analysis to incorporate a multitude of variables, including two related but distinct exposure variables and various sociodemographic, social, health, and lifestyle covariates. Investigating two measures of anxiety contributed to the reproducibility of findings, not only within, but *across* studies, as most previous research examined the related concept of neuroticism and fewer have studied the impact of anxiety disorders or anxiety symptoms on social support and/or cognitive function. Importantly, the analyses were able to adjust for numerous covariates to control for the presence of confounding. The choice of the K10 scale specifically allowed for the adjustment of depressive symptoms that matched the 30-day reference window of reported anxiety symptoms, which is longer than other screening tools used in the CLSA, such as the Center for Epidemiological Studies Short Depression Scale (CES-D-10) (Andresen et al., 1994; Radloff, 1977) that measures symptoms experienced only in the last seven days.

Lastly, this study was able to probe and mitigate issues related to attrition and missingness that are common in longitudinal analyses. First, imputing up to one missing item on the measure of FSS using mean imputation allowed the main analysis to retain more participants in the sample, thus increasing statistical power and generalisability of the findings. Second, while conducting multiple imputation on covariates as a sensitivity analysis also aided in retaining more participants, the sensitivity analysis offered additional value in enabling a comparison between the imputed data and the complete-case

data, which in turn provided insight into the extent to which attrition and selection biases may have impacted the complete-case analysis. Another sensitivity analysis tested the robustness of the main analysis by omitting T1 executive function and introducing more cognitive change into models. Overall, these procedures shed light on the characteristics of participants who are included in longitudinal analyses, an understanding that ultimately informs the conclusions that can be drawn from these data and the generalisability of findings to the larger population.

6.5 Limitations of the Study

This study has several limitations pertaining to response biases, missingness, generalisability, and construct validity of the anxiety symptoms measure. First, social desirability and recall biases are commonly recognised in survey research as sources of inaccurate reporting, which can reduce the validity of a study's findings. On a sensitive topic such as mental health for which stigma may exist, participants may be more reluctant to disclose details, or they may respond in a manner that is viewed more favourably by others. Misclassification of participants can obscure actual differences between groups and bias parameter estimates toward the null. However, use of the K10 anxiety symptoms subscale helped mitigate responses biases associated with mental health stigma, as the K10 is a measure of general psychological distress and uses more neutral language (e.g., “restless”, “nervous”) to assess functioning. Compared to measures that overtly assess mental health conditions and incorporate clinical terms such as “anxiety disorder”, the K10 may be seen as less stigmatising and therefore encourages more honest reporting.

A second limitation of the study is the selective nature of the sample. At recruitment, CLSA participants were already more likely to be Canadian-born, healthy, educated, and affluent compared to the overall Canadian population (Raina et al., 2019). Recruitment of the Comprehensive cohort was additionally restricted to individuals who lived within 25 to 50 kilometres of 11 data collection sites across Canada. With subsequent restrictions applied to create the analytic sample (detailed in Table C1), participants were included if they completed relevant follow-up assessments in person, meaning participants who transferred to telephone interviews during the COVID-19 pandemic or were missing responses on anxiety, FSS, executive function, and covariate measures were excluded. The pattern of attrition on key analytic variables is likely non-random, as even re-including participants with missing covariates in the sample resulted in a decrease in mean executive function scores and a shift within other variables to an increased proportion of less healthy individuals (Table F1). When considering

additional missingness on mental health, social support, and cognitive variables which, in other CLSA research, have also been linked to poorer executive function (Iacono et al., 2024), it may be reasonable to postulate that the participants included in the final analytic sample were among the healthiest participants in the CLSA. Although there are no grounds to suspect that participants interviewed pre-COVID-19 would be systematically different from participants interviewed during COVID-19 (besides the impact of COVID-19 itself), there are potentially unmeasured differences between these groups that were not accounted for by the study covariates. Taken together, the process of selecting participants for inclusion in the study may have led to an underestimation of mediated effects, as those remaining at the end may be less likely to report lower FSS or experience changes in executive function over time.

The healthiness of the analytic sample limits the ability to generalise study findings to the overall population of middle-aged and older adults. Although an inability to incorporate survey weights into PROCESS may have further reduced generalisability and comparability of results with other weighted CLSA analyses, previous research suggests the use of survey weights may not impact analyses of FSS or cognition in the CLSA (O’Connell et al., 2019; Oremus et al., 2022). Furthermore, there are certain limitations associated with the implementation of survey weights themselves, such as weights being based on all CLSA participants at each timepoint and not the analytic sample and weights for the Comprehensive cohort being based on the regions where participants were recruited rather than being truly national in scope (Raina et al., 2019). Thus, regardless of whether survey weights are applied in analyses, care should still be taken when generalising results based on the Comprehensive cohort to the general population.

The last limitation relates to the construct validity of the anxiety symptoms measure, namely that the scale it was based on, the K10, was not necessarily designed to capture anxiety symptoms. Rather, the K10 is intended to measure nonspecific psychological distress (Kessler et al., 2003), and there is a lack of consensus on which items in the K10 relate to depressive or anxiety symptoms (Brooks et al., 2006; Lace et al., 2019; Peixoto et al., 2021; Sunderland et al., 2012). While dividing the K10 into anxiety and depression subscales has seen some clinical application (Buchanan, 2020), it has been noted that the four-item anxiety subscale—the same used in this study—is less sensitive and accurate than the depression subscale comprising the remaining six items (Lace et al., 2019). While this study was conservative in its choice of an anxiety subscale, selecting items which most factor analyses agreed were related to anxiety, four questions may not be adequate to capture the full spectrum of

anxiety symptoms that may be experienced by older adults, a population within which anxiety symptoms are known to manifest differently compared to younger adults. That is, some symptoms of anxiety may potentially be more related to an older individual's social or cognitive functioning than those captured by the current measure.

6.6 Implications and Future Directions

The current study may be the first to longitudinally examine FSS as a mediator between anxiety and executive function within subgroups defined simultaneously by age and sex. Whether referring to clinical anxiety or anxiety symptoms as the exposure, FSS does not appear to play a significant role in explaining the link between anxiety and executive function, for any age or sex subgroup. These findings suggest that executive function remains relatively resilient to the impacts of anxiety through FSS, to the extent that the small effects of clinical anxiety and anxiety symptoms on FSS, observed in the overall sample and in men aged 45 to 54 years respectively, are not, in turn, associated with subsequent changes in executive function. This is an encouraging finding from a therapeutic standpoint, given the potential of anxiety-related social impairment to affect health in a myriad of ways and the preexisting challenges in targeting other mechanisms of cognitive decline in older adulthood. For example, the impact of anxiety on cognitive function through stress and inflammation may occur over a number of years and necessitate sustained efforts to address stress appraisal and management (Da Silva Coelho et al., 2022). Treatment may be complicated by low compliance, which is common in individuals with anxiety, as well as the presence of cognitive impairment in those who are older. Knowledge of which pathways to target or not target is useful for designing interventions that are both effective and capable of utilising allocated resources efficiently. Given the present finding that FSS does not transmit adverse effects from anxiety to executive function, interventions may be better placed to direct resources to targeting other processes of cognitive decline in aging adults experiencing anxiety. However, these findings should still be interpreted with caution, as biases related to missing data may have led to an underestimation of mediated effects.

This study provided a broad overview of the mechanistic role FSS may play in the association between anxiety and executive function. Future studies examining subtypes of FSS may be particularly relevant given the possibility provided by this study that anxiety symptoms may increase, rather than decrease, FSS in certain subgroups. For example, middle-aged men may not require tangible support while they are still functionally capable of handling physical tasks themselves, but

may elect to seek more emotional support to help cope with the stresses and anxieties in their life. Help-seeking behaviour that leverages certain subtypes of social support may potentially facilitate anxiety-related coping, but further research is needed to determine whether this can protect cognitive function in the long term. Additionally, other possible mediators beyond social factors should be considered. Anxiety's close affiliation with stress may affect cardiovascular health, sleep quality, or even lifestyle behaviours such as substance use, which also have ties to cognitive function. Although age and sex did not moderate the mediated effect of anxiety and executive function in this study, it would be worth revisiting these moderators with other mediators. SSS, which was partially adjusted for as a covariate in this study, could also be re-examined as a moderator given the potential buffering role it may have in mitigating the impact of anxiety on cognitive function (D'Amico et al., 2024; Liu et al., 2024).

The K10 scale was used to assess baseline anxiety symptoms, but more dedicated instruments of anxiety may be available for analysis. For instance, the GAD-7 (Spitzer et al., 2006), a widely adopted tool for screening generalised anxiety symptoms, was introduced as an additional measure of anxiety in the CLSA starting at second follow-up. With subsequent follow-up assessments, it may be possible to examine the GAD-7 as an exposure variable. Relatedly, it may be worthwhile to explore anxiety as a mediator and FSS as the exposure, given the bidirectional associations between these factors. The inclusion of additional follow-up assessments may also enable the incorporation of greater cognitive change into analyses and a larger sample utilising the entire Comprehensive cohort, unimpacted by COVID-19 pandemic restrictions. Finally, future studies might also consider refining the use of MI for variables beyond covariates to minimise the extent of missing data in CLSA analyses, particularly on cognitive tests.

6.7 Conclusion

A global aging population and the recognition that more attention should be paid to mental health conditions outside of depression has spurred a heightened interest in studying anxiety in older adults. As knowledge on how anxiety influences cognition continues to develop, a common theme emerges in the literature stressing the need for more longitudinal studies and an examination of mediators to clarify the causal nature of the relationship (Gulpers et al., 2016). The current study examined FSS as a mediator of the association between anxiety and executive function, a cognitive domain that plays a crucial role in maintaining independence in later life. The finding that FSS did not significantly

mediate the association between a self-reported clinical diagnosis of anxiety or self-reported anxiety symptoms and executive function suggests that additional provision of FSS may not be required to counteract the impact of anxiety and maintain executive function in middle-aged and older adults. Future studies can build upon these findings by examining subtypes of FSS, other stress-related mediators, additional moderators such as SSS, and alternative measures of anxiety, as well as incorporating additional follow-up assessments or advanced missing data strategies to enhance the retention of participants in longitudinal analyses. By taking preliminary steps to investigate FSS as a mechanism that links anxiety and executive function, specifically within subgroups of age and sex, this study contributes to an emergent understanding of how anxiety impacts executive function over time, which in turn informs the development of programmes and interventions aimed at promoting the cognitive health of aging adults, particularly those with anxiety.

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Appendix A

Literature Search Strategy

Systematic literature searches were conducted using PubMed (1950 to present) and PsycINFO (1840 to present) and combined on August 30, 2023. Results using a revised search strategy were updated on January 11, 2025. The revised search strategy eliminated several keywords to improve identification of relevant articles. These terms were related to post-traumatic stress disorder and were excluded in the revised search to reflect the DSM-5's categorisation of trauma and stress-related disorders as distinct from anxiety disorders. Tables A1 and A2 present the revised search strategies using the updated keywords. Figure A1, which depicts the PRISMA flowchart, captures the search results of both the original and revised search strategies. The combined search of PubMed and PsycINFO databases, using both original and revised search strategies, produced 2464 articles after removing duplicates, which reduced to 29 after applying exclusion criteria.

Articles were excluded if studies were conducted on non-human populations, children, or adolescents. Articles were also excluded if they were case reports, case series, opinion pieces, lectures or perspectives; were not available in English; included trauma- or stress-related disorders as the main exposure; did not include either global cognition or executive function as an outcome; did not include anxiety or social support (functional or structural) as an exposure; or were retracted.

Studies were included if they explicitly assessed social support (functional or structural) as a mediator between anxiety and cognitive function. Due to a limited number of mediation studies on anxiety, FSS, or executive function, inclusion criteria were expanded to include anxiety-related terms such as fear and neuroticism, SSS and related social factors, and global cognition including subdomains other than executive function. Also included were studies that examined the association between social support and cognitive function while accounting for anxiety (e.g., through adjustment, stratification, matching, standardisation, or restriction), consistent with approaches of estimating Path II in mediation models where the exposure is controlled for as a covariate (Baron & Kenny, 1986; Hayes, 2022).

Table A1. PubMed Search Strategy

| | Predictor | Outcome | Population | Additional Variable (Mediator) |
|------------------------|---|---|--|---|
| Concept | Anxiety | Executive Function | Middle Age OR Older Adult (45+) | Functional Social Support (FSS) |
| Author Keywords | Anxiety OR Anxiety Disorder* OR Worry OR Fear OR Phobi* OR Neuroticism | Cognitive Function*[tiab] OR Executive Function*[tiab] OR Cognitive Abilit*[tiab] OR Cognitive Impairment*[tiab] OR Cognitive Deficit*[tiab] OR Cognitive Performance[tiab] OR Executive Control*[tiab] OR Executive Dysfunction[tiab] | Older Adult* OR Elderly[T W] OR Middle Age | Social Support*[tiab] OR Perceived Support*[tiab] OR Emotional Support*[tiab] OR MOS- SSS[tiab] OR Social Interaction OR Social Connection OR Support Relations* OR Social Engagement OR Social Participation OR Social Withdrawal OR Loneliness OR Social Isolation OR Interpersonal Relations* OR Social Capital |
| MeSH Terms | Anxiety[MeSH] OR Psychological Distress[MeSH] OR Fear[MeSH] OR Anxiety Disorders[MeSH:n oexp] OR Neuroticism[MeSH] | Cognition[MeSH:noexp] OR Cognition Disorders[MeSH] OR Executive Function[MeSH] OR Dementia[MeSH] | Aged[MeS H] OR Middle Aged[MeS H] | Social Support[MeSH] OR Interpersonal Relations[MeSH] OR Social Interaction[MeSH] OR Social Isolation[MeSH] |

Search strategy: (Anxiety OR Anxiety Disorder* OR Worry OR Fear OR Phobi* OR Neuroticism OR Anxiety[MeSH] OR Psychological Distress[MeSH] OR Fear[MeSH] OR Anxiety Disorders[MeSH:noexp] Neuroticism[MeSH]) AND (Cognitive Function*[tiab] OR Executive Function*[tiab] OR Cognitive Abilit*[tiab] OR Cognitive Impairment*[tiab] OR Cognitive Deficit*[tiab] OR Cognitive Performance[tiab] OR Executive Control*[tiab] OR Executive Dysfunction[tiab] OR Cognition[MeSH:noexp] OR Cognition Disorders[MeSH] OR Executive Function[MeSH] OR Dementia[MeSH]) AND (Older Adult* OR Elderly[TW] OR Middle Age OR Aged[MeSH] OR Middle Aged[MeSH]) AND (Social Support*[tiab] OR Perceived Support*[tiab] OR Emotional Support*[tiab] OR Social Interaction OR Social Connection OR Support Relations* OR Social Engagement OR Social Participation OR Social Withdrawal OR Loneliness OR Social Isolation OR Interpersonal Relations* OR Social Capital OR Social Support[MeSH] OR Interpersonal Relations[MeSH] OR Social Interaction[MeSH] OR Social Isolation[MeSH])

