EXAMINING SOCIAL NETWORKS, SOCIAL SUPPORT, AND RELATIONSHIP QUALITY AS PROTECTIVE FACTORS FOR NEUROCOGNITIVE HEALTH IN INDIVIDUALS WHO ARE HOMELESS OR PRECARIOUSLY HOUSED

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Abstract

Social connectedness has been posited to contribute to cognitive reserve (CR) in the context of aging, but its role in predicting neurocognitive functioning in adults who are unstably housed is unknown. This study examined cross-sectional associations between dimensions of social connectedness and domains of neurocognitive functioning in a sample of adults experiencing homelessness or precarious housing. HIV infection, history of traumatic brain injury (TBI), and psychotic disorder were analyzed as moderating clinical risk factors that may weaken any associations observed. Hierarchical multiple regression analyses revealed that available social network size significantly predicted verbal memory performance. HIV moderated the association between average relationship conflict and verbal memory and the association between available social network size and cognitive flexibility. Neither social support nor relationship conflict predicted neurocognitive functioning. Neither TBI nor psychotic disorder moderated any associations. The present findings elucidate the role of social connectedness in contributing to CR in this marginalized population and provide initial support for the potential for social-oriented interventions to preserve their neurocognitive health.

Keywords: Social connectedness, neurocognitive functioning, cognitive reserve, homelessness, precarious housing

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1. Introduction

Homelessness and precarious housing are associated with a range of multimorbid adverse health outcomes, including substance use disorder, brain injury, and mental and physical illnesses (Aldridge et al., 2018; Gutwinski et al., 2021; Krausz et al., 2013; Schütz et al., 2019). Paired with socioeconomic risk factors such as lack of stable income and access to healthcare, individuals who are homeless or precariously housed experience higher rates of neurocognitive dysfunction (Depp et al., 2015; Hurstak et al., 2017; Stergiopoulos et al., 2015; Stone et al., 2019). In turn, these neurocognitive difficulties result in poor functional outcomes such as vocational challenges, lack of interpersonal skills, and impairments in performing activities of daily living (R. T. Brown et al., 2012, 2013; Stone et al., 2019). This is indicated by the higher prevalence of neurocognitive impairments, among other geriatric conditions, in older adults experiencing homelessness relative to domiciled adults who are 20 years older (Booth et al., 2024; R. T. Brown et al., 2016).

Among individuals who are homeless or precariously housed, there may be protective lifestyle and environmental factors that contribute to their resilience to pathological changes in neurocognition, as posited by the cognitive reserve (CR) hypothesis (Satz, 1993; Stern, 2002; Stern et al., 2020). CR reflects the brain's flexibility and efficiency in coping with the pathological mechanisms of brain illness and injury by deploying available brain resources to offset neurocognitive decline (Alvares Pereira et al., 2022; Whalley et al., 2004). As such, greater CR allows for the maintenance of neurocognitive abilities in daily living to minimize the impact of brain injury on functional outcomes (Pettigrew & Soldan, 2019; Stern, 2021). Common markers of CR include levels of education and occupational attainment (Katzman, 1993; Thow et al., 2018; Wilson et al., 2019). As persons who are homeless and precariously housed face poorer outcomes for these dimensions of socioeconomic status, in addition to higher

incidence of functional deficits and increased risk of mortality (Aldridge et al., 2018; Gutwinski et al., 2021; S. W. Hwang, Wilkins, et al., 2009), it is critical to identify modifiable protective factors that reinforce CR and improve health outcomes in this population. These may include dimensions of social connectedness (Cohen, 2004; Fratiglioni et al., 2004; Kelly et al., 2017), which is defined as the subjective bond and sense of belonging that one feels with other individuals or groups (Haslam et al., 2015).

1.1 Social Network Size and Neurocognitive Functioning

Social connectedness encompasses a myriad of dimensions including social network size. This consists of the number of individuals with whom one has had recent, regular contact. Evidence from the neurocognitive aging literature suggests that large social network sizes may protect against the adverse physiological and psychological health outcomes associated with social isolation (Nicholson, 2012), which in turn offsets neurocognitive decline (Béland et al., 2005; Bennett et al., 2006; Crooks et al., 2008; Evans et al., 2019; Fratiglioni et al., 2004, 2020; Penninkilampi et al., 2018; Röhr et al., 2020; Zhang et al., 2021; Zunzunegui et al., 2003).

Cross-sectional studies with healthy older adults have found an association between larger social network sizes and greater performance in neurocognitive domains such as processing speed (Nie et al., 2021; Sharifian et al., 2021), executive functioning (Casey et al., 2021; Sharifian et al., 2021), immediate verbal recall (Nie et al., 2021), visual memory (Casey et al., 2021), verbal fluency (Nie et al., 2021), visuoconstructional skills (Meister & Zahodne, 2022), and global neurocognition (Ali et al., 2018; Evans et al., 2019; Kelly et al., 2017; Sharifian et al., 2019). Empirical support for the protective nature of larger social network sizes on global neurocognition at single timepoints has additionally been observed in clinical populations, including older adults with Alzheimer’s Disease pathology (Bennett et al., 2006)

and stroke survivors 6 months into recovery (Glymour et al., 2008). These neurocognitive benefits may be present as larger network sizes facilitate social interaction (Dykstra et al., 2005) and confer more opportunities to engage in cognitively stimulating activities than smaller network sizes would, such as encountering novel situations, planning tasks, and maintaining language proficiency (Fratiglioni et al., 2020; Glymour et al., 2008; Meister & Zahodne, 2022; Penninkilampi et al., 2018; Sharifian et al., 2019). This aligns with previous research positing that social engagement offsets neurocognitive decline associated with aging (Hertzog et al., 2008; Hultsch et al., 1999; J. Li et al., 2019; McDonough et al., 2017; Williams & Kemper, 2010; Wilson, 2002), thereby positioning social network size as a potential contributor to CR.

Cross-sectional associations between social network size and neurocognitive functioning have been well established, but supportive evidence on the longitudinal nature of the relationship has been mixed. While certain studies have found that larger social network sizes remain positively associated with global neurocognition in middle-aged and older adults after initial assessment (Bennett et al., 2006; Crooks et al., 2008; Evans et al., 2019; Sörman et al., 2017), others have not found any association to be maintained at follow-up sessions conducted 2 to 4 years later (Kuiper et al., 2020; Nie et al., 2021). It is possible that these discrepancies are due to differences in selected measures of neurocognitive abilities and definitions of social networks across these studies.

Most literature has examined social network size as a predictor for neurocognition. However, the association may be bidirectional. An individual's number of social ties may impact their neurocognitive abilities, but these may in turn influence their capacity to maintain numerous social relationships (Collins et al., 2022; Cornwell, 2009). Indeed, a longitudinal study found that older adults experiencing neurocognitive decline see a reduction in social network

size (Aartsen et al., 2004). As such, further exploration of the nature of the effect of social network size on neurocognitive functioning is needed, specifically as it has never been examined in the context of unstable housing.

Current knowledge of homeless and precariously housed individuals' social network sizes is scant relative to the general population. Previous research has found that individuals in supportive housing and women who are homeless have no significant differences in social network size compared to the general population (Addo et al., 2022; Rattelade & Farrell, 2020; Toohey et al., 2004). In samples of youth experiencing homelessness, small social network sizes were reported by adolescent participants (S. M. Brown et al., 2020; Johnson et al., 2005) as well as those who had experienced caretaker abuse and who reported depressive symptoms (Johnson et al., 2005). In a qualitative study, Bower et al. (2018) found that smaller social network sizes in a sample of adults experiencing homelessness are the result of social marginalization, rejection from family, lack of companionship, and precarious relationships with other individuals who are homeless. No research to date has examined social network sizes of adults experiencing homelessness or precarious housing in relation to their neurocognitive functioning. Given that this population is vulnerable to fragmented social networks (Hawkins & Abrams, 2007) and neurocognitive impairment (Depp et al., 2015; Hurstak et al., 2017; Stergiopoulos et al., 2015; Stone et al., 2019), examining this association would bridge a gap in knowledge of the nature of social connectedness as a positive contributor to this high-risk population's CR.

1.2 Relationship Quality and Neurocognitive Functioning

It has been theorized that the quality of ties in a social network is more important than the size of the network itself (Amieva et al., 2010; Kuiper et al., 2020; Meister & Zahodne, 2022; Zunzunegui et al., 2003), especially in populations who are homeless and consequently

experience higher rates of social marginalization (Jafry et al., 2021; Schütz et al., 2019). As such, relationship quality is another significant dimension of social connectedness and may play a unique role in predicting neurocognitive functioning.

Cross-sectional research in the context of aging has found that a higher frequency of positive interactions across all relationships (a marker of relationship quality) is associated with greater global neurocognition (Pillemer & Holtzer, 2016). Longitudinal research conducted with older adults who are stably housed has suggested that negative social interactions predict a higher incidence of mild cognitive impairment and more rapid decline in episodic, semantic, and working memory (Wilson et al., 2015).

Findings for the relevance of relationship quality can be further delineated by specific types of relationships, as conflict with spouses, family members, and friends each have a differential impact on neurocognitive functioning in older adults. At the longitudinal level, Scholes & Liao (2022) found that older adults reporting spousal and friendship strain exhibit faster decline in verbal memory at a 16-year follow-up from study baseline. At the cross-sectional level, Meister & Zahodne (2022) observed an association between older adults' higher spousal and friendship strain with poorer processing speed, executive functioning, visuoconstructional skills, and language. When assessing older adults at a single timepoint, La Fleur & Salthouse (2016) found negative correlations between friendship strain and visuoconstructional skills, processing speed, language, reasoning, and a global index of memory. M. Li et al. (2021) additionally observed negative correlations between family strain and global neurocognition, episodic memory, working memory at the cross-sectional level. Cross-sectional research with middle-aged adults indicate similar patterns, where lower social strain across relationships with one's spouse, family, and friends was associated with greater performance in

executive functioning (Tun et al., 2013). The negative relationship consistently observed between relational conflict and neurocognition may be explained through stress mechanisms. As social conflict and strain are sources of chronic interpersonal stress, they may have a detrimental effect on neural processes important for neurocognitive functioning (Lupien et al., 2009; Vyas et al., 2016). Indeed, exposure to chronic stress can contribute to the conversion from neurocognitive decline to pathological neurodegenerative disorders such as Alzheimer's and Parkinson's Disease (Esch et al., 2002; Justice, 2018; Peña-Bautista et al., 2020).

The association between relationship quality and neurocognitive functioning has yet to be investigated in the context of homelessness and precarious housing. As individuals who are homeless are prone to experiencing conflict in lower-quality relationships (Bower et al., 2018), in addition to chronic and acute interpersonal stressors related to housing insecurity such as stigma and discrimination (Banyard & Graham-Bermann, 1998; Durbin et al., 2019; Reilly et al., 2022; Skosireva et al., 2014; Stergiopoulos et al., 2015), investigating whether relationship quality is linked to this vulnerable population's neurocognitive abilities would provide greater insight into its potential role in contributing to CR and improving functional outcomes.

