SOCIAL DETERMINANTS AND BIOSOCIAL CONSEQUENCES OF DEPRESSIVE SYMPTOMS: ANALYZING SOCIAL CAPITAL, DEPRESSION, AND COGNITION IN LATER LIFE

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ABSTRACT

This dissertation explores the complex relationship between social capital and depressive symptoms across the life course, focusing particularly on the increasing prevalence of depression from mid-to-late life. Using a life course perspective, this research investigates how social determinants, such as social capital, shape the emergence, increase, and decrease of depressive symptoms as individuals age. Drawing from both the tangible and psychological dimensions of social capital, this dissertation examines how changes in social connections and networks influence depressive symptom outcomes, including the biosocial consequences of depression on cognitive function in later life. The research is divided into three analytical papers. The first paper analyzes longitudinal data to assess the association between social capital and depressive symptoms, distinguishing between within- and between-person effects. The second paper explores how depressive symptom subtypes evolve from midlife to later life, identifying distinct subtypes and examining the stability and transitions between them over time. The third paper investigates the relationship between depressive symptom subtypes, social capital, and cognitive function, exploring how depressive symptom subtypes may mediate this

association. Findings across these studies emphasize the pivotal role of social capital in shaping depression outcomes, highlighting how social isolation and disconnection may exacerbate depressive symptoms in later life. This dissertation contributes to the sociology of mental health and aging by offering new insights into the social mechanisms underlying depression and its long-term impacts on cognitive function. Through this work, policymakers and health professionals may gain a deeper understanding of how targeted interventions aimed at enhancing social capital could mitigate the global burden of depression in aging populations.

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CHAPTER 1: INTRODUCTION

As the leading cause of disability worldwide, depression is a major contributor to the overall global burden of disease ("WHO | Depression," 2019). With more than 246 million people affected worldwide ("WHO | Depression," 2019), depression exists as both a private, or individual problem, as well as a public issue deserving of sociological investigation. As C. Wright Mills (1959) outlined in *The Sociological Imagination,* social scientists aim to translate private troubles into public issues. Specifically, the sociological imagination discusses the relation between self (e.g., private troubles) and society (e.g., public issues) – or more simply put, a framework for how to examine the relationship between society and individual experiences (Mills, 1959). Thus, a sociologist would not view depression from a biological or individualistic perspective but rather would use the sociological imagination to acknowledge how private troubles may result in an individual being diagnosed with a mental illness. For example, an individual experiencing high levels of stressful life events may be more likely to be diagnosed with depression. However, stopping there only acknowledges the individual experience of depression – the stressful life events – but fails to connect this to society. In this example, the sociological imagination would also point to the common larger societal processes and conditions that impact all those living with a mental illness (Clarke et al., 2011; Fitzgerald et al., 2016; Karp, 1997; Manning, 2019; Warner, 2009).

Scholars contributing to the sociology of mental health have used the sociological imagination to analyze and pinpoint how social structures and relationships are associated with poor mental health. While sociologists examining mental health have made significant strides in understanding the social structures impacting mental health (Allen et

al., 2014; Barrett & Turner, 2005; Brown, 2002; Roth, 2020; Sinkewicz et al., 2022; Williams, 2018; Wong & Waite, 2015), there still remain many unanswered questions, especially when focusing on specific types of mental illness, such as depression across the life course.

At present, the United States ranks as the third most "depressed" country in the world, with an estimated one in every six Americans diagnosed with clinical depression in their lifetime ("WHO | Depressive symptoms," 2019). This high prevalence of clinical depression, as well as undiagnosed depression and subclinical depressive symptoms, is an increasingly important public health issue when considering the disabling components of the illness (Liu et al., 2020; Moussavi et al., 2007; Yu et al., 2020). Beyond the disabling components of depression, in the most dire circumstances, depression can lead to suicide (Ferrari et al., 2013; Mueller & Abrutyn, 2016; Stanley et al., 2016). It is also a predictor of physical disability (Prince et al., 2007), cognitive decline in older adults (Brailean et al., 2017; Dlugaj et al., 2015; Graziane et al., 2016; van den Kommer et al., 2013), and lifelong mental health problems (Fergusson et al., 2005; Johnson et al., 2018; Naicker et al., 2013). As such, despite the individual lived experience of depression, which makes this illness a private trouble for millions, the global rise of the illness and the social consequences of living with this mental illness have transformed depression into a leading contributor to the global burden of disease, or rather, a public issue (Ferrari et al., 2013; Karp, 1997; Liu et al., 2020).

The overarching aim of this dissertation is to investigate depressive symptoms as an embodied consequence of social troubles, or disadvantages, accumulated over the life course and why there is an increase in depressive symptoms from mid-to-late life for

aging adults. Specifically, this dissertation will examine the accumulation of social capital, in three separate but related papers which will be discussed more in-depth later in this introduction. While this dissertation does not investigate the lived experiences or illness narratives of those living with depression, these narratives have informed this dissertation through examining the key social factors that are common among those living with depression. It is important to first discuss my reasons for focusing on social capital as a major contributor to depressive symptoms in later life.

In David Karp's pivotal work, *Speaking of Sadness: Depression, Disconnection, and the Meaning of Illness*, Karp describes depression as "an illness of isolation" (1997: pp. 7). After analyzing over 100+ narratives of individuals living with depression as well as living with depression himself, Karp reports a link between depression and feelings of social isolation. Karp describes this social isolation as both a *cause* and a *symptom* of depression. By *cause* and *symptom*, Karp refers to the idea that feeling socially isolated can bring about depressive symptoms (e.g., a cause), while those living with depression also tend to isolate themselves, whether intentionally or unintentionally (e.g., a symptom). As such, this pivotal work has sparked my interest in my aim to understand what brings about these feelings of isolation that amount to experiencing depressive symptoms, specifically for aging adults.

I hypothesize that these depressive symptoms may be a response to an accumulation of disadvantages which result in an individual feeling isolated and thus, depressed. A number of sociologists have expanded upon studying depression by approaching it from this collective standpoint in an effort to understand the social environmental causes associated with increases in depressive symptoms (Assari, 2017;

Brown, 2002; Sinkewicz et al., 2022). Prior studies have examined social determinants such as educational attainment (Bjelland et al., 2008; J. Lee, 2011), income (Miech & Shanahan, 2000; Zimmerman & Katon, 2005), gender (Rodgers et al., 2014; Ross & Mirowsky, 2006) and how each of these social factors impact depressive symptoms. Another common theme among these scholars is the association between depressive symptoms and age (Abrams & Mehta, 2019; Chi & Chou, 2000; Clarke et al., 2011; Forsman et al., 2012; Mirowsky & Ross, 1992; Yang, 2007). Some of these scholars have suggested that a curvilinear relationship exists between age and depressive symptoms, where symptoms are high in adolescence then are lowest during middle age, but once again rise as the individual ages into later life (Miech & Shanahan, 2000; Mirowsky & Ross, 1992; Sutin et al., 2013; Tampubolon & Maharani, 2017; Yang, 2007). While this curvilinear relationship has been well-documented, less research explains *why* this curvilinear relationship exists. Prior quantitative studies in sociology that have attempted to explain this relationship have typically focused on socioeconomic status, most commonly measured as income, which is typically highest in midlife and declines throughout later life (Miech & Shanahan, 2000; Mirowsky & Ross, 2001; Xue et al., 2021). Scholars have primarily focused on socioeconomic status because this was an indicator of independence, which has been shown to be closely related to depressive symptoms (Miech & Shanahan, 2000; Sinkewicz et al., 2022; Xue et al., 2021). However, when examining the qualitative literature about those living with depression, scholars are not reporting respondents focusing on their own socioeconomic status, but rather on their losses of social connection (Haroz et al., 2017; Karp, 1994, 1997; Kokanovic et al., 2013; Kotliar, 2016). As such, there appears to be a disconnect between these qualitative and

quantitative findings. I want to look at social connection, but quantitatively, to better understand this rise in depressive symptoms from mid-to-later life.

Depression is not only found in later life, but also common throughout the life course (Achterbergh et al., 2020). Therefore, depression is not framed as an illness of age despite this finding that depressive symptoms increase from mid-to-late life (Miech & Shanahan, 2000; Mirowsky & Ross, 1992; Tampubolon & Maharani, 2017; Yang, 2007). Thus, there appear to be social factors associated with depression, specifically in later life, that must be contributing to this increased risk in depressive symptoms after midlife. Based upon the illness narratives of those living with depression, one of these social factors is associated with social connections that seem to decline, on average, as adults age (Haroz et al., 2017; Karp, 1997; Kokanovic et al., 2013). In this dissertation, I will focus on depression in later life and social capital – the social determinant I hypothesize as contributing to this increase in depression for older adults.

This dissertation will contribute to the current research in the sociology of mental health and the sociology of aging, by examining the two dimensions of social capital as social determinants of depression as well as how these two dimensions influence subtypes of depressive symptoms that may exist as individuals age from mid-to-late life. Through these chapters I aim to understand how social capital may influence this relationship for aging adults when levels of dependency on social others typically increases (Cannuscio et al., 2003). Furthermore, I analyze the biosocial repercussions of living with depression in mid-to-late life on later life cognition. While scholars have investigated the association between depression and cognition (Beaujean et al., 2013; Brailean et al., 2017; Butters et al., 2008), less research has examined how other social

determinants may contribute to their association. This dissertation will expand upon this literature by analyzing how the accumulation, or loss, of social capital may impact the association between depression and cognition in later life.

Chapter two, the first of three empirical studies, examines the association between two dimensions of social capital, structural (e.g., tangible) and cognitive (e.g., psychological), and depressive symptoms, as well as investigating their within- and between persons effects. Due to the multifaceted nature of social capital, it is imperative to include both dimensions in an analysis as these dimensions and their pathways are interconnected rather than mutually exclusive (Bassett & Moore, 2013; Forsman et al., 2012; Nakamine et al., 2017). These two dimensions, structural and cognitive, address the tangible resources one can garner from their social networks as well as the psychological aspects of having social networks (Cohen-Cline et al., 2018; Forsman et al., 2012; Hamano et al., 2010). Including both dimensions in the same analysis will potentially identify specific factors to target for change, providing policymakers with key data to construct policies to reduce depressive symptoms. However, previous research has largely examined these dimensions separately, which inaccurately suggests they are independent of one another (Kawachi & Berkman, 2001; Webber et al., 2011). Furthermore, previous studies that included both dimensions of social capital used different numbers of variables to represent these dimensions. For example, studies including structural social capital often focus on singular variables such as civic engagement (Hamano et al., 2010) or social network size (Forsman et al., 2012) to explain the pathway to improved mental health outcomes. This narrow scope may contribute to the inconsistent findings in the literature (Ehsan & De Silva, 2015). Unlike

studies examining structural social capital, many researchers measuring cognitive social capital have used numerous variables to measure the concept. Yet, scholars examining depressive symptoms and cognitive social capital have also reported inconsistent results, depending on exactly which social capital variables were included in the analysis (Fujiwara & Kawachi, 2008; Harpham et al., 2002).

Moreover, longitudinal studies are pivotal to understanding how social capital affects the change an individual experiences over time (Curran & Bauer, 2011). While prior longitudinal research provides valuable insights, I am aware of only four studies examining depressive symptoms and both dimensions of social capital (Cohen-Cline et al., 2018; Kim et al., 2012; Landstedt et al., 2016; Nakamine et al., 2017). Of these four longitudinal studies, only one of them examines differences between persons versus intraindividual change over time (Cohen-Cline et al., 2018). The first study aims to address these gaps in the current literature by examining both between-person and within-person changes and analyzing the association between social capital and depressive symptoms at individual and collective levels.

For this first study, as well as the other two empirical studies in this dissertation, I use data from the Wisconsin Longitudinal Study (WLS). The WLS is a comprehensive, cohort study that began in 1957 with a random sample of one-third of the Wisconsin high school graduating class (N = 10,317). Following the initial survey in participants' senior year, the WLS conducted follow-up assessments in 1964, 1975, 1992, 2004, 2011, and 2020, collecting extensive data on participants' social, economic, and psychological well-being across decades (Herd et al., 2014). For this dissertation, the WLS provides a robust set of data to examine how social capital impacts depressive symptoms over time within

an aging cohort. The WLS's longitudinal structure makes it possible to analyze both between-person differences and within-person changes in depressive symptoms, addressing key gaps in prior research that have primarily relied on single-wave or cross-sectional analyses, often overlooking the dynamic interactions between social capital and mental health across the life course (Cohen-Cline et al., 2018; Kim et al., 2012).

In this chapter I conducted a multi-level 2-wave longitudinal analysis using Stata version 14.2 to examine both within- and between-person variance in depressive symptoms (Snidjers & Bosker, 1999). The models utilized logged Center for Epidemiological Studies- Depression (CES-D) as the outcome variable with time represented by age centered at 65, incorporating random intercepts for each participant and fixed slopes in two-wave models. Model I examined age as an independent variable, Model II included structural social capital variables and covariates, Model III contained cognitive social capital variables and covariates, and Model IV integrated both structural and cognitive social capital variables along with covariates. Model V incorporated structural and cognitive social capital variables, covariates, and assessed differences between within- and between-person effects for social capital variables. I performed imputation for missing data, with 70% of cases having complete data and 95% providing valid responses to more than half of the variables. Multiple imputation by chained equations was performed for each wave, with regression results combining estimates from thirty imputed datasets using Rubin's rules (Bodner, 2008; Rubin, 1987).

In the third chapter I investigate how social capital is linked to different subtypes of depressive symptoms in older adults. This analysis is crucial because, while social capital is known to affect depression broadly (Forsman et al., 2012; Landstedt et al.,

2016), its specific impact on different clusters of depressive symptoms remains underexplored. The present study fills this gap by examining how social capital might exacerbate or mitigate certain types of depressive symptoms rather than treating depression as a homogenous illness. Moreover, this study will build on previous literature that has mostly focused on social determinants of mental health in a general sense, lacking a nuanced understanding of how specific social factors, such as social capital, influence the heterogeneity of depression (Mezuk & Kendler, 2012; Singham et al., 2022). My research contributes to the growing field of sociology of mental health by identifying possible social mechanisms that may impact specific depressive symptom clusters in later life, offering potential avenues for targeted interventions.

The Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), defines depression as Major Depressive Disorder (MDD) with various specifiers that have specific features such as anxious distress, but it treats depression as a homogenous illness despite potential differences in symptom presentation across age groups (American Psychological Association, 2013). While traditional diagnostic criteria rely on symptom counts, research suggests that depressive symptoms may manifest differently in older adults compared to younger adults, highlighting the need for a nuanced approach to diagnosis and intervention. For example, Gallo and colleagues (1994) found that older adults tend to experience depression without sadness (i.e., somatic symptoms), while middle-aged individuals experience depression with sadness (i.e., a mix of somatic and depressed affect symptoms). Similarly, Hybels and colleagues (2011) argued that the DSM diagnosis of MDD only requires a symptom count of five or more symptoms during

the same two week period that are a change from previous functioning, potentially overlooking age-specific symptom patterns.

Using statistical methods such as latent class analysis (LCA) and latent transition analysis (LTA), researchers have identified distinct subtypes of depression based on common symptom patterns. Hybels and colleagues (2013) used LCA to identify three classes of depressive symptoms in older adults, including a class endorsing no symptoms, another endorsing negative affect and somatic symptoms, and a third endorsing negative affect, somatic symptoms, and low positive affect symptoms. Similarly, Lee et al. (2012) and Veltman et al. (2017) reported similar findings with a 3-class solution. However, inconsistencies exist in these models, likely due to variations in samples, scales used, and inclusion criteria. Furthermore, longitudinal studies using LTA have revealed the heterogeneity of depression experience over time, with some individuals transitioning between symptom classes as they age (Lamers et al., 2012; Rodgers et al., 2014; Ulbricht et al., 2018). By analyzing these depressive symptom subtypes and the impact of covariates, including tangible social capital measures, I aim to provide empirical evidence for possible interventions targeting specific symptoms and social factors associated with depression in later life.

The analysis in chapter three employed a three-step LTA approach using MPlus version 5 (Asparouhov & Muthén, 2014; Collins & Lanza, 2010). Initially, LCA was conducted at each time point to identify latent groups based on respondents' observations of several variables, referred to as latent statuses in this study to account for potential temporary class membership changes (Collins & Lanza, 2010; Vermunt, 2010). Following the identification of the best fitting LCA models, measurement invariance

between them was assessed to determine if depressive symptom classes remained consistent across a 19-year period. Finally, an LTA model estimated the probabilities of individuals transitioning or retaining their status between the two time points. MPlus provided probabilities for each individual's classification, which were then used in Stata to conduct multinomial logistic regression, considering covariates such as tangible social capital, respondents sex, education, total assets, self-rated health, and polygenic score for depression, to explore the influence of these factors on baseline depression status and transitions over time.

In the final analytical chapter, I examine how social capital and depressive symptoms impact cognitive ability in later life. Over the past two decades, scholars have investigated the association between depression and cognitive decline to determine if depression is a risk factor but have reported inconsistent results (Beaujean et al., 2013; Brailean et al., 2017; Butters et al., 2008; Sachs-Ericsson et al., 2005; van den Kommer et al., 2013). Yet, some studies have shown that cognitive abilities improve when depression is treated (Beaujean et al., 2013; van den Kommer et al., 2013). As such, it is pivotal to examine how depressive symptoms impacts cognition in late life. More recently scholars have begun to investigate early life social factors and their impact on cognition in later life (Greenfield & Moorman, 2019; Moorman et al., 2018). Yet, research has been limited in understanding factors impacting depression over the life course and its effect on cognition. Prior research has examined depression in later life and its impact on cognition without examining how other social factors may be influencing this association (Beaujean et al., 2013; Brailean et al., 2017; Butters et al., 2008). Furthermore, this chapter builds on the research from Chapter 3 by using the depressive

symptom subtypes to examine if certain clusters of symptoms are associated with cognitive function in later life.

In this study, I used multiple regression analysis to investigate the relationship between cognitive function and various factors including depressive symptom subtypes, tangible social capital, and psychological social capital. Multiple regression offers a robust method to examine these relationships, considering the interplay of numerous factors influencing cognitive functioning in later life (Allison, 1999). Moreover, it allows for the exploration of potential mediating effects. Following Karlson, Holm, and Breen (KHB) method for mediation analysis, I assessed the statistical significance of associations between (1) the independent variable (IV) and dependent variable (DV), (2) the IV and the mediation variable (M), (3) the M and DV, and finally (4) a multiple regression model with IV and M predicting the DV (Breen et al., 2021). The KHB approach offers a more precise distinction between direct and indirect effects, enabling the quantification of how much of the independent variable's impact on the dependent variable is mediated and providing a formal test for statistical significance. Thus, the study initially conducted multiple regression analysis and subsequently tested for mediation, where social capital variables served as the independent variable (IV), depression as the mediation variable, and cognitive function as the dependent variable (DV).

In the final chapter, I summarize the findings from the three empirical chapters and discuss their implications for examining the interconnections between depressive symptoms and the two dimensions of social capital. Findings from this dissertation emphasize the need to understand depression as a heterogenous illness where different

clusters of symptoms represent different types of depression. Furthermore, the findings advance the literature on the sociology of mental health and aging to better understand the curvilinear nature of depressive symptoms and later life and structural causes that may impact individual's depression. Additionally in this chapter, I outline some limitations of the research presented in this dissertation and discuss how these findings inform opportunities for future research. Overall, this dissertation seeks to emphasize the pivotal importance of social capital as a plausible area of intervention for policymakers to aid in these increasing levels of depression from mid-to-late life.

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CHAPTER 2: A COHORT LONGITUDINAL STUDY OF INDIVIDUAL LEVEL SOCIAL CAPITAL AND DEPRESSIVE SYMPTOMS IN THE WISCONSIN LONGITUDINAL STUDY

ABSTRACT

This study examined the association between two dimensions of social capital, structural and cognitive, and depression, as well as investigating their within-person and between-person effects. Using the Wisconsin Longitudinal Study, I applied a multi-level 2-wave longitudinal analysis, over a 19-year period, to examine the effects of these two dimensions of social capital influence on individuals' depressive symptoms at both the between- and within-person levels. Results suggest both dimensions of social capital are negatively related with levels of depressive symptoms. The within-person changes for both self-efficacy and sense of belonging were larger than the estimates of between-effects, while trust and structural social capital effects were equal. These findings add to the growing body of literature examining depressive symptoms in late life, while also providing evidence for policymakers to hone in on key areas that can address depressive symptoms with social capital interventions.

INTRODUCTION

Despite much previous research that demonstrates a clear association between social capital and mental health, numerous questions remain about the mechanisms by which social capital influences mental health and more specifically, depressive symptoms (Fujiwara & Kawachi, 2008; Giordano & Lindstrom, 2010). According to the World Health Organization, mental health has become a "global burden," with depression accounting for the majority of this burden (Ferrari et al., 2013; Funk, 2016; Liu et al.,

2020). At present, one in every six Americans is diagnosed with clinical depression in his or her lifetime ("WHO | Depressive symptoms," n.d.). This high prevalence of clinical depression, as well as undiagnosed depression and subclinical depressive symptoms, is an increasingly important public health issue when considering the disabling components of the illness. In the most dire circumstances, depression can lead to suicide (Ferrari et al., 2013). It is also a predictor of physical disability (Prince et al., 2007) and lifelong mental health problems (Fergusson et al., 2005). Therefore, it is imperative to understand the underlying mechanisms at play in combating depressive symptoms to better promote both the physical and mental health of the general population.

Social capital, or the social resources produced, consumed, and exchanged in interpersonal relationships, is of particular importance in understanding the high levels of depressive symptoms (Cohen-Cline et al., 2018; Landstedt et al., 2016; B. Turner, 2003; Wu et al., 2015). Previous research has posited that one's social capital influences one's overall physical health, mental health, and well-being (Giordano & Lindström, 2011; Hamano et al., 2010; Wu et al., 2015). For example, one meta-analysis suggests that poor social relationships can be as, if not more, harmful than excessive drinking, obesity, and lack of exercise when predicting an individual's physical health status (Holt-Lunstad et al., 2010). Similarly, social relationships have been found to protect against various forms of mental illness for individuals across the life course (Ehsan & Silva, 2015; Wu et al., 2015). Yet, while social capital has been associated with mental health broadly, this paper will hone in on depressive symptoms by examining how they are influenced by change in two dimensions of social capital.

As public health policy gradually gains momentum, shifting discussions of depression away from a curative model towards a preventive route, understanding the social mechanisms at play will aid in identifying how social capital shapes some of this "global burden." To better understand how social capital is associated with levels of depressive symptoms, this study examines both cognitive and structural social capital over a 19-year period from 1992-2011 using data from the Wisconsin Longitudinal Study (WLS).

SOCIAL CAPITAL: COMPETING DEFINITIONS, MEASUREMENTS, AND PATHWAYS

The study of social capital and its connection to health and well-being dates at least to Durkheim's seminal work on suicide (1897)**.** As Durkheim's theory suggests, social integration protects individuals against suicide. Thus, suicide rates were low when there was high social integration and regulation, and high when individuals experienced isolation. While Durkheim never explicitly employed the concept of social capital in his work, Turner (B. S. Turner & Turner, 2004) examines how Durkheim's views of social solidarity evoke a parallel to social capital as they "refer to levels of social support that a person has and feelings of belonging that promote a sense of well-being."

The concept of social capital has now expanded into numerous fields, especially social policy and sociology, which focus on determinants of health at the individual- and community-levels. A number of studies support Durkheim's general hypothesis that larger social networks and higher levels of social support lead to better health (Berkman et al., 2000; Berkman & Syme, 1979; House et al., 1988; Mueller & Abrutyn, 2016). However, debates surrounding the association between social capital and health remain,

stemming from contention over the specific definition of social capital, how social capital is best quantified and measured, as well as the pathways for how social capital influences health outcomes (Giordano et al., 2012; Landstedt et al., 2016; Webber et al., 2011).

Bourdieu, Coleman, Portes, and Putnam, four major social capital theorists, vary in their framing of what social capital is and how it accumulates (Bourdieu, 1986; Coleman, 1988; Portes, 1998; Putnam, 1995). Scholars focusing on either Bourdieu's, Coleman's or Portes's definition theorize social capital as the presence of networks, relationships, and associations that connect individuals (Webber et al., 2011). This dimension is known as structural social capital and has been theorized to influence health via a social support pathway. When individuals expand their social networks through involvement in group activities, such as volunteering or sports, they gain a broader social support system of people who affect their own health behaviors (Giordano & Lindstrom, 2010).

Researchers focusing on Putnam's definition understand social capital as one's perception of trust, sense of belonging, and self-efficacy from the community-level (Harpham et al., 2002). This dimension is known as cognitive social capital. Cognitive social capital has been theorized to influence health via a psychosocial pathway where the quality of relationships matters in influencing health through biological processes. Previously this pathway has been discussed by Wilkinson (2002) who found that low levels of trust can lead to increased levels of blood cortisol, resulting in poor health outcomes like depression.

Yet, it is important to note that social capital is a multidimensional concept and therefore needs to be examined with both dimensions in the same analysis. These two

dimensions of social capital and their pathways are not mutually exclusive. However, previous research has typically modeled these dimensions of social capital separately.