Number of results: 2345

Date of search: January 11, 2025

Table A2. PsycINFO Search Strategy

| | Predictor | Outcome | Population | Additional Variable of Interest |
|------------------------|---|---|--|--|
| Concept | Anxiety | Executive Function | Middle Age OR Older Adult (45+) | Functional Social Support |
| Author Keywords | Anxiety OR Anxious OR Worry OR Fear OR Phobi* OR "Psychological Distress" OR Neuroticism OR "Emotional Stability" | "Cognitive Function*" OR "Cognitive Abilit*" OR "Cognition" OR "Cognitive Disorder*" OR "Cognitive Impairment" OR "Cognitive Performance" OR "Executive Function*" OR "Executive Control*" OR "Executive Process*" OR "Executive Dysfunction" OR "Dementia" | "Middle Age*" OR "Older Adult*" OR Elderly | "Social Support*" OR "Perceived Support" OR "Emotional Support" OR "MOS-SSS" OR "Social Connection" OR "Social Relations*" OR "Interpersonal Relations*" OR "Social Interaction" OR "Social Engagement" OR "Social Participation" OR "Social Capital" OR "Social Withdrawal" OR Loneliness OR "Social Isolation" |

Search Strategy: ((Keywords: (Anxiety) OR Keywords: (Anxious) OR Keywords: (Worry) OR Keywords: (Fear) OR Keywords: (Phobi*) OR Keywords: ("Psychological Distress") OR Keywords: (Neuroticism) OR Keywords: ("Emotional Stability")) OR (abstract: (Anxiety) OR abstract: (Anxious) OR abstract: (Worry) OR abstract: (Fear) OR abstract: (Phobi*) OR abstract: ("Psychological Distress") OR abstract: (Neuroticism) OR abstract: ("Emotional Stability"))) AND ((Keywords: ("Cognitive Function*") OR Keywords: ("Cognitive Abilit*") OR Keywords: ("Cognition") OR Keywords: ("Cognitive Disorder*") OR Keywords: ("Cognitive Impairment") OR Keywords: ("Cognitive Performance") OR Keywords: ("Executive Function*") OR Keywords: ("Executive Control*") OR Keywords: ("Executive Process*") OR Keywords: ("Executive Dysfunction") OR Keywords: ("Dementia")) OR (abstract: ("Cognitive Function*") OR abstract: ("Cognitive Abilit*") OR abstract: ("Cognition") OR abstract: ("Cognitive Disorder*") OR abstract: ("Cognitive Impairment") OR abstract: ("Cognitive Performance") OR abstract: ("Executive Function*") OR abstract: ("Executive Control*") OR abstract: ("Executive Process*") OR abstract: ("Executive Dysfunction") OR abstract: ("Dementia"))) AND ((Keywords: ("Middle Age*") OR Keywords: ("Older Adult*") OR Keywords: (Elderly)) OR (abstract: ("Middle Age*") OR abstract: ("Older Adult*") OR abstract: (Elderly))) AND ((Keywords: ("Social Support*") OR Keywords: ("Perceived Support") OR Keywords: ("Emotional Support") OR Keywords: ("MOS-SSS") OR

Keywords: ("Social Connection") OR Keywords: ("Social Relations*") OR Keywords: ("Interpersonal Relations*") OR Keywords: ("Social Interaction") OR Keywords: ("Social Engagement") OR Keywords: ("Social Participation") OR Keywords: ("Social Capital") OR Keywords: ("Social Withdrawal") OR Keywords: (Loneliness) OR Keywords: ("Social Isolation")) OR (abstract: ("Social Support*") OR abstract: ("Perceived Support") OR abstract: ("Emotional Support") OR abstract: ("MOS-SSS") OR abstract: ("Social Connection") OR abstract: ("Social Relations*") OR abstract: ("Interpersonal Relations*") OR abstract: ("Social Interaction") OR abstract: ("Social Engagement") OR abstract: ("Social Participation") OR abstract: ("Social Capital") OR abstract: ("Social Withdrawal") OR abstract: (Loneliness) OR abstract: ("Social Isolation"))

Number of results: 472

Date of search: January 11, 2025

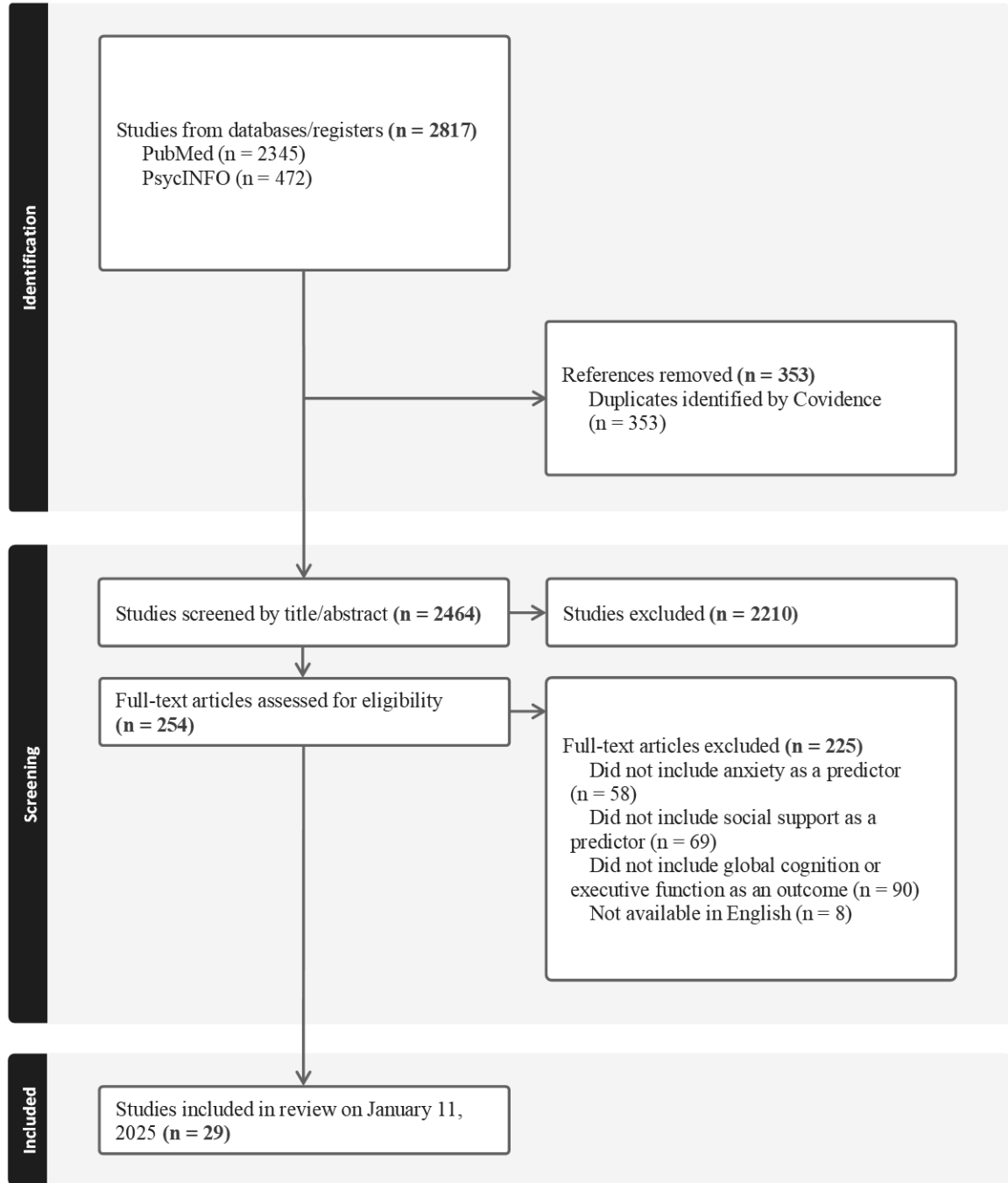


Figure A1. PRISMA Flowchart

Additional exclusions: Non-human studies, studies conducted solely on children or adolescents; dissertations, case reports, case series, opinion pieces, lectures or perspectives; included trauma- or stress-related disorders as the main exposure; retracted papers

Appendix B

Summary of Key Literature

Table B1. Summary of Relevant Literature

| Study | Population, Design, Sample Characteristics | Independent Variables | Dependent Variables | Analysis | Moderation by Age or Sex/Gender? | Results and Conclusions |
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| <p>Alper, H. E., Tuly, R. A., Seil, K., & Brite, J. (2020). Post-9/11 Mental Health Comorbidity Predicts Self-Reported Confusion or Memory Loss in World Trade Center Health Registry Enrollees. Int J Environ Res Public Health, 17(19).</p> <p>CATEGORIES: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>10766 enrollees in the World Trade Center Health Registry (aged 35–64 at wave 4).</p> <p>Longitudinal design.</p> <p>United States.</p> | <p>Exposure: Mental health comorbidity (probable PTSD, depression, and generalized anxiety disorder) at wave 3.</p> <p>Covariates: Gender (male/female), age at wave 3, race/ethnicity, educational attainment, marital status at wave 3, social support at wave 3, rescue/recovery workers (RRW) status at wave 1.</p> | <p>Outcome: Self-reported confusion or memory loss (CML) at wave 4.</p> | <p>Analysis: Log-binomial regression.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>CML exhibited a dose-response relationship with mental health comorbidity, after controlling for gender, race/ethnicity, age at wave 3, education, marital status, and social support, and RRW status.</p> <p>Worsening CML exhibited a moderate to strong association with mental health comorbidity among enrollees who reported CML, after controlling for gender, race/ethnicity, age at wave 3, education, marital status, and social support.</p> <p>Conclusions: Those who have multiple mental health conditions are at greater risk of developing confusion or memory loss.</p> <p>Considerations: Unadjusted RRs were not reported, and anxiety independent of PTSD and/or depression was not examined.</p> |