1.3 Social Support and Neurocognitive Functioning

Social connectedness additionally includes social support, which consists of the amount of care and assistance one receives from their social network. It encompasses a variety of facets, such as emotional support, instrumental support, advice or information, and guidance with a directive quality (Stokes & Wilson, 1984). Unless otherwise specified, the current paper will refer to the construct of social support as a combination of these four facets as not all studies in the literature examine each one individually, and many refer to social support as a general measure of multiple facets.

Previous research in the field of neurocognitive aging has indicated that social support is positively associated with global neurocognition (Costa-Cordella et al., 2021; Kelly et al., 2017; Seeman et al., 2001) and reduces the risk of dementia and Alzheimer's Disease (Amieva et al., 2010). The nature of its relationship with individual neurocognitive domains varies at the cross-sectional level. When assessing older adults at one timepoint, emotional and informational support positively predicted global neurocognition (Pillemer & Holtzer, 2016), language, processing speed, reasoning, visuoconstructional skills, and a composite measure of memory (La Fleur & Salthouse, 2016). Higher social support in the form of supportive listening is cross-sectionally associated with greater global neurocognition in participants aged 45 years or older (Salinas et al., 2021), in addition to greater executive functioning in a sample of older adults at risk for dementia (Zuelsdorff et al., 2019). Meanwhile, older adults aged 70 years old and above who reported high levels of social support exhibited better global neurocognition with no link to the general domain of memory. It must be noted that this association was no longer present when symptoms of depression were included as a covariate (Gow et al., 2013). Overall, higher levels of social support are associated with greater neurocognitive functioning in older adults at the cross-sectional level.

While cross-sectional findings indicate that social support is positively associated with neurocognitive functioning, longitudinal research has been inconclusive. A number of observational studies have found that higher social support is associated with a slower decline in verbal memory over the course of 16 years (Scholes & Liao, 2022), and reduces the risk of neurocognitive impairment (Amieva et al., 2010; Ma et al., 2024; Seeman et al., 2001). Others did not find a significant impact of social support on neurocognitive decline over time. For instance, Moreno et al. (2022) observed an association between higher social support and greater

global neurocognition in a sample of older women at baseline, but this effect did not hold at the six year follow-up. Such findings suggest that social support as a standalone factor may be insufficient in offsetting neurocognitive impairment (Eisele et al., 2012; Joyce et al., 2022). Discrepant results on the impact of social support in longitudinal studies may be attributable to differences in the operationalization of the construct of social support, the administered neurocognitive measures, and the timeframe of data collection. Indeed, the studies that did not find a positive influence of social support had available data for shorter observation periods (18 months in Eisele et al. (2012), 5 years in Joyce et al. (2022), 6 years in Moreno et al. (2022)) than the studies that did (7 years in Seeman et al. (2001) and 15 years in Amieva et al. (2010)).

Although social support has been found to have a generally positive association with neurocognitive functioning, the opposite effect has been observed depending on the support provider. For instance, a longitudinal study by Meister & Zahodne (2022) conducted with older adults found that support from children was associated with lower performance on episodic memory, executive function, visuoconstructional skills, language, and processing speed, while support from friends was positively correlated with episodic memory, language, and processing speed. In the same study, spousal support revealed mixed effects with positive correlations with episodic memory, executive function, and processing speed, and negative correlations with language and visuoconstructional skills. This stands in contrast to previous findings that family support is associated with better neurocognitive functioning at the cross-sectional level (Zhu et al., 2012). Explanations for these results include the suggestion that an individual's emerging cognitive deficits may lead to them relying on support from their children and spouse rather than their friends. This aligns with evidence that social support received from friends has stronger

positive associations for overall neurocognitive functioning than support received from family (Fiori & Jager, 2012; J. Li et al., 2019).

The relationship between social support and neurocognitive functioning may additionally be bidirectional, as an individual's social support may decrease with emerging neurocognitive deficits. When using neurocognitive performance as a predictor of social connectedness, Dyer et al. (2021) found that greater cognitive impairment in older adults with mild-to-moderate Alzheimer's Disease was associated with poorer quality of social networks (which included lower social support). Moreover, greater dementia severity at baseline predicted a greater decline in social network at the 18-month follow-up assessment. Another longitudinal study conducted with middle-aged adults over the course of 10 years found that neurocognition affected the amount of social support received, but social support did not have a significant impact on neurocognition (Liao et al., 2016). Further research is needed to disentangle the causal nature of the relationship between social support and neurocognition.

Nonetheless, social support appears to be positively associated with greater neurocognitive functioning. It may be a protective factor for neurocognitive decline as it promotes cognitive engagement, which in turn contributes to CR (Fratiglioni et al., 2004) and allows for improved neurocognitive health (Holt-Lunstad et al., 2010, 2015; Pillemer & Holtzer, 2016; Reblin & Uchino, 2008; Zhu et al., 2012) and functional outcomes (Cummings et al., 2022). Others have suggested that social support may enhance CR by buffering against the deleterious effects of chronic stress (Cohen, 2004; Cohen & Wills, 1985; Eisenberger et al., 2007; Thoits, 2010).

Positive effects of social support have been observed in populations experiencing unstable housing, who have been found to receive low levels of social support (Addo & Ivey,

2022; S. M. Brown et al., 2020; Chassman et al., 2023; Wright et al., 2017). In this context, social support mitigates the association between stress and poor health (Durbin et al., 2019; S. W. Hwang, Kirst, et al., 2009; Jafry et al., 2021), in addition to correlating positively with subjective well-being (Addo et al., 2022), and increased resilience (Durbin et al., 2019). However, its relationship to neurocognitive functioning in the context of homelessness and precarious housing has yet to be investigated.

1.3 Clinical Risk Factors

Clinical conditions commonly overrepresented in individuals who are homeless or precariously housed may impede the capacity for social connectedness to be a protective factor for their neurocognitive functioning. These conditions include human immunodeficiency virus (HIV) infection (Arum et al., 2021; Kidder et al., 2008; Klinkenberg et al., 2003; Milloy et al., 2012), which has been found to be present in up to 21.1% of adults experiencing unstable housing (Fazel et al., 2014). Similarly, traumatic brain injury (TBI) is a highly prevalent pathology in this population (Cusimano et al., 2021; S. W. Hwang et al., 2008; Stubbs et al., 2020; Zeiler et al., 2021), affecting 53.1% of participants across prior studies (Stubbs et al., 2020). Moreover, relative to the general population, psychotic disorders such as schizophrenia and schizoaffective disorder disproportionately impact individuals who are homeless (Llerena et al., 2018) with prevalence rates ranging up to 21.2% (Ayano et al., 2019).

HIV infection has been associated with altered levels of social connectedness in adults who are stably housed. Experiences of stigma related to the condition (Brener et al., 2013; Takada et al., 2014; Yoo-Jeong et al., 2023) and an increased need for social support (Y. Li et al., 2021; Tam et al., 2023) may underlie changes in subjective sense of interpersonal connection when living with HIV. The detrimental effects of an HIV infection and its accompanying social

stressors are magnified in the context of unstable housing (Riley et al., 2012; Wolitski et al., 2007). Given HIV patients' poorer performance on neurocognitive tests relative to those without an HIV infection (Clifford & Ances, 2013) and the potential for mild HIV-associated neurocognitive disorder (HAND) to persist even with antiretroviral therapy (Heaton et al., 2010; Sanmarti et al., 2014), HIV infection may play a role in the relationship between social connectedness and neurocognitive functioning in adults who are homeless or precariously housed.

Social connectedness has been found to be disrupted post-TBI. Previous research has suggested that stably housed adults who have sustained a TBI experience a decrease in social network size (Douglas, 2020), reduced social participation (Flynn et al., 2018), and difficulties with forming new social connections (Salas et al., 2018). In the context of precarious housing, TBI rates are higher than in stably housed populations (Stubbs et al., 2020; Topolovec-Vranic et al., 2012), and have been associated with poorer physical and mental health, as well as memory concerns (Stubbs et al., 2020; Zeiler et al., 2021). While CR has been posited as a protective factor against neurocognitive impairment associated with TBI in stably housed individuals (Stenberg et al., 2020), the contributions of social connectedness to CR and the influence of TBI on these associations is unknown in individuals who are homeless or precariously housed.

Psychotic disorders represent a category of clinical diagnoses that may impact levels of social connectedness in adults experiencing unstable housing. Loneliness, lower social network size (Badcock et al., 2015; Giacco et al., 2016), and poorer quality of interpersonal interactions (Heering & Van Haren, 2016) are common in individuals with psychotic disorders who are stably housed. As a high risk for psychosis has been linked to neurocognitive deficits (Zheng et al., 2018), psychotic disorders remain an important factor to consider when examining the

relationship between social connectedness and neurocognitive functioning in the context of homelessness and precarious housing.

Understanding the roles that various physical and psychological conditions such as HIV infection, TBI, and psychotic disorders play in the associations between social connectedness and neurocognitive functioning may aid in further elucidating the nature of these in adults who are homeless and precariously housed. If there are conditions that either preclude or facilitate the ability for social connectedness to contribute to CR, examining these would provide further insight into the complexity of this vulnerable population's neurocognitive health.

1.5 Present Study

The relationship between social connectedness and neurocognitive functioning has been extensively researched in the general population. However, to our knowledge, no research on social network size, relationship quality, and social support in relation to neurocognitive functioning has been conducted in adults who are homeless or precariously housed. Investigating these associations in a vulnerable population that experiences low social connectedness (Bower et al., 2018; Chassman et al., 2023; Gültekin et al., 2014; Hawkins & Abrams, 2007), higher rates of neurocognitive impairment (Depp et al., 2015; Hurstak et al., 2017; Stergiopoulos et al., 2015), and poorer functional outcomes than individuals who are stably housed (R. T. Brown et al., 2012, 2013, 2016; Stone et al., 2019) would provide insight as to whether dimensions of social connectedness are potential contributors to CR. This in turn may help bridge a gap in knowledge on modifiable lifestyle factors that can offset neurocognitive impairment and improve long-term functional outcomes in this socially marginalized group.