Many studies examining structural social capital have focused on only one variable, such as level of civic engagement (Hamano et al., 2010), number of social group membership (Cruwys et al., 2013), or frequency of social contacts with family and friends (Forsman et al., 2012). In each of these papers, the scholars focused on the social support pathway and how the size of social networks led to better health outcomes. However, this narrow focus may explain why inconsistent findings remain in the literature. One cross-sectional study in Australia reported that community participation was not strongly associated to psychological well-being (Phongsavan et al., 2006). Similarly, Ehsan and De Silva (2015) performed a systematic review and discuss finding weak to non-significant associations among mental health, depressive symptoms and civic engagement. These competing results make it unknown whether the sole measure a study identifies is most representative of the construct of structural social capital or if this measure is specific to the population or dataset they are using.

Unlike studies examining structural social capital, many researchers measuring cognitive social capital have used numerous variables to measure the concept. Cognitive social capital has been shown to have a strong association with self-rated health (Snelgrove et al., 2009) as well as an inverse relationship with mental health (De Silva et al., 2007; Hamano et al., 2010). When analyzing cognitive social capital and depressive symptoms, Fujiwara and Kawachi (2008) reported that trust of neighbors lowers the risk of developing depressive symptoms. Similarly, Aneshensel and Sucoff (1996) and Ross

(2000) also discuss findings supporting the notion that cognitive social capital lowers risks of depressive symptoms.

Yet, there remain inconsistent results even within this literature. For example, Fujiwara and Kawachi (2008) reported an association between trust and depressive symptoms but found no association for two other measures of cognitive social capital – sense of belonging and mutual aid. Similarly, Harpham and colleagues (2002) examined social capital and mental health in Cali, Columbia and concluded there was a significant association between trust and mental health, but not between social cohesion and mental health. However, the majority of these studies use cross-sectional data, which limits conclusions to those comparing individuals who have different levels of cognitive social capital at one point in time. As such, further research is needed to examine this association using a large longitudinal sample to understand if changes in in cognitive social capital result in lower levels of depressive symptoms within an individual over time.

Longitudinal studies are pivotal to understanding how social capital affects depression both between people as well as the change an individual experiences over time (Curran & Bauer, 2011). While a plethora of prior studies provides valuable insights into the association between social capital and depressive symptoms cross-sectionally, there are limited numbers of longitudinal studies. I am aware of only three. One longitudinal study by Nakamine and colleagues (2017) studied 17,768 middle-aged adults in Japan over a 9-year period and examined the association between "bridging" (i.e., relationships among heterogenous individuals) and "bonding" (i.e., relationships among homogenous individuals) social capital and depression. They used latent growth

modelling and concluded that increasing one's bonding social capital is related with diminished depression, while bridging had no association. A second longitudinal study by Kim and colleagues (2012) focused on two measures of cognitive social capital and analyzed data from the ongoing Korean Welfare Panel Study (2006-2008). Using logistic regression, they reported that low interpersonal trust is an independent risk factor for developing clinical depression one year later. Finally, Landstedt and colleagues (2016) examined the directional associations between structural social capital and depressive symptoms. They used cross-lagged structural equation modeling on a study of Northern Swedish men and women to understand whether civic engagement was related to changes in depressive symptoms. Their study found that for men, youth civic engagement was associated with declines in depressive symptoms while no such longitudinal relations were found for women. Overall, these three longitudinal studies posit that higher levels for both dimensions of social capital lowers levels of depressive symptoms. Yet, it is still difficult to make meaningful comparisons across these studies due to the variables they chose to use. For example, Kim and colleagues (2012) as well as Landstedt and colleagues (2016) only used one dimension of social capital in their study's while Nakamine and colleagues (2017) estimated models using bridging and bonding social capital. Furthermore, each of these studies used samples from three different countries. The current study will add to the social capital literature by examining both structural and cognitive social capital with a large longitudinal sample based in the United States to understand how these two dimensions of social capital influence depressive symptoms.

Furthermore, none of the three longitudinal studies previously mentioned has scrutinized differences between persons versus intraindividual change over time,

although a strength of longitudinal design is that both estimates are available. By assuming that these effects are equal, these scholars may in fact have misspecified the within-person effects for social capital on depressive symptoms. Within-person effects are of greatest interest from an interventionist perspective. While between-person effects suggest how one's current level of social capital is associated with one's current level of depressive symptoms, within-person effects give insight into how changes in either dimension of social capital may influence changes in levels of depressive symptoms. A study by Cohen-Cline and colleagues (2018) used two-wave twin data to examine whether a difference existed at these two levels of measurement. They found structural social capital to have no associations with depression, while cognitive social capital was associated with fewer depressive symptoms between-twin pairs as well as decreasing depressive symptoms over time within-twin pairs. Their findings identified the within-twin effects as significantly stronger than the between-twin effects. As such, it is possible that social capital affects depressive symptoms differently when examining between- and within-person levels. For example, increasing levels of trust may decrease depressive symptoms for that individual, but individuals with high levels of trust may not have lower levels of depressive symptoms than individuals with lower levels of trust. The current study aims to examine these between- and within-person effects. I estimate the between-effects to examine how they correspond with the results of prior studies examining social capital and depression, as well as to determine whether any of the within-person effects are significantly different.

AIMS OF THIS STUDY

I estimate a growth curve model to better understand the connections among cognitive and structural social capital and depressive symptoms. Growth curve models allow me to investigate the differences, if any, that exist between the two dimensions of social capital at the level of between- and within-persons among cohort members, and also intra-individual change across this 19-year period. I anticipate that cognitive and structural social capital will have an inverse association with depressive symptoms at both the level of between- and within-persons. I will also examine whether between-person and within-person effects are equal or unequal.

DATA AND METHODS

DATA

The Wisconsin Longitudinal Study (WLS) is a random sample of one-third of the men and women who graduated from Wisconsin high schools in 1957 (*N*=10,317). The cohort was first surveyed in 1957 (age 18) and was followed up in 1964 (age 25), 1975 (age 36), 1992 (age 53), 2004 (age 65), and 2011 (age 72). The WLS has used a combination of follow up techniques including in-person, telephone, and mail surveys. The data used in this longitudinal analysis come from the years 1992, 2004, and 2011 because 1992 was the first wave that included the depressive symptoms battery. Overall, retention for the WLS is high, with response rates from the 1992 wave (87%), 2004 wave (86%), and 2011 wave (74%) all at or above 74%, excluding deaths. For the present analyses, individuals who left the study before 1992 (n = 1,471) and those whose participation continued but who did not provide any valid responses to the depressive symptoms battery across the three waves (n = 1,052) were omitted. The final analytic sample was 7,794.

It is important to note that the WLS reflects the population of Wisconsin in the 1950s, meaning that there are only a small number of African-Americans, Hispanics, or Asians. In this study less than 1% of individuals identified as non-white. Further, all respondents are high school graduates and so have above average levels of educational attainment, occupational status, and income compared to nationally representative studies. Despite this, the WLS provides valuable data on white non-Hispanic high school graduates across the life course. As Piliavin and Siegl noted, "These characteristics describe nearly two-thirds of Americans in this age cohort" (2007, p.454). Therefore, these data remain a valuable source of information for examining numerous life course questions.

OUTCOME VARIABLE

Depressive symptoms were measured in 1992, 2004, and 2011 using the Center for Epidemiological Studies-Depression scale (CES-D) (Radloff, 1977). However, the WLS used a slightly modified version of the original response scale. Instead of the typical 8-point range reporting frequency of symptoms in the past week (0 days to 7 days), the WLS collapsed the response scale to range from 0 to 3 (less than 1 day, 1-2 days, 3-4 days, and 5-7 days). I created a sum score; the lowest reliability (Cronbach's alpha) was 0.83 in 2004. To reduce the positive skewness of the variable, I log transformed it for regression analyses.

STRUCTURAL SOCIAL CAPITAL VARIABLES

Number of volunteering activities, social support, and social integration were all used as measures of structural social capital. Number of volunteering activities was first measured in 2004. To retain the three-wave design, participants' 2004 values were

imputed for 1992. The module asked about specific types of organizations at which the individual might have volunteered over the past 12 months. These volunteer organizations included religious; school or education; political group or labor union; senior citizen group or related organization; national or local organization; and other. Each of these questions was asked with a 'yes' or 'no' response. I added together the 'yes' responses to construct an index of the number of volunteering activities an individual had been involved in during the past 12 months. The range for responses was between 0 and 6. This is a measure of general engagement in volunteering activities; unfortunately, the WLS did not have measures to determine intensity of volunteering in a single type of organization.

Social support was measured, during all three waves, as an index of two questions that asked whether a respondent had a person in 'your family' and then 'a friend' with whom they could 'share [your] very private feelings and concerns.' The items were measured dichotomously with either a 'yes' (1) or 'no' (0) response. I summed the values to create an index ranging from 0 to 2 with 2 referring to sources of social support both inside and outside the family, and 0 meaning no social support.

Previous scholars have analyzed social integration by determining how large an individual's social network is based on the number of connections they make. Measures have included time spent with family and friends, as well as an individual's marital status (Berkman & Syme, 1979; Wong & Waite, 2015). Research suggests that married individuals are more socially integrated than single individuals due to their access to their spouse's network. These three measures were included in all three waves. Time spent with family was ascertained with the question "How many times in the past four weeks

have you gotten together with family?" Time spent with friends was ascertained with the question "How many times in the past four weeks have you gotten together with friends?" I scored each question as 'spent no time' (0); 'spent some time' (1) if they spent up to a total of four times in the past four weeks with their family/friends; and 'spent a lot of time' (2) if they spent more than four times in the past four weeks with family/friends. Marital status was a simple indicator of either 'single' (0) or 'married' (1).

COGNITIVE SOCIAL CAPITAL VARIABLES

Self-efficacy, trust, and sense of belonging were measures for cognitive social capital and were only measured in the 2004 and 2011 waves. To retain the three-wave design, I duplicated participants' 2004 values for the 1992 wave. Self-efficacy, as captured by the environmental mastery dimension of the Ryff (1989) psychological well-being scale, reflects an individual's perceived ability to effectively manage life's demands. Conceptualizing self-efficacy as a dimension of social capital aligns with literature identifying it as a resource that is often shaped and supported by social relationships. While autonomy and personal control can be seen as individual traits, self-efficacy within the context of social capital emphasizes the extent to which a person's belief in their control is reinforced through their social networks (Forsman et al., 2012). Framing self-efficacy in this way allows the analysis to disentangle its social dimensions from purely autonomous traits, reflecting its broader social role as a resource that can buffer against depressive symptoms over time. The scale used in this measure is comprised of five questions, including "To what extent do you feel in charge of the situation in which you live?" Each of these items was measured on a 6-point Likert scale

ranging from ‘disagree strongly’ to ‘agree strongly.’ Sum scores ranged from 0 to 30 with higher scores representing more self-efficacy; the lowest reliability was 0.72 in 2004.

Neither trust nor sense of belonging were included as multi-item scales in the WLS and therefore I selected a single item from the Ryff (1989) psychological well-being instrument for each of these concepts. Both items were measured on a 6-point Likert scale ranging from ‘disagree strongly’ to ‘agree strongly.’ Trust was measured by asking ‘To what extent do you agree that you have not experienced any warm and trusting relationships with others?’ Sense of belonging was measured by asking respondents ‘To what extent do you agree that you often feel lonely because you have few close friends with to whom to share your concerns?’

COVARIATES

The following covariates were selected due to their potential to be confounders because of their association with both depressive symptoms and social capital (Fujiwara & Kawachi, 2008; Murayama et al., 2013; Webber et al., 2011). Self-rated health was measured in each of the three waves. The original item in the WLS was measured on a 5-point scale ranging from ‘very poor’ to ‘excellent.’ I dichotomized the item (very poor, poor, or fair vs. good or excellent) to account for skewness.

Extraversion was originally measured on a 6-point scale in 1992 with 2 questions and then again in 2004 and 2011, but with 6 questions (John et al., 2008). The 6-point scale for all the time points ranged from ‘strongly disagree’ to ‘strongly agree.’ To adjust for these measurement differences, I took the average of the available extraversion variables at each wave. Of the three scales, 1992 had the lowest reliability at 0.59.

Educational attainment was originally a continuous variable, but for analysis I dichotomized it into high school degree (0) and some college or beyond (1). A high school degree, 12 years of education, was the lowest possible education since all individuals in the WLS had graduated from a Wisconsin high school.

Additionally, sex of respondent and total assets from 1992, 2004, and 2011 were also included as independent variables. Total assets was an index of the respondent's home equity; respondent's and spouse's retirement plans; respondent's and spouse's checking accounts, savings accounts, or money market funds; respondent's and spouse's CDs, government saving bonds, or treasury bills; and the total cash value of life insurance policies**.** Total assets from each of the three years was skewed and therefore I divided it into three roughly equal categories (33%) of 'low,' 'medium,' and 'high' assets. I chose total assets as a proxy for socioeconomic status because assets encompass all the possible financial resources individuals could draw upon during times of need (Ruggles & Williams, 1989).

DATA ANALYSIS

I conducted a growth curve analysis using the statistical software package Stata version 14.2 to test for both within- and between-person variance in depressive symptoms (Duncan et al., 2013). This decomposition allows for a more nuanced understanding of social capital's influence by distinguishing between stable, cross-sectional effects across individuals and dynamic, longitudinal changes within individuals over time. The between-person effect captures how mean differences in social capital across individuals are associated with depressive symptoms, reflecting, for instance, how individuals with higher social capital tend to experience differing depressive symptom levels compared to

those with lower levels. The within-person effect, on the other hand, captures how changes in an individual's own social capital over the study period correlate with changes in their depressive symptoms, providing insights into whether increases or decreases in social capital may influence depressive outcomes for that individual. By isolating these distinct effects, the multilevel model provides an analysis of whether social capital's impact on depressive symptoms arises primarily from stable interindividual differences or from intraindividual changes over time. Each of the models had the logged CES-D as the outcome variable while time was represented by age and age-squared. A curvilinear model produced better fit than a linear model. Therefore, all models that follow include both linear and quadratic slopes. I allowed the linear effect of age to vary across individuals while all other slopes, including that of age-squared, were fixed.

Model I included age, centered at age 53, and age-squared, centered at age 53. Model II contained all structural social capital variables and all covariates. Model III contained all cognitive social capital variables and all covariates. Model IV included both structural and cognitive social capital variables, and all covariates. Model V included both structural and cognitive social capital variables, as well as all covariates, and also examined whether any differences existed among the within- and between-person effects for social capital variables. Therefore, this final model estimates all within- and between-person effects separately. The between effects in Table 2.3 represent the total between-person effects, or the sum of the observation-level coefficient and the person-level coefficient, as shown below:

$$Y_{ti} = \gamma_{00} + \gamma_{10}age_{ti} + \gamma_{20}age_{ti}^2 + \gamma_{30}socialcapital_{ti} + \gamma_{N0}othercovariates_{ti} + \gamma_{01}socialcapital_i + \gamma_{02}gender_i + \gamma_{03}education_i + \mu_{0i} + \mu_{1i}age_{ti} + \varepsilon_{ti}$$

Within = γ_{30}

Between = $\gamma_{30} + \gamma_{01}$

Where:

Y_{ti}: The dependent variable representing depressive symptoms for individual *i* at time *t*.

γ_{00}: The intercept term, representing the expected value of depressive symptoms when all predictors are held at zero. This term captures the average baseline level of depressive symptoms across individuals.

$\gamma_{10}age_{ti}$: The fixed effect of age, capturing the linear change in depressive symptoms over time within each individual.

$\gamma_{20}age_{ti}^2$: The fixed effect of age squared, allowing for a curvilinear trend in depressive symptoms as individuals age. This term enables the model to account for potential accelerations or decelerations in the rate of change in depressive symptoms at different points in the life course.

$\gamma_{30}socialcapital_{ti}$: The within-person effect of social capital, representing the association between changes in an individual's social capital over time and their depressive symptoms. This term isolates the effect of fluctuations in social capital within the same individual, holding other variables constant.

$\gamma_{N0}othercovariates_{ti}$: This term represents the fixed effects for any time-varying covariates included in the model, such as other social determinants of health that might vary across time and affect depressive symptoms.

γ_{01}socialcapital$_i$: The between-person effect of social capital, indicating how differences in average social capital levels across individuals are associated with differences in depressive symptoms. Together with γ_{30}, this term contributes to the overall between-person effect on depressive symptoms.

γ_{02}gender$_i$: The fixed effect of gender, accounting for any baseline differences in depressive symptoms between genders across the sample.

γ_{03}education$_i$: The fixed effect of education, representing how educational attainment is associated with depressive symptoms across individuals.

μ_{0i}: The random intercept for individual *i*, allowing each person to have their own baseline level of depressive symptoms, thereby capturing individual differences in depressive symptoms not explained by the fixed effects.

μ_{1i}age$_{ti}$: The random slope for age, which allows the rate of change in depressive symptoms over time to vary across individuals, capturing individual-specific growth trajectories.

ε_{ti}: The residual error term, representing individual *i*'s deviation from the predicted level of depressive symptoms at time *t*, accounting for unexplained variance.

γ_{30}: This term represents the effect of social capital changes within an individual over time on their depressive symptoms. It isolates how increases or decreases in social capital over time within the same person are associated with corresponding changes in depressive symptoms.

$\gamma_{30} + \gamma_{01}$: This combined effect represents how differences in average levels of social capital between individuals are associated with depressive symptoms. This effect captures the cross-sectional, or baseline, differences in depressive symptoms across individuals who vary in their social capital.

ATTRITION

As with all longitudinal studies, attrition of respondents was present in the WLS. Analyses were consistent with documentation on dropout rates in the WLS where respondents who were male, had lower adolescent cognitive ability, lower parental SES in adolescence, and poorer self-reported health in adulthood had higher levels of dropout, as discussed by Herd and colleagues (2014). Additionally, for respondents who attrited after 1992, I tested each of the key social capital variables as well as depressive symptoms, to determine whether they were predictive of dropout. None of these variables predicted dropout.

IMPUTATION

Within the analytical sample of 7,794, 62% of cases had complete data on all the variables of interest, and 84% of participants provided valid responses to more than half of the independent variables. Therefore, to account for missing data in the three waves analyzed, I performed multiple imputation by chained equations separately for each wave. If an individual did not answer any questions in a wave, then data were not imputed for that individual in that wave. I included the dependent variable in the imputation phase as well as the analyses. The regression results presented here combine the estimates from the five imputed datasets using Rubin's (1987) rules.

RESULTS

Table 2.2.1 shows the descriptive statistics, including means, standard deviations, and range, of all variables pooled across waves. The average score for depressive symptoms logged was 2.49, which corresponds to low levels of depressive symptoms. Logged depressive symptoms decreased on average 6.5% from wave 1 to wave 2 and then increased on average 4.5% from wave 2 to wave 3. This suggests that at age 65 respondents experienced, on average, their lowest depressive symptoms scores over the 19-year period.

Table 2.2 displays correlations among all social capital variables and depressive symptoms. Correlations among the social capital variables were generally small to moderate in magnitude, with the largest correlation between sense of belonging and trust (0.498, $p<0.001$) followed by self-efficacy and sense of belonging (0.492, $p<0.001$) and depressive symptoms and self-efficacy (-0.446, $p<0.001$).

GROWTH CURVE MODELS

The results of the growth curve models are shown in Table 2.3. All coefficients were standardized for ease of interpretation. Model I included age and age squared. The fixed slopes were statistically significant (age: $\gamma = -0.034$, $p<.001$; age squared $\gamma = 0.002$, $p<.001$). This indicated a U-shaped trajectory where the lowest levels of depressive symptoms occurred in the early 60s, on average. The random slope on the linear term was also statistically significant, indicating that individuals varied in their trajectories of depressive symptoms ($\mu = 0.014$, $p<.05$).

Research question 1 first aimed to determine if structural and cognitive social capital has an inverse association with depressive symptoms. Model II included structural

social capital variables. All five variables measuring structural social capital – volunteering activities, social integration (marital status, time spent with family and friends), and social support (from a family member and friend) – were inversely related to an individual's level of depressive symptoms. Of the five significant coefficients, marital status was largest ($\gamma = -0.172$, $p<.001$), followed by social support ($\gamma = -0.102$, $p<.001$), time spent with friends ($\gamma = -0.049$, $p<.001$), time spent with relatives ($\gamma = -0.031$, $p<.001$), and volunteering activities ($\gamma = -0.016$, $p<.05$). I performed Wald tests between each pair of structural social capital variables to test equality among the standardized coefficients. All pairs were significantly different ($p<0.001$), except time spent with friends and time spent with relatives. These results suggest that the effects for time spent with friends and time spent with relatives were equal contributors to a decrease in depressive symptoms.

Model III included cognitive social capital variables: self-efficacy, trust, and sense of belonging. Each of these variables was inversely associated with depressive symptoms levels ($p<.001$). Self-efficacy had the largest coefficient of these three variables ($\gamma = -0.289$, $p<.001$). Sense of belonging had the second largest coefficient ($\gamma = -0.15$, $p<.001$) of the three cognitive social capital measures, followed by trust ($\gamma = -0.048$, $p<.001$). Wald tests were performed to test coefficient equality between each of the cognitive social capital coefficient pairs. Each comparison produced significant results suggesting coefficients are unequal.

Model IV combined structural and cognitive social capital, as well as all covariates. This model was used to determine if any significance was lost from either structural or cognitive social capital when including them in a single model. All variables

retained their statistical significance, with the exception of time spent with friends and volunteering activities. Model V then combined both dimensions of social capital, all covariates, and also tested the longitudinal random coefficient model's assumption that longitudinal and cross-sectional effects are equal. Unlike the previous four models, Model V examined between- and within-person effects separately, whereas the previous models (Model I-IV) did not distinguish between these effects. When examining the structural social capital variables in Model V, this assumption held true: The longitudinal and cross-sectional effects were equal. The overall effects of social support ($\gamma = -0.042$), marital status ($\gamma = -0.185$), time spent with friends ($\gamma = -0.019$), and time spent with relatives ($\gamma = -0.022$) were all statistically significant ($p<0.05$), while volunteering continued to have a statistically non-significant relationship with depressive symptoms. The cognitive social capital variables in Model V showed that a difference existed between- and within-person effects for sense of belonging (within: $\gamma = -0.105$; between: $\gamma = -0.151$) and self-efficacy (within: $\gamma = -0.20$; between: $\gamma = -0.35$) (all four effects: $p<0.01$). Furthermore, Model V displayed that the cross-sectional effect of trust was equivalent to the longitudinal effect of trust ($\gamma = -0.041$, $p<0.01$).

These findings show an inverse association among both dimensions of social capital and depression. Furthermore, these results provide clarity in understanding if the effects for both dimensions of social capital have equal or unequal effects. Structural social capital variables were found to be associated with depressive symptoms equally at the within- and between-person levels, while cognitive social capital had different effects on depressive symptoms at these two levels of measurement. Trust was equally associated with decreasing depressive symptoms at both levels of measurement while the

other two cognitive social capital variables, self-efficacy and sense of belonging, presented a difference when analyzing the between- and within-person levels. In both instances, the between-person effects were larger than the within-person effects. When comparing the coefficients from Model IV and V I note the importance of parsing out these between- and within-person effects. If I continued to treat the longitudinal and cross-sectional effects as equal, then I would have lost this significant evidence of between-effects having a larger effect than the within-person effects for these two cognitive social capital variables.

DISCUSSION AND CONCLUSION

The purpose of this longitudinal study was to first examine whether both structural and cognitive social capital were associated with depressive symptoms among individuals in midlife. The results suggest that both dimensions of social capital, cognitive and structural, are inversely related with levels of depressive symptoms for individuals in midlife, even after controlling for a number of health determinants known to impact depressive symptoms. These results add further evidence to the previous line of research examining the association between social capital and depressive symptoms (Cao et al., 2015; Fujiwara & Kawachi, 2008; Webber et al., 2011). Furthermore, results from this study are consistent with previous studies using growth curve models positing a curvilinear relationship between depression and age where levels of depression are lowest during middle adulthood and then increase as individuals age (Mirowsky & Ross, 1992; Yang, 2007).

However, unlike many of the previous studies, I also examined if there were any differences in the association among the two dimensions of social capital and depressive

symptoms when considering the between- and within-person levels. Within-persons, increases in structural social capital were shown to be associated with decreasing depressive symptoms. Additionally, when comparing people, individuals with higher levels of structural social capital had lower levels of depressive symptoms than individuals with lower levels of structural social capital. Both the cross-sectional and the longitudinal effects for structural social capital are equal in size. These dual interpretations also held true for one of the three cognitive social capital variables, trust; however, the other two cognitive social capital variables, self-efficacy and sense of belonging, suggested a difference in their effects on depressive symptoms when analyzing individual change versus differences between individuals. In both cases, differences between individuals were stronger than change within an individual.

ASSESSING STRUCTURAL AND COGNITIVE SOCIAL CAPITAL

It is pivotal to examine both dimensions of social capital to understand their unique effects to better assist policymakers in creating interventions. While all aspects of social capital have been shown to be associated with lower depression, both in separate analyses (Kim et al., 2012; Landstedt et al., 2016; Ross, 2000) and jointly (Cohen-Cline et al., 2018; Fujiwara & Kawachi, 2008; Hamano et al., 2010), it is crucial from a policy perspective to understand the nuances of these relationships. Specifically, it is imperative to determine which of these factors is most effective in decreasing levels of depressive symptoms. Furthermore, due to social capital being a multidimensional concept, both dimensions need to be examined simultaneously: These two dimensions of social capital are not mutually exclusive. Through including these dual dimensions, researchers of social capital can understand how these elements of social capital each uniquely

contribute to the decreasing of depressive symptoms in relation to each other, and further clarify which factor or combination of factors are most effective. This will aid policymakers with specific targets of how best to address this "global burden" of high levels of depression, rather than focusing on either cognitive or structural social capital more generally.