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| <p>Best, R. D., Cruitt, P. J., Oltmanns, T. F., & Hill, P. L. (2021). Neuroticism predicts informant reported cognitive problems through health behaviors. <i>Aging Ment Health, 25</i>(12), 2191–2199.</p> <p>CATEGORIES: Social support as a mediator between anxiety and cognitive function</p> | <p>829 participants from the St. Louis Personality and Aging Network (SPAN) cohort study (aged 60–73 at baseline).</p> <p>Longitudinal design.</p> <p>United States.</p> | <p>Exposure: Personality (conscientiousness, openness, agreeableness, extraversion, and neuroticism) (wave 1).</p> <p>Covariates: Age, gender, race, years of education.</p> <p>Mediators: Health behaviours, social engagement (wave 2).</p> | <p>Outcome: Informant reported cognitive problems (wave 3).</p> | <p>Analysis: Bootstrapped mediation analysis.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Neuroticism was the only personality trait that predicted informant reported cognitive problems. Health behaviours, specifically wellness maintenance, partially mediated the relationship between neuroticism and cognitive problems. Social engagement did not mediate the relationship between personality and cognitive problems.</p> <p>After controlling for covariates, the indirect effect of wellness maintenance was no longer significant, although the estimate remained similar, suggesting a small effect.</p> <p>Conclusions: Wellness maintenance partially explained the association between neuroticism and informant reported cognitive problems, whereas social engagement did not.</p> <p>Considerations: Some items on the social engagement measure were excluded (e.g., subjective items related to personality).</p> |
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| <p>Bethell, J., Andrew, M. K., Hothi, S., Mick, P., Morgan, D., O’Connell, M. E., ... McGilton, K. S. (2023). Does social connection mediate the association between neuroticism and cognition? Cross-sectional analysis of the Canadian Longitudinal Study on Aging. <i>Aging & Mental Health</i>, 28(3), 482–490.</p> <p>CATEGORY: Social support as a mediator between anxiety and cognitive function</p> | <p>27,765 Canadians aged 45–85 years at baseline from the CLSA Comprehensive Cohort.</p> <p>Cross-sectional design.</p> <p>Canada.</p> | <p>Exposure: Neuroticism.</p> <p>Covariates: Age, educational attainment, hearing.</p> <p>Mediators: Social connection (social isolation and loneliness).</p> <p>Moderators: Sex.</p> | <p>Outcome: Cognition (Rey Auditory Verbal Learning Test immediate and delayed recall, Animal Fluency Test, Mental Alternation Test, Controlled Oral Word Association Test, and Stroop Test).</p> | <p>Analysis: Structural equation modelling (SEM).</p> | <p>Females reported higher mean levels of neuroticism, loneliness, and social isolation. Females also had higher mean scores on memory, higher scores on COWAT, lower scores on AFT and MAT, and similar scores on Stroop.</p> <p>The partial mediation of neuroticism and cognition through social connection did not differ by sex.</p> | <p>Neuroticism was inversely associated with all measures of cognition except Stroop. Social connection partially mediated the association between neuroticism and cognition in both males and females.</p> <p>Conclusions: Social connection may partially mediate the association between neuroticism and cognition, and this mediation does not appear to differ between males and females.</p> |
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| <p>D’Amico, D., Alter, U., Laurin, D., Ferland, G., & Fiocco, A. J. (2024). Examining a healthy lifestyle as a moderator of the relationship between psychological distress and cognitive decline among older adults in the NuAge study. <i>Gerontology</i>, 70(4), 418–428.</p> <p>CATEGORY: Interaction of anxiety and social support</p> | <p>1,272 cognitively intact older adults aged 67+ at baseline from the Quebec Longitudinal Study on Nutrition and Successful Aging (NuAge).</p> <p>Longitudinal design.</p> <p>Canada.</p> | <p>Exposure: Psychological distress.</p> <p>Covariates: Age, sex, educational attainment, daily energy intake, marital status, BMI, diabetes, hypertension, smoking status.</p> <p>Moderators: Healthy lifestyle indicator (HLI) composite score of Mediterranean diet intake, physical activity, and social engagement.</p> | <p>Outcome: Cognitive function (Modified Mini-Mental Examination [3MS]).</p> | <p>Analysis: Linear mixed-effects modelling.</p> | <p>Psychological distress was associated with steeper decline among males but not in females, but women experienced less cognitive decline overall, so ability to detect an effect in females may be limited.</p> <p>There were no associations found between distress and baseline cognitive function in either males or females.</p> | <p>HLI did not significantly moderate distress and cognitive function. However, social engagement was a significant moderator of distress and cognitive decline. Specifically, social engagement buffered the effects of higher levels of distress on cognitive decline.</p> <p>Conclusions: The longitudinal association between psychological distress and cognitive decline may be sex dependent. Social engagement may buffer the effects of distress on cognitive decline.</p> <p>Considerations: Distress and social engagement were entered simultaneously in models; unadjusted estimates where either variable was excluded was not provided.</p> |
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| <p>Evans, I. E. M., Llewellyn, D. J., Matthews, F. E., Woods, R. T., Brayne, C., & Clare, L. (2019). Social isolation, cognitive reserve, and cognition in older people with depression and anxiety. <i>Aging Ment Health, 23</i>(12), 1691–1700.</p> <p>CATEGORIES: Social support predicting cognitive function in populations with anxiety</p> | <p>123 adults with depression or anxiety (aged 65+).</p> <p>Longitudinal design.</p> <p>Wales.</p> | <p>Exposure: Social isolation (structural and functional).</p> <p>Covariates: Age, gender, education, cardiovascular risk factors.</p> <p>Moderators: Cognitive reserve.</p> | <p>Outcome: Cognitive function.</p> | <p>Analysis: Linear regression.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>In bivariate analyses, older people with depression or anxiety were more likely to report higher feelings of loneliness despite having the same amount of social contact as those with depression or anxiety.</p> <p>In older people with depression or anxiety, social isolation was associated with cognitive function at baseline but not change in cognition over two-year follow-up controlling for all covariates. Cognitive reserve was not a moderator at either baseline or follow-up.</p> <p>Conclusions: In older adults with depression or anxiety, social isolation may be associated with cognitive function cross-sectionally but not longitudinally.</p> <p>Considerations: The cross-sectional and longitudinal differences in findings may be due to a reduction in mood symptoms at follow-up as well as a lack of significant cognitive change at follow-up.</p> |
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| <p>Foong, H. F., Hamid, T. A., Ibrahim, R., & Haron, S. A. (2018). Mediating role of neuroticism in the association between loneliness and cognitive function among community-dwelling older adults. <i>Australas J Ageing</i>, 37(4), 283–287.</p> <p>CATEGORY: Anxiety as a mediator between social support and cognitive function</p> | <p>2322 community-dwelling older adults (aged 60–92).</p> <p>Cross-sectional design.</p> <p>Malaysia.</p> | <p>Exposure: Loneliness.</p> <p>Covariates: Age, sex, marital status, educational achievement, household income.</p> <p>Mediators: Neuroticism.</p> | <p>Outcome: Cognition (MMSE).</p> | <p>Analysis: Hierarchical multiple linear regression.</p> | <p>In bivariate analyses, women scored higher in loneliness and neuroticism, but lower on MMSE, compared to men.</p> <p>Moderation in multivariable analyses was not pursued.</p> | <p>Controlling for covariates, loneliness and neuroticism both negatively predicted cognitive function in older adults. When neuroticism was added into the model with loneliness, the impact of loneliness was reduced and no longer significant.</p> <p>Conclusions: Neuroticism fully mediated the relationship between loneliness and cognition.</p> <p>Considerations: Due to the cross-sectional design, temporality cannot be ascertained.</p> |
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| <p>Foong, H. F., Hamid, T. A., Ibrahim, R., & Haron, S. A. (2018). Information processing speed as a mediator between psychosocial stress and global cognition in older adults. Psychogeriatrics, 18(1), 21–29.</p> <p>CATEGORIES: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>2322 community-dwelling older adults (aged 60+).</p> <p>Cross-sectional design.</p> <p>Malaysia.</p> | <p>Exposure: Psychosocial stress (stress, depression, loneliness, and neuroticism).</p> <p>Covariates: Age, sex, marital status, years of education, household income.</p> <p>Mediators: Information processing speed.</p> <p>Moderators: Gender.</p> | <p>Outcome: Global cognition.</p> | <p>Analysis: Structural equation modelling (SEM).</p> | <p>In bivariate analyses, men scored significantly higher on global cognition, whereas women scored significantly higher on all psychosocial stress variables.</p> <p>In multivariable analyses, the relationship between psychosocial stress and global cognition was significant in men but not in women.</p> | <p>Controlling for covariates, there was a negative association between psychosocial stress and cognitive function, with this association being partially mediated by information processing speed.</p> <p>Sex, age, years of education, household income, marital status, psychosocial stress, and information processing speed were all independently associated with global cognition.</p> <p>Conclusions: Information processing speed partially explains the association between psychosocial stress and cognitive function.</p> <p>Considerations: The four indicators of psychosocial stress were not examined separately. Unadjusted estimates were not provided.</p> |
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| <p>Fung, A. W. T., & Lam, L. C. W. (2017). A cross-sectional study on clinical correlates of anxiety disorders in 613 community living older adults in Hong Kong. Int J Geriatr Psychiatry, 32(7), 742–749.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>613 community-dwelling, non-demented older adults (aged 60+).</p> <p>Cross-sectional design.</p> <p>Hong Kong.</p> | <p>Exposure: Anxiety disorders.</p> <p>Covariates: Lifestyle, social factors (functional and structural social support), health burdens, sociodemographic characteristics.</p> | <p>Outcome: Cognitive function.</p> | <p>Analysis: Binary logistic regression.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>In bivariate analyses, presence of anxiety disorders was associated with the quality (functional) but not the quantity (structural) of social relationships. Participants with anxiety disorders reported fewer confidantes.</p> <p>After adjustment for age, gender, education, health-related and social covariates, anxiety independent of depression was related to poor delayed recall and impaired judgment.</p> <p>Conclusions: Those with late-life anxiety report fewer people they can confide in. Late life anxiety disorders are associated with early cognitive decline in non-demented participants without clinical depression.</p> <p>Considerations: Unadjusted estimates were not provided.</p> |
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| <p>Hsieh, N., Liu, H., & Lai, W. H. (2021). Elevated Risk of Cognitive Impairment Among Older Sexual Minorities: Do Health Conditions, Health Behaviors, and Social Connections Matter? Gerontologist, 61(3), 352–362.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>3567 community-dwelling older adults (aged 50–97).</p> <p>Cross-sectional design.</p> <p>United States.</p> | <p>Exposure: Sexual orientation, mental health (depressive symptoms, anxiety symptoms), physical comorbidity, health behaviours (smoking, drinking, exercise), social connection (marital status, number of close family members, living arrangement, community participation).</p> <p>Covariates: Age, gender, education, race/ethnicity.</p> <p>Mediators: Mental health, physical comorbidity, health behaviours, social connections, sociodemographic characteristics.</p> <p>Moderators: Sexual orientation.</p> | <p>Outcome: Cognitive health.</p> | <p>Analysis: Ordinal logit regression.</p> <p>Karolson-Holm-Breen (KHB) mediation.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Anxiety symptoms were not a significant predictor of cognitive impairment when added to a model consisting of sexual orientation, age, gender, education, race/ethnicity, and depressive symptoms.</p> <p>This association did not change when indicators of physical comorbidity, health behaviours, and social connection were added to the model.</p> <p>Social connection was also not significant with cognitive health after all covariates were added to the model.</p> <p>Depressive symptoms were the only mechanism that, partially, accounted for the cognitive health disparity by sexual orientation in the analyses.</p> <p>Conclusions: Anxiety symptoms and social connection do not significantly increase the odds of cognitive impairment independent of other cited risk factors of cognitive impairment.</p> |
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| <p>James, B. D., Wilson, R. S., Barnes, L. L., & Bennett, D. A. (2011). Late-life social activity and cognitive decline in old age. J Int Neuropsychol Soc, 17(6), 998–1005.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>1138 community-based older adults without dementia enrolled in the Rush Memory and Aging Project (aged 65+).</p> <p>Longitudinal design.</p> <p>Other: United States.</p> | <p>Exposure: Social activity.</p> <p>Covariates: Age, sex, education, race, social network size, depression, chronic conditions, disability, neuroticism, extraversion, cognitive activity, physical activity, income.</p> | <p>Outcome: Cognitive function.</p> | <p>Analysis: Linear mixed effects modelling.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Adjusted for socioeconomic status, social network size, health, disability, affect, personality, and physical and cognitive activity, more frequent social activity was associated with reduced rates of cognitive decline over an average of 5 years of follow-up.</p> <p>This association held after excluding the most cognitively impaired from the sample to account for the possibility of reverse causation.</p> <p>Conclusions: Social activity may slow cognitive decline in late life.</p> <p>Considerations: Unadjusted estimates were not provided, and the independent influence of neuroticism as a covariate was not reported on.</p> |
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| <p>Kassem, A. M., Ganguli, M., Yaffe, K., Hanlon, J. T., Lopez, O. L., Wilson, J. W., Ensrud, K., & Cauley, J. A. (2018). Anxiety symptoms and risk of dementia and mild cognitive impairment in the oldest old women. <i>Aging Ment Health, 22</i>(4), 474–482.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>1425 community-dwelling older women (aged 65+).</p> <p>Longitudinal design.</p> <p>United States.</p> | <p>Exposure: Anxiety symptoms.</p> <p>Covariates: Age, education, race, marital status, medical history, smoking status, alcohol use, caffeine intake, exercise (walking), self-rated health status, functional status, depressive symptoms, sleep quality, medication use.</p> | <p>Outcome: Cognitive decline, cognitive impairment.</p> | <p>Analysis: Logistic regression.</p> | <p>Analyses were conducted on a sample of oldest old women.</p> | <p>Women with mild anxiety symptoms at baseline were more likely to develop dementia at follow-up compared to women with no anxiety symptoms at baseline (unadjusted OR = 1.58). This association remained statistically significant after adjusting for potential confounders (adjusted OR = 1.66).</p> <p>Moderate/severe anxiety symptoms were not significantly associated with risk of dementia; however, women who had moderate/severe anxiety symptoms and no anxiety symptoms at follow-up were more likely to have dementia at follow-up (unadjusted OR = 2.81), which remained significant even after adjusting for confounders (adjusted OR = 3.80).</p> <p>Any level of anxiety symptoms was not significantly associated with risk of MCI. Anxiety symptoms were also not significantly associated with cognitive decline after adjusting for confounders.</p> <p>Conclusions: Mild symptoms of anxiety predicted dementia but not more severe symptoms of anxiety which may suggest that anxiety is an early symptom of cognitive decline.</p> <p>Considerations: The individual influence of social support-related covariates (e.g., marital status) was not examined.</p> |
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| <p>Kobayashi, L. C., O’Shea, B. Q., Joseph, C., & Finlay, J. M. (2022). Acute relationships between mental health and cognitive function during the COVID-19 pandemic: Longitudinal evidence from middle-aged and older US adults. SSM Ment Health, 2, 100097.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>Multi-frame, nonprobability sample of 2262 adults residing in all 50 U.S. states, the District of Columbia, and Puerto Rico (aged 55+).</p> <p>Longitudinal design.</p> <p>United States.</p> | <p>Exposure: Loneliness, anxiety, worry about COVID-19.</p> <p>Covariates: Age, sex, race/ethnicity, highest educational attainment, pre-COVID-19 employment status, relationship status, previous physical diagnosis of a physical or mental health condition, use of a mobility aid, pre-COVID-19 social isolation (all baseline).</p> <p>Additionally, depressive symptoms, loneliness, anxiety symptoms, and worry about COVID-19 were measured monthly and treated as time-varying confounders.</p> | <p>Outcome: Self-reported cognitive function and abilities.</p> | <p>Analysis: Logistic regression, marginal structural modelling.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Loneliness and anxiety symptoms were significantly associated with worse perceived cognitive function and abilities but worry about COVID-19 was not.</p> <p>These results held after the other exposures were accounted for in analyses. In particular, after adjusting for loneliness and worry about COVID-10, the between-persons and within-persons estimate for anxiety symptoms and perceived cognitive function reduced from -5.48 to -5.45 and -4.22 to -4.12, respectively. Reductions were similar for perceived cognitive abilities.</p> <p>For all three exposure variables, between-persons associations were stronger in magnitude than within-persons associations, and the strongest magnitudes of association were observed for anxiety symptoms and cognitive outcomes.</p> <p>Conclusions: Elevated loneliness and anxiety symptoms were associated with worse perceived cognitive outcomes, and these associations were observed in adults of similar age and to one's usual levels.</p> <p>Considerations: The independent influences of loneliness and worry about COVID-19 on the association between anxiety symptoms and</p> |
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| | | | | | | perceived cognitive outcomes cannot be determined. |
| <p>Li, W., Sun, H., Xu, W., Ma, W., Yuan, X., Wu, H., & Kou, C. (2021). Leisure activity and cognitive function among Chinese old adults: The multiple mediation effect of anxiety and loneliness. Journal of Affective Disorders, 294, 137–142.</p> <p>CATEGORY: Social support as a mediator between anxiety and cognitive function</p> | <p>6525 community-dwelling older adults enrolled in the Chinese Longitudinal Healthy Longevity Survey (aged 65+).</p> <p>Cross-sectional design.</p> <p>China.</p> | <p>Exposure: Leisure activity.</p> <p>Covariates: Age, gender, current residence, marital status, co-residence of respondent, self-rated health, smoking, drinking.</p> <p>Mediators: Anxiety symptoms, loneliness.</p> | <p>Outcome: Cognitive function.</p> | <p>Analysis: Serial multiple mediation analysis using PROCESS macro.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Anxiety and loneliness independently and serially mediated the effect of leisure activity on cognitive function. In the serial mediation analysis, anxiety had a significant direct effect of 0.3023 on loneliness, which in turn had a direct effect of -0.0272 on cognitive function.</p> <p>Conclusions: Greater leisure activity is associated with lower anxiety, helping to reduce feelings of loneliness, which in turn is related to higher cognitive function.</p> |

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| <p>Liu, Y., Chang, J., Zhao, Y., & Tang, Y. (2024). Neuroticism personality, social contact, and dementia risk: A prospective cohort study. Journal of Affective Disorders, 358, 391–398.</p> <p>CATEGORY: Interaction of anxiety and social support</p> | <p>393,939 participants with a mean (SD) age of 56.4 (8.1) from the UK Biobank, assessed from 2006 to 2010 and followed up until December 2022.</p> <p>Longitudinal design.</p> <p>UK.</p> | <p>Exposure: Neuroticism.</p> <p>Covariates: Age, sex, ethnicity, educational attainment, socioeconomic status, smoking status, alcohol intake, physical activity, sleep duration, healthy diet score, Body Mass Index (BMI), hypertension status, diabetes status, high cholesterol status, cardiovascular disease status, APOE allele status.</p> <p>Moderators: Social contact.</p> | <p>Outcome: All-cause dementia.</p> | <p>Analysis: Cox proportional hazards regression.</p> | <p>No notable interactions emerged through stratification by sex, age, or APOE status.</p> | <p>Increasing social contact was associated with lower risk of dementia. Neuroticism was associated with higher dementia risk.</p> <p>Compared to those with low neuroticism, those with high neuroticism and high social contact had little to no excess dementia risk. In contrast, the association of social contact with all-cause dementia was not significantly modified by neuroticism.</p> <p>Conclusions: Social contact is generally protective against dementia, particularly for those with high neuroticism. The association between social contact and dementia risk is not moderated by neuroticism.</p> <p>Considerations: Neuroticism and social contact were simultaneously entered into models, and HRs unadjusted for either variable were not reported.</p> |
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| <p>McHugh Power, J. E., Lawlor, B. A., & Kee, F. (2017). Social support mediates the relationships between extraversion, neuroticism, and cognitive function in older adults. Public Health, 147, 144–152.</p> <p>CATEGORIES: Social support as a mediator between anxiety and cognitive function</p> | <p>Convenience sample of 624 community-dwelling adults (60+).</p> <p>Half-longitudinal mediation design.</p> <p>Ireland.</p> | <p>Exposure: Neuroticism.</p> <p>Covariates: Age, gender, education.</p> <p>Mediators: Social support (functional and structural).</p> | <p>Outcome: Global cognitive function.</p> | <p>Analysis: Structural equation modelling.</p> | <p>Moderation by age or gender was not considered or reported on.</p> | <p>Controlling for covariates, neuroticism was not directly associated with cognitive function at follow-up. Social support partially mediated this relationship.</p> <p>The total effect of neuroticism on cognitive function (including the mediator) was positive. Neuroticism had a negative (trending, small) association with social support (path <i>a</i>: X→M) and social support had a positive relationship with cognitive function (path <i>b</i>: M→Y).</p> <p>Conclusions: Neuroticism may lead to lower levels of social support, although social support may still exert a net positive effect on cognitive function.</p> <p>Considerations: Only two timepoints were available for analyses, so an assumption of stationarity was made. Path <i>a</i> modelled the mediator at time 2 and X at time 1, controlling for the mediator at time 1. Path <i>b</i> modelled Y at time 2 and the mediator at time 1 controlling for Y at time 1.</p> |
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| <p>Nakanishi, N., Tatara, K., Shinsho, F., Takatorige, T., Murakami, S., & Fukuda, H. (1998). Prevalence of intellectual dysfunction and its correlates in a community-residing elderly population. Scandinavian Journal of Social Medicine, 26(3), 198–203.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>Random selection of 1405 community-dwelling older adults (aged 65+).</p> <p>Cross-sectional design.</p> <p>Japan.</p> | <p>Exposure: Sex, age over 75, poor general health, current medical treatment, no participation in social activities, lack of a sense that life is worth living, anxiety about the future.</p> <p>Covariates: Exposure variables were adjusted for in analyses.</p> | <p>Outcome: Intellectual dysfunction (minor, moderate, appreciable).</p> | <p>Analysis: Logistic regression.</p> | <p>Distribution of intellectual dysfunction was examined by age and sex. More severe forms of intellectual dysfunction were more prevalent in older age for both men and women. There were no significant differences between men and women in terms of intellectual dysfunction across the age groups.</p> | <p><i>Anxiety for the future</i> was independently associated with mild and moderate intellectual dysfunction after adjusting for all other risk factors. <i>No participation in social activities</i> was independently associated with any level of intellectual dysfunction after adjusting for all other risk factors.</p> <p>Conclusions: Both anxiety and social participation are independently associated with intellectual dysfunction.</p> <p>Considerations: Unadjusted odds ratios were not reported, nor were models with and without anxiety/social participation compared.</p> |
| <p>Nayak, S., Mohapatra, M. K., & Panda, B. (2019). Prevalence of and factors contributing to anxiety, depression and cognitive disorders among urban elderly in Odisha – A study through the health systems’ Lens. Archives of Gerontology and Geriatrics, 80, 38–45.</p> <p>CATEGORY: Anxiety and social support in the</p> | <p>244 randomly selected older adults from Berhampur city (aged 60+).</p> <p>Cross-sectional design.</p> <p>India.</p> | <p>Exposure: Age, gender, marital status, educational level, economical dependency, family type, physical ability, daily working hours, personal monthly income, perception of social support, substance abuse, morbidity status, mental health status (depression, anxiety, cognitive impairment).</p> <p>Covariates: Models adjusted for exposure variables that were significant in unadjusted analyses.</p> | <p>Outcome: Depression, anxiety, cognitive impairment.</p> | <p>Analysis: Linear regression.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>In adjusted models, increased age, physical inactivity, and geriatric depression score were independently associated with decreased MMSE scores.</p> <p>In unadjusted models, anxiety was independently associated with MMSE score, but this association was no longer significant when the model adjusted for other social and behavioural variables (including social support). The pattern is similar for social support and MMSE score.</p> |