The current study uses data from the Hotel Study, a 20-year longitudinal investigation of mental health and neurocognitive functioning in over 500 adults experiencing precarious housing

in an impoverished neighbourhood of Vancouver, Canada (Honer et al., 2017; Vila-Rodriguez et al., 2013). We aimed to investigate 1) whether dimensions of social connectedness are cross-sectionally associated with neurocognitive functioning in a sample of adults who are homeless or precariously housed, and 2) whether clinical risk factors common in unhoused populations moderate these cross-sectional associations. It was hypothesized that larger social networks, lower relationship conflict, and higher levels of global social support at study entry would each be independently associated with neurocognitive health, as indicated by greater global neurocognitive performance as well as greater performance on tasks probing four domains of neurocognition: verbal memory, inhibitory control, sustained attention, cognitive flexibility. Verbal memory and inhibitory control were selected as decreased performance in these two domains has been previously found to be associated with a higher risk of mortality in the current sample (see Gicas et al., 2020). Sustained attention and cognitive flexibility were included to provide a more comprehensive understanding of neurocognitive functioning. The four domains have been consistently measured throughout the course of the study and are vulnerable to disruption in neurological and psychiatric illness. Moreover, as HIV, a prior TBI, and psychotic disorders are prevalent clinical risk factors in the current sample (see Honer et al., 2017) and have been found to be associated with social connectedness, we further hypothesized that they would influence cross-sectional relationships between social connectedness and neurocognitive functioning. We predicted that the presence of an HIV infection, TBI history, or psychotic disorder would disrupt the protective effects of social connectedness on neurocognitive functioning, weakening any observed associations. Specifically, participants without an HIV infection, TBI history, or psychotic disorder would exhibit stronger associations between social connectedness and neurocognitive functioning, compared to those who do have one of these

clinical conditions. It should be noted that although prior research has revealed potential bidirectional associations between social connectedness and neurocognitive functioning, the majority of literature supports the positioning of social connectedness as a predictor of neurocognitive functioning through the CR hypothesis. Given this and the fact that the current data does not involve the use of multiple timepoints, the examination of bidirectionality was not pursued in the present study.

2. Methods

2.1 Study Design & Participants

Homelessness is defined as a lack of stable, safe, permanent, and appropriate housing (Gaetz et al., 2012). Precarious housing involves the risk of homelessness in the immediate or near future due to factors such as financial poverty, external or personal hardship, discrimination, lack of other available housing, or inappropriateness of current housing (Echenberg & Munn-Rivard, 2020; Gaetz et al., 2012). Based on these definitions, in the Hotel Study, participants were recruited from single-room occupancy hotels and a community courthouse located in Vancouver's Downtown East Side (DTES), a low-income urban neighbourhood. Additional recruitment was conducted via youth-supported single-room occupancy hotels and St. Paul's Hospital Emergency Services. These recruitment processes allowed for a more representative sample of the precariously housed and homeless population. Fluent English speakers aged 18 years and older who were able to provide written informed consent were eligible for enrollment. As participants were recruited into the study on a rolling basis, data collection took place during baseline interviews and testing sessions between November 2008 to April 2017. All participants provided written informed consent and received monetary compensation after each study session. Testing sessions were conducted in either 1) the Hotel Study's main DTES research office if the

participant was not undergoing a neuroimaging scan, or 2) a laboratory at the University of British Columbia's Vancouver campus if they were undergoing a neuroimaging scan. The study was approved by the Clinical Research Ethics Boards of the University of British Columbia and Simon Fraser University. Additional approval from York University's Research Ethics Board was provided for the current analysis.

2.2 Measures of Social Connectedness

Information on available social network size was collected via the Arizona Social Support Interview Schedule (ASSIS) (Barrera et al., 1980). The ASSIS consists of a structured interview with six questions assessing six social support categories: 1) material aid, 2) physical assistance, 3) intimate interaction, 4) guidance, 5) feedback, and 6) positive social interaction. Within each question, there are two measures of the number of social network connections: a) the number of available individuals who could provide that form of support, and b) the number of individuals who provided that form of support in the past month. These two measures were summed across the six social support categories, yielding two scores: total available network size and total actual social support network size. Total available network size was used in the current study to understand whether the number of social connections, regardless of whether they were supportive or not, was associated with neurocognitive functioning.

To assess relationship conflict, the Maudsley Addiction Profile (MAP) was administered. In the MAP, four domains are assessed: 1) substance use, 2) health risk behaviour, 3) physical and psychological health, and 4) personal/social functioning (Marsden et al., 1998). Within the personal/social functioning domain, social contacts and conflicts are reported. Social contacts consist of face-to-face interactions or telephone calls with partner(s), relatives, children up to the age of 18, friends, or acquaintances. Conflicts consist of serious arguments which may include

verbal abuse, violence, and atypical differences of opinion. As per the MAP protocol, relationship conflict scores are calculated as the total number of days in conflict divided by the total number of days in contact in the past 30 days, multiplied by 100 to yield a percentage. These relationship conflict scores are derived for three categories of social connections: 1) partner, 2) relatives, and 3) friends. They were averaged to have one global relationship conflict score across all types of social connections.

Participants' social support was measured via the Inventory of Socially Supportive Behaviors (ISSB) (Barrera et al., 1981; Barrera & Ainlay, 1983). The ISSB consists of 40 items measuring social support in the following four categories: 1) directive guidance, 2) tangible assistance, 3) positive social exchange, and 4) nondirective support. Participants were asked to rate the frequency of occurrence in the past month of each of the 40 items on a 5-point Likert scale where 1 = Not at all and 5 = About every day. The 40 items were averaged to yield a global measure of social support received. The measure displayed excellent internal consistency, with a Cronbach's α of 0.97 [95% CI: 0.97, 0.98].

2.3 Measures of Neurocognitive Functioning

A battery of neuropsychological tests assessing a range of neurocognitive processes was administered at study baseline by trained research assistants under the supervision of a clinical neuropsychologist. The following four domains of neurocognitive functioning were among those assessed: verbal memory, inhibitory control, sustained attention, and cognitive flexibility. Verbal memory was assessed using Form 1 of the Hopkins Verbal Learning Test-Revised (HVLT-R) (Benedict et al., 1998). The HVLT-R consists of a 12-item word list that is read aloud to the participant and must subsequently be recalled in learning, recognition, and delayed recall trials. The learning and recognition trials take place first over the course of approximately 5 minutes.

Unrelated tasks are then administered for approximately 25 minutes until the delayed recall trial. The total number of words recalled in the delayed recall trial served as the measure of verbal memory in the current study.

Inhibitory control, a key executive functioning process, was measured via the Stroop colour-word subtest (Stroop, 1992). This subtest involves a table where names of colours are printed in a different colour of ink than the name indicates. Participants were required to name the colour of the ink rather than reading the word, thereby demanding the ability to suppress the automatic reading response. This in turn acts as an index of poor inhibitory control. The subtest consists of 100 items which must be read with a maximum time limit of 45 seconds (Scarpina & Tagini, 2017). The total number of correctly answered items served to indicate inhibitory control performance.

Sustained attention was assessed via the Rapid Visual Information Processing (RVP) A' subtest on the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Robbins et al., 1994). The RVP A' subtest involves the pseudo-random presentation of single digits ranging from 2 to 9 in a white box on computer screen, with the digits changing at a rate of 100 digits per minute. The participant is requested to detect target sequences of digits (e.g. 2-3-8) by clicking the button on screen as quickly as possible once they believe the target sequence has been presented. Digit sequence length ranges between one to three digits. The task completion time is 4 minutes. The total correct score consisted of the measure of sustained attention.

Cognitive flexibility was measured using the total number of adjusted errors on the Intra-Extra Dimensional (IED) Set Shift subtest on the CANTAB (Robbins et al., 1994). The IED involves the presentation of various combinations of white lines and pink shapes to participants who must use feedback from the administrator to work out a rule to determine the pattern before

the stimuli set changes. The ability to deduce the new rules as the stimuli set changes is considered as the marker of greater cognitive flexibility. Perseveration as per the inability to use administrator feedback to shift rules is reflective of poor cognitive flexibility. The task completion time is 5 minutes. To correct a non-normal distribution, the scores underwent a logarithmic transformation and were subsequently multiplied by -1 to facilitate interpretation in line with other measures whereby higher scores represent better performance.

The fifth neurocognitive outcome in this study was a measure of global neurocognition. It was computed by first transforming the raw scores for each neurocognitive domain into *z*-scores. The *z*-scores for each neurocognitive domain were then averaged to yield one global neurocognition score.

2.4 Measures of Clinical Risk Factors

Demographic information, including age, sex, ethnicity, number of years of education, and number of years homeless were self-reported in a structured interview at study entry. Clinical data was collected by trained research assistants and psychiatrists in a separate session from neurocognitive testing. Diagnoses for psychiatric and neurological illnesses were made according to criteria in the text-revised 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association, 2000). The diagnostic process included a review of medical records, a mental status examination with a psychiatrist, the Best Estimate Clinical Evaluation and Diagnosis (BECED) (Endicott, 1988), and a Mini-International Neuropsychiatric Psychotic Interview (MINI) (Sheehan et al., 1998).

The psychotic disorder variable consisted of participants who received a diagnosis of any of the following: schizophrenia, schizoaffective disorder, substance-induced psychosis, major depressive disorder with psychotic features, bipolar disorder with psychotic features, or

psychosis not otherwise specified. If they had a psychotic disorder diagnosis, they would be coded as a "1" for this clinical risk factor. If they had no diagnosis, they would be coded as a "0." Viral infection was assessed by blood samples drawn and tested for serologies. Participants with HIV-seropositive results were classified as having HIV and were coded as a "1." Those who did not test HIV-seropositive were coded as a "0." TBI status was defined as participants responding "Yes" to having previously sustained a TBI and endorsing any of the following post-injury symptoms: loss of consciousness, memory loss, or confusion. They received a code of "1" for this clinical condition. If participants responded "No" to experiencing a prior TBI and did not endorse any post-injury symptoms, they were considered to not have a history of TBI and were coded as a "0."

2.5 Statistical Analysis

All statistical analyses and data visualizations were conducted in R Statistical Software (version 4.3.2) (R Core Team, 2023) using *dplyr* (Wickham et al., 2023), *psych* (Revelle, 2023), *reghelper* (Hughes & Beiner, 2023), and *ggplot2* (Wickham, 2016) packages. Correlation analyses were conducted between the dimensions of social connectedness to identify any collinearity and gain a broad understanding of how these variables are related to one another. A series of hierarchical multiple regression models were subsequently constructed for each of the five neurocognitive outcomes at: global neurocognition, verbal memory, inhibitory control, sustained attention, and cognitive flexibility. To examine the first hypothesis, the main predictors of available social network size, average relationship conflict, and social support scores at study entry were mean-centered and added to the base model while controlling for demographic factors of age, sex, and years of education. Age and years of education were mean-centered. The following clinical risk factors were added to the second model: HIV infection, TBI status, and

psychotic disorder. To examine the second hypothesis and conduct the moderation analyses, the third model was built using an iterative method, where one interaction term between the social connectedness variables and the demographic or clinical risk factors was included at a time. An interaction term would only be kept in the model if it was significant at $p < .05$. Any significant interaction terms were further investigated by conducting a simple slopes analysis at both levels of the categorical moderator. If there were no significant interaction terms, only the first and second models were reported.

Sensitivity analyses were performed by conducting a multiple imputation on the dataset to account for missing data. Additional sets of hierarchical multiple regression models were constructed using the imputed dataset and compared to the initial models in order to see if the missing data was biasing the results.

3. Results

3.1 Sample Characteristics and Clinical Risk Factors

The current sample initially consisted of 484 precariously housed adults. The analytic sample size for each series of hierarchical multiple regression models varied depending on available data for the neurocognitive functioning and social connectedness variables. The pattern of demographic and clinical characteristics for the overall sample in Table 1 remained consistent across the subsamples.

Table 1.