The current study suggests that the three most effective social capital variables in decreasing depression are self-efficacy, marital status, and sense of belonging. Uncovering these three variables as the most effective in decreasing levels of depressive symptoms provides policymakers with both a route for possible targeted intervention as well as possible risk factors that should be examined throughout the life course. For example, while marital status is not easily intervenable, it does act as a potential risk factor for where policymakers could be intervening. Both self-efficacy and sense of belonging are prime areas of intervention. Additionally, these results should be used as both a remedial process once depression is diagnosed as well as a preventive measure (Cruwys et al., 2013). By policymakers disseminating the positive effects of these three effective social capital variables in decreasing depressive symptoms, health affiliates can more appropriately begin to manage this current "global burden" (Centers for Disease Control, 2014).

BETWEEN- AND WITHIN-PERSON EFFECTS

In this current study I also report that key differences exist in the between- and within-person effects for cognitive social capital, while effects are equal for structural social capital. In longitudinal random coefficient models, between- and within-person effects are assumed to be equal. However, this assumption may not be true in many cases,

such as in this study. By neglecting to test this assumption, scholars may unintentionally present findings that are inaccurate. If this current study stopped with Model IV, then I would have assumed a standard deviation increase in self-efficacy reduces depressive symptoms by 0.29. However, when this assumption was tested, I report that a within-person increase in self-efficacy only reduces depressive symptoms by 0.20. While this difference does not appear large on face-value, when scaling this to a population intervention, this would equate to only getting 66% of the benefits from an anticipated intervention.

The lack of significant between-person effects for structural social capital, contrasted with the significant effects observed for cognitive social capital, excluding trust, suggests that the benefits of structural social capital, such as social network size or frequency of interactions, may operate more strongly through changes within an individual's social environment rather than as stable, cross-sectional differences across individuals. Structural social capital, often tied to the quantity of social connections (Forsman et al., 2012; Landstedt et al., 2016), may not provide uniform benefits for mental health because merely having social contacts does not guarantee access to meaningful support or positive social interactions. In contrast, cognitive social capital components, such as perceived self-efficacy and sense of belonging, reflect an individual's internalized sense of connection and social support that likely contribute to a baseline sense of psychological resilience and emotional well-being. These cognitive aspects are inherently more personal and subjective, meaning they may exert a more consistent and cross-sectional protective effect against depressive symptoms, irrespective of specific changes in an individual's social network size or engagement. This distinction

highlights the idea that while structural social capital may fluctuate based on situational or contextual factors, cognitive social capital captures a more stable internalized resource that impacts individuals' well-being across varying social environments.

To my knowledge the only other study that investigates social capital and depression at these two levels of measurement is Cohen-Cline and colleagues (2018), who used a two-wave twin-study design. In their study, the overall level of structural social capital was not linked to lower depressive symptoms at either measurement level. A possible explanation for this could be the variables they used to measure structural social capital. Whereas this study used social interactions with friends and family, Cohen-Cline and colleagues (2018) focused on interactions with neighbors. This difference in variables may suggest that neighborhood interactions are not the most appropriate measure of structural social capital; rather, neighborhood interactions could be seen as a more appropriate measure of bonding social capital due to many neighborhoods being homogenous when examining their residents' characteristics. Thus, this measure would not account for relationships outside of the neighborhood or interactions with family members. Cohen-Cline and colleagues (2018) also included a measurement for volunteering activities, which was found not to be associated with lowering depressive symptoms in their study as well as the current study. However, in the current study, with the exception of volunteering activities, all other structural social capital variables were reported to equally influence levels of depression symptoms both across individuals and changes within individuals.

Cohen-Cline and colleagues (2018) reported significant results when examining cognitive social capital and levels of depressive symptoms. In their study, researchers

suggested a significant association both between twin-pairs as well as within-twin pairs, where the within-twin effects had a stronger association on depressive symptoms than the between-pair effects. However, in the present study, the reverse was true: The between-effects for both self-efficacy and sense of belonging were larger than the estimates of within-person change. However, this divergence in results could be due to a difference in the age of the two samples. Cohen-Cline and colleagues (2018) had an average age of around 45 across their three groups while this sample examined results from individuals starting at age 53 and in the final wave participants were 72. As such, more research is needed in examining social capital and levels of depressive symptoms to garner a better understanding of how this multidimensional concept influence levels of depressive symptoms both between individuals as well as within individuals.

LIMITATIONS

This study has several notable weaknesses. While such a homogenous sample helps to rule out unobserved bias, the sample is representative only of white high school graduates who were born in 1939. This sample does represent two-thirds of the current U.S. population in this age group in terms of race/ethnicity and educational attainment (Piliavin & Siegl, 2007), but says less about future cohorts, which are increasingly well educated and racially and ethnically diverse (Everett et al., 2011)

While this current study used growth curve models specifically to estimate both the cross-sectional and longitudinal effects for how both dimensions of social capital influence and individual's depressive symptoms, the question of causality is an interesting but separate investigation deserving of mention. Scholars have begun to work towards understanding this directional question, such as a study by Landstedt and

colleagues (2016). In this study, Landstedt and associates examined the directional association between structural social capital and depression for individuals aged 16-42 years old using structural equation modelling. Their findings report higher levels of civic engagement for men predicted lower levels of depression, but did not predict this for women. Future research should consider using instrumental variables to better decipher this problem of directionality.

There are also limitations related to the decision of which social capital proxies were included in the analysis and how they were measured. First, there is no fully agreed upon definition of social capital, which leads to issues in how researchers aim to measure the concept in their work. Due to social capital being a multidimensional concept, scholars are uncertain about what measures comprise cognitive and structural social capital (Giordano & Lindström, 2011). The second problem is specific to the limitations of how these constructs are measured in the WLS. For example, it would be beneficial to have a measure of involvement in and commitment to volunteer activities. Having more thorough measures for the level of involvement will aid in better grasping if it is truly the level of involvement or the commitment.

CONCLUSION

In conclusion, attempting to focus on either cognitive or structural social capital is inefficient and removes the nuances of how there are specific variables most effective in decreasing depressive symptoms. Furthermore, this study provides evidence of an inverse relationship between both cognitive and structural social capital and depressive symptoms, but that a difference exists between the association among these two dimensions of social capital and depressive symptoms. Structural social capital (social

support, marital status, time spent with friends, and time spent with family) is associated with depressive symptoms equally at the between- and within-person levels, whereas cognitive social capital (self-efficacy and sense of belonging) is associated with depressive symptoms unequally at the between- and within-person levels, where the cross-sectional effects had a stronger association.

These findings provide benefits for policymakers aiming to decrease levels of depression for middle-aged individuals living in the United States. Results from this study provide evidence for policymakers to target individuals' marital status, self-efficacy and sense of belonging. Policymakers should continue integrating social capital, specifically these three variables, into their policies to assist in combating this "global burden." These targeted interventions will potentially also produce what is known as collateral benefits, which are positive health benefits that move beyond those targeted by these social capital intervention to those who are connected to the individual (Villalonga-Olives et al., 2018). By discussing the inverse association between these two dimensions of social capital and levels of depression, social policymakers can begin enacting a proactive model rather than a reactive model. However, social relationships are fostered over a number of years and through presenting the positive benefits of social relationships, with an emphasis on self-efficacy, marital status and sense of belonging, policymakers can propose their importance early in life so that in mid-to-late life those needing to draw upon their social capital are able to do so.

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TABLES

Table 2.1

Descriptive Statistics

Variable	Mean	Std. Dev.	Minimum	Maximum
Depression (logged) 0 = low; 4.88 = high	2.42	0.95	0	4.88
Volunteering activities 0 = no activities; 6 = 6 activities	0.95	1.29	0	6
Social support 0 = none; 2 = high	1.24	0.85	0	2
Marital status 0 = single; 1 = married	0.78	0.41	0	1
Time spent with friends 0 = none; 2 = high	1.11	0.62	0	2
Time spent with relatives 0 = none; 2 = high	1.09	0.59	0	2
Self-efficacy 2 = low; 30 = high	24.44	3.94	2	30
Trust 1 = low; 6 = high	4.76	1.43	1	6
Sense of belonging 1 = low; 6 = high	4.76	1.37	1	6
Self rated health 0 = poor, 1 = good	0.86	0.35	0	1
Extraversion (mean scored) 1 = low; 6 = high	3.96	1.10	1	6
Education 0 = high school degree; 1 = some college and beyond	0.45	0.50	0	1

Sex of respondent 0 = male; 1 = female	0.53	0.50	0	1
Assets 0 = low; 2 = high	1.02	0.81	0	2

Table 2.2

Intercorrelations among depressive symptoms and social capital variables

Variable	1	2	3	4	5	6	7	8
1. Depression (logged)	-							
2. Volunteering activities	-0.10	-						
3. Social support	-0.14	0.13	-					
4. Marital status	-0.11	-0.17	-0.02†	-				
5. Time spent w/ friends	-0.11	-0.13	0.20	-0.08	-			
6. Time spent w/ relatives	-0.06	-0.05	0.13	0.04	0.21	-		
7. Self-efficacy	-0.45	0.10	0.16	0.01†	0.14	0.06	-	
8. Trust	-0.25	0.12	0.24	0.07	0.16	0.10	0.36	-
9. Sense of belonging	-0.37	-0.37	0.23	0.08	0.19	0.09	0.49	0.50

Note: All correlations were significant at $p<0.001$ unless marked by † which indicates the correlation is non-significant $p>0.05$.

Table 2.3

Hierarchical linear models predicting depressive symptoms

	Model I	Model II	Model III	Model IV	Model V
	γ (SE)	*γ (SE)*	*γ (SE)*	*γ (SE)*	*γ (SE)*
Fixed Components					
Age 53	-0.034*** (0.003)	-0.038*** (0.003)	-0.38*** (0.003)	-0.038*** (0.003)	-0.038*** (0.003)
Age 53 (squared)	0.002*** (0.001)	0.002*** (0.001)	0.002*** (0.001)	0.002*** (0.001)	0.002*** (0.001)
Structural Social Capital					
Volunteering Activities 0 = no activities; 6 = 6 activities		-0.023* (0.009)		-0.005 (0.008)	
Within-Person Effects					-0.008 (0.013)
Between-Person Effects					-0.015 (0.016)
Social Support 0 = none; 2 = high		-0.102*** (0.008)		-0.042*** (0.008)	
Within-Person Effects					-0.042*** (0.01)
Between-Person Effects					-0.038 (0.017)
Marital Status 0 = single; 1 = married		-0.172*** (0.018)		-0.166*** (0.017)	
Within-Person Effects					-0.185*** (0.036)
Between-Person Effects					0.015 (0.046)
Time spent with friends 0 = none; 2 = high		-0.049*** (0.007)		-0.016 (0.008)	
Within-Person Effects					-0.019* (0.009)
Between-Person Effects					-0.009 (0.014)
Time spent with relatives 0 = none; 2 = high		-0.03*** (0.007)		-0.02*** (0.006)	
Within-Person Effects					-0.022** (0.009)
Between-Person Effects					-0.016 (0.014)

Cognitive Social Capital					
Self-efficacy			-0.289***	-0.291***	
2 = low; 30 = high			(0.008)	(0.008)	
					-0.20***
Within-Person Effects					(0.013)
					-0.35***
Between-Person Effects					(0.016)
Trust			-0.048***	0.038***	
1 = low; 6 = high			(0.008)	(0.008)	
					-0.041**
Within-Person Effects					(0.012)
					0.054
Between-Person Effects					(0.017)
Sense of belonging			-0.153***	-0.139***	
1 = low; 6 = high			(0.009)	(0.139)	
					-0.105***
Within-Person Effects					(0.013)
					-0.151***
Between-Person Effects					(0.016)
Covariates					
Self Rated Health		-0.444***	0.30***	-0.289***	-0.278***
0 = poor; 1 = good		(0.02)	(0.02)	(0.02)	(0.02)
Extraversion (mean scored)		-0.119***	-0.043***	-0.34***	-0.03**
1 = low; 6 = high		(0.009)	(0.007)	(0.008)	(0.008)
Education					
0 = high school degree; 1 = some college and beyond		-0.10*** (0.018)	-0.041** (0.016)	-0.048** (0.017)	-0.033* (0.016)
Sex of Respondent		0.132***	0.128***	0.128***	0.126***
0 = male; 1 = female		(0.017)	(0.017)	(0.18)	(0.017)
Assets		-0.068***	-0.041***	-0.28***	-0.023**
0 = low; 2 = high		(0.01)	(0.008)	(0.008)	(0.008)
Random Components					
	0.014	0.012	0.005	0.006	0.001
Random Slope (age)	(0.002)	(0.002)	(0.005)	(0.004)	(0.005)
	0.564	0.49	0.405	0.402	0.4305
Person-level intercept	(0.009)	(0.009)	(0.01)	(0.01)	(0.009)

Observation-level intercept	0.748 (0.006)	0.732 (0.006)	0.712 (0.007)	0.708 (0.006)	0.706 (0.006)
Fit Statistics					
BIC	53483.59	48985.5	44177.1	43301.51	43206.86
AIC	53436.09	48867.55	44067.66	43161.12	43011.87
n	7,794	7,794	7,794	7,794	7,794

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

Note: Predictors are all standardized.

CHAPTER 3: SOCIAL CAPITAL AND DEPRESSIVE SYMPTOM SUBTYPES: A LATENT CLASS AND LATENT TRANSITION ANALYSIS

ABSTRACT

This study examines depressive symptom subtypes and subtype transitions from midlife to later life using data from the Wisconsin Longitudinal Study. Using latent class analysis (LCA) and latent transition analysis (LTA), the research identifies distinct depressive symptom subtypes at ages 54 and 72 and explores the stability and transitions between these subtypes over time. The findings reveal four subtypes of depression: *Depressed Affect*, *Somatic Symptoms*, *Depressed Affect and Somatic Symptoms*, and *Not Depressed*. Notably, while three subtypes remain consistent across the life course, somatic symptoms worsen as individuals age, further highlighting the need to understand depression as a heterogeneous illness that may change with age. Additionally, the study investigates how social capital—measured through social support, social involvement, and social integration—interacts with depressive symptom subtypes and the potential predictors of stability and transition across these statuses. Findings indicate that both social capital and the covariate, self-rated health, significantly impact depressive transitions, with higher social support and good health increasing the likelihood of improvement in depressive symptoms status later in life. These results contribute to our understanding of depression as a heterogenous illness that has specific clusters of symptoms as well as how these subtypes transition overtime.

INTRODUCTION

Depression in later life is a significant public health concern, affecting around 6% of the aging population aged 65+ (Hooker et al., 2019; Reynolds III et al., 2012; Zivin et al., 2010). Unlike in younger adults, depression in older adults often presents unique challenges, including its frequent overlap with physical illness and cognitive decline, which can complicate diagnosis and treatment (Chew-Graham et al., 2012; Massa et al., 2021; Prince et al., 2007). The prevalence of depression in later life rising is particularly concerning where rates in older adults were at 3.8% in 2019 and 6% in 2023 (B. Lee, 2023), given its association with increased disability, diminished quality of life, and heightened mortality risk (Arthur et al., 2020; Ferrari et al., 2013; Zivin et al., 2013). Moreover, the social dimensions of aging, such as the possible loss of social roles, reduced social networks, and increased isolation, may further exacerbate the risk of depression among older adults (Karp, 1997). As such, understanding the complex interplay between biological, psychological, and social factors in later-life depression is essential for developing effective interventions and improving the well-being of this vulnerable population.

In this study I examine how the tangible dimension of social capital, meaning resources acquired through social networks and social participation, is linked to different subtypes of depressive symptoms in older adults. It is critical to examine how social capital, a social determinant known to impact depression generally, may actually be contributing to increases in specific depressive symptoms rather than affecting all symptoms similarly, with some symptom clusters being potentially more disabling than others (Forsman et al., 2012; Landstedt et al., 2016; Lu & Peng, 2019). To my knowledge, no studies have previously analyzed this association in understanding how

tangible social capital may be linked with different clusters of depressive symptoms. Furthermore, current literature examining different depressive symptom classes, meaning various types of depression, over the life course has primarily been exploratory when analyzing these different classes and their association with social determinants, such as gender, race, ethnicity, socioeconomic status, and education (Li et al., 2014; Mezuk & Kendler, 2012; Singham et al., 2022; Veltman et al., 2017).

The Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), currently includes a single diagnosis of depression – Major Depressive Disorder (MDD). The DSM-5 details a range of identifiers for MDD such as severity (mild, moderate, severe) or remission status, as well as nine other different specifiers, meaning extensions of a diagnosis such as chronic, melancholic features or atypical features. While these indicators broaden the diagnostic scope of MDD, depression is still treated as a homogenous illness where the underlying symptoms all point to the same diagnosis (Hybels et al., 2011). However, depressive symptoms may present themselves differently in older adults compared to middle-aged or young adults. For example, Gallo and colleagues (1994) first reported that older adults tend to experience depression without sadness (i.e., somatic symptoms) while middle-aged individuals experience depression with sadness (i.e., a mix of somatic and depressed affect symptoms). Similarly, another study reports older adults with depression are more inclined to exhibit cognitive changes, physical symptoms, and a loss of interest, while being less likely to report affective symptoms compared to younger adults with depression (Fiske et al., 2009). As such, these different age-graded clusters of symptoms may suggest various types of depression exist (Chen et al., 2000).

Research suggests that tangible social capital may uniquely influence the manifestation of depressive symptoms, particularly in older adults (Cao et al., 2015; Forsman et al., 2012; Landstedt et al., 2016; Lu & Peng, 2019). Lower levels of social engagement can disrupt routines and reduce access to social support, contributing to increased somatic symptoms, such as fatigue and sleep disturbances, while the absence of regular social interaction or spousal support may heighten affective symptoms like loneliness and hopelessness (Clarke et al., 2011; Cohen-Cline et al., 2018; Forsman et al., 2012). Additionally, social capital may influence transitions between depressive states by increasing or reducing access to key social resources that buffer against stress, support coping strategies, or foster a sense of purpose. For example, diminished participation in community activities may trigger more severe depressive subtypes, while maintaining strong social ties can promote resilience and mitigate the increase of symptoms (Fujiwara & Kawachi, 2008; Landstedt et al., 2016; Nakamine et al., 2017). This current study aims to explore how tangible aspects of social capital, such as social participation and support networks, may be associated with specific depressive symptoms in later life.

In what follows, I begin by outlining research that discusses depression as a heterogenous illness and how clusters of symptoms may suggest that different types of depression exist. I next examine research that investigates the connection between the social determinant, tangible social capital, and depressive symptoms, while presenting an argument to why tangible social capital may be associated with specific depressive symptoms. I then discuss the aims I will be analyzing in this paper and how this research will contribute to existing literature on the social causes of depression in later life.

DEPRESSION AS A HETEROGENOUS MENTAL ILLNESS ACROSS THE LIFE COURSE

There are several hypotheses for why depressive symptoms present themselves differently across the life course, including biological, psychological, and social factors. For example, as individuals age, they are more likely to suffer from chronic illnesses and other physical ailments. These conditions can overlap or even mask certain depressive symptoms, especially if physical symptoms (e.g., somatic symptoms) are more present than affective symptoms (Gallo et al., 1994; Penninx et al., 2013). A possible psychological factor would be that older adults are more likely to focus on physical health than mental health symptoms due to cultural norms. This tendency would also result in older adults being less likely to receive a depression diagnosis (Chen et al., 2000; Penninx et al., 2013). A social factor that could further contribute to older adults experiencing somatic symptoms could be the stigma associated with mental health and fearing reporting these symptoms or health care professionals not seeing them as a "real" problem (Conner et al., 2010; Leung et al., 2023).

Although survey instruments designed to measure depressive symptoms, such as the Center for Epidemiological Studies Depression Scale (CES-D) (Radloff, 1977), ask about distinct symptoms, the DSM diagnosis of MDD only requires a count of symptoms – specifically requiring the individual to experience 5 or more symptoms over the course of two weeks (American Psychological Association, 2013). While the CES-D is not a clinical instrument, but rather used in population-based surveys to assess depression, it is a meaningful example that points to possible clinical problems with how depression is diagnosed (Veltman et al., 2017). Both the CES-D scale and the DSM diagnosis require

the summing of symptoms; however, in doing so, researchers and clinicians, respectively, may fail to account for the various patterns of symptoms that may be present at different stages in the life course and how these patterns may require specific interventions (Harald & Gordon, 2012). Numerous public health scholars argue that clinicians must begin examining the specific symptoms and not treating depression as a homogenous illness (Collins & Lanza, 2010; Li et al., 2014; Veltman et al., 2017). These scholars suggest that differences in the endorsement of symptoms may result in fewer older adults being diagnosed with depression. This can be dependent on the depression scale a clinician uses, but if clinicians only add symptoms, they may miss possible diagnoses of older adults who do not present the *typical* symptoms of depression, which are far more centered around symptoms middle-age adults present (Gallo et al., 1994; Hybels et al., 2011). Furthermore, studies suggest that depression interventions may be ineffective if we do not analyze the specific depressive symptoms present and use more tailored strategies (Carragher et al., 2009; C.-T. Lee et al., 2012). Interventions may be more effective in aiding individuals if they were curated to address these age-specific symptoms.

The list of symptoms asked in the CES-D range in whether they are referring to somatic symptoms, those related to appetite, sleep disturbance or other physical facets of depression, rather than those related to mood. Previously, these symptoms have been clustered together when assessing if an individual is exhibiting depressive symptoms *generally*. However, prior studies have suggested that these clusters of symptoms may rather be delineating certain types of depression (C.-T. Lee et al., 2012; Mezuk & Kendler, 2012; Ni et al., 2017; Rodgers et al., 2014). Researchers thus far examining

these subtypes primarily rely on latent class analysis (LCA), a statistical method which locates categories of depression based on the patterns of how participants answer questions on a specific depression scale, such as the CES-D (Hybels et al., 2013; Ulbricht et al., 2018; Veltman et al., 2017).

Hybels and colleagues (2013) used LCA and examined 3,000 older adults in a community-dwelling sample. Based on fit statistics, these scholars reported a 3-class model, which includes a class that endorses *no symptoms*, a second class that endorses *negative affect and somatic symptoms*, and finally a third class that endorses *negative affect, somatic symptoms and low positive affect symptoms*. In this study some of the negative affect symptoms included felt depressed, felt fearful, felt lonely, while some somatic symptoms included didn't feel like eating, felt everything was an effort, sleep was restless, could not get going, and a couple positive affect symptoms were felt happy and enjoyed life. Similarly two other studies examining older adults reported a 3-class solution (C.-T. Lee et al., 2012; Veltman et al., 2017). However, there appear to be inconsistencies, at least in studies analyzing older adults, surrounding the qualitative nature of these 3-class models. The inconsistencies are likely attributed to these scholars focusing on different samples (clinical (Veltman et al., 2017) vs. community (Hybels et al., 2013; C.-T. Lee et al., 2012)), using different depressive symptom scales (original CES-D (Hybels et al., 2013), revised CES-D (C.-T. Lee et al., 2012), or other depressive symptom scale (Veltman et al., 2017)) and varying in whether they include (Veltman et al., 2017) or exclude (Hybels et al., 2013; C.-T. Lee et al., 2012) individuals with a previous diagnosis of depression.

Studies have also begun to examine these depression subtypes longitudinally (Lamers et al., 2012, 2016; Ni et al., 2017; Rodgers et al., 2014; Ulbricht et al., 2018). It is essential to understand how individuals may transition or retain their depressive status as they age due to the curvilinear nature of depressive symptoms over the life course (Clarke et al., 2011; Mirowsky & Ross, 1992; Yang, 2007). By examining the dynamics of individuals transitioning between or remaining within specific depressive states, researchers and health practitioners can better identify strategies for mitigating depressive symptoms across the life course. Understanding the social factors that facilitate the movement from a depressive subtype to a non-depressive state, for instance, is crucial for the development of targeted health interventions aimed at preventing and alleviating depression.

Latent transition analysis (LTA) is the longitudinal extension of LCA, which estimates the probability of individuals remaining stable or transitioning between statuses (Collins & Lanza, 2010). Due to the heterogeneity of depression as well as the idiosyncrasy of how symptoms present themselves over the life course (Chen et al., 2000), this statistical method has been used to provide estimates to examine patterns of depressive symptoms longitudinally in older adults. A few studies investigate these subtypes of depression longitudinally in older adults with many relying on small clinical samples of fewer than 500 respondents that analyze transitions anywhere from 12 weeks to 2 years after baseline (Lamers et al., 2012; Rodgers et al., 2014; Ulbricht et al., 2018). Understanding transition and status retention of depressive statuses over a longer period may provide greater insight into why depression worsens, on average, for individuals entering later life. Beyond the limited time ranges, a modest number of studies

investigate how covariates influence these subtypes (Lamers et al., 2012; Ni et al., 2017) or only examine one covariate (Rodgers et al., 2014). However, it is essential to understand how predictors of depression impact depression statuses longitudinally to better understand how it is a heterogenous illness and to design appropriate interventions. Overall, these longitudinal subtype studies concluded that transition did occur for some adults. However, due to the difference in identified subgroups between these studies, it is more difficult to determine if any of these transitions or stability was common between studies.

In parsing out these subtypes, we may better understand the different types of depression depending on the symptoms present. These findings could lead to more tailored interventions that focus on specific symptoms and how they connect, compared to traditional methods of treating depression as a single illness (Carragher et al., 2009; C.-T. Lee et al., 2012).