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| <p>same model with cognitive function as the dependent variable</p> | | | | | | <p>Conclusions: Anxiety and social support are not associated with cognitive function independent of other social and behavioural determinants of health.</p> <p>Considerations: The individual contribution of anxiety or social support as a covariate cannot be assessed.</p> |
| <p>Olaru, G., Laukka, E. J., Dekhtyar, S., Sarwary, A., & Brehmer, Y. (2023). Association between personality traits, leisure activities, and cognitive levels and decline across 12 years in older adults. Psychology and Aging, 38(4), 277–290.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>Age-stratified sample of 1609 young-old (aged 60–72) and 1085 old-old (aged 78+) adults from the Swedish National Study on Aging and Care in Kungsholmen (SNAC-K) followed over 12 years.</p> <p>Longitudinal design.</p> <p>Sweden.</p> | <p>Exposure: Personality (neuroticism, extraversion, openness), activities (social, mental, physical).</p> <p>Covariates: Age, gender, years of education, number of chronic diseases.</p> <p>Moderators: Age group (young-old versus old-old).</p> | <p>Outcome: Cognitive abilities (episodic memory, perceptual speed, semantic memory, letter fluency, category fluency).</p> | <p>Analysis: Latent growth curve modelling, regression analysis.</p> | <p>The old-old age group was more neurotic than the young-old age group.</p> <p>Additionally, they had lower cognitive ability at baseline, were less active socially, were more likely to be female, and experienced higher attrition and faster cognitive decline.</p> <p>The associations between personality and cognitive decline, and stronger activity engagement and slower cognitive decline, were only</p> | <p>The correlation between personality traits and activities were similar for both age groups, with neuroticism being negatively associated with all activity engagement indexes after adjusting for covariates.</p> <p>In both age groups, participants with higher cognitive ability were less neurotic, more extroverted, and more open, as well as being more engaged in all types of activities.</p> <p>When personality and activity engagement were entered as predictors in a regression model, they explained an additional 9.3% of the variance in cognitive decline in the old-old age group beyond the control variables. Individually, personality explained 6.2% of the variance, and activity engagement 5.2%. In contrast, they did not increase the explained variance noticeably for the young-old cohort.</p> <p>Conclusions: Neuroticism is associated with reduced social, mental, and physical activity,</p> |

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| | | | | | significant in the old-old age group. | and both personality and activity engagement predict cognitive decline in the oldest adults. Considerations: The variance in cognitive abilities explained by neuroticism and social activity, independent of the other traits and activities, was not reported. |
| Patel, M., Bhardwaj, P., Nebhinani, N., Goel, A. D., & Patel, K. (2020). Prevalence of psychiatric disorders among older adults in Jodhpur and stakeholders perspective on responsive health system. Journal of Family Medicine and Primary Care, 9(2), 714–720. CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable | 330 community-dwelling older adults (aged 60+). Cross-sectional design. India. | Exposure: Anxiety, depression, cognitive impairment, sociodemographic variables (age, gender, religion, marital status, education level, occupation, urban/rural residence). Covariates: Anxiety, depression, cognitive impairment, sociodemographic variables (as stated above). | Outcome: Anxiety, depression, cognitive impairment. | Analysis: Logistic regression. | Older females have more severe anxiety than older males. | Anxiety was not significantly associated with cognitive impairment, controlling for all other variables in the model, which include age, gender, urban/rural residence, education level, marital status, and depression. In contrast, marital status was significantly associated with cognitive impairment after adjusting for covariates. Conclusions: Marital status, but not anxiety, is associated with cognitive impairment cross-sectionally. Considerations: Unadjusted ORs were not reported, nor was the individual contribution of marital status to the association between anxiety and cognitive impairment examined. |
| Peeters, G., Leahy, S., Kennelly, S., & Kenny, R. A. (2018). Is Fear of Falling Associated With Decline in Global Cognitive Functioning in Older Adults: Findings From the Irish | 4931 adults enrolled in the Irish Longitudinal Study on Ageing (aged 50–98). Half-longitudinal design. Ireland. | Exposure: Fear of falling (FoF). Covariates: Age, sex, educational attainment, chronic conditions, depressive symptoms, anxiety symptoms, medication use, self-rated vision and hearing, functional limitations, number of falls, | Outcome: Global cognitive functioning. | Analysis: Linear regression, logistic regression. | Those with FoF were more likely to be older, be female, be of lower education level, have health problems, report a fall, and have lower physical and social | In cross-sectional models, FoF was associated with lower cognitive function on the MoCA and MMSE. After adjusting for age, sex, education, ADLs, IADLs, depressive symptoms, anxiety, and falls in the past year, these associations were no longer statistically significant. |

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| <p>Longitudinal Study on Ageing. Journal of the American Medical Directors Association, 19(3), 248–254.e3.</p> <p>CATEGORY: Social support as a mediator between anxiety and cognitive function</p> | | <p>smoking history, alcohol intake, baseline cognitive functioning.</p> <p>Mediators: Physical activity, social activity.</p> <p>Moderators: Age.</p> | | | <p>activity than those without FoF.</p> <p>Age was not a significant modifier of the association between FoF and cognitive function.</p> | <p>In prospective models, FoF was associated with higher odds of cognitive decline on the MoCA and MMSE. After adjustment for covariates, these associations attenuated but remained statistically significant, but only for the MoCA.</p> <p>FoF was significantly associated with the mediators. The associations between the mediators and decline in cognitive functioning were not statistically significant, except physical activity was associated with lower odds of decline in MMSE. Adjustment for physical or social activity did not alter the results.</p> <p>Conclusions: The current research supports the association between FoF and cognitive function as being prospective in nature and explained by demographic factors, mood, and daily functioning. Physical and social activity are not likely to play a mediating role, nor is age likely to play a modifying role, in the association.</p> <p>Considerations: The mediators were measured at baseline.</p> |
| <p>Peeters, G., Romero-Ortuno, R., Lawlor, B., Kenny, R. A., & McHugh-Power, J. (2020). Clustering of Behavioral</p> | <p>4571 adults enrolled in the Irish Longitudinal Study on Ageing (aged 50+).</p> <p>Longitudinal design.</p> | <p>Exposure: Change in social withdrawal, physical activity withdrawal, and reduced falls-efficacy (fear of falling).</p> | <p>Outcome: Change in cognitive functioning (immediate recall,</p> | <p>Analysis: Random effects mixed models.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Concurrent withdrawal from social/physical activities or reduced falls-efficacy was associated with poorer cognitive functioning at baseline and greater declines in cognitive functioning over time.</p> |

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| <p>Changes and Their Associations with Cognitive Decline in Older Adults. Journal of the American Medical Directors Association, 21(11), 1689–1695.e1.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>Ireland.</p> | <p>Covariates: Age, sex, educational attainment, self-reported health, chronic conditions, depressive symptoms, medication use, self-rated vision and hearing, functional limitations, global cognitive function (MMSE).</p> | <p>delayed recall, verbal fluency).</p> | | | <p>In concurrent models, reduced falls-efficacy, but not social withdrawal or physical activity withdrawal, was statistically associated with greater cognitive decline. In prospective models, social withdrawal only and physical activity withdrawal only, but not reduced fall-efficacy only, were statistically associated with greater cognitive decline.</p> <p>Conclusions: Clustering of more withdrawal behaviours predicts greater cognitive decline, suggesting a cumulative effect of these behaviours on cognitive function. The findings also suggest that cognitive decline predicts falls, whereas social and physical activity predict cognitive functioning in aging populations.</p> |
| <p>Segel-Karpas, D., & Lachman, M. E. (2018). Social contact and cognitive functioning: The role of personality. Journals of Gerontology: Psychological Sciences, 73(6), 974–984.</p> <p>CATEGORY: Interaction of anxiety and social support</p> | <p>3524 community-dwelling adults enrolled in the Midlife in the U.S. (MIDUS) study (aged 32–84).</p> <p>Cross-sectional design.</p> <p>United States.</p> | <p>Exposure: Social contact.</p> <p>Covariates: Age, gender, education, race, marital status, employment status, functional limitations, perceived social support.</p> <p>Moderators: Five Big personality traits (extraversion, openness to experience, neuroticism, agreeableness, conscientiousness).</p> | <p>Outcome: Episode memory (immediate and delayed recall), executive functioning (working memory, verbal fluency, inductive reasoning, processing speed, attention-switching).</p> | <p>Analysis: Regression analysis.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>All five personality traits (except conscientiousness) moderated the association between social contact and episodic memory, and extraversion and openness moderated the relationship between social contact and executive functioning.</p> <p>Regardless of social contact, neuroticism was associated with lower levels of episodic memory, though the effect of social contact on cognitive function was stronger for those with low neuroticism.</p> |

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| | | | | | | <p>Social contact contributed to executive functioning regardless of levels of neuroticism.</p> <p>Conclusions: Neuroticism reduced individuals' ability to cognitively benefit from social contact.</p> |
| <p>Stephan, Y., Sutin, A. R., Luchetti, M., Aschwanden, D., & Terracciano, A. (2024). Physical, cognitive, and social activities as mediators between personality and cognition: evidence from four prospective samples. <i>Aging & Mental Health, 28(9), 1294–1303.</i></p> <p>CATEGORY: Social support as a mediator between anxiety and cognitive function</p> | <p>2318 adults aged 20–75 from the Midlife in the United States Survey (MIDUS), 6831 adults aged 50+ from the Health and Retirement Study (HRS), 4228 adults aged 50+ from the English Longitudinal Study of Aging (ELSA), 3499 adults aged 34–82 from the Wisconsin Longitudinal Study (WLS).</p> <p>Longitudinal design.</p> <p>United States, UK.</p> | <p>Exposure: Personality (Neuroticism, Extraversion, Openness, Agreeableness, Conscientiousness).</p> <p>Covariates: Age, sex, education. Race was controlled for in HRS, ELSA, and MIDUS. Ethnicity was controlled for in HRS. Baseline disease burden and depressive symptoms were included as additional covariates in supplemental analyses.</p> <p>Mediators: Cognitive activity, social activity, physical activity.</p> | <p>Outcome: Cognition.</p> | <p>Analysis: PROCESS macro using 5000 bootstrapped samples and 95% bias-corrected confidence intervals. Results were combined in a random-effect meta-analysis.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>The meta-analysis showed that higher neuroticism was associated with worse cognition and less engagement in physical, cognitive, and social activities.</p> <p>Lower engagement in physical and cognitive activities partially mediated the association between higher neuroticism and worse cognition. Physical and cognitive activities explained 7% and 14% of the association between neuroticism and cognition, respectively. Social activity did not mediate the association between neuroticism and cognition.</p> <p>When disease burden and depressive symptoms were included as additional covariates, physical activity was no longer a significant mediator of neuroticism and cognition in the ELSA cohort.</p> <p>Patterns of mediation remained consistent when all five traits were included simultaneously.</p> <p>Conclusions: The association between neuroticism and cognition is partially mediated</p> |

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| | | | | | | by physical and cognitive activity, but not social activity. |
| <p>Sutin, A. R., Stephan, Y., & Terracciano, A. (2019). Self-Reported Personality Traits and Informant-Rated Cognition: A 10-Year Prospective Study. J Alzheimers Dis, 72(1), 181–190.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>2536 participants from the Health and Retirement Study (aged 50+).</p> <p>Longitudinal design.</p> <p>United States.</p> | <p>Exposure: Personality (Neuroticism, Extraversion, Openness, Agreeableness, Conscientiousness).</p> <p>Covariates: Age at baseline, year of personality assessment, gender, race, education in years, baseline cognitive function or cognitive status. Informant covariates: age, gender, education in years, length of time informant knew participant, relationship to participant.</p> <p>Moderators: Participant age, gender, baseline cognitive function, concurrent cognitive status, and informant type (spousal versus other).</p> | <p>Outcome: Informant-rated cognition.</p> | <p>Analysis: Linear regression, logistic regression.</p> | <p>Neither participant age nor gender moderated the association between neuroticism and informant-rated cognition.</p> | <p>Adjusting for covariates, baseline neuroticism was associated with greater informant-rated decline in cognition over ten years and worse current cognitive function. This association remained significant when all personality traits were included in the model, when baseline cognitive function was included as a covariate, when the sample was limited to participants with intact cognition, and when the sample was limited to complete data on all measures.</p> <p>Conclusions: Neuroticism is associated with informant-reported cognitive decline.</p> <p>Considerations: While models adjusted for social support-related covariates, unadjusted versus adjusted estimates were not reported.</p> |
| <p>Tomaszewski Farias, S., De Leon, F. S., Gavett, B. E., Fletcher, E., Meyer, O. L., Whitmer, R. A., DeCarli, C., & Mungas, D. (2024). Associations between personality and psychological characteristics and cognitive outcomes among older adults. Psychology</p> | <p>157 older adults from the University of California Davis Alzheimer's Disease Research Center's Longitudinal Diversity Cohort (aged 60+ at baseline).</p> <p>Longitudinal design.</p> <p>United States.</p> | <p>Exposure: Personality (Extraversion, Neuroticism, Openness to New Experiences, Conscientiousness), sense of purpose in life, self-efficacy, sadness, anger, positive affect, loneliness.</p> <p>Covariates: Age at baseline, gender, race/ethnicity, recruitment source (clinic vs. community),</p> | <p>Outcome: Cognition (executive function, semantic memory, episodic memory, spatial ability).</p> | <p>Analysis: Mixed effects and parallel process longitudinal analysis using multilevel modelling, Bayesian modelling.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Only openness independently predicted cognitive trajectories; neuroticism and loneliness did not.</p> <p>Neuroticism was independently associated with spatial ability, even after measures of brain volume was included as covariates. Loneliness was associated with episodic memory, but not after brain volume was included as a covariate.</p> |