Sample Demographic and Clinical Characteristics

Characteristic	N (%)	Mean (SD)	Median (IQR)	Range
Age, years	484	40.92 (11.53)	42 (18)	20-75
Education, years	484	10.50 (2.38)	10 (3)	2-16
Homeless, years	347	12.70 (10.78)	9.42 (15.47)	0.11-45.97

Monthly income (Canadian dollars)	475	844.96 (382.11)	875 (390)	0-3600
Available social network size	455	4.21 (2.85)	4 (3.50)	0-18
Global social support	479	1.05 (0.84)	0.85 (1.11)	0-3.90
Average relationship conflict	419	8.51 (19)	0 (6.67)	0-100
Sex				
Male	363 (75.0)			
Female	121 (25.0)			
Ethnicity				
White	290 (60.2)			
Indigenous	166 (34.4)			
Other/mixed	26 (5.4)			
Ever homeless	348 (71.9)			
History of traumatic brain injury	199 (41.1)			
Any psychotic disorder	260 (53.7)			
HIV-seropositive	65 (14.9)			
Alcohol dependence	95 (19.6)			
Cannabis dependence	176 (36.4)			
Opioid dependence	206 (42.7)			
Stimulant dependence	379 (78.3)			

Note. SD = Standard deviation. IQR = Interquartile range. Substance dependence was based on DSM-IV-TR diagnostic criteria used in the clinical assessment.

3.2 Correlations Between Dimensions of Social Connectedness

To identify any collinearity and understand the broad relationships between the three dimensions of social connectedness of interest, Pearson's correlation analyses were conducted (see Table 2). Available social network size and global social support were positively correlated. As social network size increases, as does the level of global social support. The correlation coefficient was statistically significant, but was low and considered to be weak (Akoglu, 2018) and thus, not a concern for collinearity. Likewise, global social support and average relationship conflict were positively correlated. As social support increases, average relationship conflict

increases. This correlation coefficient was statistically significant, but the size of the coefficient itself indicated a weak relationship between the two variables. Thus, there were no indications of collinearity between any of the dimensions of social connectedness.

Table 2

Pearson's Correlation Coefficients for Available Social Network Size, Global Social Support, and Average Relationship Conflict

	Available social network size (n = 455)	Global social support (n = 479)	Average relationship conflict (n = 419)
Available social network size (n = 455)	-	0.201***	0.069
Global social support (n = 479)	0.201***	-	0.101*
Average relationship conflict (n = 419)	0.069	0.101*	-

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

3.3 Hierarchical Multiple Regression Results

3.3.1 Global Neurocognition and Social Connectedness

The final analytic sample for global neurocognition consisted of 252 participants. The hierarchical multiple regression analysis revealed that neither social network size, social support, nor relationship conflict were associated with global neurocognition in the base model (see Table 3). There was a significant main effect of age where younger participants had greater global neurocognition. An additional main effect of education was observed where participants with a higher number of years of education exhibited greater global neurocognition. In the second model, the addition of HIV, TBI history, and psychotic disorder did not significantly predict the proportion of variance in global neurocognition above and beyond the predictors in the initial

model. The main effects of age and education remained. For the third model, there were no significant interactions between HIV, TBI history, nor psychotic disorder and any of the social connectedness variables, indicating that these clinical risk factors did not have any significant moderating effects on the associations between social connectedness and global neurocognition (all interactions were at $p > .05$).

Table 3

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Global Neurocognition

Global Neurocognition (*n* = 252)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.10	-	-	< .001***
Age	-0.02	0.00	-4.96	< .001***	[-0.02, -0.01]				
Sex	0.07	0.09	0.81	.421	[-0.10, 0.25]				
Education	0.06	0.02	3.63	< .001***	[0.03, 0.09]				
Available social network size	0.03	0.01	1.90	.058	[-0.00, 0.05]				
Global social support	-0.00	0.00	-0.41	.684	[-0.00, 0.00]				
Average relationship conflict	0.00	0.00	0.35	.726	[-0.00, 0.00]				
Model 2: Clinical risk factors						0.08	-0.02	1.72	0.164
Age	-0.02	0.00	-3.73	< .001***	[-0.03, -0.01]				
Sex	0.04	0.10	0.38	.705	[-0.16, 0.24]				
Education	0.06	0.02	3.18	.002**	[0.02, 0.10]				
Available social network size	0.03	0.01	1.82	.070	[-0.00, 0.06]				
Global social support	-0.00	0.00	-0.54	.587	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.15	.881	[-0.00, 0.00]				
HIV	-0.16	0.11	-1.40	.162	[-0.38, 0.06]				
TBI	0.09	0.09	1.02	.310	[-0.08, 0.26]				
Psychotic disorder	-0.12	0.09	-1.36	.176	[-0.28, 0.05]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
*$p < .05$. **$p < .01$. ***$p < .001$.

3.3.2 Verbal Memory and Social Connectedness

The analytic sample for verbal memory included 298 participants. The initial hierarchical multiple regression model revealed that larger social network size, younger age, and a higher

number of years of education were associated with greater verbal memory performance (see Table 4). The inclusion of the clinical risk factors in the second model predicted a significant proportion of variance in verbal memory above and beyond the dimensions of social connectedness and demographic factors. In addition to the main effects of age, education, and social network size, main effects of HIV infection and psychotic disorder emerged. Specifically, the presence of an HIV infection and psychotic disorder each independently predicted poorer verbal memory performance. When constructing the third model, a significant interaction between HIV infection and relationship conflict was observed. No other significant interactions between social connectedness and the clinical risk factors emerged ($p > .05$). The third model explained a significant proportion of variance above and beyond the dimensions of social connectedness, demographic characteristics and clinical risk factors. As such, HIV moderated the association between relationship conflict and verbal memory performance (see Figure 1). A simple slopes analysis revealed that the association between relationship conflict and verbal memory was negative for those with HIV ($\beta = -0.08$, $SE = 0.04$, $p = .062$) and was positive for those without HIV ($\beta = 0.01$, $SE = 0.01$, $p = .186$), but neither slope reached statistical significance.

Table 4

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Verbal Memory

Verbal Memory (*n* = 298)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.05	-	-	< .001***
Age	-0.07	0.01	-5.46	< .001***	[-0.09, -0.04]				
Sex	0.35	0.33	1.05	.293	[-0.30, 1.01]				
Education	0.20	0.06	3.22	.001**	[0.08, 0.32]				
Total social network size	0.14	0.05	2.72	.007**	[0.04, 0.24]				
Global social support	-0.01	0.00	-1.15	.251	[-0.01, 0.00]				

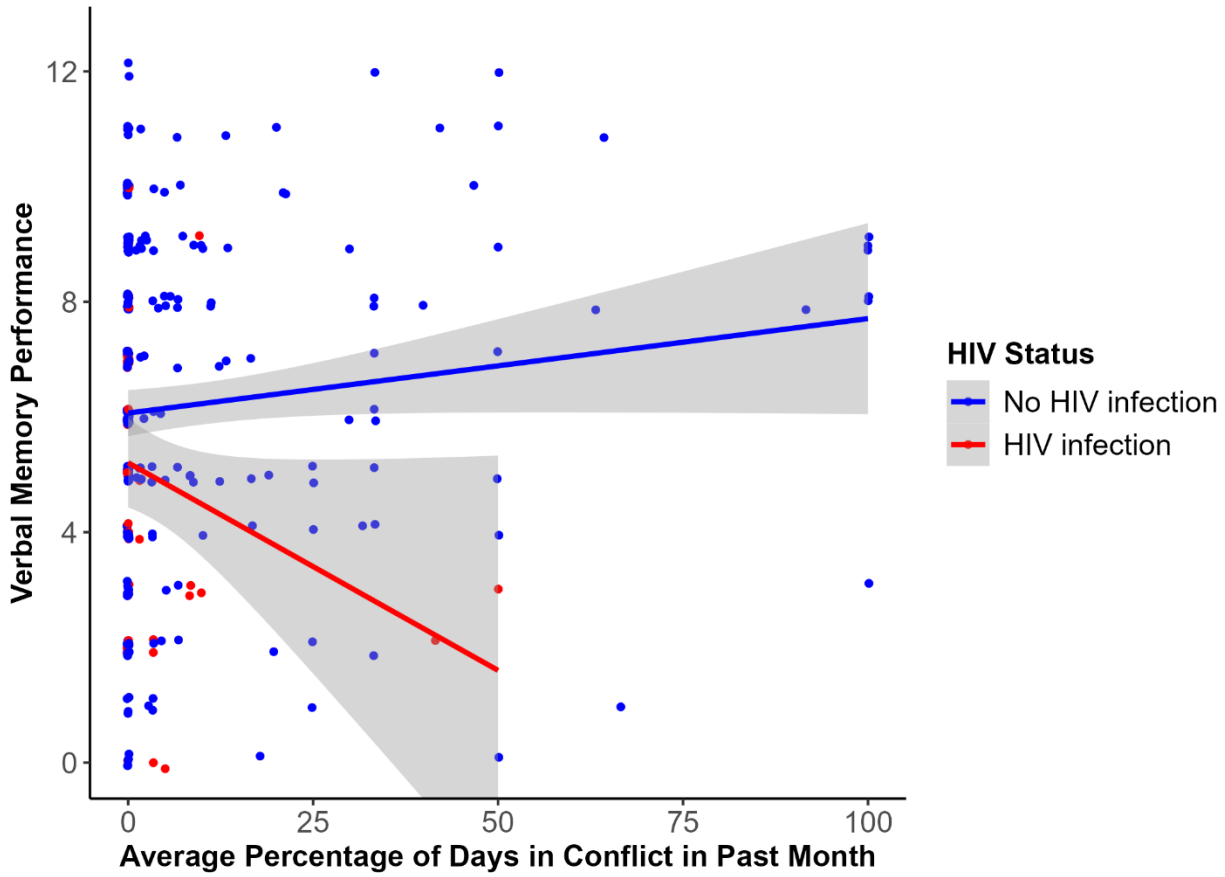
Average relationship conflict	0.01	0.01	1.68	.094	[-0.00, 0.03]				
Model 2: Clinical risk factors						0.07	0.03	3.36	.019*
Age	-0.06	0.02	-3.43	.001***	[-0.09, -0.02]				
Sex	0.25	0.40	0.63	.529	[-0.54, 1.05]				
Education	0.13	0.07	1.83	.068	[-0.01, 0.27]				
Available social network size	0.13	0.06	2.21	.028*	[0.01, 0.25]				
Global social support	-0.01	0.01	-1.01	.313	[-0.02, 0.01]				
Average relationship conflict	0.01	0.01	0.92	.358	[-0.01, 0.02]				
HIV	-1.05	0.45	-2.33	.021*	[-1.94, -0.16]				
TBI	-0.11	0.33	-0.34	.732	[-0.77, 0.54]				
Psychotic disorder	-0.68	0.33	-2.07	.039*	[-1.34, -0.03]				
Model 3: Interaction between average relationship conflict and HIV						0.08	0.01	4.39	.037*
Age	-0.06	0.02	-3.46	.001***	[-0.09, -0.02]				
Sex	0.20	0.40	0.50	.619	[-0.59, 0.99]				
Education	0.12	0.07	1.77	.078	[-0.01, 0.26]				
Available social network size	0.14	0.06	2.41	.017*	[0.03, 0.26]				
Global social support	-0.01	0.01	-1.10	.272	[-0.02, 0.00]				
Average relationship conflict	0.01	0.01	1.33	.186	[-0.01, 0.03]				
HIV	-1.52	0.50	-3.03	.003**	[-2.51, -0.53]				
TBI	-0.10	0.33	-0.30	.762	[-0.75, 0.55]				
Psychotic disorder	-0.68	0.33	-2.05	.041*	[-1.32, -0.03]				
Average relationship conflict x HIV	-0.09	0.04	-2.10	.037*	[-0.18, -0.01]				

Note. β = Standardized regression coefficient; *SE* = Standard error of the mean; CI = Confidence intervals; TBI = Traumatic brain injury.