TANGIBLE SOCIAL CAPITAL AND DEPRESSIVE SYMPTOM SUBTYPES

While examining how patterns of depressive symptoms may indicate distinct types of depression in mid- and later life, I also explored how tangible social capital may be linked to both specific depressive symptoms subtypes, and transitions between these types over time. I hypothesize that tangible social capital is most closely associated with affective symptoms of depression—those related to emotions and mood, such as feelings of loneliness, hopelessness, and fear (American Psychological Association, 2013).

The association between tangible social capital and depressive symptoms is often explained through a social support pathway (Berkman et al., 2000; Santini et al., 2015;

Webber et al., 2011). This pathway suggests that when individuals expand their social networks by engaging in group activities, they gain access to a broader social support system that includes family, friends, neighbors, community groups, and organizations. These networks can provide emotional, informational, and practical support, fostering a sense of belonging and enhancing overall well-being. Additionally, a broader social support system can have social influence on individuals through shared norms and behaviors, encouraging healthier actions such as better eating habits, regular exercise, and self-care practices (Domènech-Abella et al., 2017; Giordano & Lindström, 2011; Villalonga-Olives et al., 2018). Beyond behavioral influence, these networks can also offer crucial resources, such as emotional support or financial assistance during times of need (Bassett & Moore, 2013; Cao et al., 2015; Webber et al., 2011). This also aligns with the stress process model, which posits that social resources can buffer the effects of stress by reducing exposure to stressors, enhancing coping capacities, and providing the necessary support to mitigate stress's impact on mental health (Drentea & Reynolds, 2015; McLeod, 2012; Turner, 2013). Thus, the stress process model serves as a valuable framework for understanding how tangible social capital not only affects specific depressive symptoms but also plays a protective role in the stress-depression pathway, underscoring the dynamic interplay between social resources and mental health outcomes.

Given the pathways through which tangible social capital influences mental health, it is plausible that it plays a significant role in alleviating affective symptoms of depression. A larger support system can help reduce feelings of loneliness by increasing opportunities for social interaction, fostering a sense of connection and belonging

(Berkman et al., 2000; Santini et al., 2015). In contrast, somatic symptoms of depression, such as fatigue and sleep disturbances, may be less influenced by social factors and more by underlying biological mechanisms or other physical health problems (Lin, 2023). The findings of this study suggest that tangible social capital may have a stronger association with affective symptoms than with somatic symptoms, supporting the need to examine clusters of depressive symptoms rather than treating depression as a homogenous illness. This is because affective symptoms are more likely to be directly impacted by social interactions and support, whereas somatic symptoms may persist regardless of changes in social environment or support levels. This approach highlights the importance of considering the unique ways that social resources influence specific depressive symptom patterns.

Thus far, scholars have examined tangible social capital's influence on depression by treating the illness homogeneously. However, it is crucial to recognize that not all forms of social capital have positive impacts on health (Villalonga-Olives & Kawachi, 2017). While social networks and interactions are often considered protective factors, they can also function as sources of harm, depending on the context and group dynamics. For example, social connections may facilitate cyberbullying, reinforce unhealthy behaviors, or perpetuate harmful norms, such as stigmatization of mental health issues or pressure to conform to detrimental habits (King et al., 2019; Offer, 2021; Villalonga-Olives et al., 2018). Additionally, dense social networks may create environments where individuals are overburdened by excessive caregiving responsibilities or financial obligations to others within their network, potentially exacerbating stress and depressive symptoms (Kabayama et al., 2017).

Moreover, strong social ties can limit personal autonomy or subject individuals to social control, as group norms may discourage seeking external help or professional mental health support, thereby entrenching depressive symptoms. For instance, cultural or familial networks that value self-reliance or view mental health challenges as personal failings may deter individuals from accessing necessary resources, increasing the risk of unaddressed mental health issues (Offer, 2021; Villalonga-Olives et al., 2018). Negative interactions, such as interpersonal conflicts or feelings of obligation and guilt within tight-knit groups, may also intensify depressive symptoms by fostering environments of tension rather than support (Rook, 2015; Smith & Christakis, 2008).

Given these complexities, this study aims to investigate how specific dimensions of tangible social capital are associated with distinct clusters of depressive symptoms rather than treating depression as a singular, uniform illness. By exploring these nuanced relationships, this research seeks to provide a deeper understanding of how tangible social capital can uniquely impact depressive symptoms, acknowledging that its effects are context-dependent and multifaceted.

SOCIAL DETERMINANTS OF HEALTH AND DEPRESSIVE SYMPTOM SUBTYPES

I also examine other social determinants of health associated with these depressive subtypes. The social determinants within this study include gender, net worth, and education. Each of these social determinants have been found to be associated with depressive symptoms and social support (Assari, 2017; Chang-Quan et al., 2010; Inaba et al., 2005; Platt et al., 2020; Ross & Mirowsky, 2006). The inclusion of these covariates is crucial as they provide a more nuanced understanding of depression beyond a single-cause mechanism. Scholars argue that depression is a heterogeneous illness, influenced

by a combination of biological, psychological, and social factors (Hybels et al., 2011; Ulbricht et al., 2018). Without accounting for the impact of multiple social determinants, we risk oversimplifying the mechanisms driving depression and overlooking the complex ways in which these factors interact with depressive symptom trajectories.

By including these social determinants, this study allows for a more thorough exploration of the complex interplay between social factors and depressive symptom subtypes. In understanding the social determinants most associated with specific clusters of depressive symptoms, policymakers may gain more insight into possible areas of intervention over the life course. Such interventions could be more effectively targeted if we understand how social determinants influence both depressive statuses and transitions between these statuses. For example, transitions between depressive statuses may be strongly affected by changes in health, social support, or socioeconomic status, offering critical information for policy interventions aimed at reducing the burden of depression among vulnerable populations (Mirowsky & Ross, 1992; Platt et al., 2020).

Of those studies examining subtypes of depression longitudinally, only two include multiple covariates (Lamers et al., 2012; Ni et al., 2017). The first study is a small clinical sample of 488 respondents who are all diagnosed with MDD. These scholars examine various clinical, psychosocial and health covariates. They report a 3-class model where 76% of their respondents retained their depression status after 2-year follow-up, the majority of them classified in the *severe atypical class* (Lamers et al., 2012). This study reports that individuals who identify as women, are more overweight, have a great number of negative life events and higher neuroticism and lower functioning scores are more likely be in either the *severe typical* or *severe atypical* class than in the

moderate class. A more recent study conducted by Ni and colleagues (2017) examines a community sample of 1,783 older adult respondents over 2-years. They report a 3-class model fitting their data as well as finding 53% of individuals remaining stable from baseline to follow-up. In this second study, scholars report that those with chronic diseases, having difficulty with activities of daily living, and those who smoke are more likely to be classified in the *severe depression* class.

In both studies, the scholars highlight the importance of covariates in understanding how social, health, and psychological factors shape depression statuses and transitions (Lamers et al., 2012; Ni et al., 2017). However, despite these insights, few studies comprehensively examine multiple social determinants in relation to depressive subtypes over time. By including numerous social determinants in this analysis, I aim to provide a more comprehensive model of depression, examining not just a single-cause mechanism but how an array of social factors interacts to influence depressive outcomes. This approach offers a broader understanding of depression that can inform more targeted interventions and ultimately help address the "global burden" of depression across different stages of life (Assari, 2017; Ferrari et al., 2013; Liu et al., 2020).

AIMS OF THIS STUDY

In the current study, I use latent class and latent transition analysis (LCA/LTA) to investigate the connection between tangible social capital—defined as resources gained through social networks and participation in social activities—and various subtypes of depressive symptoms in older adults. In this study I first estimate a latent class analysis (LCA) at age 54, representing middle age, and then again in early later life, at age 72. Based upon previous literature, my first aim states that I will find more than one type of

manifestation of depressive symptoms at each of the two points. Based on prior research supporting the curvilinear nature of depression from mid-to-late life (Clarke et al., 2011; Mirowsky & Ross, 1992; Yang, 2007), my second aim states that the nature of the depressive symptom classes will worsen between midlife and early later life. In the case of LCA/LTA, worsening here would refer to a greater proportion of members of the class reporting specific symptoms. After modeling these two LCAs, my third aim is to estimate a latent transition analysis (LTA) to examine how individuals either remain in their current depressive statuses or transition between statuses from age 54 to age 72. Less research has focused on understanding these stabilizations and transitions. Finally, I will investigate how tangible social capital and other covariates impact the LTA models to capture the social and behavioral characteristics associated with baseline depressive status as well as stable or transitional statuses. I hypothesize that tangible social capital measures will be associated with affective symptoms of depression at baseline as well as during depressive symptom transitions from mid-to-early late life. These findings will contribute both empirically and theoretically to the literature on social capital and depression as well as possibly identify different types of depressive symptom statuses and social factors associated with them.

DATA AND METHODS

DATA

The data used in this study come from the Wisconsin Longitudinal Study (WLS), which is a long-term study of a random sample of men and women who graduated from Wisconsin high schools in 1957 (*N*=10,317). Respondents were first interviewed their senior year in 1957 (age 18). The cohort was subsequently followed up in 1964 (age 25),

1975 (age 36), 1992 (age 53), 2004 (age 65), and 2011 (age 72) using a combination of follow up techniques including in-person, telephone, and mail surveys. The data used in this analysis come from the years 1992 and 2011. The 1992 was the first wave that included the depressive symptoms battery. Overall, retention for the WLS is high, with response rates from the 1992 wave (87%) and 2011 wave (74%) both at or above 74%, excluding deaths. After removing individuals who did not remain in the study in 1992 (*N*= 1,824), those who did not have valid data on all depressive symptom variables in 1992 (*N*= 2,002), individuals dropped out of the study before 2011 as well as those who did not have valid data on all depressive symptom variables in 2011 (*N*= 2,480) and finally those who had valid data on all covariates of interest including genetic data to end up with the final analytic sample of 3,197 (See Appendix 3.1 A).

It is important to note that the WLS does not represent all strata of the U.S. population. Due to its design, all members of the WLS have graduated high school. Additionally, almost all participants in the study are non-Hispanic Whites. In the late 1950s there were fewer than 3% of Wisconsin residents identifying as nonwhite (Wisconsin Legislative Reference Bureau, 2015). As such, the following results cannot be generalized to the U.S. older population.

OUTCOME VARIABLE

This study uses the Centre for Epidemiological Studies-Depression scale (CES-D) for the outcome variable as a measurement of depressive symptoms (Radloff, 1977). This was measured by mail survey in 1992 and 2011. I used the modified version of the CES-D which only asks 10 of the initial 20 question index (Van Dam & Earleywine, 2011). These 10 questions originally had a 4-point range from 0 to 3 in the WLS (less than 1

day, 1-2 days, 3-4 days, and 5-7 days). I chose to dichotomize all answers: (1) 1 day or more and (0) less than 1 day. While dichotomizing the outcome variable may result in losing out on the variance of depressive symptoms, I am most interested in understanding if an individual did or did not experience a certain depressive symptom. In eliminating the various levels of magnitude, I can more readily examine the various subgroups of depressive symptoms using latent class analysis.

SOCIAL CAPITAL

Tangible social capital was measured using three variables: social support, social involvement, and social integration. Social support was created as an index of two questions that asked whether a respondent had a person in 'your family' and then 'a friend' with whom they could 'share [your] very private feelings and concerns.' Each of these questions were measured by mail survey and dichotomized 'no' (0) and 'yes' (1). I then summed them to construct an index so that a higher value refers to sources of social support both inside and outside the family.

Social involvement was measured by mail survey as a series of 17 questions that asked if an individual participated in various social activities such as church, parent-teachers' associations, and community centers. I dichotomized each individual question as 'no' (0) and 'yes' (1) and then summed all 17 questions to construct an index. Higher values suggested greater levels of social involvement in the community. Previous studies have similarly constructed an index measure (Veenstra, 2000).

There were three measures of social integration. Two questions in the mail survey asked the respondent, "How many times in the past four weeks have you gotten together with family?" and then "with friends?" I recoded both questions so that (0) spent no time,

(1) if they spent up to a total of four times, and (2) if they spent more than four times in the past four weeks. Marital status was the third measurement of social integration. Marital status was a simple indicator that asked an individual if they were married, not married, widowed, or divorced. The original variable also asked if individuals were separated, but I combined separated and divorced as one indicator. Prior research suggests that married individuals are more socially integrated than single individuals due to access to their spouse's network (Wong & Waite, 2015).

COVARIATES

The following covariates were chosen based on their association with depression: educational attainment (J. Lee, 2011), total assets (Assari, 2017), self-rated health (Han, 2002), and the polygenic score for depression (Turley et al., 2018). Each of these variables were measured in 1992, besides the polygenic score for depression, and were used to predict baseline depression status and depression status transitions, through latent transition analysis (LTA). The polygenic score for depression was constructed in 2018 using the second wave of genetic data that the WLS collected in 2007-8 as well as 2010 (*WLS Genetic Data Analysis*, n.d.).

Educational attainment was measured by phone survey. I used dichotomous indicators for the participant's highest level of attainment, which included graduation from high school, completion of some college, and graduation from college. These three variables were constructed using the highest level of education as reported in 1992.

Total assets was obtained by phone survey as an index of the respondent's home equity; respondent's and spouse's retirement plans; respondent's and spouse's checking accounts, savings accounts, or money market funds; respondent's and spouse's CDs,

government saving bonds, or treasury bills; and the total cash value of life insurance policies**.** Due to the skewness of total assets, I divided it into three roughly equal categories (33%) of 'low,' 'medium,' and 'high' assets, which had a range from 1 to 3.

Self-rated health was obtained by mail survey and was dichotomized where 0 equals poor health (very poor, poor, and fair health) and 1 equals good health (good and excellent health). Sex of respondents was a dichotomous variable (1=female).

Finally, the polygenic risk score for depression was also included as a covariate. A polygenic risk score is an aggregate of DNA variants used to estimate the likelihood of a specific disease outcome (Sugrue & Desikan, 2019). The two types of polygenic risk scores are the Genome-Wide Association Score (GWAS) and the Multi-Trait Analysis of GWAS (MTAG), which are both available in the WLS. The GWAS scans markers across complete sets of DNA of many individuals to examine genetic variations associated with a specific disease. Meanwhile, MTAG score allows for analysis of multiple diseases across complete sets of DNA of many individuals, which boosts the statistical power for detecting genetic association (Turley et al., 2018). As such, this study will use the MTAG depression scores in the analysis. This variable was standardized for ease of interpretation.

DATA ANALYSIS

The current analysis used a three-step latent transition analysis (LTA) approach (Asparouhov & Muthén, 2014; Collins & Lanza, 2010), using Mplus version 5. This three-step approach involves first running an initial latent class analysis (LCA) at each time point, then testing for measurement invariance between the two LCA models, and

finally estimating a latent transition analysis (LTA) to examine how class membership changes overtime.

LCA is a method that uses respondents' answers to a number of items to uncover latent groups (Collins & Lanza, 2010; Vermunt, 2010). Instead of the term latent class, latent status is used in this article to indicate that class membership might be temporary and change across waves. In this paper I ran an LCA using the 1992 data from the revised CES-D to determine if there were distinct and unique statuses. I began by estimating a 1-status model, meaning one classification was fitting for all individuals, and then continued estimating various status models until they were no longer able to separate qualitative differences in classes by subjective interpretation. Throughout this process I tracked Bayesian information criterion (BIC) as the fit statistic to determine the number of statuses at each wave (Collins & Lanza, 2010). BIC is a model selection fit statistic that adjusts the model log-likelihood for both sample size and number of parameters estimated (Schwarz, 1978). I then determined the best fitting number of latent statuses for both time points by comparing the models and choosing the model with the lowest BIC value. When comparing models a BIC difference between 0 and 2 indicates weak evidence, 2 to 6 indicates positive evidence, 6 to 10 strong evidence, and 10 and above indicates very strong evidence (Raftery, 1995).

The second-step includes testing for measurement invariance between the best fitting LCA models at the two time points, as recommended by Collins and Lanza (2010) when examining classes across time. As such, I assessed whether the depressive symptoms classes from 1992 were the same as the depressive symptom classes in 2011. This required me to examine quantitatively if these models were identical to determine

whether the characteristics of these statuses changed over this 19-year period or they remained the same. Therefore, I examined various types of measurement invariance to determine if any classes were identical or if any or all adjusted during this 19-year period. BIC was used as the fit statistic to determine the best fitting model when considering measurement invariance.

The third step included estimating an LTA model. LTA is the longitudinal extension of LCA that examines the probabilities of an individual either transitioning or retaining their status between time points (Collins & Lanza, 2010). In this current model, the LTA estimates the probabilities of both the transitions and retentions of individuals from time point 1 (1992) to time point 2 (2011) for the best fitting number of classes.

MPlus estimates probabilities for each individual to determine which group they are most likely to be classified in at midlife and early later-life. Using these estimates, I assigned each respondent to the class they had the highest probability of being classified in by creating categorical latent group variables in Stata. I then used Stata to estimate a multinomial logistic regression using the categories acquired from the MPlus probability estimates. The primary independent variables of interest were measures of tangible social capital and other covariates included sex of respondents, educational attainment, total assets, self-rated health, - all from the year 1992 - and the polygenic score for depression. Each of the covariates was permitted to affect the probability of baseline depression status as well as the probabilities of transitioning during this 19-year period.

RESULTS

LATENT CLASS ANALYSIS (LCA)

After examining baseline data for midlife, BIC favored a four-class model, which is displayed in Table 3.1. As Table 3.2 displays, the four-class model includes classes which I labeled *Depressed Affect and Somatic Symptoms, Depressed Affect, Somatic Symptoms,* and *Not Depressed*. The first status – *Depressed Affect and Somatic Symptoms* – experienced a lot of different symptoms. I labeled the second status *Depressed Affect,* which included participants who were likely to state that they felt depressed (61%) and also indicated that they were lonely (52%). The third status I labeled *Somatic Symptoms*. Unlike the first two classes, members of this third class did not identify themselves as depressed; rather, these individuals reported having trouble keeping their mind on tasks (61%), finding that everything was an effort (55%), sleeping poorly (59%), and being unable to get going each day (59%). The final status, *Not Depressed*, experienced few, if any, of the symptoms.

When analyzing the data for individuals in later life, BIC favored the 5-class model over the 4-class model by about 3.81 units, corresponding to weak positive evidence. However, I ultimately chose the 4-class model since the 5-class model did not have distinctive statuses of depression qualitatively (Table 3.2A). This means, when examining the data of the 5-class model there was no qualitative explanation to clearly differentiate the various classes. Data presented in Appendix B shows Class 3 is only 1% of respondents and is very similar to Class 1 besides the question, "Are you hopeful for the future?" Due to these classes being similar as well as Class 3 being so small, I followed previous scholars using LTA who have also chosen the best fitting model based on both BIC values and qualitative interpretability among classes (Mezuk & Kendler, 2012). Therefore, the four-class model carried over to the 2011 wave and included the

same statuses *Depressed Affect and Somatic Symptoms, Depressed Affect, Somatic Symptoms,* and *Not Depressed*.

Interestingly, in both mid-life and early later-life the *Not Depressed* status was the largest group. In mid-life this classification accounted for 35% of participants, which then grew to 45% in early later-life. *Somatic Symptoms* was the only other depressive symptom classification that increased from mid-life to early later-life. In mid-life this depressive symptom class accounted for 25% of respondents, which then increased to 34%. Meanwhile, both *Depressed Affect and Somatic Symptoms* as well as *Depressed Affect* statuses decreased from mid-life to early later-life.

MEASUREMENT INVARIANCE

The second step of this three-step process was to test whether the four classes identified at age 72 were quantitively identical to those at age 54. Table 3.1 contains the fit statistics for the various equality-constrained models. The best-fitting model according to BIC suggests a model that constrains 3- classes. *Depressed Affect and Somatic Symptoms, Depressed Affect, and Not Depressed* to be invariant over time, meaning these statuses were identical in terms of item-response probabilities at age 54 and age 72. The status *Somatic Symptoms*, however, adjusted from age 54 to age 72 where individuals experienced a slightly higher probability of somatic symptoms. When comparing the results in Table 3.2 and 3.3 we can see that while trouble keeping one's mind on a task improved from mid-life to later life, finding that everything was an effort, bad sleep, and being unable to get going each day each increased by around 10 or more percentage points each. Overall, these results support aim 1 that more than one depression subtype would be found at the two time points.

LATENT TRANSISTION ANALYSIS (LTA)

After determining measurement invariance, the third step is to estimate an LTA model without covariates to examine if participants were more likely to remain stable or transition between these depressive symptom statuses. As Table 3.4 shows, the majority of respondents were more likely to remain stable. Of the three depressive symptom statuses that remained the same between midlife (age 54) and later life (age 72), two of the three statuses had above a 50% chance of remaining stable. Respondents in the *Not Depressed* status in midlife had a 78% chance of remaining in that status in later life and respondents in the *Depressed Affect and Somatic Symptoms* status in midlife had a 59% chance of remaining in that status in later life. Finally, respondents in the *Depressed Affect* in midlife had a 43% chance of remaining in that status in later life. However, there were a few key transitions that occurred between midlife and later life. Interestingly, one of these transitions suggest a worsening in depressive symptom status while the other three transitions coincide with an improvement in depressive symptoms.

I will begin with the transition of those who experienced a worsening in depressive symptoms. Those in the *Not Depressed* status at age 54 had a 16% chance of moving to the *Worsening Somatic Symptoms* status at age 72, indicating specifically that these individuals experienced higher levels of somatic symptoms in later life. The next three transitions point to an improvement in depressive symptom status. First, those in the *Depressed Affect* status at age 54 had a 37% chance of transitioning to the *Not Depressed Status* at age 72. Similarly, those in the *Somatic Symptoms* status at age 54 had a 25% chance of moving to the *Not Depressed* status at age 72. The final transition of interest was that those in the *Depressed Affect and Somatic Symptoms* status at age 54 had a 24%

chance of transitioning to the *Worsening Somatic Symptoms* status at age 72, indicating that while their depressed affect symptoms may have subsided, their somatic symptoms remained.

BASELINE PREDICTORS

I then examined how covariates impacted the baseline depressive status at age 54. In performing multinomial logistic regression, I used *Not Depressed* status as the reference to compare against the three different statuses of depressive symptoms. These results can be found in Table 3.5. Females were most likely to be in the three depressive symptom baseline statues as opposed to being in the *Not Depressed* status. Females had the greatest odds of being classified in the *Depressed Affect and Somatic Symptoms* status (OR= 1.73; $p<0.001$) followed by *Depressed Affect s*tatus (OR=1.68; $p<0.001$) and finally odds of 1.31 ($p<0.01$) of being in the *Somatic Symptoms* status. Self-rated health was predictive of two of the depressive symptom statuses – *Depressed Affect and Somatic Symptoms* and *Somatic Symptoms* – compared to being in the *Not Depressed* status at baseline. Those who rated their health as good/excellent at age 54 had lower odds (OR=0.25; $p<0.001$) of being classified in the *Depressed Affect and Somatic Symptoms* status as well as lower odds (OR=0.56; $p<0.01$) for being grouped in the *Somatic Symptoms* status opposed to the *Not Depressed* status at baseline. Individuals had lower odds of being in the *Somatic Symptoms* status opposed to the *Not Depressed* status (OR=0.10; $p<0.01$) if they reported higher logged net worth. Similarly, those who reported higher logged net worth also had lower odds of being in the *Depressed Affect and Somatic Symptoms* status opposed to the *Not Depressed* (OR=0.15; $p<0.001$). Education was only significantly associated with one depressive status at age 54.

Individuals with some college education opposed to having a college degree had lower odds of being classified in the *Somatic Symptoms* status opposed to the *Not Depressed* status (OR=0.35; $p<0.05$).

A number of the tangible social capital variables were also predictive of baseline depression status. Individuals who spent a lot of time with friends had lower odds of being classified in the *Depressed Affect and Somatic Symptoms* status compared to the *Not Depressed* (OR=0.61; $p<0.01$). Marital status, one of the measures of social integration, presented a series of interesting results. Individuals who identified as never married as opposed to married had greater odds of being classified in the *Depressed Affect and Somatic Symptoms* status compared to the *Not Depressed* status at age 54 (OR=2.23; $p<0.01$). Divorced individuals had greater odds of being classified in either the *Depressed Affect* status (OR=1.45; $p<0.05$) or the *Depressed Affect and Somatic Symptoms* status (OR=1.51; $p<0.05$) compared to the *Not Depressed* status at age 54, where married is the reference category. Additionally, respondents who were widowed had greater odds of being classified in either the *Depressed Affect and Somatic Symptoms* status (OR=4.09; $p<0.001$) or the *Depressed Affect* status (OR=2.46; $p<0.05$) compared to the *Not Depressed* status at age 54, where married is the reference category. Social support was also predictive of depressive symptom statuses at age 54. Those who stated they had some level of social support had lower odds of being classified *Depressed Affect and Somatic Symptoms* status opposed to the *Not Depressed* status (OR=0.48; $p<0.001$). Similarly, those who stated they had high levels of social support were also at lower odds of being classified *Depressed Affect and Somatic Symptoms* status opposed to the *Not Depressed* status (OR=0.38; $p<0.001$). Additionally, those who once again rated their

social support as high were less likely to be classified in the *Depressed Affect* status compared to the Not Depressed status (OR=0.55; p<0.01). Finally, individuals with greater social involvement were more likely to be classified in either the *Somatic Symptoms* status (OR=1.29; p<0.001) or the *Depressed Affect and Somatic Symptoms* status (OR=1.19; p<0.05) compared to the *Not Depressed* status at age 54.