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| <p>and Aging, 39(2), 188–198.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | | <p>baseline MRI measures (total gray matter, hippocampus, and white matter hyperintensity volumes).</p> | | | | <p>Conclusions: Neuroticism and loneliness are cross-sectionally, but not longitudinally, associated with cognition.</p> <p>Considerations: Neuroticism and loneliness were simultaneously entered into models; unadjusted estimates that excluded one or the other from models were not provided.</p> |
| <p>Verma, M., Grover, S., Singh, T., Dahiya, N., & Nehra, R. (2020). Screening for cognitive impairment among the elderly attending the noncommunicable diseases clinics in a rural area of Punjab, North India. Asian Journal of Psychiatry, 50, 102001.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>297 older adults living with either diabetes mellitus and/or hypertension (aged 60+).</p> <p>Cross-sectional design.</p> <p>India.</p> | <p>Exposure: Age group (60–69, 70+), gender, marital status, education, employment status, per capita income, family type, urban/rural residence, living alone, living away, duration of disease, depression, anxiety, vulnerability to abuse, loneliness, non-communicable disease.</p> | <p>Outcome: Cognitive impairment.</p> | <p>Analysis: ANCOVA, multivariable logistic regression analysis.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Anxiety was not associated with cognitive impairment in unadjusted or adjusted models. Loneliness was significantly associated with cognitive impairment in adjusted models.</p> <p>Although not statistically significant, the odds ratio for anxiety was reduced after adjusting for other predictors, including loneliness.</p> <p>Conclusions: Loneliness, but not anxiety, is significantly associated with cognitive impairment when controlling for the other predictors in the model.</p> |
| <p>Wang, Q., Zan, C., Jiang, F., Shimpuku, Y., & Chen, S. (2022). Association between loneliness and its components and cognitive function among older Chinese adults living in</p> | <p>228 older adults living in nursing homes free of dementia/psychiatric/somatic diseases (aged 65+).</p> <p>Cross-sectional design.</p> | <p>Exposure: Loneliness (personal feelings of isolation, lack of relational connectedness, and lack of collective connectedness).</p> <p>Covariates: Age, sex, education level, current smoking and</p> | <p>Outcome: Global cognitive function.</p> | <p>Analysis: Linear regression, mediation analysis with PROCESS.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Loneliness was significantly associated with worse global cognitive function. After additional adjustment for depressive symptoms, anxiety symptoms, and sleep disturbances, the association between loneliness and global cognitive function</p> |

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| <p>nursing homes: A mediation of depressive symptoms, anxiety symptoms, and sleep disturbances. BMC Geriatr, 22(1), 959.</p> <p>CATEGORY: Anxiety as a mediator between social support and cognitive function</p> | <p>China.</p> | <p>drinking, physical activity, social involvement, living arrangement, body weight and height, body mass index, medical history of physician diagnosed diseases, comorbidities, hearing impairment.</p> <p>Mediators: Depressive symptoms, anxiety symptoms, sleep disturbances.</p> | | | | <p>attenuated, but only personal feelings of isolation remained significant.</p> <p>Anxiety symptoms mediated the association between loneliness and cognitive function via sleeping disturbances.</p> <p>Conclusions: Loneliness may decrease cognitive function by increasing anxiety, which in turn lowers sleep quality.</p> |
| <p>Wang, Y., Li, J., Fu, P., Jing, Z., Zhao, D., & Zhou, C. (2022). Social support and subsequent cognitive frailty during a 1-year follow-up of older people: The mediating role of psychological distress. BMC Geriatr, 22(1), 162.</p> <p>CATEGORY: Anxiety as a mediator between social support and cognitive function</p> | <p>2785 older adults enrolled in the Shandong Rural Elderly Health Cohort (60+).</p> <p>Longitudinal design.</p> <p>China.</p> | <p>Exposure: Social support.</p> <p>Covariates: Baseline sex, age, education, marital status, economic status, smoking status, alcohol drinking status, chronic conditions, functional disability, psychological distress, and cognitive frailty.</p> <p>Mediators: Psychological distress.</p> | <p>Outcome: Cognitive frailty (physical frailty and cognitive impairment without dementia).</p> | <p>Analysis: Logistic regression.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>After controlling for baseline covariates (excluding baseline psychological distress), social support was negatively associated with cognitive frailty.</p> <p>When further adjusting for baseline psychological distress, social support was also negatively associated with psychological distress in path <i>a</i>, and the direct effect of social support on cognitive frailty was reduced.</p> <p>Psychological distress partially mediated the association between baseline social support and consequent cognitive frailty.</p> <p>Conclusions: Social support is associated with decreased risk of psychological distress and cognitive frailty over 1-year follow-up.</p> |

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| | | | | | | Considerations: Psychological distress was treated as a unified concept and examination of anxiety versus depressive symptoms was not conducted. |
| <p>Watfa, G., Husson, N., Buatois, S., Laurain, M. C., Miget, P., & Benetos, A. (2011). Study of Mini-Mental State Exam evolution in community-dwelling subjects aged over 60 years without dementia. Journal of Nutrition, Health & Aging, 15(10), 901–904.</p> <p>CATEGORY: Anxiety and social support in the same model with cognitive function as the dependent variable</p> | <p>687 community-dwelling older adults without dementia (aged 60+).</p> <p>Longitudinal design.</p> <p>France.</p> | <p>Exposure: Age, gender, education level, blood pressure, heart rate, BMI, physical activity, smoking, alcohol consumption, glucose level, cholesterol level, triglycerides, psychological parameters (solitude, insomnia, difficulty in social relations, nervousness, inability to decide, painful event, life without interest, critics and malice in your life, despair, feelings of being surrounded by strange “incomprehensible” things, sense of being elderly, memory problems, psychological following-up, stable psychological state).</p> <p>Covariates: Significant correlates of cognitive function beyond age and gender.</p> | <p>Outcome: Cognitive function (MMSE).</p> | <p>Analysis: Multiple regression analyses.</p> | <p>Moderation by age or sex/gender was not considered or reported on.</p> | <p>Nervousness was associated with baseline MMSE score, independent of education level, despair, and regular physical activity, which altogether accounted for 13% of the total variance in MMSE score.</p> <p>Nervousness was correlated with annual changes in MMSE, but when entered in multivariable analyses, only MMSE score at baseline, education level, and difficulty in social relations remained significant, accounting for 26% of the total variance in annual change in MMSE score.</p> <p>Conclusions: Anxiety is associated with baseline MMSE but not change in MMSE, and social support is associated with change in MMSE but not baseline MMSE.</p> |

Appendix C

Derivation of Analytic Sample

Figure C1 is a flowchart depicting the derivation of the analytic sample. Table C1 provides the number of missing observations for the executive function tests (AFT, COWAT, MAT, Stroop-VV, TMT) at each timepoint based on the participants who returned for assessments at T2. The analytic sample was derived from a subset of T2 participants who completed their DCS visit in person and were thus able to provide data on all tests of executive function (pre-COVID-19 sample). Missing data on executive function measures in the pre-COVID-19 sample are provided alongside missingness in the total T2 sample, which includes participants who were not able to complete all assessments of executive function due to COVID-19 pandemic restrictions impacting data collection procedures. Additionally, Table C2 provides the number of missing observations for covariates (measured at T0) in the pre-COVID-19 sample and the total T2 sample.

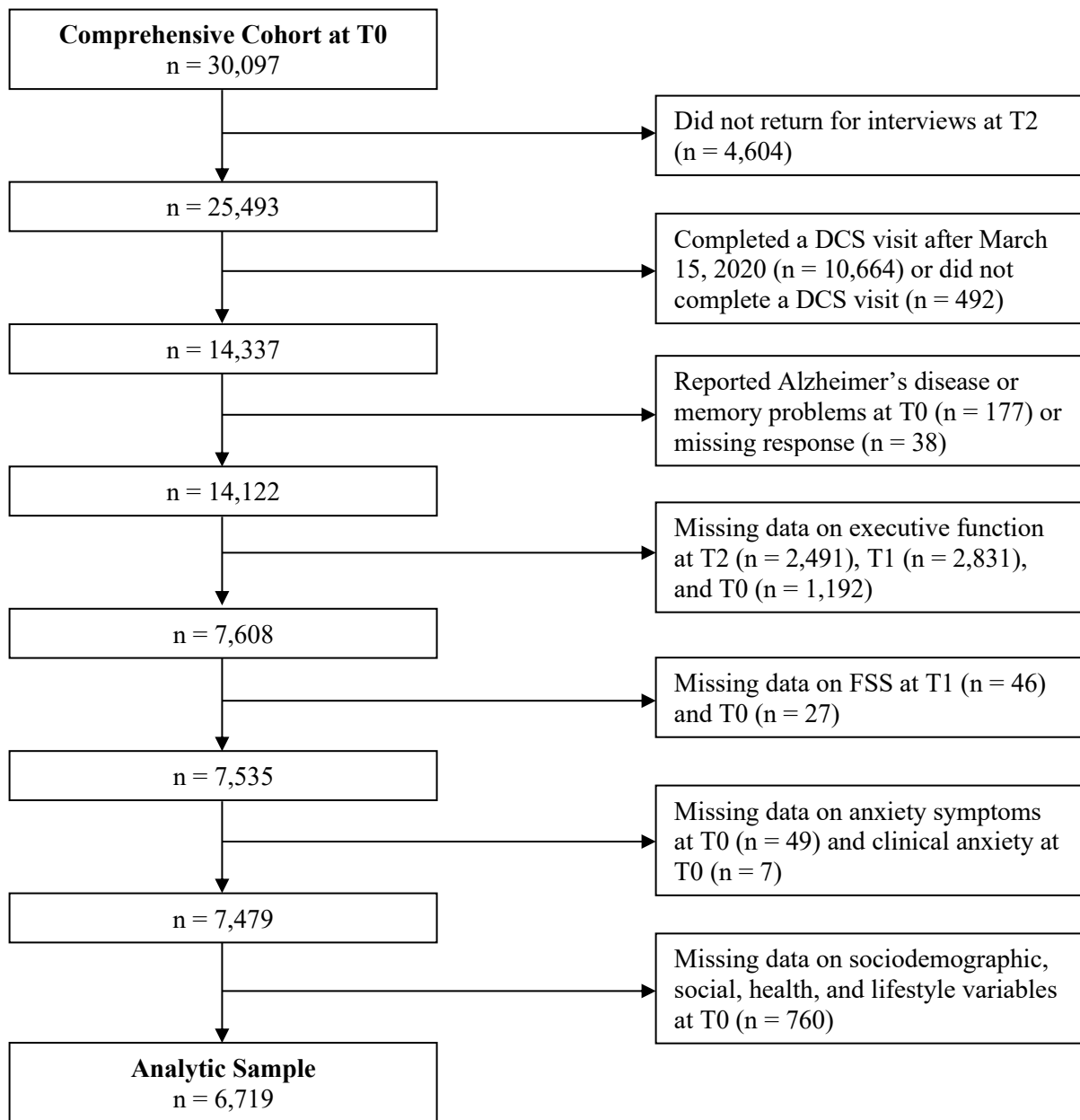


Figure C1. Derivation of the Analytic Sample

Note. DCS = data collection site; FSS = functional social support; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2.

Table C1. Incomplete Data on Executive Function Tests at Each Timepoint, by Pre-COVID-19 Sample (n=14,337) and Total T2 Sample (n=25,493)

| Executive Function Test | Missing at T0 | | Missing at T1 | | Missing at T2 | |
|---|--------------------------|-----------------|-----------------|-----------------|-----------------|------------------|
| | Pre-COVID-19 | Total T2 | Pre-COVID-19 | Total T2 | Pre-COVID-19 | Total T2 |
| | Total Missing (n) (%) | | | | | |
| Animal Fluency Test (AFT) | 609 (4.25) | 1010 (3.96) | 2669 (18.62) | 3616 (14.18) | 948 (6.61) | 2040 (8.00) |
| Controlled Oral Word Association Test (COWAT) | 1059 (7.39) | 1919 (7.53) | 691 (4.82) | 2106 (8.26) | 767 (5.35) | 11923 (46.77) |
| Mental Alternation Test (MAT) | 991 (6.91) | 1622 (6.36) | 3147 (21.95) | 4486 (17.60) | 1364 (9.51) | 2905 (11.40) |
| Stroop Neuropsychological Screening Test-Victoria Version (Stroop-VV) | 115 (0.80) | 295 (1.16) | 304 (2.12) | 1402 (5.50) | 438 (3.06) | 11594 (45.48) |
| Time-Based Prospective Memory Test (TMT) | 131 (0.91) | 343 (1.35) | 338 (2.36) | 1882 (7.38) | 566 (3.95) | 11722 (45.98) |
| Executive Function Composite Measure | 2340 (16.32) | 4003 (15.70) | 3915 (27.31) | 6911 (27.11) | 2533 (17.67) | 13689 (53.70) |

Note. Participants are missing scores if they did not complete a cognitive test or completed their test bilingually in English and French.

Table C2. Incomplete Data on Baseline (T0) Covariates in the Follow-up 2 (T2) pre-COVID-19 (n=14,337) and Total Samples (n=25,493)

| Covariates (T0) | Pre-COVID-19 Missing (n) | Total T2 Missing (n) |
|---------------------------|---------------------------------|-----------------------------|
| <i>Sociodemographic</i> | | |
| Age group | 0 | 0 |
| Sex | 0 | 0 |
| Province | 0 | 0 |
| Education | 20 | 35 |
| Total household income | 863 | 1509 |
| Income meets needs | 162 | 521 |
| <i>Social</i> | | |
| Marital status | 6 | 8 |
| Living arrangements | 6 | 14 |
| Pet ownership | 11 | 30 |
| <i>Health</i> | | |
| Self-rated general health | 7 | 14 |
| Functional impairment | 43 | 66 |
| Chronic conditions | 467 | 905 |
| Depressive symptoms (K10) | 160 | 496 |
| Clinical depression | 54 | 111 |
| <i>Lifestyle</i> | | |
| Alcohol use | 328 | 569 |
| Smoking use | 0 | 1 |

Appendix D

Measurement Instruments

Anxiety and Depressive Symptoms

Anxiety and depressive symptoms were derived from the overall Kessler Psychological Distress Scale (K10) (Kessler et al., 2003), which measures nonspecific symptoms of distress experienced in the past 30 days. Each item is rated on a 5-point scale. The items are given below:

1. How often did you feel tired out for no good reason?
2. How often did you feel nervous?
3. How often did you feel so nervous that nothing could calm you down?
4. How often did you feel hopeless?
5. How often did you feel restless or fidgety?
6. How often did you feel so restless that you could not sit still?
7. How often did you feel depressed?
8. How often did you feel that everything was an effort?
9. How often did you feel so depressed that nothing could cheer you up?
10. How often did you feel worthless?