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

Figure 1

Association Between Average Relationship Conflict and Verbal Memory Performance Based On HIV Infection Status



Note. Grey areas around the regression lines represent the 95% confidence intervals.

3.3.3 Inhibitory Control and Social Connectedness

The analytic sample for inhibitory control consisted of 295 participants. The initial hierarchical multiple regression model revealed that none of the three dimensions of social connectedness were significantly associated with inhibitory control, but younger age and a higher number of years of education both independently predicted greater inhibitory control (see Table 5). The inclusion of HIV, TBI history, and psychotic disorder in the second model did not explain a significant proportion of variance beyond the dimensions of social connectedness and demographic factors. The main effects of age and education remained consistent. In constructing the third model, there were no significant interactions between the dimensions of social connectedness and the clinical risk factors, as all *p*-values > .05. There were there no significant

moderating effects of HIV, TBI history, and psychotic disorder on the associations between social connectedness and inhibitory control.

Table 5

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Inhibitory Control

Inhibitory Control (*n* = 295)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.08	-	-	< .001***
Age	-0.02	0.00	-3.48	.001***	[-0.02, -0.01]				
Sex	0.03	0.10	0.32	.746	[-0.17, 0.23]				
Education	0.06	0.02	3.37	.001***	[0.03, 0.10]				
Available social network size	0.03	0.01	1.98	.049*	[0.00, 0.06]				
Global social support	-0.00	0.00	-0.63	.529	[-0.00, 0.00]				
Average relationship conflict	0.00	0.00	0.08	.935	[-0.00, 0.00]				
Model 2: Clinical risk factors						0.09	0.01	1.48	.219
Age	-0.02	0.00	-3.73	< .001***	[-0.03, -0.01]				
Sex	0.04	0.10	0.38	.705	[-0.16, 0.24]				
Education	0.06	0.02	3.18	.002**	[0.02, 0.10]				
Available social network size	0.03	0.01	1.82	.070	[-0.00, 0.06]				
Global social support	-0.00	0.00	-0.54	.587	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.15	.881	[-0.00, 0.00]				
HIV	-0.16	0.11	-1.40	.162	[-0.38, 0.06]				
TBI	0.09	0.09	1.02	.310	[-0.08, 0.26]				
Psychotic disorder	-0.12	0.09	-1.36	.176	[-0.28, 0.05]				

Note. B = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
*p < .05. **p < .01. ***p < .001.

3.3.4 Sustained Attention and Social Connectedness

The analytic sample for sustained attention consisted of 269 participants. Results from the hierarchical multiple regression analysis revealed that social network size, social support, and relationship conflict did not significantly predict sustained attention performance in the first model (see Table 6). Younger age and a higher number of years of education were both independently associated with greater sustained attention. The addition of HIV, TBI history, and

psychotic disorder as predictors in the second model did not contribute to a significant proportion of variance in sustained attention performance in comparison to the first model. No significant interactions between any clinical risk factors and dimensions of social connectedness were detected, suggesting that neither HIV, TBI history, nor psychotic disorder moderated any associations between dimensions of social connectedness and sustained attention (all interactions were at $p > .05$).

Table 6

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Sustained Attention

	Sustained Attention (*n* = 269)								
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.04	-	-	.015*
Age	-0.00	0.00	-2.45	.015*	[-0.00, -0.00]				
Sex	0.01	0.01	1.28	.202	[-0.01, 0.03]				
Education	0.00	0.00	2.96	.003**	[0.00, 0.01]				
Available social network size	0.00	0.00	0.90	.367	[-0.00, 0.00]				
Global social support	-0.00	0.00	-0.79	.430	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.03	.980	[-0.00, 0.00]				
Model 2: Clinical risk factors						0.03	-0.01	0.84	.475
Age	-0.00	0.00	-2.33	.021*	[-0.00, -0.00]				
Sex	0.01	0.01	1.40	.163	[-0.01, 0.03]				
Education	0.00	0.00	2.96	.003**	[0.00, 0.01]				
Available social network size	0.00	0.00	0.84	.400	[-0.00, 0.00]				
Global social support	-0.00	0.00	-0.75	.452	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.16	.872	[-0.00, 0.00]				
HIV	-0.02	0.01	-1.56	.120	[-0.04, 0.00]				
TBI	0.00	0.01	0.19	.850	[-0.01, 0.02]				
Psychotic disorder	-0.00	0.01	-0.05	.961	[-0.02, 0.02]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
*$p < .05$. **$p < .01$. ***$p < .001$.

3.3.5 Cognitive Flexibility and Social Connectedness

To examine the associations between cognitive flexibility and social connectedness, 283 participants were included in the analytic sample. The initial hierarchical multiple regression model revealed that social network size, social support and relationship conflict did not significantly predict cognitive flexibility (see Table 7). However, younger age and a higher number of years of education each independently predicted greater cognitive flexibility. There was an additional main effect of sex where male participants (M = -3.65, SE = 0.07) exhibited greater cognitive flexibility than female participants (M = -3.93, SE = 0.12). In the second model, HIV, TBI history, and psychotic disorder did not independently predict cognitive flexibility. Moreover, the model did not explain a significant proportion of variance in cognitive flexibility above and beyond the first model. When constructing the third model, a significant interaction between social network size and HIV was observed, indicating that HIV significantly moderated the association between social network size and cognitive flexibility. The simple slopes analysis revealed that the association between social network size and cognitive flexibility was negative for individuals with HIV (β = -0.08, SE = 0.05, p = .088), and positive for those without HIV (β = 0.02, SE = 0.02, p = .218). As such, larger social network size was associated with poorer cognitive flexibility among participants who were HIV-seropositive and greater cognitive flexibility among participants with no HIV infection (see Figure 2). However, neither slope reached statistical significance. There were no other significant interactions between the social connectedness variables and clinical risk factors (p > .05). The third model explained a significant proportion of variance above and beyond the inclusion of clinical risk factors in the second model.

Table 7

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Cognitive Flexibility

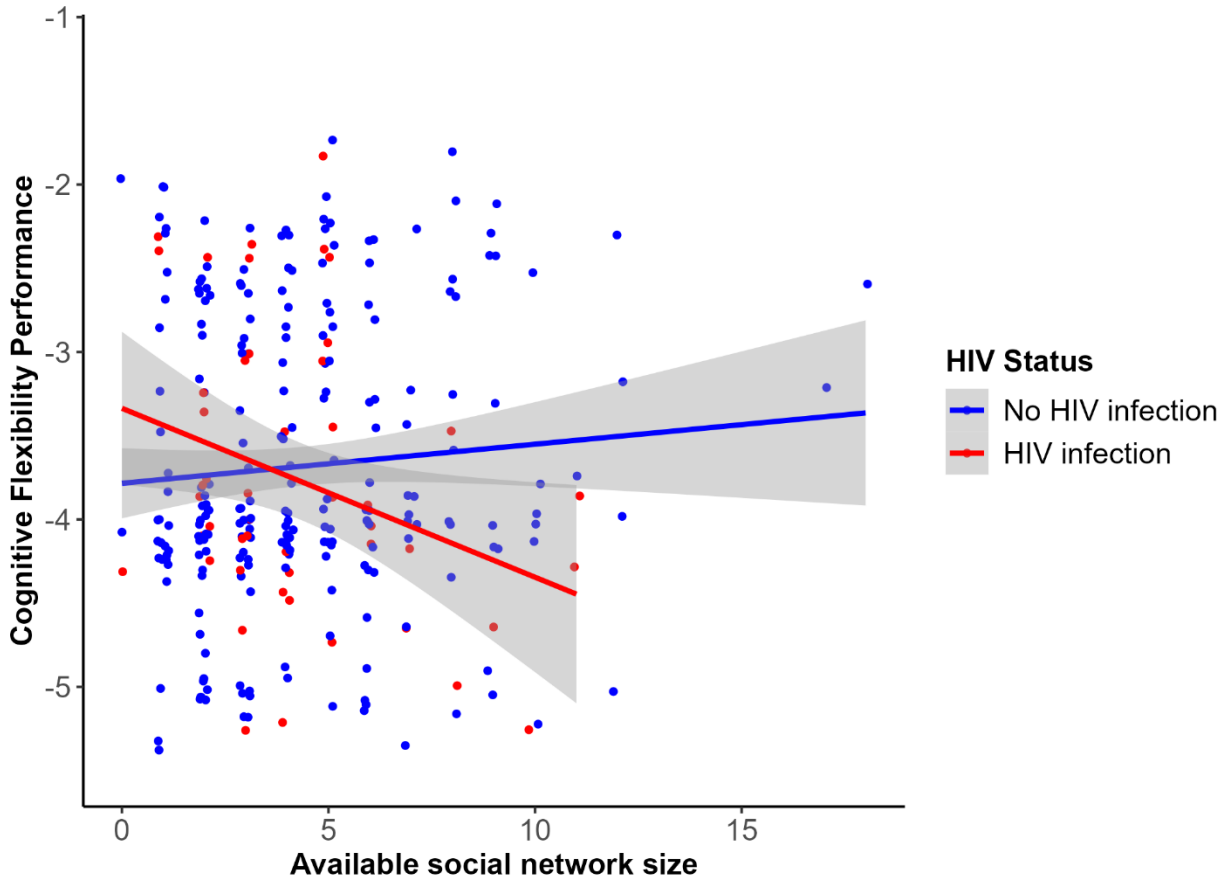
Cognitive Flexibility (*n* = 283)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.09	-	-	< .001***
Age	-0.02	0.00	-4.90	< .001***	[-0.03, -0.01]				
Sex	-0.28	0.12	-2.23	.026*	[-0.52, -0.03]				
Education	0.05	0.02	2.42	.016*	[0.01, 0.10]				
Available social network size	0.01	0.02	0.58	.565	[-0.03, 0.05]				
Global social support	0.00	0.00	0.10	.924	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.83	.405	[-0.01, 0.00]				
Model 2: Clinical risk factors						0.09	-0.01	0.96	.413
Age	-0.03	0.01	-5.12	< .001***	[-0.04, -0.02]				
Sex	-0.28	0.13	-2.20	.029*	[-0.52, -0.03]				
Education	0.05	0.02	2.30	.022*	[0.01, 0.09]				
Available social network size	0.01	0.02	0.53	.599	[-0.03, 0.05]				
Global social support	0.00	0.00	0.12	.908	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.85	.396	[-0.01, 0.00]				
HIV	-0.00	0.13	-0.02	.981	[-0.27, 0.26]				
TBI	0.09	0.10	0.92	.359	[-0.11, 0.30]				
Psychotic disorder	-0.14	0.10	-1.40	.164	[-0.35, 0.06]				
Model 3: Interaction between social network size and HIV						0.10	0.01	4.25	.040*
Age	-0.03	0.01	-5.05	< .001***	[-0.04, -0.02]				
Sex	-0.28	0.13	-2.23	.027*	[-0.52, -0.03]				
Education	0.05	0.02	2.17	.031*	[0.00, 0.09]				
Available social network size	0.02	0.02	1.24	.218	[-0.01, 0.06]				
Global social support	0.00	0.00	0.03	.976	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.73	.467	[-0.01, 0.00]				
HIV	0.01	0.13	0.09	.929	[-0.25, 0.28]				
TBI	0.11	0.10	1.09	.275	[-0.09, 0.32]				
Psychotic disorder	-0.14	0.10	-1.35	.179	[-0.34, 0.06]				
Available social network size x HIV	-0.11	0.05	-2.06	.040*	[-0.21, -0.00]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.