Overall, these findings suggest those who spend less time with friends, are never married, divorced, or widowed, report lower levels of social support, and who have high levels of social involvement were more likely to be classified as *Depressed Affect and Somatic Symptoms* status opposed to the *Not Depressed* status. Individuals who report they are divorced, widowed, or do not have high levels of social support were more likely to be classified as *Depressed Affect* status opposed to the *Not Depressed* status. The only tangible social capital variable associated with greater odds of being classified in the *Somatic Symptoms* status opposed to the *Not Depressed* status was increased social involvement. As such, tangible social capital variables are each predictive of the different types of depressive symptoms.

This study also considered how genetics influence depression subtypes in mid-life and later life. Polygenic score of depression was another key predictor for depressive symptom statuses at age 54. For both *Depressed Affect and Somatic Symptoms* (OR=0.89; p<0.05) and the *Somatic Symptoms* (OR=0.90; p<0.05) statuses, individuals were less likely to be classified in either of those depressive symptom statuses opposed to the *Not Depressed* status if their polygenic score was a higher score. However, this is surprising given that a higher score for the polygenic variable should indicate more risk of depression.

STABILITY AND TRANSITION PREDICTORS

Following the examination of baseline predictors, I analyzed how covariates were associated with individuals remaining in their depressive statuses or transitioning from midlife to early later-life. However, it is important to note that these covariates were measured in midlife and thus these things could have changed by later life. As such, this analysis is only accounting for these covariates retrospectively.

When analyzing how these covariates were associated with individuals who remained stable in the three depressive status, there was only one significant covariate in one of the statuses. Given this, I have included this table in Appendix Table 3.4 A. For both the *Not Depressed* and *Depressed Affect*, there were no significant covariates for remaining stable from midlife to later life. However, for *Depressed Affect and Somatic Symptoms* status there was one significant covariate predictive of remaining stable, which was self-rated health in midlife. Individuals who rated their health as good/excellent had a lower chance of remaining in this status in later life.

Table 3.6 displays each of the more common transitions - those with a conditional probability of 15% or greater - and the key predictors of these transition statuses. There are a total of 4 different depressive symptom statuses experiencing above 15% transitions. Three of these transitional statuses result in individuals experiencing an improvement in reported depressive symptoms: *Depressed Affect and Somatic Symptoms* transition to the *Not Depressed* status in later life as well as those being classified in *Depressed Affect and Somatic Symptoms* status at midlife transitioning to *Worsening Somatic Symptoms* in later life. The final transitional status both resulted in individuals

being classified in the *Not Depressed* status in midlife to then transitioning to *Worsening Somatic Symptoms*.

In general, covariates were not predictive of these depression status changes from mid-to-later life. As you will see in Table 3.6, the majority of covariates were not statistically significant in being associated with these transitions. However, I will discuss the few covariates that were significantly associated with these depressive status transitions. Individuals who were initially classified in the *Depressed Affect* and *Somatic Symptoms* status in midlife had greater odds (OR=1.87; $p<0.06$) to transition to *Worsening Somatic Symptoms* in later life if they rated their health as good/excellent, rather than poor in midlife. Similarly, individuals who rated their health as good/excellent in midlife had greater odds (OR=2.10; $p<0.05$) of transitioning from *Somatic Symptoms* status in midlife to *Not Depressed* status in later life. In both cases, rating one's health as good/excellent was associated with an improvement in depressive symptoms status in later life. The transitional group of moving from *Somatic Symptoms* status in midlife to *Not Depressed* status in later life also had two key tangible social capital variables associated with it. Individuals who reported high levels of social support had greater odds (OR=2.40; $p<0.05$) for experiencing this transition. Individuals who reported greater logged levels of social involvement also had lower odds (OR= 0.71; $p<0.05$) of remaining in the *Somatic Symptoms* status in later life compared to transitioning to the *Not Depressed* status.

DISCUSSION AND CONCLUSION

This study examined depression as a heterogenous illness to better understand how depressive symptom patterns may present themselves over the life course. As such, I

analyzed depressive symptom statuses at mid-and early-late life, stability and transitions in these statuses, as well as how tangible social capital and other key social factors related to depression are associated with both baseline and transitions among Wisconsin Longitudinal Study participants. My first aim stated I would find more than one manifestation of depressive symptoms at each of the two time points. My data confirmed this as I found four depressive statuses. These depressive symptom statuses at both mid- and (age 54) early later-life (age 72) were based on respondents' responses to the Center for Epidemiological Studies-Depression revised scale (CESD-R). My second aim states that the nature of the depressive symptom classes will worsen between midlife and early later life. Of these four statuses, three remained constant while one, somatic symptoms, adjusted at age 72 where the item-response probabilities suggested an increase in somatic symptoms in early later-life. This aligns with the broader literature and the observation discussed in the introduction that somatic symptoms are more prevalent among older adults. These findings suggest that as individuals age, physical health challenges and the overlap of biological and social changes may contribute to the prominence of somatic symptoms in depressive presentations (Gallo et al., 1994). Finally, my third aim was to examine how tangible social capital and other social factors were associated with baseline as well as stable or transition statuses in depressive symptoms. As the findings section discussed, these results are more mixed. However, the overarching results of this study indicate the importance of continuing to examine different types of depressive symptoms present from mid-to early later-life as well as improve our understanding of how tangible social capital and other social determinants of health can result in increased depressive symptoms.

DEPRESSION AND THE LIFE COURSE

Previous scholars have highlighted the relationship between depressive symptoms and age as one characterized by a U-shape (Clarke et al., 2011; Mirowsky & Ross, 1992; Yang, 2007). However, prior literature examining this relationship has solely focused on depressive symptoms as homogenous, meaning these studies either identified respondents as depressed or not depressed, and did not examine if different types of depressive symptoms act differently across the life course. Instead of treating depression as homogenous, the current research hones in on analyzing how different statuses, or groupings, of depressive symptoms are associated with age from midlife to early later-life.

When thinking about the curvilinear characterization of depression over the life course, midlife would represent a time on the curve when depression is at its lowest and early later-life would be when depression starts to rise. Thus, the second aim of this research suggested that the nature of these depressive symptom statuses would worsen. This would result in classes in 2011 representing worsening depressive symptoms, similar to how in midlife there was a *Somatic Symptoms* class and in 2011 there was a *Worsening Somatic Symptom* class. However, the current study found far more stability than expected when examining the nature of depressive symptom classes between midlife and early later-life. When comparing Tables 2 and 3, three of the four depressive symptom latent statuses qualitatively represented the same classification at these two time points. As such, this research raises a few interesting questions for future studies to continue assessing if these different types of depressive symptoms better represent the association between depression and age across the life course.

Future research should explore the mechanisms that drive the stability of depressive symptom subtypes across different life stages and investigate whether certain factors, such as social capital, health status, or life events, contribute to this stability. Additionally, longitudinal studies examining how transitions between different depressive symptom statuses occur over time could provide insights into the triggers or protective factors that influence these shifts. Understanding the role of biological, psychological, and social determinants in shaping specific symptom patterns may help tailor interventions to target the unique needs of individuals experiencing different depressive trajectories across the life course.

DEPRESSIVE SYMPTOM STATUSES AND LATENT CLASS/TRANSITION ANALYSIS

The results in this paper support prior research that has examined depression as a heterogenous illness (Hybels et al., 2011) and try and address the critique of depression scales simply adding up symptoms and disregarding the qualitative nature of which symptoms are present (Harald & Gordon, 2012). Like previous studies, this study analyzed these patterns of depressive symptoms using latent class and latent transition analysis (Lamers et al., 2012, 2016; Ni et al., 2017; Rodgers et al., 2014; Ulbricht et al., 2018). This current study examined depressive symptoms of individuals at age 54 and 72. The current study found four depressive symptom classes at both age 54 and 72. These results suggest four subtypes of depression exist in mid-life (age 54) and early later-life (age 72). The four subtypes of depressive symptoms reported at mid-life include *Depressed Affect and Somatic Symptoms*, *Depressed Affect*, *Somatic Symptoms*, and *Not Depressed*. Similarly, these four subtypes remain the same in early later-life except for *Somatic Symptoms*, which is found to worsen at age 72, meaning individuals report an

increase in somatic symptoms. However, when comparing these depressive symptom statuses to prior research, a clear pattern of depressive symptom subtypes is not present. Given those previous scholars have examined both different age groups, there is not enough evidence to conclude exact subtypes of depression. Additionally, cohort effects could be a possible reason for the difference in depressive symptom classes between these studies. Previous studies examining depression as a homogenous illness have reported differences in depression rates between cohorts (Blazer et al., 1991; Platt et al., 2020). These differences may reflect cohort effects driven by generational shifts in social, economic, and cultural conditions that shape depressive symptom outcomes. For instance, more recent cohorts have experienced greater educational and employment opportunities for women, as well as shifts in the division of domestic labor, which may reduce stressors historically linked to higher depression rates among women (Belle & Doucet, 2003; Platt et al., 2020). Additionally, changing societal attitudes toward mental health, increased awareness, and improved access to mental health care could influence how depressive symptoms are recognized and reported across generations (Corcoran et al., 2013; Nair et al., 2020). These cohort effects highlight how structural and societal factors intersect with age-related patterns of depressive symptoms, highlighting the need to examine depression through a life course and generational lens.

One area of interest in scholarship examining transitions in depressive symptom subtypes is whether there is much transition that exists (Ni et al., 2017). These prior studies have reported very little transition occurring between these different subtypes of depressive symptoms (Ni et al., 2017; Rodgers et al., 2014; Ulbricht et al., 2018). The current study also found very little transition between these different subtypes across a

much longer period of time. As such, this may suggest clinicians better understand the patterns of depressive symptoms present and to focus on treating those given that these tend to remain present with the individual. Yet, given the discrepancies between these studies based on using a broader range of ages (18-65) as well as if it was a community or clinical sample, it is hard to make true comparisons.

Understanding these depressive symptom patterns can allow for more targeted outreach to aging adults as well as more targeted intervention. While Lee and colleagues (S. Y. Lee et al., 2014) did not examine depressive subtypes for aging adults, their research did emphasize the importance of targeted interventions based on racial and ethnic differences in depressive symptoms. Racial, ethnic, and/or cultural differences might be why somatic symptoms manifest in others despite stating they are not depressed. As such, further research needs to delineate how depressive symptoms might be more prevalent between different populations.

DEPRESSIVE SYMPTOM STATUSES AND TANGIBLE SOCIAL CAPITAL

The association between depressive symptoms and tangible social capital has previously been examined when analyzing depressive symptoms as a homogenous illness (Carr, 2020; Forsman et al., 2012; Webber et al., 2011). These prior studies revealed tangible social capital to be inversely associated with depressive symptoms both at midlife (Giordano & Lindström, 2011; Nakamine et al., 2017) as well as in later life (Cao et al., 2015; Forsman et al., 2012, 2013). This association is theorized to be due to older adults becoming more dependent on others due to age-structural changes. These age-structural changes such as moving to live with family members or an assisted living center result in older adults' social networks dwindling, which results in these individuals

being unable to pull from their social resources. However, these studies have not examined how tangible social capital is associated with different subtypes of depression. It is necessary to parse out potential differences between types of depression to more effectively intervene at different stages of life, particularly in later life when individuals experience a decline in tangible social capital.

Results from both the baseline depressive statuses as well as the stable and transitional statuses highlight the different impact various forms of tangible social capital can have on depression. In general, baseline data points to more forms of tangible social capital being associated with the *Depressed Affect and Somatic Symptoms* status, as compared to the *Not Depressed* status. This is particularly true when examining how one's social support impacts depression. When analyzing stability and transitions from midlife to early later-life, the findings were less revealing when focusing on tangible social capital. When considering the depressive symptom statuses that remained stable between these two time points, tangible social capital was not associated with them. However, when analyzing the transitions, two forms of tangible social capital, social support and logged social involvement, were significantly associated with transitioning from the *Somatic Symptoms* status to the *Not Depressed* status. Individuals who reported high levels of social support had greater odds (OR=2.40; $p<0.05$) of experiencing this transition. Individuals who reported greater logged levels of social involvement also had lower odds (OR= 0.71; $p<0.05$) of remaining in the *Somatic Symptoms* status in later life compared to transitioning to the *Not Depressed* status. Despite these limited results, this suggests scholars must continue examining different types of depressive symptom statuses and their association with tangible social capital. This is further emphasized

when comparing results from prior studies that found many forms of tangible social capital to be significantly associated with depressive symptoms (Carr, 2020; Forsman et al., 2012; Landstedt et al., 2016). Yet, as we see in this paper, when analyzing forms of tangible social capital between different statuses, these associations differ. In parsing out these associations, policymakers will potentially be able to individualize treatment based on the type of depression rather than continuing to treat and care for it as a homogenous mental illness.

The results of this study may reflect the complex and context-dependent nature of tangible social capital's influence on specific depressive symptom subtypes. For instance, the lack of association between tangible social capital and stable depressive statuses suggests that while social capital variables like social support and involvement can facilitate transitions out of depressive states, they may be less effective in preventing the persistence of depressive symptoms once established. This may occur because stable depressive symptoms, whether affective or somatic, could be more strongly driven by entrenched biological or psychological factors that are less responsive to changes in social structures. In contrast, the observed associations with transitions suggest that tangible social capital, such as increased social support, may be particularly beneficial during critical periods of change, when individuals are more vulnerable to shifts in their mental health status.

Critical periods of change may be provoked by significant life events or stressors, such as retirement, bereavement, or changes in health status, which can disrupt established routines and increase susceptibility to depressive symptoms (Fiske et al., 2009; Leung et al., 2023). Individuals who transition out of depressive statuses may differ

from those who remain stable in their ability to leverage social resources during these periods. For example, those with stronger social networks or higher self-efficacy might be better positioned to access and benefit from social support during times of vulnerability. Conversely, individuals who remain in stable depressive status may face additional barriers, such as lower socioeconomic status, chronic physical illness, or a lack of perceived support, which prevent them from utilizing social capital effectively. The finding that greater social involvement lowers the odds of remaining in the *Somatic Symptoms* status implies that tangible engagement in social activities might help disrupt the cycle of physical symptoms, possibly by fostering a sense of routine and reducing social isolation. It is important to note, however, that while this study highlights the potential impact of critical life events on depressive symptom transitions, it did not directly test whether events such as retirement, bereavement, or health changes predict these transitions. Future research should explore these dynamics to better understand how specific stressors contribute to depressive symptom trajectories (Carr, 2020; Forsman et al., 2012; Landstedt et al., 2016).

These findings highlight the importance of considering the timing and specific nature of social interventions, suggesting that enhancing tangible social capital factors could be more effective during transitional phases rather than as a sustained buffer against ongoing depressive symptoms. Overall, the differential impact of tangible social capital across depressive symptom statuses emphasizes the need to move beyond a one-size-fits-all approach, recognizing that the influence of social resources can vary considerably depending on the type and stability of depressive symptoms experienced.

LIMITATIONS

This study is limited in several ways. The sample size of the Wisconsin Longitudinal Study is limited to white adults who all graduated high school and is only one birth cohort. Thus, the current depressive symptom subtypes may not be applicable to non-white adults as well as adults with less than a high school diploma. Both educational attainment and race have been connected to levels of depressive symptoms (Chang-Quan et al., 2010; Ladin, 2008; S. Y. Lee et al., 2014). Lower educational attainment has previously been found to be associated with increased depressive symptoms in later life due to these individuals having less self-efficacy. As such, this prior research has found adults with lower levels of education feel as though they have less control over their life, which may result in increased depressive symptoms. Additionally, racial and ethnic minorities have been found to report more physical symptoms than psychological symptoms in the U.S. when comparing them to whites (Deisenhammer et al., 2012; S. Y. Lee et al., 2014; Nadeem et al., 2009). The inclusion of a more diverse sample, both when accounting for education and race, may result in different depressive symptom patterns given these associations found in prior research.

Second, there are limitations regarding the method of assessing these classes at age 54 and 72. For example, I relied on using the revised Centre for Epidemiological Studies-Depression scale (CES-D) when performing LCA. While the CES-D is a highly regarded and consistently used measure of depressive symptoms, it is possible that if I used another depression scale, such as the Beck Depression Inventory, the EQ-5D, or the Hamilton Depression Rating Scale, I may have obtained different results for these depressive symptoms classes. This can help explain while previous studies have been unable to find consistent depressive symptom subtypes (Hybels et al., 2013; Ulbricht et

al., 2018; Veltman et al., 2017). Future studies should examine if using different depression scales results in a series of different depressive symptom classes at these two ages.

Third, a key limitation of this study is that it does not account for potential confounders, such as changes in employment circumstances, health status, or major life events, which are known to significantly influence both depressive symptoms and social capital across the life course (Cao et al., 2015; Cohen-Cline et al., 2018; O'Connor et al., 2011; Webber et al., 2011). Transitions such as retirement, bereavement, caregiving responsibilities, or the onset of chronic illnesses are particularly common between mid and later life and can profoundly shape an individual's mental health and access to social resources (Clarke et al., 2011; Moen & Sweet, 2004). However, this study intentionally limited its analysis to predictors measured no later than the time point of the first depressive symptom measurement at age 54. This analytical decision, aimed at establishing the temporal sequence of events and changes, excluded factors such as retirement or spousal caregiving that typically occur after this age. While this approach provides clarity on the relationship between earlier predictors and depressive symptom statuses, it does not capture the effects of later-life transitions on tangible social capital or mental health outcomes. Future research should incorporate longitudinal measures of tangible social capital that span mid-to-later life to better assess how shifting life circumstances influence social resources and depressive symptoms over time.

Finally, LTA models, similar to other methods used to assess intraindividual change, make it difficult to evaluate selective attrition (Moorman et al., 2020). As previously stated, LCA/LTA require participation in all waves, which means data may be

missing not at random (MNAR) since those participants who were more depressed may have dropped out of the study. Unfortunately, I am unable to assess if this is true for individuals who did not have data on depression, as 1992 was the first wave when the WLS requested information on mental health. However, I was able to assess if there was a difference between individuals who provided depression data at both midlife and early later-life. Upon analyzing these results, there were no statistically significant differences in any of the 10 depressive symptom variables when comparing means of those who provided data in both waves as compared to those who only provided data in 1992. Additionally, due to the inclusion of using genomic data as a covariate of interest from 1992, there may also be selective attrition based on healthier participants being more willing to provide genetic data. Unfortunately, I am unable to fully evaluate MNAR due to these statistical method requirements. As such, selective attrition may have impacted these results and should be further assessed in future studies.

CONCLUSION

Findings from this study contribute to a growing body of literature analyzing depressive symptom subtypes in mid- and early later-life (Hybels et al., 2013; Ulbricht et al., 2018; Veltman et al., 2017). These findings add further evidence for researchers to continue to examine the complexities of depression across the life course. This allows sociologists and other health affiliates to not only understand depression as a heterogenous illness (Hybels et al., 2011) but also to better assess the curvilinear nature of depressive symptoms across the life course by specifically accounting for the heterogeneity of depression. As this study revealed, when analyzing depression as heterogenous, there is far more stability between mid- and early later-life suggesting there

is not a U-shaped curve relationship. Furthermore, this research adds to scholarship examining social capital and depressive symptoms. Understanding depression as an illness partly affected by one's social resources allows individuals to better approach treatments and become less reliant on pharmaceutical solutions.

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TABLES

Table 3.1

Summary of Fit Statistics for Model Selection (Wisconsin Longitudinal Study, N *= 3,197)*

1992, Age 54					
Number of latent classes	Number of parameters estimated	G^2	*df*	BIC	ℓ
2	21	1116.1	997	32666.1	-16248.3
3	32	816.4	987	32450.6	-16096.2
4	43	684.2	977	**32390.5**	-16021.7
5	54	670.5	969	32418.9	-15991.6
6	65	607.8	957	32454.2	-15964.8
2011, Age 72					
Number of latent classes	Number of parameters estimated	G^2	*df*	BIC	ℓ
2	21	1204.6	996	30505.3	-15167.9
3	32	861.1	988	30221.7	-14981.7
4	43	762.1	979	30182.7	-14917.8
5	54	659.9	967	**30178.9**	-14871.6
6	65	627.3	958	30216.2	-14845.8
Measurement Invariance					
Number of constrained classes	Number of parameters estimated	G^2	*df*	BIC	ℓ
0	95			61605.1	-30419.2
1	85			61546.3	-30430.2
2	75			61511.1	-30452.9

3	65	**61476.2**	-30475.8
4	55	61514.9	-30535.5

Table 3.2

Four Latent Class Model of Depressive Symptoms Item-Responses Probabilities at Age 54 (Wisconsin Longitudinal Study, 1992, N = *3,197)*

	Latent Class			
Assigned Label	Depressed Affect and Somatic Symptoms	Depressed Affect	Not Depressed	Somatic Symptoms
Latent Class Prevalences	0.23	0.14	0.35	0.28
Item Response				
Bothered	0.68	0.39	0.10	0.31
Happy*	0.03	0.02	0.02	0.00
Lonely	0.70	0.52	0.07	0.18
Depressed	0.83	0.61	0.01	0.13
Trouble Keeping Mind	0.80	0.28	0.15	0.61
Effort	0.91	0.26	0.08	0.55
Hopeful Future*	0.07	0.08	0.08	0.04
Fear	0.71	0.32	0.08	0.23
Bad Sleep	0.79	0.64	0.31	0.59
Not Get Going	0.86	0.36	0.07	0.59

** Responses for Happy and Hopeful Future were both reverse coded.*

Table 3.3

Four Latent Class Model of Depressive Symptoms Item-Responses Probabilities at Age 72 (Wisconsin Longitudinal Study, 2011, N = *3,197)*

	Latent Class			
Assigned Label	Depressed Affect and Somatic Symptoms	Depressed Affect	Not Depressed	Worsening Somatic Symptoms
Latent Class Prevalences	0.14	0.07	0.45	0.34
Item Response				
Bothered	0.71	0.36	0.07	0.32
Happy*	0.02	0.05	0.03	0.01
Lonely	0.80	0.66	0.09	0.21
Depressed	0.75	0.69	0.02	0.09
Trouble Keeping Mind	0.88	0.37	0.12	0.56
Effort	0.97	0.42	0.11	0.65
Hopeful Future*	0.05	0.16	0.09	0.04
Fear	0.68	0.36	0.05	0.24
Bad Sleep	0.89	0.71	0.41	0.77
Not Get Going	0.94	0.51	0.10	0.71

** Responses for Happy and Hopeful Future were both reverse coded.*

Table 3.4

Probability of Transitioning to Age 72 Class Conditional on Age 54 Class

	Age 72 Class			
Age 54 Class	Not Depressed	Depressed Affect	Worsening Somatic Symptoms	Depressed Affect and Somatic Symptoms
Not Depressed	0.78	0.05	0.16	0.01
Depressed Affect	0.37	0.43	0.06	0.14
Somatic Symptoms	0.25	0.00	0.69	0.06
Depressed Affect and Somatic Symptoms	0.10	0.07	0.24	0.59

Table 3.5

Odds Ratios for covariates predicting baseline latent statuses of depression.

		Depressed Affect and Somatic Symptoms		Somatic Symptoms		Depressed Affect	
Covariates		*OR*	*SE*	*OR*	*SE*	*OR*	*SE*
Female		1.73***	0.19	1.31**	0.13	1.68***	0.19
Self-Rated Health		0.25***	0.04	0.56**	0.11	0.79	0.18
Logged Net worth		0.86***	0.04	0.90*	0.04	0.94	0.05
High School		1.10	0.13	1.20	0.13	0.81	0.10
Some College		1.19	0.19	1.42*	0.20	0.91	0.15
College		Ref.		Ref.		Ref.	
Friends							
	1	0.81	0.13	0.78	0.12	0.94	0.17
	2	0.61**	0.11	0.78	0.13	1.11	0.22
Relatives							
	1	1.34	0.21	1.14	0.16	1.13	0.19
	2	1.20	0.23	1.08	0.18	0.91	0.18
Not Married		2.23**	0.68	1.46	0.44	1.80	0.59
Divorced		1.51*	0.26	0.87	0.15	1.45*	0.26
Widowed		4.09***	1.37	0.61	0.28	2.46*	0.91
Married		Ref.		Ref.		Ref.	
Social Support							
	1	0.48***	0.10	0.80	0.17	0.82	0.19
	2	0.38***	0.07	0.76	0.15	0.55**	0.12
Logged Social Involvement		1.19*	0.10	1.29***	0.10	1.08	0.10
Polygenic Score for Depression		0.89*	0.05	1.02	0.05	0.90*	0.05

Notes: Not depressed class was used as reference category.

Table 3.6

Odds Ratios for covariates predicting transitions between latent statuses of depression.