Note: Item 3 was skipped if item 2 was “none of the time”. Similarly, item 6 was skipped if item 5 was “none of the time”.

Possible answers:

- None of the time
- A little of the time
- Some of the time
- Most of the time
- All of the time

Based on the confirmatory factor analyses of Brooks et al. (2009) and Lace et al. (2019), items 2, 3, 5, and 6 were summed to create an anxiety symptoms subscale. The remaining items 1, 4, 7–10 were summed to create a depressive symptoms subscale.

Functional Social Support

The 19 items from the Medical Outcomes Survey-Social Support Survey (MOS-SSS) (Sherbourne & Stewart, 1991) are listed below. The items are responses to the question: "How often is each of the following kinds of support available to you if you need it?"

Items:

1. Someone to help if you were confined to bed.
2. Someone you can count on to listen to you when you need to talk.
3. Someone to give you advice about a crisis.
4. Someone to take you to the doctor if needed.
5. Someone who shows you love and affection.
6. Someone to have a good time with.
7. Someone to give you information in order to help you.
8. Someone to confide in or talk to about yourself or your problems.
9. Someone who hugs you.
10. Someone to get together with for relaxation.
11. Someone to prepare your meals if you were unable to do it yourself.
12. Someone whose advice you really want.
13. Someone to do things with to help you get your mind off things.
14. Someone to help with daily chores if you were sick.
15. Someone to share your most private worries and fears with.
16. Someone to turn to for suggestions about how to deal with a personal problem.
17. Someone to do something enjoyable with.
18. Someone who understands your problems.
19. Someone to love you and make you feel wanted.

Possible answers:

- None of the time (1)
- A little of the time (2)
- Some of the time (3)
- Most of the time (4)
- All of the time (5)

Additionally, there are four subscales in the MOS-SSS:

- Emotional/informational support (items 2, 3, 7, 8, 12, 15, 16, and 18)
- Affectionate support (items 5, 9, and 19)
- Tangible support (items 1, 4, 11, and 14)
- Positive social interactions (items 6, 10, and 17)

Item 13 is not included in any of the subscales.

Overall functional social support (FSS) was calculated as the mean of all 19 items in the MOS-SSS. Up to one item was allowed to be missing, and the missing item was replaced using mean imputation. If the missing item belonged to a subscale, it was imputed using the mean of the subscale. If the missing item did not belong to a subscale (i.e., item 13), it was imputed using the mean of the other 18 items. See Lupoi (2024) for documentation on how overall FSS was derived.

Executive Function

The list below provides an overview of the tests used to assess executive function in the CLSA (Raina et al., 2008). Detailed information on the administration of cognitive tests is available elsewhere (CLSA, 2019). Scores on each test were converted to z-scores based on the language of administration (English and French) and then summed to create a composite score. Note that z-scores were derived using the subset of T2 participants who completed their cognitive assessments before March 16, 2020 (pre-COVID-19).

| Executive Function Test | Description |
|---|---|
| Animal Fluency Test (AFT) | A test of verbal fluency that asked participants to name as many animals as possible in 60 seconds. |
| Controlled Oral Word Association Test (COWAT) | A test of phonological fluency that asks participants to name words starting with a specific letter. Usually, three letters are used, for a total of three one-minute trials. |
| Mental Alternation Test (MAT) | A test of cognitive flexibility that asks first asks participants to count aloud from 1 to 20 and say the alphabet as quickly as possible, then to alternate between number and letter (i.e. 1-A, 2-B, 3-C ...) as quickly as possible for 30 seconds. |
| Stroop Neuropsychological Screening Test-Victoria Version (Stroop-VV) | A test of inhibition, attention, processing speed, and cognitive control that asks participants to name the colour of three types of stimuli: coloured dots, common words printed in the same colour as the dots, and colour words printed in non-corresponding colours of ink. |
| Time-Based Prospective Memory Test (TMT) | A test of prospective memory that asks participants to interrupt themselves at a designated time and show a card with a predetermined number to the interviewer. |

Covariates

| Covariates | Classification | Details |
|-------------------------|---|--|
| <i>Sociodemographic</i> | | |
| Age group | <ul style="list-style-type: none"> • 45–54 (ref) • 55–64 • 65–74 • 75+ | Age (years) |
| Sex | <ul style="list-style-type: none"> • Male (ref) • Female | “Are you male or female?” |
| Province | <ul style="list-style-type: none"> • Ontario (ref) • Alberta • British Columbia • Manitoba • Newfoundland and Labrador • Nova Scotia • Quebec | Province at recruitment |
| Education | <ul style="list-style-type: none"> • Less than secondary school graduation (ref) • Secondary school graduation, no-post secondary education • Some post-secondary education • Post-secondary degree/diploma | Highest educational level obtained |
| Income | <ul style="list-style-type: none"> • < \$20,000 (ref) • ≥ \$20,000 to < \$50,000 • ≥ \$50,000 to < \$100,000 • ≥ \$100,000 to < \$150,000 • ≥ \$150,000 | Total household income |
| Income adequacy | <ul style="list-style-type: none"> • Totally inadequately (ref) • Not very well • With some difficulty • Adequately • Very well | How well income currently satisfies basic needs “Totally inadequately”, “not very well”, and “with some difficulty” were collapsed in multivariable analyses. |

| Covariates | Classification | Details |
|------------------------------|---|--|
| <i>Social</i> | | |
| Marital status | <ul style="list-style-type: none"> • Married/living with a partner in a common-law relationship (ref) • Single, never married or never lived with a partner • Widowed • Divorced • Separated | “Divorced” and “separated” were combined in multivariable analyses. |
| Living arrangements | <ul style="list-style-type: none"> • Lives with others (ref) • Lives alone | Derived from number of people living in the household |
| Pet ownership | <ul style="list-style-type: none"> • No (ref) • Yes | Own a household pet for companionship |
| <i>Health</i> | | |
| Self-rated general health | <ul style="list-style-type: none"> • Poor (ref) • Fair • Good • Very good • Excellent | CLSA derived variable “Poor” and “fair” were combined in multivariable analyses. |
| Number of chronic conditions | <ul style="list-style-type: none"> • 0 (ref) • 1 • 2 • 3+ | Self-reported diagnosis of peripheral vascular disease/poor circulation in limbs; back problems excluding fibromyalgia and arthritis; high-blood pressure/hypertension; kidney disease/kidney failure; under-active thyroid/hypothyroidism/myxedema; chronic obstructive pulmonary disease/emphysema/chronic bronchitis; diabetes/borderline high blood sugar; angina; heart attack/myocardial infarction; stroke; Parkinsonism/Parkinson’s disease; experienced a ministroke/transient ischemic attack; epilepsy; or multiple sclerosis |
| Functional impairment | <ul style="list-style-type: none"> • No (ref) • Yes | Derived from a modified Older Americans Resources and Services |

| Covariates | Classification | Details |
|---------------------|---|---|
| | | Multidimensional Assessment Questionnaire |
| Clinical depression | <ul style="list-style-type: none"> • Absence (ref) • Presence | Self-reported diagnosis of depression |
| Depressive symptoms | Sum of six items from the K10 | See documentation in Appendix D on derivation of the anxiety and depressive symptom subscales |
| <i>Lifestyle</i> | | |
| Smoking use | <ul style="list-style-type: none"> • No (I don't smoke, and I never have) (ref) • Former (I don't smoke now but I have in the past) • Yes (I currently smoke) | Smoking status self-report measure |
| Alcohol use | <ul style="list-style-type: none"> • Did not drink in the last 12 months (ref) • Occasional drinker (less than once a month) • Regular drinker (at least once a month) | CLSA derived self-report measure |

Appendix E

Covariate Effects for Paths I and II

The tables below provide estimates for the independent effects of covariates on Path I (anxiety to FSS) and Path II (FSS to executive function) that were described in Section 5.2.4. Estimates for interaction terms were omitted as they are included in Section 5.2.3.

Table E1. Effect of Covariates on Functional Social Support and Executive Function in the Model with Clinical Anxiety, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Independent Variables | Path I: Anxiety (T0) → FSS (T1) R ² = 0.3815 b (95% CI) | Path II: FSS (T1) → Executive Function (T2) R ² = 0.5843 b (95% CI) |
|---|---|---|
| Main variables | | |
| <i>Exposure</i> | | |
| Clinical anxiety (T0) | -0.0433 (-0.0843, -0.0024)* | -0.0402 (-0.2042, 0.1239) |
| <i>Mediator</i> | | |
| FSS ¹ (T1) | — | FSS*age group ³ |
| <i>Antecedent outcome variables</i> | | |
| FSS ¹ (T0) | 0.6780 (0.6601, 0.6959)*** | 0.0935 (-0.0035, 0.1904) |
| Executive function ² (T0) | -0.0008 (-0.0051, 0.0035) | 0.3195 (0.2961, 0.3428)*** |
| Executive function ² (T1) | — | 0.5112 (0.4879, 0.5346)*** |
| Sociodemographic covariates | | |
| <i>Age group (ref: 45–54 years)</i> | | |
| 55–64 | -0.0192 (-0.0458, 0.0073) | FSS*age group ³ |
| 65–74 | -0.0309 (-0.0637, 0.0019) | FSS*age group ³ |
| 75+ | -0.0875 (-0.1310, -0.0439)*** | FSS*age group ³ |
| <i>Sex (ref: male)</i> | | |
| Female | 0.0072 (-0.0144, 0.0289) | 0.0551 (-0.0318, 0.1419) |
| <i>Province (ref: Ontario)</i> | | |
| Alberta | 0.0063 (-0.0353, 0.0478) | -0.1723 (-0.3386, -0.0059)* |
| British Columbia | 0.0339 (0.0016, 0.0661)* | -0.0906 (-0.2197, 0.0384) |
| Manitoba | -0.0121 (-0.0502, 0.0260) | 0.2731 (0.1205, 0.4258)*** |
| Newfoundland | 0.0519 (0.0118, 0.0921)* | 0.0504 (-0.1105, 0.2113) |
| Nova Scotia | 0.0244 (-0.0138, 0.0626) | -0.0954 (-0.2485, 0.0576) |
| Quebec | 0.0159 (-0.0189, 0.0508) | -0.1127 (-0.2523, 0.0269) |
| <i>Education (ref: less than high school)</i> | | |
| High school graduate | -0.0040 (-0.0716, 0.0637) | 0.2057 (-0.0652, 0.4766) |
| Some post-secondary education | -0.0103 (-0.0813, 0.0607) | 0.2859 (0.0015, 0.5703)* |
| Post-secondary degree/diploma | -0.0105 (-0.0707, 0.0498) | 0.2797 (0.0382, 0.5211)* |
| <i>Total household income (ref: < \$20,000)</i> | | |
| ≥ \$20,000 to < \$50,000 | 0.0138 (-0.0504, 0.0781) | 0.0692 (-0.1878, 0.3262) |
| ≥ \$50,000 to < \$100,000 | 0.0362 (-0.0294, 0.1019) | 0.1077 (-0.1551, 0.3705) |
| ≥ \$100,000 to < \$150,000 | 0.0462 (-0.0236, 0.1159) | 0.1869 (-0.0925, 0.4664) |
| ≥ \$150,000 | 0.0735 (0.0010, 0.1460)* | 0.1902 (-0.1003, 0.4808) |
| <i>Income meets needs (ref: with some or more difficulty)</i> | | |
| Adequately | 0.0254 (-0.0188, 0.0696) | -0.0869 (-0.2640, 0.0901) |
| Very well | 0.0889 (0.0440, 0.1337)*** | -0.1185 (-0.2985, 0.0615) |
| Social covariates | | |
| <i>Marital status (ref: married/common-law)</i> | | |
| Single/never married | -0.1072 (-0.1608, -0.0536)*** | 0.0490 (-0.1661, 0.2642) |
| Widowed | -0.0519 (-0.1073, 0.0034) | 0.0061 (-0.2155, 0.2277) |
| Divorced/separated | -0.0674 (-0.1125, -0.0224)** | 0.1650 (-0.0154, 0.3454) |
| <i>Living arrangements</i> | | |

| | | |
|--|------------------------------|-----------------------------|
| <i>(ref: lives with others)</i> | | |
| Lives alone | 0.0030 (-0.0414, 0.0475) | 0.0265 (-0.1517, 0.2046) |
| <i>Pet ownership (ref: no)</i> | | |
| Yes | 0.0102 (-0.0115, 0.0318) | 0.0175 (-0.0694, 0.1043) |
| Health covariates | | |
| <i>Self-rated general health</i> | | |
| <i>(ref: poor/fair)</i> | | |
| Good | 0.0056 (-0.0415, 0.0528) | -0.0776 (-0.2666, 0.1113) |
| Very good | 0.0558 (0.0088, 0.1028)* | -0.0481 (-0.2363, 0.1401) |
| Excellent | 0.0699 (0.0195, 0.1204)** | -0.0031 (-0.2051, 0.1989) |
| <i>Number of chronic conditions (ref: 0)</i> | | |
| 1 | 0.0071 (-0.0179, 0.0322) | -0.0073 (-0.1076, 0.0929) |
| 2 | 0.0184 (-0.0122, 0.0491) | -0.0563 (-0.1791, 0.0665) |
| 3+ | -0.0013 (-0.0398, 0.0373) | -0.1803 (-0.3346, -0.0260)* |
| <i>Functional impairment (ref: no)</i> | | |
| Yes | -0.0203 (-0.0655, 0.0250) | -0.1438 (-0.3251, 0.0375) |
| <i>Clinical depression (ref: absence)</i> | | |
| Presence | 0.0057 (-0.0251, 0.0365) | -0.0627 (-0.1860, 0.0605) |
| Lifestyle covariates | | |
| <i>Smoking status (ref: never smoked)</i> | | |
| Former smoker | -0.0107 (-0.0325, 0.0111) | -0.0735 (-0.1607, 0.0137) |
| Current smoker | -0.0664 (-0.1089, -0.0238)** | -0.1410 (-0.3114, 0.0295) |
| <i>Alcohol use (ref: no)</i> | | |
| Occasional | 0.0314 (-0.0148, 0.0776) | -0.1558 (-0.3408, 0.0291) |
| Regular | 0.0499 (0.0137, 0.0862)** | 0.0052 (-0.1400, 0.1504) |

Note. *b* = unstandardised regression coefficient; CI = confidence interval; FSS = functional social support; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; ref = reference category.

¹Functional social support ranged from 1 to 5, with higher scores indicating higher levels of functional social support.

²Executive function is a standardised and summed score of five neurocognitive tests of executive function.

³Effects of interactions are included in Table 7 and omitted in this table.