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

Figure 2.

Association Between Available Social Network Size and Cognitive Flexibility Performance Based On HIV Infection Status



Note. Shaded grey areas around the regression lines represent the 95% confidence intervals.

3.4 Sensitivity Analysis

To understand the influence that missing data may have on the current results, a sensitivity analysis was conducted using multiple imputation. See Appendix A for the results of the hierarchical multiple regression models that were constructed using the imputed dataset. The pattern of results from the imputed dataset did not diverge from the main results, with the exception of the verbal memory models where the main effect of social network size and the interaction between relationship conflict and HIV did not reach significance. Moreover, a main effect of HIV emerged for global neurocognition and sustained attention, and a main effect of psychotic disorder was observed for global neurocognition and cognitive flexibility. These

differing significant effects inform the nature of the missing data and should be considered when interpreting the results thus far.

4. Discussion

4.1 Overall Findings

The goal of this study was to examine associations between dimensions of social connectedness to various domains of neurocognitive functioning in a sample of adults experiencing homelessness or precarious housing. To our knowledge, this is the first study to have investigated such associations in this vulnerable population that is prone to low social connectedness (Bower et al., 2018; Chassman et al., 2023; Gültekin et al., 2014; Hawkins & Abrams, 2007) and higher rates of neurocognitive dysfunction (Depp et al., 2015; Hurstak et al., 2017; Stergiopoulos et al., 2015). As social connectedness has been identified as an important contributor to CR in the context of neurocognitive aging (Cohen, 2004; Fratiglioni et al., 2004; Kelly et al., 2017), we aimed to understand whether its protective effects could be extended to conditions of homelessness and precarious housing and potentially inform its utility in contributing to CR in this population. Consistent with the initial hypothesis, it was found that available social network size significantly predicted verbal memory performance. Specifically, larger social network size was associated with greater verbal memory performance. Neither social support nor relationship conflict were significant predictors of neurocognitive functioning. Moreover, aligned with our second hypothesis, HIV infection emerged as a moderator of the association between relationship conflict and verbal memory, as well as the association between available social network size and cognitive flexibility. Neither psychotic disorder nor TBI emerged as moderators of any associations between dimensions of social connectedness and neurocognitive functioning.

Available social network size significantly predicted verbal memory performance, thereby supporting our first hypothesis. This is consistent with previous research that has suggested that a higher number of social connections buffers against social isolation and allows one to engage in cognitively stimulating activities that requires use of verbal memory abilities (Glymour et al., 2008; Hertzog et al., 2008; Nie et al., 2021; Penninkilampi et al., 2018; Sharifian et al., 2019). However, this result also stands in contrast to prior studies that have found no effect of social network size on memory in older adults (Kuiper et al., 2020; Meister & Zahodne, 2022). While the effect of social network size is relatively mixed in the field of neurocognitive aging, social network size may be an important contributor to verbal memory in the context of homelessness and precarious housing as isolation and loneliness are highly prevalent (Addo & Ivey, 2022). As such, homeless and precariously housed individuals may particularly benefit from having a higher number of social ties to partake in activities which require the practice of verbal memory abilities, including engaging in conversation and managing everyday occurrences, which require one to retain information for future action (e.g. receiving directions, providing guidance on a task, discussing goals to accomplish). However, as the current finding is cross-sectional, the presence of a bidirectional effect is unclear. It is possible that greater verbal memory abilities predict the capacity to maintain a larger social network, as has been suggested by prior literature in the field of neurocognitive aging (Aartsen et al., 2004; Collins et al., 2022; Cornwell, 2009). Additional longitudinal research is needed to deepen understanding of the current finding and elucidate any potential bidirectional associations in adults who are homeless or precariously housed.

Contrary to our initial hypothesis, global social support did not significantly predict any neurocognitive outcome of interest. It may be that although social support may not have any

significant cross-sectional associations with participants' neurocognitive functioning, its positive effects may be cumulative and emerge over time. Given that prior longitudinal studies have found evidence that higher levels of social support predict slower decline in verbal memory over the course of 16 years (Scholes & Liao, 2022) and a reduced risk of dementia and Alzheimer's Disease (Amieva et al., 2010; Seeman et al., 2001) in older adults, it is likely that longitudinal analyses are needed to delineate the true impact of social support on neurocognitive functioning in adults experiencing homelessness or precarious housing. The lack of significant associations between social support and neurocognitive functioning observed in the present study may be further attributable to the fact that a global measure of social support was used as a predictor, rather than individual domains of social support. As specific types of social support such as emotional support and informational support can have stronger protective effects on neurocognitive functioning (La Fleur & Salthouse, 2016; Mogic et al., 2023), it may be that using an average level of social support masked potential significant associations.

Moreover, global relationship conflict did not independently predict neurocognitive performance in any domain, contrary to our initial hypothesis. Although prior studies have found relationship quality to be an independent contributor to neurocognitive functioning (La Fleur & Salthouse, 2016; Meister & Zahodne, 2022; Pillemer & Holtzer, 2016; Tun et al., 2013), this was not observed in the current sample of participants who are homeless or precariously housed. This may be due to the nature of the MAP relationship conflict measure. Several participants did not yield conflict scores in specific relationship categories due to the lack of those particular connections in their lives. The average relationship conflict score was not composed of the same number of relationships for all participants. One participant may have had an average relationship conflict score derived from their level of conflict with their friends and relatives, but

no partner, and another may have had an average relationship conflict score derived from their level of conflict with their partner only, as they do not have friends or relatives. The global measure of relationship conflict was thus heterogeneous and may have affected the detection of associations with neurocognitive functioning. Moreover, as the variable was an average of up to three available relationship types, it may not be a significant predictor of neurocognitive functioning when considered as a standalone factor. It should also be noted that sample size limitations may have also masked important effects of relationship conflict, specifically as it pertains to verbal memory in the context of an HIV infection. As the slope for the association between relationship conflict and verbal memory in the subsample of participants with an HIV infection was only approaching significance at $p = .062$. it may be that greater statistical power is required to detect more modest effects of relationship conflict on neurocognitive functioning.

In addition to being an overall significant predictor of verbal memory, HIV infection moderated the association between relationship conflict and verbal memory. As such, HIV infection interrupts the apparent protective mechanism of relationship conflict on verbal memory for individuals who are homeless or precariously housed. It has been previously found that individuals who are HIV-seropositive experience greater levels of loneliness, interpersonal conflict, and poor conflict management (Harris et al., 2020; Ninnoni et al., 2023; Straits-tröster et al., 1994), thereby providing supportive evidence of the detrimental effects of HIV on social connectedness. The current results extend these findings to the field of neurocognitive functioning and housing insecurity. Relationship conflict may not be a contributor to CR for individuals with an HIV infection experiencing unstable housing. HIV infection further served as a moderator of the relationship between available social network size and cognitive flexibility. Larger social network size does not predict better cognitive flexibility in the context of an HIV

infection. As smaller social networks, loneliness, and higher rates of stigma have been found in stably housed individuals with HIV (Yoo-Jeong et al., 2023), the results from the present study suggest that this has important implications for neurocognitive functioning in individuals experiencing unstable housing or homelessness. It should be noted that although these two significant interactions emerged, the simple slopes analyses comparing those with an HIV infection to those without HIV did not reach statistical significance for either interaction. Statistically significant differences may be detectable with a larger sample size in future analyses and may provide insight into why the current pattern of results was observed.

Neither psychotic disorder nor TBI were significant moderating clinical risk factors, although psychotic disorder significantly predicted verbal memory performance independent of social connectedness. This is consistent with prior research that psychotic illness is associated with neurocognitive dysfunction and rapid cognitive aging, specifically memory impairment, among individuals who are stably housed (Aleman et al., 1999; Fett et al., 2020; Sheffield et al., 2018). The present findings suggest that while psychotic disorder is not specifically involved in the associations between social connectedness and neurocognitive functioning in adults who are homeless or precariously housed, it remains a prominent clinical condition to consider with regards to their verbal memory abilities.

Significant associations were observed between demographic factors and neurocognitive functioning. Consistent with prior findings in the neurocognitive aging literature (Murman, 2015), age was a significant predictor of neurocognitive performance in all domains, with younger age being associated with better performance. The current results further establish years of education as a contributor to CR as a higher number of years of education significantly predicted greater global neurocognition, inhibitory control, sustained attention, and cognitive

flexibility, but not verbal memory performance. Cognitive flexibility was the only neurocognitive domain that was significantly associated with sex, with male participants demonstrating greater performance than female participants.

The sensitivity analyses revealed a largely similar pattern of results using a version of the dataset that underwent multiple imputation to account for missing data. However, there were diversions from the main analyses, which included the absence of a significant main effect of social network size and significant interaction between relationship conflict and HIV infection for verbal memory. As such, the missing data may have influenced the main results presented, highlighting the need for further research with a larger sample size and comprehensive dataset to solidify whether social network size predicts verbal memory and whether HIV infection moderates the association between relationship conflict and verbal memory. There was an additional main effect of HIV infection on global neurocognition and sustained attention, and a main effect of psychotic disorder on global neurocognition and cognitive flexibility which were observed in the sensitivity analysis. The emergence of new main effects is to be expected with the increase of statistical power that comes with multiple imputation, and thus, does not detract from the main results of the clinical risk factors. Overall, the results from the sensitivity analyses are to be acknowledged when interpreting the main results but are not cause for major concern as most observations remained consistent across both the imputed and non-imputed datasets.