Transitions		**Depressed Affect and Somatic Symptoms → Worsening Somati Symptoms**		**Not Depressed→ Worsening Somatic Symptoms**		**Depressed Affect → Not Depressed**		**Somatic Symptoms → Not Depressed**	
Covariates		*OR*	*SE*	*OR*	*SE*	*OR*	*SE*	*OR*	*SE*
Female		0.81	0.16	1.29	0.23	1.03	0.22	1.05	0.18
Self-Rated Health		1.87*	0.51	1.36	0.61	0.81	0.34	2.10*	0.75
Logged Net worth		0.90	0.07	1.15	0.10	1.02	0.09	1.07	0.08
High School		0.79	0.18	1.15	0.23	0.96	0.22	0.81	0.16
Some College		1.24	0.35	1.29	0.33	0.77	0.23	0.81	0.20
College		Ref.		Ref.		Ref.		Ref.	
Friends									
	1	1.20	0.37	0.99	0.29	1.17	0.40	1.57	0.43
	2	1.35	0.48	1.10	0.35	0.91	0.34	1.33	0.41
Relatives									
	1	0.92	0.27	0.89	0.22	1.18	0.34	1.09	0.28
	2	0.90	0.32	0.65	0.20	0.76	0.27	0.93	0.28
Not Married		0.32	0.21	1.67	0.88	0.68	0.41	0.66	0.30
Divorced		0.90	0.26	0.78	0.27	0.84	0.27	0.53	0.29
Widowed		0.98	0.46	0.97	0.75	1.25	0.66	0.28	0.33
Married		Ref.		Ref.		Ref.		Ref.	
Social Support									
	1	0.97	0.31	1.32	0.67	0.59	0.25	2.33	1.03
	2	0.88	0.26	1.06	0.16	0.63	0.25	2.40*	1.02
Logged Social Involvement		1.13	0.18	1.04	0.15	0.90	0.15	0.71*	0.10

Polygenic Score for Depression	0.90	0.82	0.89	0.08	1.11	0.11	1.05	0.08

Note: Transitions that only had a 15% chance or above are included.

APPENDIX

Table 3.1 A

Selection of the Analytic Sample

	N (% of previous row)
Participated in Wisconsin Longitudinal Study in 1957	10,317 (100)
…and remained in study in 1992	8,493 (72.3)
…and had valid data on all depressive symptoms variables in 1992	6,491 (76.4)
…and remained in the study, with valid data on all depressive symptom variables in 2011	4,001 (61.6)
…and have valid data on all covariates of interest in 1992 and Genomic Data	3,197 (79.9)

Table 3.2 A

Five Latent Class Model of Depressive Symptoms at Age 72 (Wisconsin Longitudinal Study, 2011, N = *3,197)*

	Latent Class				
Assigned Label	Not Depressed	Depressed Affect	Not Depressed/Hopeful for Future	Depressed Affect and Somatic Symptoms	Somatic Symptoms
Latent Class Prevalences	0.44	0.07	0.01	0.14	0.33
Item Response					
Bothered	0.07	0.37	0.08	0.71	0.31
Happy*	0.01	0.04	0.01	0.02	0.01
Lonely	0.09	0.66	0.07	0.79	0.20
Depressed	0.02	0.66	0.05	0.75	0.09
Trouble Keeping Mind	0.12	0.37	0.14	0.89	0.56
Effort	0.11	0.43	0.01	0.97	0.64
Hopeful Future*	0.05	0.15	1.00	0.05	0.04
Fear	0.05	0.36	0.01	0.68	0.23
Bad Sleep	0.41	0.72	0.24	0.89	0.77
Not Get Going	0.09	0.51	0.10	0.94	0.71

Table 3.3 A

Distribution of Highest Posterior Probabilities for Final Models

	Highest Posterior Probability	*n* (%)
2004 4-class LCA		
	0.3-0.39	32 (1.00%)
	0.4-0.49	281 (8.79%)
	0.5-0.59	337 (10.54%)
	0.6-0.69	445 (13.92%)
	0.7-0.79	362 (11.32%)
	0.8-0.89	772 (24.15%)
	0.9-0.99	968 (30.28%)
Entropy: 0.60		
2011 4-class LCA		
	0.3-0.39	12 (0.38%)
	0.4-0.49	92 (2.88%)
	0.5-0.59	394 (12.32%)
	0.6-0.69	309 (9.67%)
	0.7-0.79	288 (9.01%)
	0.8-0.89	557 (17.42%)
	0.9-0.99	1,545 (48.33%)
Entropy: 0.68		
LTA with three invariant classes		
	0.2-0.29	48 (1.50%)
	0.3-0.39	222 (6.94%)

0.4-0.49	415 (12.98%)
0.5-0.59	375 (11.73%)
0.6-0.69	425 (13.29%)
0.7-0.79	450 (14.08%)
0.8-0.89	436 (13.64%)
0.9-0.99	826 (25.84%)

Entropy: 0.70

Table 3.4 A

Odds Ratios for covariates predicting stability for latent statuses of depression.

Transitions		**Depressed Affect and Somatic Symptoms**		**Depressed Affect**		**Not Depressed**	
Covariates		*OR*	*SE*	*OR*	*SE*	*OR*	*SE*
Female		1.23	0.25	0.97	0.21	0.77	0.14
Self-Rated Health		0.53*	0.15	1.24	0.52	0.74	0.33
Logged Net worth		1.11	0.08	0.98	0.09	0.87	0.07
High School		1.27	0.29	1.04	0.24	0.87	0.18
Some College		0.81	0.23	1.30	0.39	0.78	0.20
College		Ref.		Ref.		Ref.	
Friends							
	1	0.83	0.26	0.86	0.29	1.01	0.29
	2	0.74	0.26	1.10	0.41	0.91	0.29
Relatives							
	1	1.09	0.32	0.84	0.24	1.13	0.28
	2	1.11	0.39	1.32	0.47	1.53	0.47
Not Married		Ref.		Ref.		Ref.	
Divorced		0.36	0.24	0.81	0.53	2.13	1.30
Widowed		0.33	0.26	0.55	0.43	1.73	1.61
Married		0.32	0.21	0.68	41	1.67	0.88
Social Support							
	1	1.03	0.33	1.70	0.72	0.76	0.51
	2	1.14	0.34	1.59	0.64	0.94	0.87
Logged Social Involvement		0.88	0.14	1.12	0.18	0.96	0.79
Polygenic Score for Depression		1.11	0.10	0.90	0.08	0.90	0.19

Notes: Not depressed class was used as reference category.

CHAPTER 4: THE ASSOCIATION OF DEPRESSIVE SYMPTOM SUBTYPES AND SOCIAL CPAITAL WITH COGNITIVE FUNCTIONING IN LATER LIFE

ABSTRACT

With increasing rates of cognitive decline and depression among older adults, it is essential to explore how these conditions interact and what factors might alleviate their effects. This study examines the associations between social capital and cognitive decline, while also investigating whether subtypes of depressive symptoms mediate these effects. Using data from the Wisconsin Longitudinal Study, I conduct multiple regression and mediation analysis to explore how the tangible and psychological dimensions of social capital, such as social networks and a sense of belonging, influence cognitive functioning in older adults. The findings reveal that, compared with those in the *Not Depressed* subtype, individuals categorized in the *Somatic Symptoms* depressive symptom subtype are significantly likely to have lower cognitive functioning as measured by the Modified Telephone Interview for Cognitive Status (TICS-M). Individuals categorized in the *Depressed Affect* and *Somatic Symptoms and Depressed Affect* subtypes show similar cognitive functioning to individuals categorized in the *Not Depressed* subtype. Surprisingly, none of the social capital variables, such as social engagement, trust, or sense of belonging, were found to have a significant direct effect on cognitive functioning in later life. Further the Karlson-Holm-Breen (KHB) mediation analysis reveals that depressive symptom statuses did not mediate the relationship between social capital and cognitive function in later life. These findings suggest that cognitive function in later life is more closely associated with specific depressive symptom clusters, particularly somatic ones, rather than broader measures of social

capital or depression as a homogenous condition. This research highlights the importance of considering depression subtypes when addressing cognitive health in aging populations.

INTRODUCTION

As the aging population in the United States continues to increase, it is imperative to understand the interrelated concerns of depressive symptoms and age-related cognitive decline individuals may need to manage (H. Lee & Ang, 2019; van den Kommer et al., 2013). This article aims to analyze modifiable factors and their association with cognitive function in later life. One potentially modifiable factor is depression, an illness characterized by persistent low mood (i.e., sadness) and/or decline of physical functions (i.e., alterations to sleep) (Zhu et al., 2022). Another modifiable factor that has been associated with both depressive symptoms (Cao et al., 2015; Cohen-Cline et al., 2018; Forsman et al., 2012) and cognitive function (Hikichi et al., 2017; Pandey et al., 2023) in later life is social capital. Social capital is defined as the social resources produced, consumed, and exchanged in interpersonal relationships (Cohen-Cline et al., 2018; Hikichi et al., 2020). In this study, I will examine how depressive symptoms and social capital are associated with cognitive functioning in later life. Specifically, this paper responds to the recent identification of social capital as a modifiable factor of interest associated with protecting cognitive functioning (Hikichi et al., 2017; Ihle et al., 2018; Wang et al., 2021).

Cognitive function is critical area for sociological research due to its significant impact on quality of life, independence, and the ability to maintain meaningful social and community connections (Ito et al., 2019; Kelly et al., 2017; Perry et al., 2022). As

individuals age, impairments in cognition can disrupt daily functioning, limiting activities such as remembering important tasks or participating in social interactions. These impairments not only diminish personal autonomy but also increase the risk of adverse outcomes, including dementia and social isolation (Livingston et al., 2024; Pillai & Verghese, 2009; Stern, 2009). Later life is a particularly vulnerable period for cognitive decline, with an estimated 10-20% of older adults over age 65 experiencing mild cognitive impairment and 5-7% developing dementia, conditions that can significantly affect their ability to remain connected to their communities and sustain a sense of purpose (*Alzheimer's Disease Facts and Figures*, 2023; Kelly et al., 2017; Perry et al., 2022). Given these risks, examining factors such as social capital and depressive symptoms provides an opportunity to identify modifiable pathways that could preserve cognitive functioning, reduce the risks associated with decline, and ultimately support healthier aging trajectories (Pandey et al., 2023; van den Kommer et al., 2013; Wang et al., 2021; Z. Wu et al., 2012). Understanding the factors that can protect or enhance cognitive functioning, such as social capital, is essential for addressing the challenges associated with aging.

I will begin by first outlining social capital theory to establish an understanding of how social capital is connected to one's health. Social capital encompasses both the perceived trust, reciprocity, and sense of belonging within a community (Coleman, 1988; Lin, 2002) and the tangible aspects of social networks, such as the size of one's social network, frequency of social interactions, and participation in group activities (Claridge, 2018). These social relationships are vital for fostering well-being and have been linked to mental health outcomes such as depressive symptoms (Cao et al., 2015; Cohen-Cline

et al., 2018; Forsman et al., 2012; Q. Wu et al., 2010). I will also discuss how social capital may buffer against cognitive decline, a critical issue as individuals age (Hikichi et al., 2017; Pandey et al., 2023; Piolatto et al., 2022; Wang et al., 2021).

Then I will discuss theories connecting depressive symptoms to cognitive functioning to understand their biological associations and explain how I plan to approach this in my study from a sociological perspective (Baer et al., 2012; Lu & Peng, 2019). Specifically, I aim to analyze whether various symptom clusters within depression have different associations with cognitive function in later life. Depressive symptoms can manifest as (a) depressed affect, such as feeling restless or hopeless, (b) somatic symptoms, such as lack of sleep or loss of energy, or (c) both types of symptoms (American Psychological Association, 2013). This study will add to existing literature that has studied how depressive symptoms may influence cognitive functioning in later life by examining depressive symptom subtypes and analyzing what influences the development of depression (Zhu et al., 2022).

Finally, I will explain how social capital has previously been associated with depressive symptoms and cognitive function. Including social capital with both depressive symptoms and cognitive functioning may help disentangle the relationship between this social determinant of health and these health outcomes. Given the established associations among these variables, it would be inadequate to ignore one of them. I will also investigate whether a potential mediation pathway exists between social capital and cognitive functioning by depression. Through examining these variables together, I aim to add to existing literature working to understand these multifaceted and complex relationships between society and our biology.

SOCIAL CAPITAL THEORY AND HEALTH

Social capital theory has increasingly been used as a theoretical framework to examine how social relationships can benefit individual and community health. This theory emphasizes how the quality and quantity of social connections can influence social outcomes (Schneider-Kamp, 2021). Durkheim (1897/1951) first discussed the connection between social networks and health in his pivotal study on suicide. While Durkheim did not name the concept "social capital," his work illuminated how social relationships can impact health outcomes. Following Durkheim, there have been several other sociologists who have examined the influence of social relationships on health outcomes, which has resulted in understanding social capital as a social determinant of health (Berkman et al., 2000; Freak-Poli et al., 2021; Piolatto et al., 2022; Wang et al., 2021). Social determinants are nonmedical conditions that impact health, either positively or negatively, such as education, marital status, food insecurity, etc. (Gutin, 2024). The research examining social capital as a social determinant of health primarily draws from Bourdieu (1986), Coleman (1988), Portes (1998) and Putnam (1995) as these theorists provide a comprehensive foundation for examining how social relationships and networks shape health outcomes by offering varied conceptualizations of social capital, from individual-level resources to community-wide structures (Fujiwara & Kawachi, 2008). I will now briefly summarize these scholars' definitions and then describe how this social determinant impacts an individual's health.

Bourdieu (1986) defined social capital as "the sum of resources, actual and virtual, that accrue to an individual or a group by virtue of possessing a durable network of less institutionalized relationships of mutual acquaintance and recognition." As such,

Bourdieu's concept of social capital emphasizes "an individual's ability to draw upon the resources within the social network" (Rouxel et al., 2015). Coleman conceptualized social capital as the structures that facilitate certain actions within a community or group. He emphasizes how relationships, trust, and shared norms enable individuals to access resources and achieve collective goals, ultimately benefiting both individuals and the community through mutual cooperation and support (Coleman, 1988). Similarly, Portes' definition of social capital emphasizes the ability of individuals to secure benefits and resources through their membership in social networks or other social structures (Portes, 1998). This definition of social capital is consistent with Bourdieu's and Coleman's initial conceptions, but expands upon it to emphasize the different types of social relationships one has and how this number is influenced through social structures such as family, work, etc. Finally, Putnam's understanding of social capital focused on "social networks and the associated norms of reciprocity and trustworthiness that arise from them." Here we can once again see agreement across these scholars' conceptions of social capital. However, Putnam included perceptions of one's social networks in terms of trust, sense of belonging, and self-efficacy. While these scholars have varying theories of social capital, there is some overlap when analyzing how these conceptions emphasize the role of social networks, trust, and reciprocity in shaping individuals' access to resources.(Islam et al., 2006). Social capital can thus be understood as having two interrelated components – the psychological elements of social relationships and the tangible resources that can come from social relationships (Bourdieu, 1986; Coleman, 1988; Lin, 2002; Portes, 1998; Putnam, 1995).

Scholars have theorized how these interrelated components influence health outcomes (Giordano & Lindström, 2011; Kelly et al., 2017; Webber et al., 2011; Wilkinson, 2002). For example, social capital can improve health outcomes by fostering emotional support, enhancing trust and reciprocity within social networks, and reducing stress, which can contribute to better mental health and resilience against illness (Ehsan & De Silva, 2015; Forsman et al., 2012; Giordano & Lindström, 2011). Additionally, social capital can contribute to better health outcomes based on the resources one can draw from those networks (Forsman et al., 2012; Landstedt et al., 2016; Lu & Peng, 2019). However, it is important to recognize that social capital does not always result in more material safety (Offer, 2021; Small et al., 2024; Villalonga-Olives et al., 2018). Larger social networks may also lead to following norms that may harm health as well as experiencing cyberbullying (Villalonga-Olives et al., 2018). This suggests it is not the larger network itself that is beneficial for health outcomes but rather the quality of the resources one can draw from their networks that positively influence health (Bourdieu, 1986). I will now discuss previous research that has shown associations between social capital and depressive symptoms.

Research examining the association between social capital and depressive symptoms focuses on how individuals or communities can use their social resources to positively impact their mental health (Cao et al., 2015; Cohen-Cline et al., 2018). However, the influence of social capital on depressive symptoms is complex and may depend on numerous factors. While inconsistent results remain in the literature (Carr, 2020), several studies have shown how social capital is associated with lower levels of depressive symptoms in later life (Cao et al., 2015; Cohen-Cline et al., 2018; Forsman et

al., 2012; Landstedt et al., 2016). These results suggest that the number of social networks and connections, as well as how one perceives their social connections, can be protective factors against depressive symptoms. This is particularly important for studies focusing on later life when depressive symptoms are on the rise and simultaneously, some older adults' social networks are shrinking (Ayalon & Levkovich, 2019; Coll-Planas et al., 2017; Cornwell et al., 2008; Q. Wu et al., 2010). However, it is important to recognize that not all older adults experience a reduction in social networks as they age. Scholars have also found that social networks can remain stable or even expand in later life, driven by factors such as increased time for social activities, relocation to communities with strong social support, or purposeful efforts to maintain connections (Baker et al., 2017; Cornwell & Laumann, 2015; Leist, 2013; Villar, 2012).

SOCIAL CAPITAL AND COGNITIVE FUNCTION

Similarly, there has been an influx of research analyzing social capital's associations with cognitive functioning. These studies have examined how social capital in both midlife (Ihle et al., 2018; Pandey et al., 2023) and later life can influence cognitive functioning (Bennett et al., 2006; Haslam et al., 2014; Hikichi et al., 2017; Wang et al., 2021). Generally, the findings from these articles suggest that having higher levels of social capital leads to less stress in life as well as more leisure time, which both have a positive effect on cognition (Wang et al., 2021). Additionally, other researchers have focused on specific types of social interactions that are associated with better cognition in later life. Specifically, Haslman and colleagues (2014) examined whether there was a difference between individual versus group engagement. Their findings suggested that group engagement may be more beneficial to cognition than individual

engagement. They hypothesize this to be due to “the level of engagement required to maintain group relationships is greater than that involved in maintaining individual relationships” (2014, p. 63).

A more recent exploration of social capital and cognitive functioning by Perry and colleagues (2022) investigated the impact of different forms of social connectedness on cognitive aging. Their findings suggest that cognitive benefits from all forms of social connectedness may operate through a cognitive reserve mechanism. A cognitive reserve hypothesis posits that varied and enriching life experiences provide individuals with a resource or buffer that helps them resist age-related cognitive decline (Ellwardt et al., 2015; Stern, 2006, 2009). This hypothesis is supported by research indicating that older adults with larger and more diverse social networks tend to be more resilient to the cognitive impacts of neurodegeneration (Kelly et al., 2017; Pillai & Verghese, 2009).

Few studies have considered both dimensions of social capital (i.e., psychological and tangible) in their analysis of cognitive function. Of those studies that have included both components of social capital, they focused on social capital and cognition following a natural disaster (Hikichi et al., 2020) or examined social capital at the community level (Hikichi et al., 2020; Jiang et al., 2020; Murayama et al., 2018). However, this article will be focusing on social capital within-persons and not between persons to examine how policymakers can possibly use individualized actions to address this association between social capital, depression, and cognitive function. Based on a review of the literature, two other studies have examined these two dimensions of social capital at the individual level and their association with cognitive function (Ito et al., 2019; Zhang et al., 2020). Zhang and colleagues (2020) reported that only the psychological dimensions of social capital

and not the tangible resources drawn from social networks was associated with cognitive functioning in later life. Interestingly, this association was only significant in the group of individuals who had high levels of education (i.e., completed secondary school education or higher) (Zhang et al., 2020). However, these scholars did not include depressive symptoms in their analysis, either as a covariate or as a mediator. Ito and colleagues (2019) found a difference in the association with social capital and cognitive function by gender. For men, there was a statistically significant association between the psychological dimension of social capital and cognitive function, whereas for women the significant association was between the tangible dimension of social capital and cognitive function. Ito and colleagues (2019) included depressive symptoms in their analysis as a covariate.

DEPRESSIVE SYMPTOMS AND COGNITIVE FUNCTION

As more studies work to understand cognitive functioning in later life, scholars have become interested in understanding what possible modifiable factors are associated with cognitive function. One of these modifiable factors that has come under scrutiny is depressive symptoms (Chodosh et al., 2007; Dotson et al., 2010; Livingston et al., 2024; van den Kommer et al., 2013). Both retrospective and prospective studies have revealed that "both recent depressive symptomatology and a lifetime of depression represent increased risk factors for cognitive decline, mild cognitive impairment, and dementia" (Baer et al., 2012). Most recently this was discussed in the Lancet Commissions by Livingston and colleagues (2024) who reported an increased risk of dementia for individuals with depression compared to those without depression. This has resulted in these scholars classifying depression as a midlife risk factor for dementia (Livingston et

al., 2024). Furthermore, there have also been studies that have found no relationship between depressive symptoms and cognitive function (Dufouil et al., 1996; McDermott & Ebmeier, 2009). As such, explanations regarding how depressive symptoms and cognitive function may be associated are still debated (Baer et al., 2012; Comijs et al., 2004; Dufouil et al., 1996; McDermott & Ebmeier, 2009; Zhu et al., 2022). I will briefly explain the hypothesized biological connection between depressive symptoms and cognitive function and then provide how I plan to approach this in my study from a sociological perspective.

There are four biological associations that are commonly discussed that vary in their implications for whether modifying depression will have any effects on cognition. The first focuses on the high levels of cortisol the body produces during stressful events, which can have adverse impacts on the brain (Dean & Keshavan, 2017). Cortisol is a steroid hormone our bodies produce and release to help regulate the body during period of stress. The more stressed an individual is, the more cortisol the body will produce and release. Over time, these high levels of cortisol in the body can damage brain structures such as the hippocampus and prefrontal cortex that have been linked to cognitive decline (Ouanes & Popp, 2019). While cortisol helps regulate the body during periods of stress, those periods of cortisol up-regulation are not supposed to be chronic, which is when these high levels of cortisol damage the body. Those with depressive symptoms typically have chronically higher levels of cortisol due to the stress involved with depressive symptoms, which is suggested to lead to cognitive decline in later life (Baer et al., 2012). This first association is one of the more directly causal explanations. In this case,

stopping depression should prevent further cognitive decline, though not reverse what decline has occurred so far.

The next explanation suggests the correlation between depression and cognitive decline is spurious. Literature suggests that "white matter and other subcortical abnormalities" could simultaneously lead to depressive symptoms and cognitive decline in older adults (Baer et al., 2012). So, it is not that depression causes cognitive decline or vice versa, but rather, white matter lesions cause both depression and cognitive decline (Ma et al., 2023). In this second association, depression is a non-modifiable factor and is not causal to cognitive decline.

Thirdly, depression could be seen as a psychological reaction to declining cognitive capacities that presents earlier than other symptoms (Baer et al., 2012). Scholars have suggested depression to be a response to cognitive decline where individuals becoming aware of declining function may close themselves off from their social connections and then experience symptoms of depression. For this third biological association, depression is also a non-modifiable factor because depression is a reaction to cognitive loss, not a cause of it.

Finally, depressive symptoms could manifest as temporary cognitive impairment. This is because of the recognized association between depression and cognitive function where cognitive impairment is among the symptoms of depression (Livingston et al., 2024; Muhammad & Meher, 2021). The relationship between depressive symptoms and cognitive functioning in later life is interconnected, with cognitive impairment potentially further exacerbating depressive symptoms. Studies have demonstrated that depression has negative effects on various cognitive domains, such as memory, executive functioning,

attention, and speed processing (Alexopoulos, 2005). In this last case, if one were to treat the depressive symptoms, the person may stop experiencing cognitive impairment.

While these four biological postulations exist for how depressive symptoms and cognitive decline may be associated, I will be focusing on the first and last assumptions, which suggest that depression produces cognitive impairment. Given the limitations of my data, I would not be able to effectively explore assumptions two and three. In this study I will not be measuring cortisol levels, but rather focusing on the sociological theory of social capital and how social support can lead to fewer stressful events as well as being associated with fewer depressive symptoms (Cao et al., 2015; Lu & Peng, 2019). Additionally, it is important to understand how time impacts the association of these variables. Focusing on the first and last conceptual frameworks places depressive symptoms sequentially before cognitive decline and could provide helpful information for those studying these associations. I am not statistically ruling out the other hypotheses, but rather just focusing on these two hypotheses to aid in explaining the possible association between depression and cognitive function. While this study focuses on these two hypotheses to explain the possible association between depression and cognitive function, it does not aim to determine whether cognitive impairments linked to depression are reversible. Given the available data, I cannot statistically assess whether improvements in depressive symptoms would lead to a reversal of cognitive decline.

While the relationship between depressive symptoms and cognitive function is complex and multifaceted, I aim to suggest one possible association by examining subtypes of depressive symptoms. A number of studies have examined the association between depressive symptoms and cognitive function (Chodosh et al., 2007; Comijs et

al., 2004; Graziane et al., 2016; van den Kommer et al., 2013). However, these studies analyze depression as a homogenous illness, where symptoms all indicate the same underlying diagnosis (Hybels et al., 2011). This current study aims to expand upon this research to examine depression as a heterogenous illness and not a homogenous illness.

The Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), currently relies on a symptom count, specifically requiring individuals to experience five or more symptoms over a two week period, to be diagnosed with Major Depressive Disorder (MDD) (American Psychological Association, 2013). However, the grouping of these depressive symptoms overlooks which symptoms may be manifesting at a given time, which could potentially reveal different types of depression (Hybels et al., 2013) or even lead to tailored interventions (Harald & Gordon, 2012). Several scholars in public health have begun to argue for a shift towards analyzing these specific groupings of symptoms rather than continuing to treat depression similarly, regardless of the symptoms a person presents (Veltman et al., 2017). This is particularly important when considering how depression symptoms vary throughout the life course, suggesting treating depression as a homogenous illness is inaccurate. Rather, it seems there are different types of depression that occur over the life course based on the clustering of certain symptoms. For example, Gallo and colleagues (1994) reported that middle-aged individuals experienced a mix of somatic and depressed affect symptoms while older adults experienced somatic symptoms without sadness. These results suggest it is important to consider the character of individual symptoms to better determine a possible type of depression an individual is experiencing. Given prior research examining depression as a

heterogeneous illness, my previous study used latent class/transition analysis to assess groupings of depressive symptoms.