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

Table E2. Effect of Covariates on Functional Social Support and Executive in the Model with Anxiety Symptoms, Canadian Longitudinal Study on Aging Comprehensive Cohort (n = 6,719)

| Independent Variables | Path I: Anxiety (T0) → FSS (T1) R ² = 0.3815 b (95% CI) | Path II: FSS (T1) → Executive Function (T2) R ² = 0.5843 b (95% CI) |
|---|---|---|
| Main variables | | |
| <i>Exposure</i> | | |
| Anxiety symptoms ¹ (T0) | anxiety symptoms*age group*sex ⁴ | 0.0090 (-0.0167, 0.0348) |
| <i>Mediator</i> | | |
| FSS ² (T1) | — | FSS*age group ⁴ |
| <i>Antecedent outcome variables</i> | | |
| FSS ² (T0) | 0.6635 (0.6453, 0.6818)*** | 0.0928 (-0.0045, 0.1901) |
| Executive function ³ (T0) | -0.0007 (-0.0049, 0.0036) | 0.3193 (0.2960, 0.3427)*** |
| Executive function ³ (T1) | — | 0.5108 (0.4874, 0.5341)*** |
| Sociodemographic covariates | | |
| <i>Age group (ref: 45–54 years)</i> | | |
| 55–64 | anxiety symptoms*age group*sex ⁴ | FSS*age group ⁴ |
| 65–74 | anxiety symptoms*age group*sex ⁴ | FSS*age group ⁴ |
| 75+ | anxiety symptoms*age group*sex ⁴ | FSS*age group ⁴ |
| <i>Sex (ref: male)</i> | | |
| Female | anxiety symptoms*age group*sex ⁴ | 0.0539 (-0.0329, 0.1408) |
| <i>Province (ref: Ontario)</i> | | |
| Alberta | 0.0081 (-0.0333, 0.0495) | -0.1696 (-0.3359, -0.0034)* |
| British Columbia | 0.0311 (-0.0010, 0.0632) | -0.0891 (-0.2181, 0.0399) |
| Manitoba | -0.0148 (-0.0527, 0.0232) | 0.2742 (0.1216, 0.4269)*** |
| Newfoundland | 0.0450 (0.0050, 0.0850)* | 0.0535 (-0.1074, 0.2144) |
| Nova Scotia | 0.0227 (-0.0154, 0.0607) | -0.0929 (-0.2458, 0.0601) |
| Quebec | 0.0247 (-0.0102, 0.0595) | -0.1116 (-0.2518, 0.0285) |
| <i>Education (ref: less than high school)</i> | | |
| High school graduate | -0.0054 (-0.0729, 0.0621) | 0.2058 (-0.0651, 0.4768) |
| Some post-secondary education | -0.0127 (-0.0836, 0.0582) | 0.2829 (-0.0015, 0.5673) |
| Post-secondary degree/diploma | -0.0100 (-0.0702, 0.0502) | 0.2778 (0.0363, 0.5193)* |
| <i>Total household income (ref: < \$20,000)</i> | | |
| ≥ \$20,000 to < \$50,000 | 0.0087 (-0.0552, 0.0727) | 0.0662 (-0.1905, 0.3230) |
| ≥ \$50,000 to < \$100,000 | 0.0323 (-0.0330, 0.0977) | 0.1079 (-0.1547, 0.3704) |
| ≥ \$100,000 to < \$150,000 | 0.0404 (-0.0291, 0.1099) | 0.1877 (-0.0915, 0.4669) |
| ≥ \$150,000 | 0.0675 (-0.0046, 0.1396) | 0.1916 (-0.0988, 0.4819) |
| <i>Income meets needs (ref: with some or more difficulty)</i> | | |
| Adequately | 0.0073 (-0.0369, 0.0515) | -0.0879 (-0.2657, 0.0899) |
| Very well | 0.0643 (0.0192, 0.1093)** | -0.1225 (-0.3039, 0.0589) |
| Social covariates | | |
| <i>Marital status (ref: married/common-law)</i> | | |
| Single/never married | -0.1050 (-0.1584, -0.0517)*** | 0.0474 (-0.1678, 0.2626) |
| Widowed | -0.0530 (-0.1086, 0.0026) | 0.0056 (-0.2160, 0.2272) |
| Divorced/separated | -0.0697 (-0.1145, -0.0248)** | 0.1607 (-0.0197, 0.3410) |
| <i>Living arrangements (ref: lives with others)</i> | | |

| | | |
|---|-------------------------------|-----------------------------|
| Lives alone | -0.0039 (-0.0482, 0.0404) | 0.0261 (-0.1521, 0.2043) |
| <i>Pet ownership (ref: no)</i> | | |
| Yes | 0.0119 (-0.0097, 0.0335) | 0.0151 (-0.0716, 0.1019) |
| Health covariates | | |
| <i>Self-rated general health (ref: poor/fair)</i> | | |
| Good | -0.0167 (-0.0640, 0.0307) | -0.0866 (-0.2770, 0.1037) |
| Very good | 0.0250 (-0.0224, 0.0724) | -0.0572 (-0.2478, 0.1333) |
| Excellent | 0.0315 (-0.0196, 0.0826) | -0.0150 (-0.2202, 0.1903) |
| <i>Number of chronic conditions (ref: 0)</i> | | |
| 1 | 0.0077 (-0.0172, 0.0326) | -0.0099 (-0.1101, 0.0903) |
| 2 | 0.0219 (-0.0086, 0.0524) | -0.0594 (-0.1820, 0.0632) |
| 3+ | 0.0074 (-0.0309, 0.0457) | -0.1851 (-0.3391, -0.0311)* |
| <i>Functional impairment (ref: no)</i> | | |
| Yes | -0.0090 (-0.0542, 0.0362) | -0.1428 (-0.3242, 0.0386) |
| <i>Depressive symptoms⁵</i> | -0.0175 (-0.0222, -0.0127)*** | -0.0119 (-0.0310, 0.0072) |
| Lifestyle covariates | | |
| <i>Smoking status (ref: never smoked)</i> | | |
| Former smoker | -0.0117 (-0.0336, 0.0101) | -0.0756 (-0.1628, 0.0117) |
| Current smoker | -0.0638 (-0.1063, -0.0212)** | -0.1425 (-0.3131, 0.0280) |
| <i>Alcohol use (ref: no)</i> | | |
| Occasional | 0.0288 (-0.0172, 0.0748) | -0.1528 (-0.3378, 0.0321) |
| Regular | 0.0468 (0.0107, 0.0829)* | 0.0080 (-0.1371, 0.1531) |

Note. *b* = unstandardised regression coefficient; CI = confidence interval; FSS = functional social support; T0 = baseline; T1 = follow-up 1; T2 = follow-up 2; ref = reference category.

¹Anxiety symptom scores were a sum of four items from the Kessler Psychological Distress Scale (K10). Scores range from 0 to 16, with higher values indicating higher levels of anxiety.

²Functional social support ranged from 1 to 5, with higher scores indicating higher levels of functional social support.

³Executive function is a standardised and summed score of five neurocognitive tests of executive function.

⁴Effects of interactions are included in Table 8 and omitted in this table.

⁵Depressive symptoms were summed across six items from the Kessler Psychological Distress Scale (K10). Scores range from 0 to 24, with higher values indicating higher levels of depression.

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

Appendix F

Comparisons of Pooled Imputed Data and Complete-Case Data

The tables below provide additional pooled results of the sensitivity analysis involving multiple imputation of missing covariates, provided alongside the results of the main (complete-case) analysis. Multiple imputation of covariates recovered 760 participants that were previously excluded due to missing data. These participants tended to be less healthy, older, and female.

Table F1. Univariate Descriptive Analyses of Variables Included in the Complete-Case Dataset (n=6,719) Versus Imputed Dataset (Pooled) (n=7,479)

| | Complete-Case Dataset n=6,719 | Imputed Dataset (Pooled) ¹ n=7,479 |
|---|----------------------------------|--|
| Continuous Variables | | |
| | | <i>MD (IQR)</i> |
| Anxiety symptoms (T0) | 1.00 (2.00) | 1.00 (2.00) |
| Depressive symptoms (T0) ² | 1.00 (3.00) | 1.00 (3.00) |
| Functional social support (T0) | 4.47 (0.89) | 4.47 (0.89) |
| Functional social support (T1) | 4.44 (0.94) | 4.44 (0.94) |
| | | <i>\bar{x} (SD)</i> |
| Executive function (T0) | 0.64 (2.66) | 0.59 (2.68) |
| Executive function (T1) | 0.54 (2.70) | 0.46 (2.82) |
| Executive function (T2) | 0.34 (2.87) | 0.28 (2.89) |
| Categorical Variables (T0) | | <i>Frequency (%)</i> |
| <i>Clinical anxiety</i> | | |
| Absence | 92.44 | 92.37 |
| Presence | 7.56 | 7.63 |
| Sociodemographic | | |
| <i>Age group (years)</i> | | |
| 45–54 | 28.93 | 28.36 |
| 55–64 | 37.21 | 36.76 |
| 65–74 | 23.01 | 23.37 |
| 75+ | 10.85 | 11.51 |
| <i>Sex</i> | | |
| Female | 51.48 | 52.49 |
| Male | 48.52 | 47.51 |
| <i>Province</i> | | |
| Ontario | 20.36 | 20.52 |
| Alberta | 8.84 | 8.89 |
| British Columbia | 21.12 | 20.94 |
| Manitoba | 11.42 | 11.37 |
| Newfoundland and Labrador | 9.97 | 9.83 |
| Nova Scotia | 11.52 | 11.74 |
| Quebec | 16.77 | 16.71 |
| <i>Education²</i> | | |
| Less than high school | 3.33 | 3.58 |
| High school graduate | 8.36 | 8.54 |
| Some post-secondary | 6.47 | 6.46 |
| Post-secondary degree/diploma | 81.83 | 81.41 |
| <i>Total household income²</i> | | |
| < \$20,000 | 3.26 | 3.46 |
| ≥ \$20,000 to < \$50,000 | 18.47 | 19.17 |
| ≥ \$50,000 to < \$100,000 | 36.94 | 37.04 |
| ≥ \$100,000 to < \$150,000 | 22.29 | 22.03 |
| ≥ \$150,000 | 19.04 | 18.29 |
| <i>Income meets needs²</i> | | |

| | | |
|---|-------|--------------|
| With some or more difficulty | 7.17 | 7.53 |
| Adequately | 34.81 | 35.10 |
| Very well | 58.01 | 57.37 |
| Social | | |
| <i>Marital status</i> ² | | |
| Married/common-law | 75.04 | 74.19 |
| Single/never married | 7.08 | 7.28 |
| Widowed | 6.53 | 6.98 |
| Divorced/separated | 11.34 | 11.55 |
| <i>Living arrangements</i> | | |
| Lives alone | 17.84 | 18.38 |
| Lives with others | 82.16 | 81.62 |
| <i>Pet ownership</i> | | |
| No | 55.08 | 55.92 |
| Yes | 44.92 | 44.08 |
| Health | | |
| <i>Self-rated general health</i> ² | | |
| Poor/fair | 6.04 | 6.40 |
| Good | 26.57 | 26.81 |
| Very good | 43.89 | 43.62 |
| Excellent | 23.50 | 23.17 |
| <i>Number of chronic conditions</i> ² | | |
| 0 | 33.92 | 33.69 |
| 1 | 35.66 | 35.54 |
| 2 | 19.24 | 19.15 |
| 3+ | 11.18 | 11.63 |
| <i>Functional impairment</i> ² | | |
| No | 93.99 | 93.59 |
| Yes | 6.01 | 6.41 |
| <i>Clinical depression (self-reported)</i> ² | | |
| Absence | 84.70 | 84.53 |
| Presence | 15.30 | 15.47 |
| Lifestyle | | |
| <i>Smoking status</i> | | |
| Never smoked | 49.31 | 49.90 |
| Former smoker | 43.80 | 43.29 |
| Current smoker | 6.89 | 6.81 |
| <i>Alcohol use</i> ² | | |
| No | 9.36 | 9.75 |
| Occasional | 10.86 | 11.21 |
| Regular | 79.77 | 79.05 |

Note. Medians and interquartile ranges were provided for skewed variables; means and standard deviations were given for executive function, which approximated a normal distribution; frequencies were provided for categorical variables.

IQR = interquartile range; MD = median; SD = standard deviation; \bar{x} = mean.

¹Imputed datasets involved multiple imputation of missing sociodemographic, social, health, or lifestyle covariates only.

²Results for imputed variables are bolded.

Table F2. Fully Adjusted Path Effects for the Complete-Case Dataset (n=6,719) Versus Imputed Dataset (Pooled) (n=7,479)

| | Complete-Case Dataset n=6,719 | Imputed Dataset (Pooled) ¹ n=7,479 |
|----------------------------|----------------------------------|--|
| Clinical Anxiety | <i>b (95% CI)</i> | |
| Path I² | | |
| Overall | -0.0433 (-0.0843, -0.0024)* | -0.0388 (-0.0778, 0.0002) |
| Path II³ | | |
| 45–54 | -0.1027 (-0.2470, 0.0416) | -0.0501 (-0.1883, 0.0881) |
| 55–64 | 0.1000 (-0.0274, 0.2274) | 0.0850 (-0.0366, 0.2065) |
| 65–74 | 0.0603 (-0.0934, 0.2140) | 0.0799 (-0.0667, 0.2264) |
| 75+ | -0.2383 (-0.4393, -0.0372)* | -0.1093 (-0.2946, 0.0760) |
| Anxiety Symptoms | <i>b (95% CI)</i> | |
| Path I² | | |
| Male 45–54 | 0.0176 (0.0034, 0.0318)* | 0.0159 (0.0024, 0.0294)* |
| Female 45–54 | -0.0020 (-0.0141, 0.0101) | -0.0017 (-0.0135, 0.0100) |
| Male 55–64 | -0.0034 (-0.0171, 0.0104) | -0.0024 (-0.0157, 0.0110) |
| Female 55–64 | -0.0071 (-0.0194, 0.0052) | -0.0072 (-0.0189, 0.0046) |
| Male 65–74 | -0.0111 (-0.0295, 0.0073) | -0.0091 (-0.0268, 0.0085) |
| Female 65–74 | 0.0154 (-0.0025, 0.0333) | 0.0124 (-0.0039, 0.0287) |
| Male 75+ | 0.0180 (-0.0091, 0.0450) | 0.0074 (-0.0179, 0.0327) |
| Female 75+ | 0.0171 (-0.0084, 0.0427) | 0.0182 (-0.0054, 0.0417) |
| Path II³ | | |
| 45–54 | -0.1079 (-0.2527, 0.0370) | -0.0587 (-0.1975, 0.0801) |
| 55–64 | 0.0955 (-0.0323, 0.2234) | 0.0776 (-0.0443, 0.1996) |
| 65–74 | 0.0564 (-0.0974, 0.2102) | 0.0732 (-0.0735, 0.2198) |
| 75+ | -0.2417 (-0.4428, -0.0407)* | -0.1133 (-0.2986, 0.0719) |

Note. *b* = unstandardised regression coefficient; CI = confidence interval; ΔR^2 = R-square change.

¹Imputed datasets involved multiple imputation of missing sociodemographic, social, health, or lifestyle covariates only.

²Path I represents the association between baseline anxiety and follow-up 1 functional social support.

³Path II represents the association between follow-up 1 functional social support and follow-up 2 executive function, controlling for baseline anxiety.

**p* < .05

Appendix G

Model Diagnostics

The figures below provide standard model diagnostic plots for linear regression. Assumptions of linear regression were not violated at either path of the mediated effect. Refer to Section 5.2.7 for a summary of these figures.