4.2 Limitations & Future Directions

There are several limitations to consider as well as potential future areas of research that stem from the current study. First, the current analysis was cross-sectional. Therefore, whether the observed effects of social connectedness on neurocognitive functioning in this sample would hold beyond study baseline has yet to be ascertained. Although social connectedness has

longitudinal associations with neurocognitive functioning in domiciled individuals (Evans et al., 2019; Scholes & Liao, 2022; Sörman et al., 2017), it is unknown whether changes in social connectedness vary concurrently with neurocognitive functioning over the course of several years in individuals who are homeless or precariously housed. It is additionally unknown whether social connectedness predicts long-term neurocognitive functioning. Empirical approaches that investigate the effects of social connectedness past one timepoint would identify the presence of any longitudinal associations and substantiate the contributions of social connectedness to CR in adults experiencing homelessness and precarious housing. Directionality between social connectedness and neurocognitive functioning may be clarified in the process, as it remains unclear whether dimensions of social connectedness predict neurocognitive functioning or vice versa.

Second, the global social support and average relationship conflict variables consisted of broad measures of their constructs. Global social support provided a general overview of social support as a starting point for examining this particular dimension of social connectedness in this sample, but domain-specific effects may have been masked as a result. Examining each of the four forms of social support on the ISSB in relation to neurocognitive functioning would clarify whether there are more nuanced dimensions of social support that may act as protective factors for neurocognitive functioning in adults who are homeless or precariously housed. Furthermore, average relationship conflict consisted of a composite mean of interpersonal conflict with the participant's partner, relatives, and friends to account for the fact that some participants did not have that particular relationship in their lives. Consequently, there may have been effects of relationship conflict on neurocognitive functioning that could have been present when examining these discrete categories of relationships separately but were not observed when averaged

together. Moreover, it should be noted that despite efforts to collect comprehensive information on participants' social connectedness, the MAP did not fully capture the complex nature of relationship conflict for this population. If participants did not have a partner, friends, or relatives, they would not receive a score for that particular relationship. This would subsequently affect their score for average relationship conflict which was based on the available number of conflict scores. The possibility that heterogeneous data for the average relationship conflict score may have biased the current results should be considered. Future analyses should consider the use of alternative measures of relationship conflict and negative interpersonal interactions to provide a more robust and accurate predictor variable.

Third, the present study did not examine certain clinical risk factors overrepresented in individuals who are homeless or precariously housed. For one, dependence on substances such as stimulants, opioids, and alcohol is highly prevalent in this population and has been found to influence their neurocognitive functioning. Moreover, previous research has established relationships between substance use and a variety of dimensions of social connectedness in the context of unstable housing (S. M. Brown et al., 2020; Stanton et al., 2022; Wenzel et al., 2010, 2012). These substances have differential impacts and associations with social connectedness. For instance, youth experiencing homelessness who report having alcohol, cigarette, or cannabis users in their social network also reported greater use of these substances themselves (Wenzel et al., 2010); social norms in the networks of youth experiencing homelessness and the social network composition of adults experiencing unstable housing have been found to be associated with stimulant and opioid use (Barman-Adhikari et al., 2015; Bohnert et al., 2009); marriage and relationship quality have predicted lower frequency of stimulant and opioid use (Heinz et al., 2009); higher frequency of interactions with family has been associated with decreased

frequency of opioid use (Tseng et al., 2010); lower levels of social support predicted more frequent stimulant use (Leach et al., 2022); and smaller social networks and empathy impairments have been found to be prevalent in stimulant users relative to stimulant non-users (Preller et al., 2014). Overall, there is a wealth of literature to suggest that substance use plays an important role in the associations between neurocognitive functioning and social connectedness in stably housed populations. These effects may extend to neurocognitive functioning in individuals experiencing unstable housing, among whom multimorbid substance use is pervasive (Aldridge et al., 2018; Krausz et al., 2013; Schütz et al., 2019). Creating a general substance dependence variable which captured use of any category of substances was considered in the present study. However, given that different substances have distinct associations with neurocognitive functioning and social connectedness, this line of investigation was not pursued. Including each category of substance dependence in the hierarchical multiple regression models presented challenges as there would be an overwhelming number of clinical risk factors in the statistical analyses. Consequently, substance use was out of the scope of the study and was not examined, but merits attention in future analyses with a larger sample to detect any of its potential moderating effects. Moreover, mood, anxiety, and stress disorders such as major depressive disorder, social anxiety disorder, and post-traumatic stress disorder were not investigated in the current analyses. Previous research has found that these disorders have differential impacts, interactions, and associations in the context of social connectedness and neurocognitive functioning in individuals who are stably housed (Cardona & Andrés, 2023; Chen et al., 2024; Hayes et al., 2012; Y. Hwang et al., 2024; Shouse et al., 2013; Sippel et al., 2021). The extension of any potential moderating roles of these psychological disorders in the

relationship between social connectedness and neurocognition in populations experiencing unstable housing warrant future research.

Fourth, the potential influence of HIV status in the association between social connectedness and neurocognitive functioning merits further investigation. The present findings provide preliminary insight on its moderating role, but a greater depth of understanding is precluded by sample size limitations. Conducting additional analyses with a higher number of HIV-seropositive individuals experiencing unstable housing would assist in clarifying how the illness impacts the association between social connectedness and neurocognitive functioning.

Fifth, although sex was included as a covariate in the hierarchical regression models, neither sex- nor gender-based analyses were pursued in the current study. As females consisted of only 25% of participants, they were not adequately represented in the sample and there was not sufficient statistical power to report results using sex as a moderating variable of the associations between social connectedness and neurocognitive functioning. However, sex and gender remain an important avenue to explore in this marginalized population as it pertains to their social connectedness and neurocognitive performance. Previous studies have suggested that there are meaningful differences in patterns of social connectedness between stably housed males and females due to socially prescribed norms and roles related to gender identity (Caetano et al., 2013; Lee & Robbins, 2000). These differences emerge across a variety of dimensions of social connectedness. For instance, certain studies have found that females across all age groups report higher levels of loneliness than males do (Kiely et al., 2021; Nicolaisen & Thorsen, 2014). However, others have suggested that being male presents a risk factor for loneliness (Hutten et al., 2022) and that living with another individual has a stronger protective effect against loneliness for males than it does for females (Altschul et al., 2021; Hansen & Slagsvold, 2016).

The effects of sex and gender on social connectedness can additionally stem from differences in life experience. A variety of life events have been found to have distinct implications for male and female loneliness, with female older adults' loneliness being related to economic problems in their childhood, and male older adults' loneliness being linked to a history of parental conflict and having been bullied (Nicolaisen & Thorsen, 2014). Furthermore, sex-based research on social connectedness has traditionally supported the notion that females of all ages are more likely to seek, receive, and provide social support than males (Bedrov & Gable, 2023), and experience more physical and mental health benefits from supportive social networks than males do (Shin & Park, 2023). In contrast, males tend to value to instrumental over emotional support (Fiori & Jager, 2012). However, qualitative research has suggested that social support among males is diverse, and that some actively seek emotional support from their social networks while others prefer social independence (McKenzie et al., 2018; Pereira et al., 2024). Social connectedness has additionally been found to have differential associations with neurocognitive functioning based on sex, where males with low social connectedness and females with strong social ties to a large network of friends had a higher risk of dementia (Htun et al., 2024). Overall, sex- and gender-based analyses on social connectedness and neurocognitive functioning are complex and worth exploring further in the context of unstable housing as this has not yet been examined. Future studies should consider recruiting a balanced, sex-representative sample to thoroughly investigate the influence of sex on the associations between social connectedness and neurocognitive functioning in individuals who are homeless or precariously housed.

Sixth, baseline assessments were conducted across the span of several years due to distinct waves of targeted recruitment from various community sources, which included single room-occupancy hotels, a community courthouse, and the emergency department at a local

hospital. There may have been differences in cohorts that influenced the associations between social connectedness and neurocognitive functioning. For instance, it is possible that the cohort of young adult participants recruited from youth-supported single-room occupancy hotels differ in their levels of social connectedness relative to the cohort recruited through the emergency department. The heterogeneity of the sample resulting from multiple cohorts should be considered when interpreting the findings of the present study.

Lastly, the current results are specific to the Hotel Study sample in Vancouver, Canada, and thus may not be generalizable to other samples of populations experiencing homelessness or precarious housing in other cities or healthcare systems. Future clinical research on homelessness and precarious housing may consider replicating the current findings along with exploring new social dimensions, neurocognitive domains, and clinical risk factors, and in doing so, contribute to supportive evidence of the role that social connectedness plays in enhancing CR in this marginalized population.

4.3 Strengths & Implications

The present study had several strengths. Notably, to our knowledge, it is the first study to provide initial evidence as to whether social connectedness can be considered a protective factor for neurocognitive health in individuals who are homeless or precariously housed. This topic has been more thoroughly investigated in the context of cognitive aging (Evans et al., 2019; Fratiglioni et al., 2004, 2020; Nicholson, 2012; Pillemer & Holtzer, 2016; Reblin & Uchino, 2008; Zhang et al., 2021; Zhu et al., 2012), but the current study extends our knowledge of social connectedness and neurocognitive functioning to a population with complex needs that faces consistent social marginalization and high rates of neurocognitive impairment. Clinical research conducted on individuals who are unstably housed often centers on a deficits-based approach,

focusing on risk factors for neurocognitive impairment rather than strengths at the individual-level that can be leveraged to maintain neurocognitive health. In the face of physical and mental health conditions and socioeconomic challenges, this population's ability to maintain personal community and meaningful interpersonal connections serves as a marker of resilience that also contributes to CR. The results of the present study lay the initial groundwork for future investigations on the benefits of social connectedness for individuals who are homeless or precariously housed, specifically with regards to their neurocognitive health.

The current findings suggest that there are important associations between larger social network sizes and greater verbal memory performance. This highlights the importance of implementing social and community-based interventions to help build CR, preserve neurocognitive functioning, and maintain functional outcomes. Indeed, community integration and social participation have been identified as important factors in improving the quality of life of people experiencing homeless or precarious housing (Gaetz, 2016; Rutenfrans-Stupar et al., 2019). Studies led among individuals experiencing homelessness or precarious housing have found that such interventions have positive impacts on their overall health and social integration. For example, a 'growth through participation' program conducted among Dutch adults living in a shelter facility targeted the development of social skills via social interaction with one another, the identification of strengths and talents, and a structured recovery plan to achieve stable housing (Rutenfrans-Stupar et al., 2020). The intervention was associated with a perceived increase of quality of life, which included the quality of their social relationships. A study on the impact of an arts community program for adults experiencing homelessness in Australia found that it provided an entry point for social participation and inclusion, served as an alternative to health-adverse activities such as substance use, and had a positive effect on participants' sense of

identity (Thomas et al., 2011). An intervention targeting the improvement of physical activity levels for individuals experiencing unstable housing in Scotland found that participation remained high as it allowed for positive social interaction and increased social support (Malden et al., 2019). As these interventions either directly or indirectly target dimensions of social connectedness which contribute to CR, their implementation in communities of individuals experiencing unstable housing in other major cities may be particularly beneficial in maintaining their neurocognitive health.