By examining depression as a heterogenous illness, this article will analyze three presentations of depression – *Depressed Affect and Somatic Symptoms, Depressed Affect, Somatic Symptoms*. These three types of depression were assessed in Chapter 3 by accounting for the types of symptoms that were commonly correlated. For example, *Depressed Affect and Somatic Symptoms* described individuals who exhibited both affective and somatic symptoms. *Depressed Affect,* which included participants who stated they felt sad, but also indicated that they were lonely. *Somatic Symptoms*, unlike the first two presentations of depression, did not identify themselves as sad; rather, these individuals reported having trouble keeping their mind on tasks, finding that everything was an effort, sleeping poorly, and being unable to get going each day. As such, some types of depression have more somatic symptoms and others have more affective symptoms. I hypothesize that those types of depression that were connected with affective symptoms (i.e., psychological) will be more associated with cognitive function compared to types of depression that have more somatic symptoms. This is because literature has found that affective symptoms of depression have been associated with greater impairment in memory and executive functioning, while somatic symptoms have not (Perini et al., 2019). Parsing out what types of depressive symptoms are more associated with cognitive functioning could potentially allow practitioners to be aware of this following a diagnosis. As empirical chapter two of this dissertation reports, lower levels of tangible social capital were associated being classified in as *Depressed Affect and Somatic Symptoms* status and the *Depressed Affect* status opposed to the *Not*

Depressed status. This suggests that having a larger support system could help reduce affective symptoms. For example, individuals with more extensive social networks might experience less loneliness due to increased opportunities for social interaction (Berkman et al., 2000; Santini et al., 2015). Furthermore, these networks may encourage optimism and diminish fear by supporting healthy behaviors and offering emotional support (Bassett & Moore, 2013; Cao et al., 2015; Webber et al., 2011). As such, this current paper is investigating whether increasing social capital would reduce depressive symptoms and in doing so, improve cognition in later life.

AIMS

Despite the number of studies that have examined how social capital is associated with depressive symptoms (Cao et al., 2015; Cohen-Cline et al., 2018; Forsman et al., 2012; Webber et al., 2011) and cognitive function (Jiang et al., 2020; Pandey et al., 2023; Wang et al., 2021; Zhang et al., 2020) in later life separately, there is only one other study that has examined all three (Ito et al., 2019). Ito and colleagues mainly focused on examining differences in the relationship between cognitive function and the two dimensions of social capital by gender. Using a sample of 491 participants from a rural area of Japan, they reported that cognitive function and the psychological dimensions of social capital were associated in men while cognitive function and the tangible dimension of social capital were associated in women. In both instances, higher levels of social capital were associated with lower levels of cognitive decline. This study also included depressive symptoms as a covariate and found a significant negative relationship with cognitive function (Ito et al., 2019). Additionally, as I have emphasized in my previous paper, it could be ineffective to treat depression as a homogenous illness. Depressive

symptoms can manifest differently across age groups. For instance, Gallo and colleagues (1994) found that older adults often experience depression primarily through somatic symptoms rather than feelings of sadness, whereas middle-aged individuals typically exhibit a combination of somatic symptoms and depressive affect. This variation across age groups may indicate the presence of distinct forms of depression (Chen et al., 2000).

My current study will examine these associations by parsing out depression into four statuses: *Depressed Affect and Somatic Symptoms, Depressed Affect, Somatic Symptoms,* and *Not Depressed*. I will use multiple regression to examine the association between social capital, depressive symptoms, and cognitive function in later life with mediation analysis. In this mediation analysis the independent variable is social capital, the mediation variable is depressive status, and the dependent variable is cognitive function. The reason for using this statistical model to better understand this association is because I want to assess the strength and direction of any relationships among cognitive function, social capital, and depressive symptoms. These findings will contribute to existing literature in sociology and public health to further understand how depressive subtypes and social capital are associated with cognitive function in later life.

DATA AND METHODS

DATA AND SAMPLE

The following study uses data from the Wisconsin Longitudinal Study (WLS). The WLS is a comprehensive longitudinal study of a randomly selected cohort of individuals who completed high school in Wisconsin in 1957 (N=10,317). Initial surveys for the WLS were conducted during participants' senior year in 1957 when they were 18

years old. Subsequent follow-ups were conducted at various intervals, in 1964 (age 25), 1975 (age 36), 1992 (age 53), 2004 (age 65), and 2011 (age 72). These follow-up assessments employed a combination of in-person interviews, telephone interviews, and mail surveys.

Most recently, the WLS has partnered with the University of Wisconsin Madison's Alzheimer Disease Research Center (ADRC) to conduct a new round of interviews that focused on the impact of Alzheimer's Disease and related dementias (Wisconsin Longitudinal Study, n.d.). The data used in the present analysis comes from the years 2011 and 2020. The 2011 data were included in this analysis to model depressive symptom subtypes in later life, at age 72, as well as to include various measures of social capital. Data from 2020 were included to assess cognition functioning of respondents using the Telephone Interview for Cognitive Status – modified (TICS-M). This provides an 8-to-9-year gap between when respondents were asked these questions to when they were assessed for cognitive decline. Generally, the WLS has a high response rate with the 2011 wave being at 74%, excluding deaths. The final analytic sample of participants with valid data was 1,545 after omitting individuals who were not asked questions on depression and TICS-M.

Due the specific design of the WLS, it is important to acknowledge it does not encompass the full spectrum of the U.S. population. All individuals who were included in the WLS sample have graduated from high school, which limits the representation of individuals who did not attain this educational degree. Furthermore, the study predominantly comprises non-Hispanic White participants. Historical data from the 1950s indicates that less than 3% of Wisconsin residents identified as nonwhite during

that time (Wisconsin Legislative Reference Bureau, 2015). Consequently, it is important to recognize that the findings presented in this study cannot be extrapolated to the older adult population of the United States as a whole.

OUTCOME VARIABLE

The outcome variable for this study uses the Telephone Interview for Cognitive Status modified version (TICS-M), which was administered by researchers over the telephone in 2020. The TICS-M is a global mental status test that is used to detect cognitive impairment and dementia in longitudinal and population based studies (Fong et al., 2009). The TICS-M includes measures of delayed recall, verbal comprehension, as well as requiring "that the respondents provide the first and last name of the U.S. President and Vice President instead of providing their own address, which was included in the original version." (Fong et al., 2009). The WLS provides the raw scores for the TICS-M as well as two adjustments, one which accounts for the prior level of education and another which accounts for whether there was evidence of prior cognitive decline (Wisconsin Longitudinal Study, n.d.). For this study I will be using the raw score, which has a range from 4 to 50. The TICS-M is a culmination of 12 question categories that each have their own range. These 12 question categories and their ranges include: State full name (2 points), Date (5 points), Age/Phone Number (2 points), Counting Backwards (2 points), Word List Learning (10 points), Subtractions (5 points), Responsive Naming (4 points), Repetition (2 points), Pres/Vice Pres (4 points), Finger Tapping (2 points), Word Opposites (2 points), and Delayed Word Recall (10 points) (Fong et al., 2009). Higher scores on the TICS-M represent better cognitive functioning.

Participants with evidence of cognitive decline prior to 2020 were not excluded for two reasons. First, cognitive measures prior to 2020 were last asked in 2011, which would be a large gap in determining prior cognitive decline. Furthermore, these measures of cognition in the WLS prior to 2020, which included phonemic verbal fluency, semantic verbal fluency, and two tests of episodic memory, are not comparable to the TICS-M measure, which was first introduced to the study in 2020.

SUBTYPES OF DEPRESSION

Latent class analysis (LCA) is a statistical method used to identify latent subgroups based on response patterns to categorical variables (Collins & Lanza, 2010). To uncover these latent groups of depressive symptoms, I used LCA to examine how participants responded to questions from the 2011 revised Center for Epidemiologic Studies Depression Scale (CES-D). Using this statistical method, I uncovered four latent groups of depressive symptom subtypes: *Depressed Affect and Somatic Symptoms, Depressed Affect, Somatic Symptoms,* and *Not Depressed*. Respondents were then each assigned to one of these depressive symptom categories based on their most probable group. *Depressed Affect and Somatic Symptoms* respondents (N= 278) scored high on all revised CES-D questions. The subsequent latent group, *Depressed Affect*, (N= 158) included participants who identified as depressed (61%) and reported feelings of loneliness (52%) and difficulties with sleep (64%). Next, *Somatic Symptoms* (N= 563) consisted of respondents who did not self-identify as depressed but reported challenges such as difficulty concentrating (61%), finding that everything was an effort (55%), poor sleep quality (59%), and difficulty with daily activities (59%). The last latent group, *Not Depressed*, (N= 837) indicated low scored across all questions on the revised CES-D. A

variable was then created that labeled each participant with one of these latent groups that ranged from 1 to 4. *Not Depressed* was represented by 1, *Depressed Affect and Somatic Symptoms* was labeled as 2, *Depressed Affect* is 3, and *Somatic Symptoms* was identified as 4.

SOCIAL CAPITAL VARIABLES

The tangible measures of social capital variables included three measures of social integration and volunteering activities. The first two measures of social integration were measured by mail survey in 2011 and asked, "How many times in the past four weeks have you gotten together with family?" and asked the same question for time spent with friends. Respondents were originally able to input a number to indicate their responses. I recoded both variables to range from 0-2 where 0 represented no social engagement with family or friends, 1 represented low social engagement (spent up to a total of four times in the past four weeks), and 2 represented a high social engagement (spent more than four times in the past four weeks). Additionally, marital status was also included as a third measure of social integration. Prior research has suggested that individuals who are married are more socially integrated due to having their own as well as their spouse's network (Barton et al., 2014; Ermer & Proulx, 2020). Simple indicators of married, never been married, divorced, and widowed were included in the analysis. It was important to differentiate these types of marital statuses to determine if key differences existed. Volunteering activities were measured by mail in 2011, and participants reported whether they had volunteered at various organizations in the past 12 months, including religious, educational, political, senior, or other local/national groups. Responses were recorded as 'yes' or 'no,' and the 'yes' answers were summed to create

an index ranging from 0 to 6, reflecting overall engagement in volunteering. Unfortunately, the WLS lacked data on the intensity of participation within individual organizations.

The psychological dimensions of social capital variables included a measure of self-efficacy, trust, and sense of belonging. These measures of psychological social capital shape individuals' perceptions of their social environment and influence their engagement in social networks, thereby contributing to overall social cohesion and collective well-being. Self-efficacy can be understood as a measure of social capital because it reflects how social relationships reinforce an individual's sense of control and agency. Rather than being solely an individual trait, self-efficacy is shaped and supported by social networks, emphasizing its role as a socially derived resource (Forsman et al., 2012). Trust is a measure of one's subjective perception of others and institutions, which strengthens social bonds. Lastly, a sense of belonging highlights the emotional bonds and shared values that bring people together and strengthen social ties.

Each of these three psychological dimensions of social capital variables were measured by mail in 2011. Single items were selected for each of these variables from the Ryff (1989) psychological well-being instrument. The correlations between these three variables are weak to moderate with the highest correlation between self-efficacy and sense of belonging at 0.49. All three items were measured on a 6-point Likert scale ranging from 'disagree strongly' to 'agree strongly.' Self-efficacy was measured by asking, 'To what extent do you agree that you are in charge of the situation in which you live?' Trust was measured by asking 'To what extent do you agree that you have not experienced any warm and trusting relationships with others?' This specific measure was

also reverse coded. Finally, sense of belonging was measured by asking respondents 'To what extend do you agree that you often feel lonely because you have few close friends with to whom to share your concerns?' Previous studies examining psychological social capital have used similar questions to measure these variables (Ehsan & De Silva, 2015; Ito et al., 2019; Webber et al., 2011).

COVARIATES

To better understand the association between cognitive functioning in later life and social capital and depression, the following covariates were selected: educational attainment, total assets, self-rated health, gender, and a cardiometabolic diagnoses (Assari, 2017; Forsman et al., 2012; Han, 2002; J. Lee, 2011; Moorman & Kobielski, 2023). Each of the covariates used in this analysis were measured in 2011. I will now outline each of these covariates and discuss any adjustments that were made.

The sex of the respondents was recorded as a dichotomous variable with a value of 1 representing female respondents.

Self-rated health was assessed via a mail survey. A value of 0 indicates poor health, encompassing very poor, poor, and fair health categories, while a value of 1 denotes good health, encompassing good and excellent health categories.

Educational attainment was assessed through a telephone survey. I used a series of dichotomous indicators to capture participants' highest level of educational achievement, including high school graduation, some college completion, and college graduation. These three variables were constructed based on the highest level of education reported by participants in 2011.

Total assets, representing the combined value of various financial resources, was obtained through a telephone survey. This index comprised the respondent's home equity, retirement plans of both the respondent and their spouse, checking accounts, savings accounts, or money market fund of both the respondent and their spouse, CDs, government saving bonds, or treasury bills of both the respondent and their spouse, as well as the total cash value of life insurance policies. Given the skewed distribution of total assets, I categorized it into three approximately equal groups, namely 'low,' 'medium,' and 'high' assets, which were defined as 33% intervals ranging from 1 to 3.

Cardiometabolic diagnoses is a sum of five different measures that asked 'Has a doctor ever told Participant that they have…' diabetes, heart disease, hypertension, high cholesterol, and stroke. Each of these five questions was measured dichotomously as either 'yes' represented by 1 or 'no' indicated by 2. These were then recoded to 0 (no) and 1 (yes) and then the five questions were summed together to construct a variable that ranges from 0 to 5, where 5 would represent that an individual received a diagnosis of all five cardiometabolic illnesses from a doctor. Prior research has also used this measure when examining cognition in later life (Moorman & Kobielski, 2023).

DATA ANALYSIS

For this study I used a multiple regression analysis to examine the relationship between cognitive function as the dependent variable and depressive symptoms and social capital as independent variables. Through using multiple regression, I can analyze these relationships as well as the other control variables in the study. Given the complexity of factors that influence cognitive functioning in later life, multiple regression allows for modeling of numerous variables to better assess these relationships (Allison,

1999). Additionally, using multiple regression allows one to examine if a possible mediation exists. To do this within the context of multiple regression I will use the Karlson, Holm, and Breen (KHB) method for mediation analysis (Breen et al., 2021). The KHB method provides an estimation of both direct and indirect effects, which allows one to quantify how much of the effect of the independent variable on the dependent variable is mediated by the mediator, with an accompanying test for statistical significance. I am unable to use other alternative tests such as the Sobel test due to having a categorical variable mediator rather than a continuous variable mediator, which impacts the Sobel test assumptions (Hayes, 2009). Therefore, this study will begin by estimating a multiple regression and then afterward, test for mediation, where the independent variable is social capital, the mediation variable is depression, and the dependent variable is cognitive function.

RESULTS

The sample exhibited a mean TICS-M score of 32.30 (SD = 5.11), with scores ranging from 11 to 50. Using latent class analysis data from empirical paper 2, individuals were assigned to one of four depressive subtypes. These subtypes included *Depressed Affect and Somatic Symptoms, Depressed Affect, Somatic Symptoms,* and *Not Depressed*. Of the respondents in this analysis 45% were categorized as *Not Depressed*, 31% were categorized as *Somatic Symptoms*, 15% were categorized as *Depressed Affect and Somatic Symptoms, Depressed Affect*, and 9% were categorized as *Depressed Affect*. All respondents had graduated from high school with 16% also having some college education and 34% having completed college. Females constituted 56% of the sample, and 90% of participants rated their health positively (SD = 0.30). Social engagement, on

average, appeared to be slightly higher with friends (M = 1.21, SD = 0.60) than with relatives (M = 1.07, SD = 0.57). In terms of marital status, 77% of participants were married, while 9% were divorced, 11% widowed, and 23% identified as not married. The mean level of logged social involvement was 2.78 (SD = 0.54). Additionally, 19% of participants reported high levels of trust, 84% expressed a strong sense of belonging, and 4% exhibited high self-efficacy. Cardiometabolic health averaged 1.15 (SD = 1.10), with scores ranging from 0 to 5. These descriptive statistics provide an overview of the demographic, health, and social capital variables of the sample, establishing a foundation for the subsequent analyses.

I began by first evaluating the model fit for the multiple regression analysis. The model's goodness of fit was evaluated using the F-statistic, which has a value of 6.93 and has a p-value < 0.001. This suggests the overall model is statistically significant. The results of the multiple regression report that 8.3% of variance in cognitive levels, as measured by TICS-M, is explained by the independent variables in this model. The root mean square error of 4.7 indicates the average difference between the actual values and the predicted values using this statistical model. Given scores range from 0-50, this would suggest there is an average error rate of about 9.4%.

Next, we will analyze the independent variables for this statistical model. When examining the four subtypes of depressive symptoms in 2011, only the *Somatic Symptoms* subtype was statistically significantly different from the reference category, *Not Depressed*. Table 4.2 reveals that those who were assigned to the *Somatic Symptoms* category compared to the *Not Depressed* category scored 0.64 points lower on the TICS-M, on average. Neither the *Depressed Affect* group nor the *Depressed Affect and Somatic*

Symptoms depressive categories were statistically significantly different from the *Not Depressed* category.

Social capital variables, including social engagement with friends, social engagement with family, marital status, and the logged value of general engagement in volunteering activities were also not statistically significant when assessing their relationship with cognitive levels in later life. Additional social capital variables, including self-efficacy, trust, and sense of belonging, were also not statistically significant.

In addition to the depressive symptom group variable and the social capital variables, several of the control variables were statistically significant ($p < 0.05$) when assessing the relationship between them and cognitive functioning in later life. Compared to individuals with a college degree, individuals with a high school degree scored 1.76 points lower on the TICS-M and those with some college scored 1.17 points lower on the TICS-M. For every logged point of net worth in 2011, individuals scored 0.26 point higher on the TICS-M. Females scored 1.56 points higher on the TICS-M in comparison to men. Respondents who self-rated their health as good compared to poor scored 0.96 points higher on the TICS-M. For every point higher on the cardiometabolic score, individuals scored 0.30 points lower on the TICS-M.

Next, I investigated if a mediation exists using the KHB approach. This method allows me to examine the size of the direct and indirect effects. The results from Table 4.3 indicate that sense of belonging has a positive and moderate to strong association with TICS-M scores, with a total effect of 1.11 points. After accounting for depressive subtypes as a potential mediator, the direct effect of sense of belonging on TICS-M scores

decreases to 1.01 points, suggesting that this relationship is primarily from the direct effect between sense of belonging and TICS-M. The indirect effect of sense of belonging on TICS-M scores through depressive subtypes is small (0.10) and not statistically significant, indicating that the mediator does not play a significant role in this relationship. Thus, the KHB method provides a statistical significance test to determine whether depressive subtypes was a mediator, which it was not.

DISCUSSION AND CONCLUSION

This study aimed to understand how examining depression as a heterogenous illness can help to further identify the association with cognitive functioning in later life while also determining how social capital influences cognitive functioning. Using multiple regression analysis to explore the association between depressive symptom subtypes and cognitive functioning in later life, I found that only the *Somatic Symptoms* depression subgroup was statistically significantly different from the reference category, *Not Depressed* and the *Not Depressed* subgroup had overall better cognitive functioning when measured by TICS-M. This emphasizes the need for future studies to examine depression as a heterogeneous illness (Bogner et al., 2009; Hybels et al., 2013; Li et al., 2014; Ulbricht et al., 2018).

Next, I assessed the association between social capital and cognitive functioning in later life. Results of the multiple regression analysis reveal that none of the variables representing social capital were statistically significant. Finally, I examined whether any of the depressive subtypes mediated the association between social capital and cognitive functioning in later life. Results from the KHB method indicate that this mediation is not statistically significant.

Currently, there is only one other study by Ito and colleagues (2019) that has examined the association between depression, social capital, and cognitive functioning in later life. They reported social capital variables were associated differently between genders: in men, cognitive function was associated with the psychological dimensions of social capital, whereas in women, cognitive function was associated with the tangible dimensions of social capital. Moreover, higher levels of social capital were associated with poorer cognitive functions for both men and women. Depressive symptoms were also considered as a covariate and showed a significant negative relationship with cognitive function. In supplemental analyses, the current study examined if this data supported these findings through analyzing multiple regressions with multiplicative interactions between gender and social capital variables (Table 4.1 A). Almost no associations were statistically significant except for sense of belonging, a psychological social capital variable, which was statistically significant for women but not for men. This finding is the opposite of what Ito and colleagues (2019) found in their study, suggesting further research should examine larger populations to understand possible gender differences.

SOCIAL CAPITAL AND COGNITIVE FUNCTIONING

Social capital and its relationship with cognitive functioning in later life have been examined by a number of scholars (Pandey et al., 2023; Wang et al., 2021), yet few studies have examined both dimensions of social capital and their association at the individual level (Ito et al., 2019; Zhang et al., 2020). Of those articles that have examined both dimensions of social capital's association with cognitive functioning there are mixed results of which variables are statistically significant. The current study adds to this

literature by further exploring the relationship between these two dimensions of social capital and cognitive functioning. Results from this study suggests that none of the social capital variables were statistically associated with cognitive levels in later life when controlling for sex, education, self-rated health, marital status, and cardiometabolic diagnoses.

DEPRESSIVE SYMPTOMS AND COGNITIVE FUNCTIONING

Previous literature studying depressive symptoms and their association with cognitive functioning in later life have found mixed results with several scholars suggesting no association (Blazer, 2003; Muhammad & Meher, 2021) while others report a statistically significant relationship (Alexopoulos, 2005; Perini et al., 2019; Sachs-Ericsson et al., 2005). These past studies have also only included depression as a homogenous illness, which further limits their results. This is because more literature has begun to explore the importance of understanding how different constellations of symptoms make up various types of depression (Hybels et al., 2013; Li et al., 2014; Ulbricht et al., 2018).

The current study may help shed more light on understanding the association between depressive symptoms and cognitive functioning in later life given the mixed results produced by the multiple regression analysis. Four different types of depressive symptoms were assessed in this study and only one of them was reported as statistically significant – *Somatic Symptoms* when compared to the reference category – *Not Depressed*.

As per the results of Chapter 2, somatic symptoms of depression include having trouble keeping their mind on tasks, finding that everything was an effort, and being unable to get going each day. These somatic symptoms may have a direct impact on health behaviors that affect cognitive health in later life. For example, if an individual is having trouble keeping their mind on tasks or if everything feels like an effort, they may be less likely to be engaging in or focusing on healthy behaviors. There has also been an established connection between depression and unhealthy behaviors that helps to support this proposed association between somatic symptoms and cognition (Saint Onge et al., 2014; Zivin et al., 2010). Unhealthy behaviors such as poor diet or lack of exercise have been found to be detrimental to cognitive function over time (Clancy et al., 2022; Y. Lee et al., 2010).

Additionally, having trouble with attention is a problem of executive function, which is a domain of cognition, meaning respondents are reporting problems with cognition (Avila et al., 2009). This aligns with the fourth biological hypothesis, where depressive symptoms may cause temporary cognitive impairment, negatively affecting domains like memory and executive functioning, but improving with treatment (Muhammad & Meher, 2021). These somatic symptoms might signify early cognitive decline, which could foster unhealthy behaviors that further exacerbate cognitive impairment. Addressing these behaviors, therefore, has the potential not only to improve cognitive functioning but also to alleviate depressive symptoms, creating a cycle of mutual benefit.

Another hypothesis for why somatic symptoms are associated with cognitive health in later life is that somatic symptoms have been reported to be closely related to

chronic stress (Glise et al., 2014). High levels of cortisol in the body that are experienced continuously can have an adverse impact on the body such as sleep disturbance, negative effects on mood and mental health, as well as cognitive problems such as issues with memory and concentration (Baer et al., 2012). Given this established relationship between somatic symptoms and chronic stress, this increased production of stress hormones could have a negative impact on cognitive health. Moreover, this increase in cortisol has been suggested to also result in neurobiological changes that may predispose individuals to cognitive decline and/or increase their vulnerability for other neurodegenerative diseases (de Souza-Talarico et al., 2011; Echouffo-Tcheugui et al., 2018).

Affective symptoms of depression include persistent sadness, hopelessness, and feeling lonely. Overall, these symptoms involve an individual's mood or feelings (Zhu et al., 2022). Affective symptoms could be hypothesized not to be associated with cognitive functioning because these symptoms can be transient and might not have lasting effects on cognitive processes (Singham et al., 2022). Moreover, mood disorders, such as Bipolar disorder, where individuals experience significant affective symptoms, have been found to not be associated with a decline in cognitive functioning (Krabbendam et al., 2005). Through parsing out these different types of depression it could suggest that cognitive levels in later life are only associated with certain types of depressive symptoms, and not with others. Further studies will need to examine these results using various datasets to see if this is a consistent finding.

LIMITATIONS

This study has notable limitations. First, although the utilization of a homogenous sample such as the Wisconsin Longitudinal Study (WLS) aids in mitigating unobserved variable bias, it is important to acknowledge the sample is only representative of white high school graduates born in the 1930s and 1940s (Herd et al., 2014). Despite these limitations, the WLS is representative of two-thirds of the current population in regard to race/ethnicity and educational attainment for individuals in this age group (Piliavin & Siegl, 2007).

There are also limitations regarding how social capital was measured in this study. Given social capital is a multidimensional concept, scholars and researchers are uncertain exactly what measures should be used to represent these different dimensions of social capital (Giordano & Lindström, 2011; Hikichi et al., 2020; Zhang et al., 2020). As such, studies use several variables to comprise social capital with the variables available in the data and this study is no different.