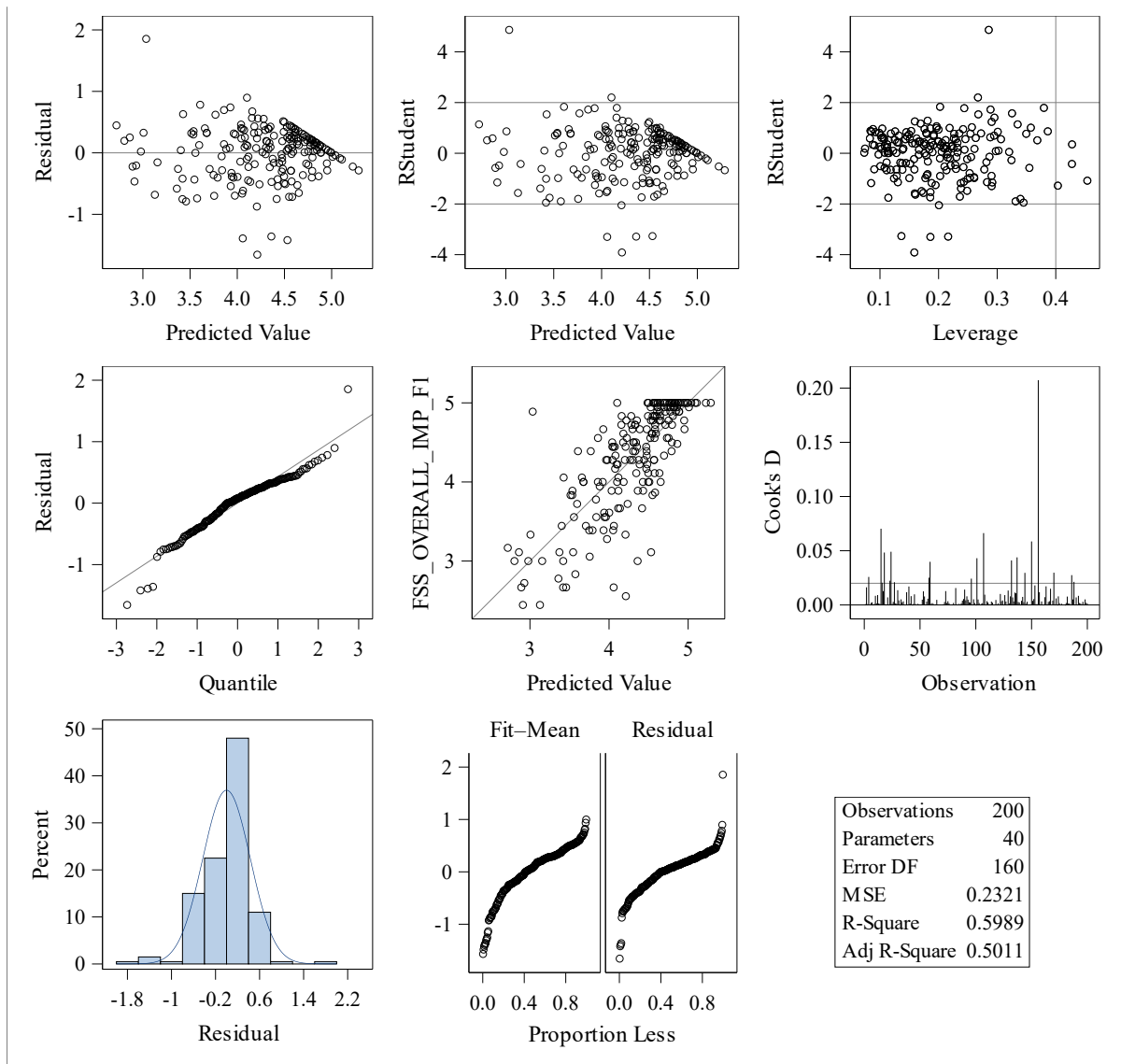


Figure G1. Fit Diagnostics for a Fully Adjusted Path I Model (Clinical Anxiety → Functional Social Support) on a Random Sample of 200 Participants

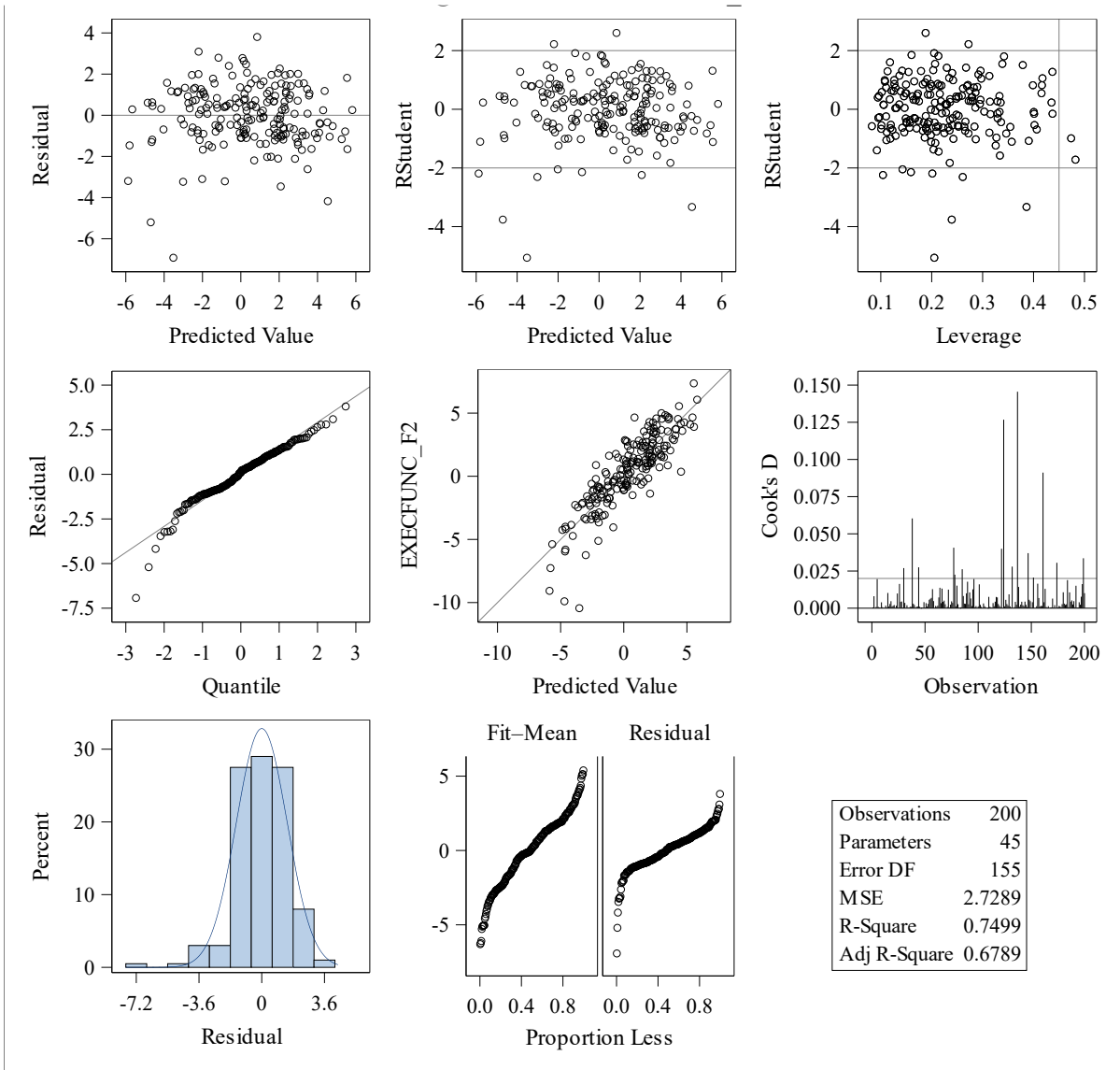


Figure G2. Fit Diagnostics for a Fully Adjusted Path II Model (Functional Social Support → Executive Function, Controlling for Clinical Anxiety) on a Random Sample of 200 Participants

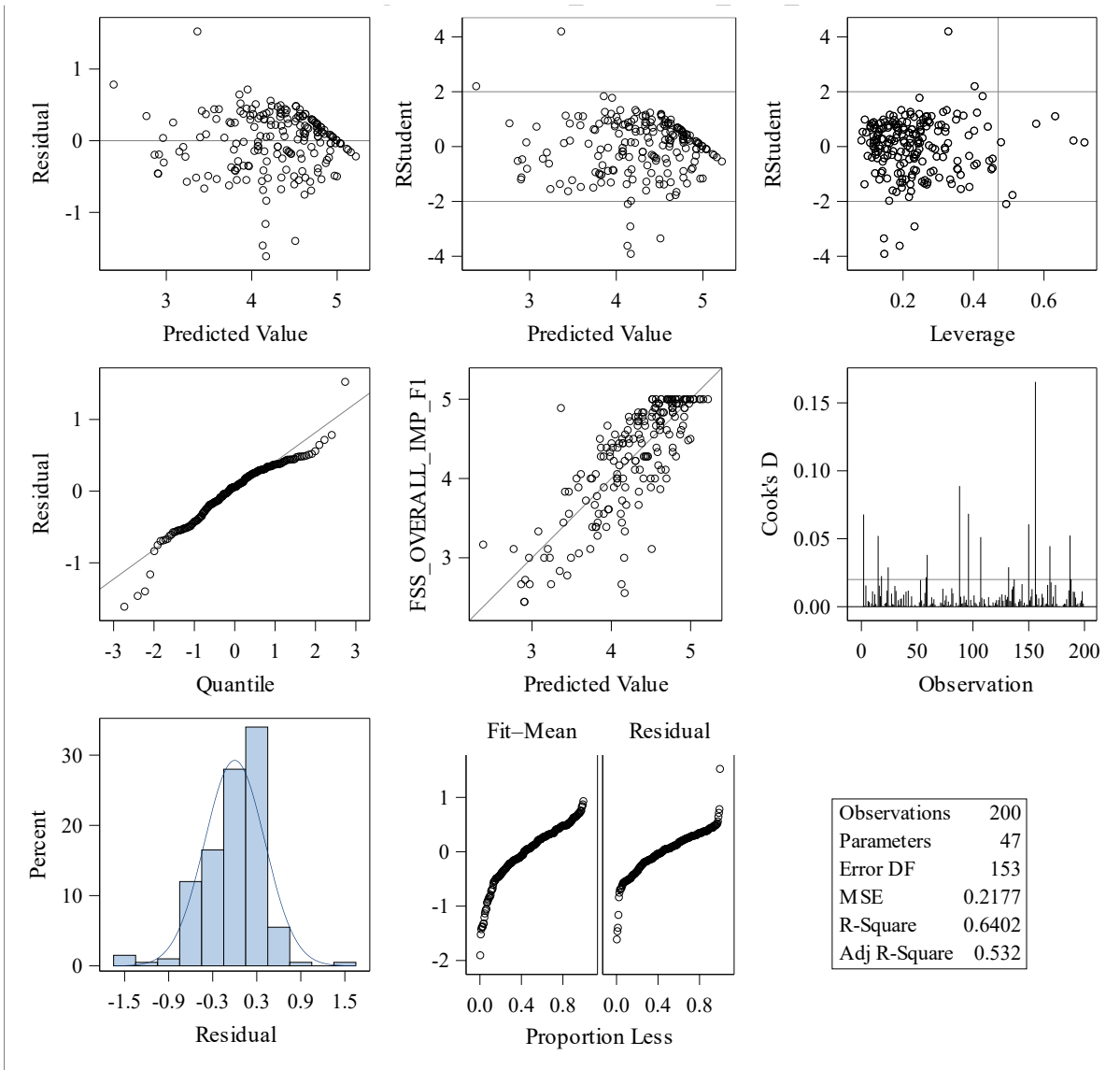


Figure G3. Fit Diagnostics for a Fully Adjusted Path I Model (Anxiety Symptoms → Functional Social Support) on a Random Sample of 200 Participants

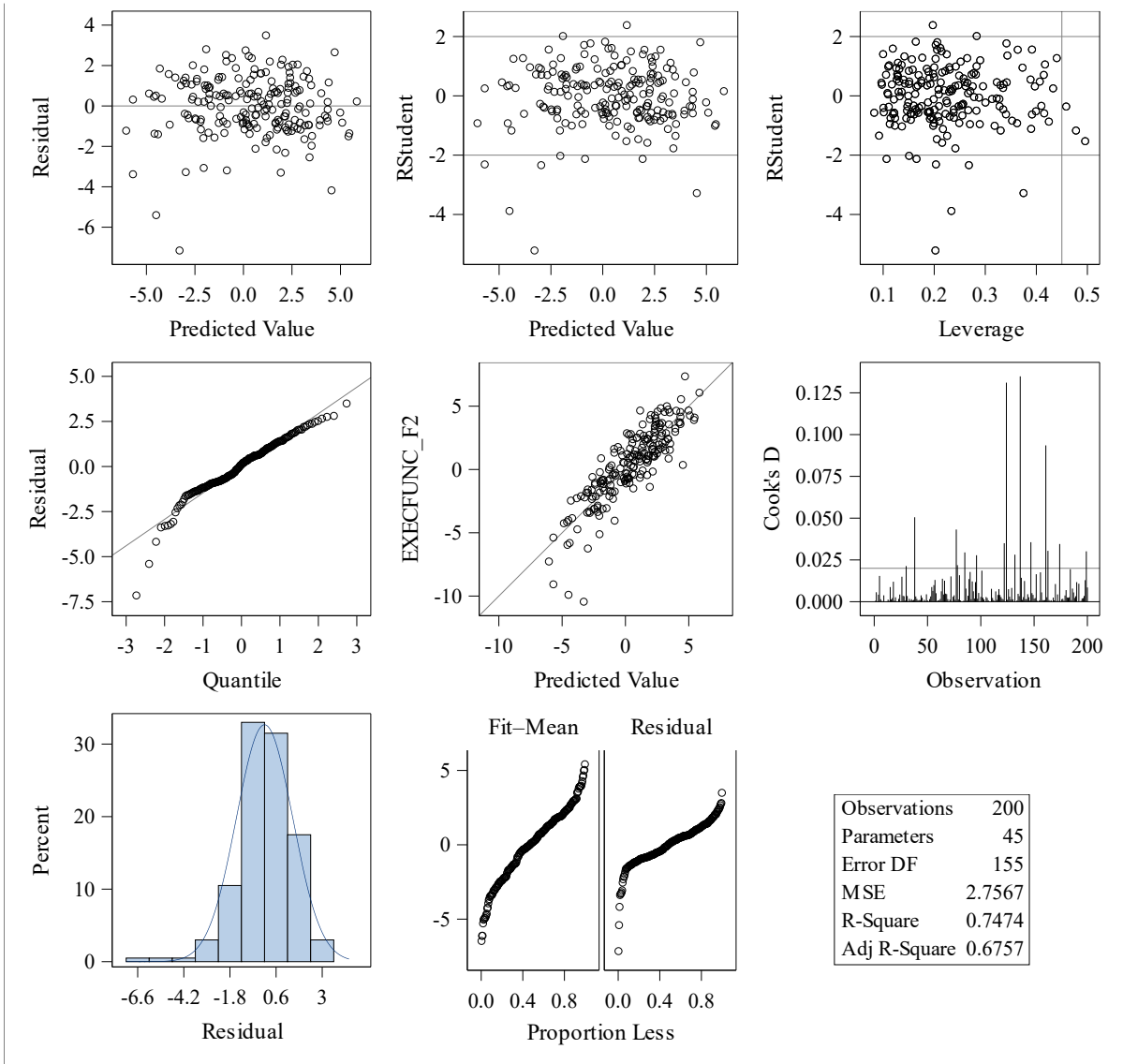


Figure G4. Fit Diagnostics for a Fully Adjusted Path II Model (Functional Social Support → Executive Function, Controlling for Anxiety Symptoms) on a Random Sample of 200 Participants