There are additional implications for the current findings that suggest that the presence of an HIV infection disrupts the protective nature of relationship conflict on verbal memory, and social network size on cognitive flexibility. It may be particularly important to consider multidimensional social interventions that benefit individuals experiencing homelessness and living with HIV, as this subgroup experiences elevated rates of dual stigma related to both their illness and housing status (Davila et al., 2018). Examples of such interventions include one which targeted the negative effects of HIV stigma through care coordination and the establishment of trusting relationships. This was found to promote social functioning and overall health in individuals with HIV who were homeless (Maskay et al., 2018). Mobilizing existing social networks to care for domiciled individuals with HIV in rural Kenya has been effective in reducing stigma and improving their sense of social support (Salmen et al., 2015). Psychosocial interventions such as symptom-oriented cognitive behavioural therapy, social support, and meditation have been posited to have small positive effects for individuals who are stably housed and living with HIV (Bhochhibhoya et al., 2021). Such interventions may be expanded upon and translated to populations living with HIV and experiencing homelessness or precarious housing

in a Canadian context. There are opportunities for future research with regards to the impact of these social-oriented programs on neurocognitive health in this vulnerable population.

4.4 Conclusion

The present findings shed light on the potential for dimensions of social connectedness to contribute to CR in adults experiencing homelessness or precarious housing. Larger social network size was found to predict greater verbal memory. The associations between social connectedness and neurocognitive functioning were unique among HIV-seropositive participants, as HIV infection influenced the effect of relationship conflict on verbal memory as well as the effect of social network size on cognitive flexibility. Neither social support nor relationship conflict independently predicted any domains of neurocognitive functioning. Likewise, neither TBI nor psychotic disorder significantly moderated any associations between the dimensions of social connectedness and neurocognitive domains. The current study underscores the importance of considering protective, modifiable social factors that promote neurocognitive health in this marginalized population that experiences high rates of social isolation and neurocognitive dysfunction. To our knowledge, it is the first to have examined dimensions of social connectedness in relation to neurocognitive functioning in the context of unstable housing. Additional research is warranted to investigate whether the current results can be replicated using longitudinal methods and elucidate whether dimensions of social connectedness have long-term protective effects on neurocognitive functioning. Future studies with larger sample sizes of HIV-seropositive individuals may also provide clarification on how HIV infection dysregulates the associations between social connectedness and neurocognitive health. Such research would lead to a more comprehensive understanding of the relationship between this population’s interpersonal and neurocognitive functioning. This may in turn inform

the implementation of social interventions that can facilitate contributions to CR, the preservation of their neurocognitive health, and maintenance of functional outcomes as a result.

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Appendix A: Sensitivity Analysis Results

Table A1

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Global Neurocognition Using Imputed Dataset

Global Neurocognition (*n* = 398)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.16	-	-	< .001***
Age	-0.02	0.00	-7.06	< .001***	[-0.03, -0.02]				
Sex	-0.04	0.08	-0.46	.644	[-0.20, 0.12]				
Education	0.07	0.01	5.16	< .001***	[0.05, 0.10]				
Available social network size	0.02	0.01	1.85	.065	[-0.00, 0.04]				
Global social support	-0.00	0.00	-0.50	.619	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.33	.744	[-0.00, 0.00]				
Model 2: Clinical risk factors						0.18	-0.03	4.88	.002**
Age	-0.02	0.00	-7.13	< .001***	[-0.03, -0.02]				
Sex	-0.02	0.08	-0.26	.798	[-0.18, 0.14]				
Education	0.07	0.01	5.05	< .001***	[0.04, 0.10]				
Available social network size	0.02	0.01	1.76	.079	[-0.00, 0.04]				
Global social support	-0.00	0.00	-0.46	.648	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-0.51	.609	[-0.00, 0.00]				
HIV	-0.23	0.09	-2.55	.011*	[-0.40, -0.05]				
TBI	0.06	0.07	0.99	.323	[-0.06, 0.19]				
Psychotic disorder	-0.17	0.07	-2.60	.010**	[-0.30, -0.04]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
*p < .05. **p < .01. ***p < .001.

Table A2

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Verbal Memory Using Imputed Dataset

Verbal Memory (*n* = 398)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.08	-	-	< .001***
Age	-0.07	0.01	-5.13	< .001***	[-0.10, -0.04]				
Gender	0.24	0.36	0.66	.511	[-0.47, 0.95]				
Education	0.23	0.06	3.66	< .001***	[0.11, 0.35]				
Available social network size	0.07	0.05	1.45	.148	[-0.03, 0.18]				
Global social support	-0.00	0.00	-0.89	.374	[-0.01, 0.01]				
Average relationship conflict	0.00	0.01	0.73	.465	[-0.01, 0.02]				
Model 2: Clinical risk factors						0.11	0.03	4.85	.003**
Age	-0.07	0.01	-5.11	< .001***	[-0.10, -0.04]				
Gender	0.31	0.36	0.87	.386	[-0.39, 1.02]				
Education	0.22	0.06	3.56	< .001***	[0.10, 0.34]				
Available social network size	0.07	0.05	1.35	.179	[-0.03, 0.17]				
Global social support	-0.00	0.00	-0.83	.406	[-0.01, 0.01]				
Average relationship conflict	0.00	0.01	0.55	.586	[-0.01, 0.01]				
HIV	-1.13	0.39	-2.89	.004**	[-1.91, -0.36]				
TBI	0.20	0.29	0.70	.482	[-0.37, 0.77]				
Psychotic disorder	-0.67	0.29	-2.30	.022*	[-1.24, -0.10]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
*p < .05. **p < .01. ***p < .001.

Table A3

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Inhibitory Control Using Imputed Dataset

Inhibitory Control (*n* = 398)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.08	-	-	< .001***
Age	-0.27	0.05	-5.73	< .001***	[-0.37, -0.18]				
Gender	1.05	1.28	0.82	.412	[-1.47, 3.57]				
Education	1.01	0.22	4.53	< .001***	[0.57, 1.44]				
Available social network size	0.27	0.18	1.50	.135	[-0.09, 0.63]				
Global social support	-0.00	0.02	-0.23	.817	[-0.04, 0.03]				
Average relationship conflict	-0.01	0.02	-0.68	.496	[-0.05, 0.03]				
Model 2: Clinical risk factors						0.13	0.02	2.62	.051
Age	-0.29	0.05	-5.84	< .001***	[-0.38, -0.19]				
Gender	1.30	1.28	1.01	.312	[-1.22, 3.82]				
Education	0.97	0.22	4.38	< .001***	[0.53, 1.41]				
Available social network size	0.26	0.18	1.42	.156	[-0.10, 0.62]				
Global social support	-0.00	0.02	-0.21	.831	[-0.04, 0.03]				
Average relationship conflict	-0.02	0.02	-0.81	.419	[-0.05, 0.02]				
HIV	-2.07	1.40	-1.47	.141	[-4.83, 0.69]				
TBI	1.36	1.04	1.31	.192	[-0.69, 3.40]				
Psychotic disorder	-2.02	1.04	-1.94	.053	[-4.07, 0.02]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
$p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Table A4

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Sustained Attention Using Imputed Dataset

Sustained Attention (n = 398)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.03	-	-	.006**
Age	-0.00	0.00	-2.40	.017*	[-0.00, -0.00]				
Gender	0.01	0.01	0.88	.380	[-0.01, 0.02]				
Education	0.00	0.00	3.41	.001***	[0.00, 0.01]				
Available social network size	0.00	0.00	1.05	.293	[-0.00, 0.00]				
Global social support	-0.00	0.00	-0.62	.536	[-0.00, 0.00]				
Average relationship conflict	0.00	0.00	0.28	.783	[-0.00, 0.00]				
Model 2: Clinical risk factors						0.04	0.02	2.11	.098
Age	-0.00	0.00	-2.10	.037*	[-0.00, -0.00]				
Gender	0.01	0.01	0.94	.349	[-0.01, 0.02]				
Education	0.00	0.00	3.42	.001***	[0.00, 0.01]				
Available social network size	0.00	0.00	0.98	.327	[-0.00, 0.00]				
Global social support	-0.00	0.00	-0.54	.588	[-0.00, 0.00]				
Average relationship conflict	0.00	0.00	0.15	.881	[-0.00, 0.00]				
HIV	-0.02	0.01	-2.31	.021*	[-0.04, -0.00]				
TBI	-0.00	0.01	-0.55	.580	[-0.02, 0.01]				
Psychotic disorder	-0.01	0.01	-0.79	.428	[-0.02, 0.01]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
$p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Table A5

Hierarchical Multiple Regression Models Examining the Associations Between Social Connectedness and Cognitive Flexibility Using Imputed Dataset

Cognitive Flexibility (n = 398)									
Term	β	*SE*	*t*	*p*	95% CI	R^2	ΔR^2	ΔF	*p*
Model 1: Demographic and social connectedness variables						0.13	-	-	< .001***
Age	-0.02	0.00	-6.49	< .001***	[-0.03, -0.02]				
Gender	-0.38	0.10	-3.71	< .001***	[-0.58, -0.18]				
Education	0.05	0.02	2.76	.006**	[0.01, 0.08]				
Available social network size	0.02	0.01	1.16	.248	[-0.01, 0.05]				
Global social support	0.00	0.00	0.40	.692	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-1.31	.191	[-0.01, 0.00]				
Model 2: Clinical risk factors						0.14	0.01	2.22	.086
Age	-0.03	0.00	-6.79	< .001***	[-0.03, -0.02]				
Gender	-0.37	0.10	-3.58	< .001***	[-0.57, -0.17]				
Education	0.05	0.02	2.60	.010**	[0.01, 0.08]				
Available social network size	0.02	0.01	1.13	.259	[-0.01, 0.05]				
Global social support	0.00	0.00	0.36	.721	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-1.36	.174	[-0.01, 0.00]				
HIV	-0.03	0.11	-0.30	.762	[-0.26, 0.19]				
TBI	0.11	0.08	1.35	.177	[-0.05, 0.28]				
Psychotic disorder	-0.18	0.08	-2.16	.031*	[-0.35, -0.02]				
Model 3: Interaction between available social network size and HIV						0.15	0.01	3.96	.047*
Age	-0.03	0.00	-6.70	< .001***	[-0.03, -0.02]				
Gender	-0.36	0.10	-3.50	.001***	[-0.56, -0.16]				
Education	0.05	0.02	2.54	.011*	[0.01, 0.08]				
Available social network size	0.03	0.02	1.72	.086	[-0.00, 0.06]				
Global social support	0.00	0.00	0.28	.779	[-0.00, 0.00]				
Average relationship conflict	-0.00	0.00	-1.35	.177	[-0.01, 0.00]				
HIV	-0.05	0.11	-0.48	.632	[-0.28, 0.17]				
TBI	0.12	0.08	1.48	.140	[-0.04, 0.29]				
Psychotic disorder	-0.17	0.08	-2.08	.038*	[-0.34, -0.01]				
Available social network size x HIV	-0.09	0.05	-1.99	.047*	[-0.18, -0.00]				

Note. β = Standardized regression coefficient. *SE* = Standard error of the mean. CI = Confidence intervals. TBI = Traumatic brain injury.
$p < .05$. $^{**}p < .01$. $^{***}p < .001$.