Additionally, this study included education as a covariate which has been found to be associated with social capital (Grenfell, 2009; Huang et al., 2009), depressive symptoms (Bjelland et al., 2008; Chang-Quan et al., 2010; J. Lee, 2011), and cognition (Allaire & Whitfield, 2004; Clouston et al., 2015; Liu & Lachman, 2020). Due to this association between this covariate and these variables of interest, it would be advantageous to have a more representative sample of educational levels. Due to how the WLS was administered, the current dataset only includes participants who graduated from high school at minimum (Herd et al., 2014).

Finally, this study uses a variable constructed from a previous paper to measure the four different types of depressive symptoms. Using latent class analysis (LCA)

(Collins & Lanza, 2010) of respondent's answers to the revised CES-D in 2004 and 2011, the current study uses responses from 2011 to classify individuals into either *Depressed Affect and Somatic Symptoms, Depressed Affect, Somatic Symptoms,* and *Not Depressed*. While previous studies have used LCA to construct different types of depressive symptom subgroups, these studies have produced varying results (Bogner et al., 2009; Li et al., 2014; Ulbricht et al., 2018; Veltman et al., 2017), which may influence the results of the current study.

CONCLUSION

The results of the multiple regression and mediation analysis aimed to reveal the relationship between depressive symptoms, social capital, and cognitive functioning in later life. These findings contribute to the body of literature examining cognitive health in later life and highlight the importance of addressing psychosocial factors such as social capital (Baer et al., 2012; Muhammad & Meher, 2021; Zhang et al., 2020). Furthermore, these results suggest the need for sociologists and health affiliates to examine depression as a heterogenous illness rather than a homogenous one to better understand what is associated with these different subtypes (Bogner et al., 2009; Hybels et al., 2013; Li et al., 2014; Ulbricht et al., 2018). As these results show, different depressive symptoms have varying associations with cognitive functioning in later life. Interrogating these relationships between the different types of depression and social capital can possibly add new avenues for how to address cognitive health in later life.

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TABLES

Table 4.1

Descriptive Statistics of Key Variables (N = 1,545)

Variable	Mean	Std. Dev.	Minimum	Maximum
TICS-M 11 = low; 50 = high	32.30	5.11	11	50
Not Depressed 0 = Other Depressed Category; 1 = Not Depressed	0.46		0	1
Somatic Symptoms and Depressive Affect 0 = Other Depressed Category; 1 = Somatic Symptoms and Depressive Affect	0.15		0	1
Depressive Affect 0 = Other Depressed Category; 1 = Depressive Affect	0.08		0	1
Somatic Symptoms 0 = Other Depressed Category; 1 = Depressive Affect	0.31		0	1
High School 0 = none; 1 = high school degree	0.50		0	1
Some College 0 = none; 1 = some college	0.16		0	1
College 0 = none; 1 = college degree	0.34		0	1
Logged Net Worth 6.91 = low; 17.65 = high	12.90	1.27	6.91	17.65
Sex of Respondent 0 = male, 1 = female	0.56		0	1
	1.21		0	2

Social Engagement with Friends 0 = low; 2 = high				
Social Engagement with Relatives 0 = low; 2 = high	1.07		0	2
Married 0 = other marital status; 1 = married	0.77		0	1
Divorced 0 = other marital status; 1 = divorced	0.09		0	1
Widowed 0 = other marital status; 1 = widowed	0.11		0	1
Not Married 0 = other marital status; 1 = not married	0.23		0	1
Logged Social Involvement 0 = low; 2.89 = high	2.78	0.54	0	2.89
Trust 0 = low; 1 = high	0.19		0	1
Sense of Belonging 0 = low; 1 = high	0.84		0	1
Self-Efficacy 0 = low; 1 = high	0.04		0	1
Cardiometabolic 0 = diagnoses; 5 = all diagnoses	1.15	1.10	0	5

Table 4.2

Multiple Regression Analysis of TICS-M Score

Measure	Estimate	SE	95% CI	
			LL	UL
Somatic Symptoms and Depressed Affect	-0.45	0.39	-1.22	0.32
Depressed Affect	-0.54	0.47	-1.46	0.38
Somatic Symptoms	-0.64*	0.29	-1.20	-0.07
High School Education	-1.76***	0.29	-2.32	-1.19
Some College Education	-1.17**	0.38	-1.91	-0.44
Net Worth	0.26**	0.10	0.06	0.47
Female	1.56***	0.27	1.04	2.09
Self-Rated Health	0.97*	0.43	0.12	1.82
Low Friend Engagement	-0.12	0.44	-0.98	0.74
High Friend Engagement	0.28	0.48	-0.66	1.23
Low Relative Engagement	-0.23	0.38	-0.96	0.51
High Relative Engagement	-0.03	0.45	-0.92	0.85
Not Married	-0.77	0.79	-2.32	0.78
Divorced	0.33	0.45	-0.55	1.21
Widowed	0.45	0.40	-0.34	1.24
Logged Social Involvement	-0.34	0.27	-0.87	0.19
Trust	0.26	0.34	-0.41	0.92

Sense of Belonging	0.49	0.38	-0.26	1.23
Self-Efficacy	-0.58	0.63	-1.81	0.66
Cardiometabolic	-0.30**	0.12	-0.53	-0.07

*Note: Table 4.2 used two-tailed tests *p < .05; **p < .01; ***p < .001*

Table 4.3

KHB Method Mediation Analysis

Models	Estimate	SE	95% CI	
			LL	UL
Reduced	1.11***	0.32	0.47	1.75
Full	1.01**	0.34	0.34	1.69
Difference	0.10	0.11	-0.11	0.31

*Note: TICS-M is the outcome variable, sense of belonging is the dependent variable, and depressive subtypes are the mediator. *p < .05; **p < .01; ***p < .001*

APPENDIX

Table 4.1A

Multiple Regression Analysis of TICS-M Score with Gender Interaction

Measure	Estimate	SE	95% CI	
			LL	UL
Somatic Symptoms and Depressed Affect	-0.45	0.39	-1.23	0.31
Depressed Affect	-0.52	0.48	-1.46	0.41
Somatic Symptoms	-0.60*	0.29	-1.17	-0.04
Some College Education	0.61	0.36	-0.09	1.31
College Education	1.76***	0.29	1.19	2.32
Net Worth	0.26*	0.11	0.06	0.47
Self-Rated Health	1.01*	0.44	0.15	1.86
Female X Low Friend Engagement	2.59	1.85	-1.05	6.22
Male X Low Friend Engagement	-0.13	0.60	-1.30	1.03
Female X High Friend Engagement	-0.58	0.41	-1.39	0.22
Male X High Friend Engagement	1.07	0.69	-0.29	2.42
Female X Low Relative Engagement	-0.21	0.61	-1.41	0.99

Male X Low Relative Engagement	0.07	0.53	-0.98	1.11
Female X High Relative Engagement	-0.58	0.41	-1.39	0.22
Male X High Relative Engagement	-0.60	0.69	-1.96	0.76
Female X Not Married	-0.21	1.01	-2.19	1.77
Male X Not Married	-2.58*	1.26	-5.06	-0.11
Female X Divorced	-0.27	0.51	-1.28	0.73
Male X Divorced	0.93	0.92	-0.88	2.74
Female X Widowed	-0.68	0.46	-1.59	0.23
Male X Widowed	-0.16	0.81	-1.74	1.42
Logged Social Involvement	-0.38	0.27	-0.92	0.15
Female X Trust	-0.29	0.50	-1.27	0.70
Male X Trust	0.25	0.46	-0.66	1.16
Female X Sense of Belonging	-1.06*	0.50	-2.04	-0.08
Male X Sense of Belonging	-0.38	0.57	-1.49	0.73
Female X Self-Efficacy	0.78	0.80	-0.79	2.35
Male X Self-Efficacy	-0.19	1.02	-2.19	1.81
Cardiometabolic	-0.28*	0.12	-0.51	-0.05

*Note: Appendix 4.1A used two-tailed tests *p < .05; **p < .01; ***p < .001*

CHAPTER 5: CONCLUSION

This dissertation examines the nuanced relationship between depressive symptoms and social capital from mid-to-late life. Using data from the Wisconsin Longitudinal Study, this research examines how variations in social capital impact depressive symptoms. Key findings from this dissertation reveal that (a) the tangible dimension of social capital, including variables such as social integration and social support, was inversely associated with depressive symptoms equally at the between- and within-person levels, (b) the psychological dimension of social capital, particularly self-efficacy and sense of belonging, was associated with depressive symptoms unequally at the between- and within-person levels, where the cross-sectional effects had a stronger association, (c) depression should be examined as a heterogenous illness, rather than a homogenous illness, where clusters of depressive symptoms appear to represent different subtypes of depression, (d) and respondents who were categorized in the *Not Depressed* subtype had overall better cognitive functioning than those categorized in the *Somatic Symptoms* depression subgroup. This concluding chapter will further discuss these relationships; contextualize them within the broader literature on the sociology of mental health and the sociology of aging; outline the broader implications for sociology and public health policy to explore the dynamic role of social capital in aging populations that impacts depressive symptoms; and highlight limitations of these three empirical studies.

DEPRESSIVE SYMTPOMS AND SOCIAL CAPITAL

Previous research on social determinants of depressive symptoms has highlighted the association between depressive symptoms and social capital, emphasizing how social networks and social support systems can positively impact mental health (Allen et al.,

2014; Almedom, 2005; Cornwell & Laumann, 2015; De Silva et al., 2005; Domènech-Abella et al., 2017). Additionally, scholars have demonstrated that individuals with higher levels of social capital tend to exhibit fewer depressive symptoms, suggesting that robust social networks and supportive relationships can provide emotional resources and support that mitigate depressive symptoms (Cao et al., 2015; Forsman et al., 2012; Fujiwara & Kawachi, 2008; Webber et al., 2011). The mechanisms for how social capital impact depressive symptoms include influencing health through either the social support pathway or the psychosocial pathway. The social support pathway involves tangible assistance from one's social network, such as emotional or financial help, while the psychosocial pathway emphasizes how positive perceptions of trust and belonging within these networks can reduce stress and improve mental health (Giordano & Lindstrom, 2010; Wilkinson, 2002). The results of this dissertation build off previous research examining the connection between social capital and depressive symptoms as well as contribute to the growing literature on the sociology of aging and sociology of mental health more generally.

The first study of this dissertation focuses on the two dimensions of social capital, the tangible and the psychological, to delineate how each dimension impacts depressive symptoms from mid-to-late life. Furthermore, this first study aimed to distinguish these effects of social capital and understand if these effects occur between or within-individuals. Findings from this initial empirical study suggest a negative association between both dimensions of social capital and depressive symptoms, even after adjusting for various social determinants of health, such as education, sex of respondent, assets, and extraversion. These results support existing research on the association between

social capital and depressive symptoms, emphasizing the significant impact of the psychological dimension of social capital, particularly self-efficacy and sense of belonging, in alleviating depressive symptoms among individuals (Forsman et al., 2012; Fujiwara & Kawachi, 2008; Nakamine et al., 2017). Furthermore, the chapter examines variations in this association at both between- and within-person levels. The analysis revealed that the effects of tangible social capital were equal at both the between- and within-person levels. In contrast, the psychological dimension of social capital, particularly self-efficacy and sense of belonging, had stronger between-person effects, indicating that differences between individuals in these variables were more impactful than changes within individuals over time. Trust, however, showed equal effects at both levels. These findings highlight the importance of distinguishing between longitudinal (within-person) and cross-sectional (between-person) effects when analyzing the impact of social capital on depressive symptoms. Making this distinction is essential for understanding whether changes in an individual's social capital over time directly affect their depressive symptoms, or if differences between individuals with varying levels of social capital are responsible for these effects. Clarifying this distinction provides a clearer foundation for developing targeted interventions (Cohen-Cline et al., 2018). Additionally, although the results from chapter two do not establish causality, the findings suggest that an increase in an individual's social capital may mitigate depressive symptoms.

Moreover, the second empirical study of this dissertation also adds to this literature by once again examining social capital's impact on depression. However, this second study analyzes these effects on different subtypes of depression, rather than

treating depression as a homogenous illness (Lamers et al., 2016; Ulbricht et al., 2018). The results of this study highlight the varying impacts of tangible social capital on depression, depending on depressive symptoms subtypes. Baseline data, meaning an individual's depressive symptom subtype at midlife (age 54), show that lower levels of tangible social capital are associated with the *Depressed Affect and Somatic Symptoms* status opposed to the *Not Depressed* status. However, when examining the stability and transitions of depressive symptom subtypes from midlife to early later life, tangible social capital had less impact on stable depressive symptom subtypes. Notably, tangible social capital variables were not associated with depressive symptom subtypes that remained stable over time. In contrast, when analyzing transitions, two forms of tangible social capital – social support and social involvement – were significantly associated with transitioning from the *Somatic Symptoms* subtype to the *Not Depressed* subtype. Individuals with high social support were more likely to transition, while those with higher social involvement had lower odds of remaining in the *Somatic Symptoms* subtype.

Findings in this second empirical study emphasize the need to further research how social capital impacts depressive symptoms, and more importantly, how social capital can affect different subtypes of depressive symptoms over the life course. Prior studies have shown statistically significant associations between tangible social capital and depressive symptoms (Carr, 2020; Forsman et al., 2012; Landstedt et al., 2016), but results from this second empirical study indicate that these associations vary depending on the cluster of depressive symptoms, which make up different subtypes. Better understanding these nuances will help policymakers and health professionals possibly

tailor treatments to specific types of depression, moving away from treating and caring for depression as a homogenous mental illness.

DEPRESSION AND SYMPTOM CLUSTERS

Another primary goal of this dissertation was to examine depression as a heterogenous illness rather than a homogenous one as is commonly done (Lee et al., 2012; Li et al., 2014; Ulbricht et al., 2018). Through treating depression as a heterogenous illness, researchers can account for how clusters of depressive symptoms present themselves as well as the variation in symptoms across different age groups (Hybels et al., 2013; Veltman et al., 2017). Older adults, for instance, have been reported to experience depression through somatic symptoms rather than the sadness (i.e., affective symptoms) commonly seen in younger individuals (Gallo et al., 1994; Chen et al., 2000). Public health scholars have supported this shift of examining specific depressive symptoms to avoid misdiagnoses and ineffective treatments, especially in older adults who may not present *typical* depressive symptoms (Carragher et al., 2009; Hybels et al., 2013; Veltman et al., 2017).

Previous literature analyzing different subtypes of depressive symptoms have used both latent class analysis (LCA) (Bogner et al., 2009; Lee et al., 2012; Li et al., 2014) and latent transition analysis (LTA) (Ni et al., 2017; Rodgers et al., 2014). LCA and LTA are statistical methods that allow for identifying and tracking depression subtypes over time, revealing the heterogeneity of depressive symptoms based on the clusters of symptoms (Collins & Lanza, 2010). Studies using LCA have identified distinct depressive subtypes in older adults, such as classes that endorse no symptoms, negative affect, somatic symptoms, and low positive affect symptoms (Hybels et al., 2013; Lee et

al., 2012; Veltman et al., 2017). However, findings from these studies highlight inconsistencies likely due to varying sample types and depressive symptom scales used to examine these subgroups. Longitudinal studies utilizing LTA further demonstrate the idiosyncratic nature of depressive symptoms, showing how depressive symptom subgroups can transition over time as well as how covariates influence these transitions (Lamers et al., 2016; Ni et al., 2017; Rodgers et al., 2014). Understanding these transitions is crucial, as it provides insight into the possible worsening of depressive symptoms in later life and highlights the need for tailored interventions that address specific depressive subtypes, rather than treating depression as a single, uniform condition (Carragher et al., 2009; Yang, 2007).

The second empirical study of this dissertation contributes to the literature by utilizing a dataset that has not previously been analyzed in this manner to examine the consistency of depressive symptom subtypes. Using data from the WLS, findings from the LCA revealed a four-class model as the best fit model for midlife data. The four depressive symptom subgroups identified were *Depressed Affect and Somatic Symptoms*, *Depressed Affect*, *Somatic Symptoms*, and *Not Depressed*. Additionally, another LCA was conducted on these same WLS respondents in later life (age 72). Once again, the 4-class model was retained due to its clearer interpretability, showing similar statuses over the 19-year period. Notably, the *Not Depressed* status was the largest group in both midlife and later life, while the *Somatic Symptoms* class increased over time, and the two others decreased. Measurement invariance testing confirmed three invariant classes across time, with the *Somatic Symptoms* class exhibiting slight worsening symptoms. LTA indicated stability in depressive symptom statuses, with most respondents remaining in their

respective statuses. However, transitions revealed both worsening and improvement in symptoms, such as individuals moving from *Depressed Affect* to *Not Depressed* status. This aligned with prior research that also found inconsistent results in transition between different depressive subtypes (Ni et al., 2017; Rodgers et al., 2014; Ulbricht et al., 2018). Baseline predictors at midlife indicated various factors associated with depressive statuses, including sex, marital status, social support, and polygenic scores for depression. Stability and transition predictors highlighted the influence of self-rated health and social support on symptom changes from midlife to later life, suggesting potential avenues for intervention and support.

These findings support emerging literature emphasizing the need to understand depression as a heterogenous illness (Ni et al., 2017; Rodgers et al., 2014; Ulbricht et al., 2018). Most sociological research previously analyzing depression across the life course has only considered depression as a homogenous mental illness. This is also how previous scholars have highlighted the relationship between depressive symptoms and age as one characterized by a U-shape (Clarke et al., 2011; Mirowsky & Ross, 1992; Yang, 2007). However, by understanding depression as a heterogenous illness and examining these clusters of depressive symptoms, this second empirical paper did not find worsening depression from mid-to-late life, as suggested by this U-shape. Instead, when analyzing these depressive symptom clusters, it suggests far more stability when examining the nature of depressive symptom classes between midlife and early later-life.

Additionally, this second empirical paper adds to the literature examining the association between tangible social capital and depressive symptoms (Forsman et al., 2012; Landstedt et al., 2016). Prior studies have established an inverse relationship

between tangible social capital and depressive symptoms in both midlife (Giordano & Lindström, 2011; Nakamine et al., 2017) and later life (Cao et al., 2015; Forsman et al., 2012, 2013), largely due to the age-structural changes that older adults experience, such as possible role loss or more dependency on family members. However, this empirical study suggests the need to differentiate between types of depression to better inform interventions at different life stages when understanding the connection between depression and social capital. My findings indicate that while baseline tangible social capital was linked to *Depressed Affect and Somatic Symptoms* status, its impact on depression during stability and transitions from midlife to later life varies. Notably, high levels of social support are associated with transitioning from *Somatic Symptoms* status to a *Not Depressed* status, while greater logged social involvement is linked to lower odds of remaining in the *Somatic Symptoms* status. These results suggest that policymakers should consider the specific type of depression when designing treatment strategies, as the effectiveness of structural social capital varies across different depressive symptom statuses.

DEPRESSION AND COGNITION

As research into cognitive functioning in later life expands, there is growing interest in identifying modifiable factors, such as depressive symptoms, that may influence cognitive health (Chodosh et al., 2007; Dotson et al., 2010; van den Kommer et al., 2013). Both retrospective and prospective studies suggest that recent depressive symptoms and a lifetime history of depression are significant risk factors for cognitive decline, mild cognitive impairment, and dementia (Baer et al., 2012). However, some studies have found no relationship between depressive symptoms and cognitive function,

leading to ongoing debates about the nature of this association (Comijs et al., 2001; Dufouil et al., 1996; Zhu et al., 2022). The results of this third empirical chapter aim to explore the connection between depressive symptoms and cognitive function from a sociological perspective, focusing on social capital and its impact on depression (Cao et al., 2015; Lu & Peng, 2019).

This study examined the association between the depressive symptom subtypes identified in Chapter 3 (*Depressed Affect and Somatic Symptoms*, *Depressed Affect*, *Somatic Symptoms*, and *Not Depressed*) and cognitive functioning in later life. Using multiple regression analysis, I observed that only the *Somatic Symptoms* subgroup exhibited a statistically significant difference from the reference category, *Not Depressed*, with the latter demonstrating higher levels of cognitive functioning as measured by the Telephone Interview for Cognitive Status-modified (TICS-M). This highlights the importance of exploring depression as a heterogeneous illness in future research (Bogner et al., 2009; Li et al., 2014; Ulbricht et al., 2018). Furthermore, analyzing the association between social capital and cognitive functioning revealed no statistically significant findings. Subsequent mediation analysis using the KHB method indicated a mediation does not exist between sense of belonging, depressive symptom subtypes, and cognitive functioning.

This study's findings contribute to the literature on depressive symptom subtypes in midlife and early later-life (Hybels et al., 2013; Ulbricht et al., 2018; Veltman et al., 2017). These results highlight the importance of examining the complexities of depression throughout the life course and further emphasize the need to study and treat depression as a heterogeneous illness (Ulbricht et al., 2018). This nuanced understanding

enables sociologists and health professionals to better assess the curvilinear nature of depressive symptoms, potentially revealing more stability between midlife and early later life. This challenges the traditional assumption of a U-shaped relationship in depression trends (Clarke et al., 2011; Mirowsky & Ross, 1992; Yang, 2007). Additionally, this research improves our understanding of how social capital influences depressive symptoms, emphasizing the importance of considering social resources in treatment approaches and potentially reducing reliance on pharmaceutical solutions.

THEORETICAL IMPLICATIONS

This research contributes to a growing body of literature that seeks to understand mental health from a sociological perspective. Through framing depressive symptoms within the context of social capital, the findings from this dissertation further add to the evidence of understanding mental health issues as deeply intertwined with social and environmental conditions (Bjelland et al., 2008; Lee et al., 2012; Rodgers et al., 2014; Zimmerman & Katon, 2005). This research moves beyond traditional individualistic or biological explanations of depression and instead highlights the pivotal role of social structures and relationships in shaping mental health outcomes. The integration of social capital into the analysis of depressive symptoms highlights the importance of viewing mental health through a sociological lens, where social determinants such as community engagement, social networks, and trust significantly impact an individual's mental well-being (Forsman et al., 2013; Lu & Peng, 2019; Nakamine et al., 2017). Furthermore, this perspective opens new pathways for prevention and intervention, suggesting that enhancing social capital could significantly reduce the burden of depression (Ferrari et al., 2013; Funk, 2016; Karp, 1997; Liu et al., 2020).

The study's emphasis on both tangible and psychological dimensions of social capital reveals that both the presence and quality of social relationships are fundamental to understanding and addressing depressive symptoms (Bassett & Moore, 2013; Forsman et al., 2012; Nakamine et al., 2017). Tangible social capital, encompassing the resources acquired through social networks and support systems, consistently shows an inverse relationship with depression. This indicates that social integration and community involvement can serve as protective factors against depressive symptoms (Forsman et al., 2013; Hamano et al., 2010). Psychological social capital, which includes trust, self-efficacy, and a sense of belonging, further informs how perceptions and experiences of social connectivity can influence depressive symptoms (Cohen-Cline et al., 2018; Giordano & Lindström, 2011). These findings suggest that fostering environments where individuals feel supported, connected, and valued can mitigate depressive symptoms, highlighting the need for community-based interventions and policies that enhance social capital.

Moreover, the research emphasizes the dynamic and evolving nature of social capital's influence on depressive symptoms over the life course. By adopting a longitudinal approach, the study demonstrates how changes in social capital can impact depressive symptoms over time, emphasizing the importance of sustained social support and community engagement throughout an individual's life (Cohen-Cline et al., 2018; Kim et al., 2012; Landstedt et al., 2016; Nakamine et al., 2017). This temporal perspective challenges static views of mental health determinants and calls for policies that promote long-term development of social networks and trust within communities (Cohen-Cline et al., 2018). Such an approach aligns with the broader sociological

understanding that mental health is not merely a private trouble but a public issue shaped by collective social conditions (Allen et al., 2014; Barrett & Turner, 2005; G. W. Brown, 2002; Karp, 1997; Sinkewicz et al., 2022; Williams, 2018). This dissertation suggests holistic public health strategies that integrate improving social capital to help address the global burden of depression.

LIMITATIONS AND FUTURE DIRECTIONS

While these empirical studies contribute the existing literature on depression symptoms, social capital, and cognitive functioning for adults in later life, there are several limitations that warrant further discussion.

A key limitation of this dissertation is the restricted generalizability of its findings due to the use of data from the WLS. The WLS cohort is composed predominantly of white individuals who completed high school in the 1950s, limiting the diversity of racial, and educational backgrounds represented. As a result, the findings may not be fully applicable to populations with different demographic characteristics, particularly those with lower educational attainment or more diverse racial and ethnic backgrounds. For example, the U-shaped curve often observed in the relationship between depression and age (Mirowsky & Ross, 1992; Yang, 2007) may not hold in this subgroup of white, well-educated participants, as this pattern is likely influenced by broader socioeconomic and cultural contexts that are underrepresented in the WLS data. WLS respondents have all graduated from high school and may have more security from depressive symptoms due to higher levels of education and socioeconomics (Barrett & Turner, 2005; Bjelland et al., 2008; Chang-Quan et al., 2010; Miech & Shanahan, 2000). Similarly, the four depression subtypes identified in this study may not fully capture the heterogeneity of

depressive symptoms in the broader population. Cultural and contextual differences may influence the clustering of symptoms or their prevalence, suggesting the need for additional research using more diverse datasets to assess the stability and relevance of these subtypes across populations (Lee et al., 2012; Li et al., 2014). These limitations highlight the importance of interpreting these findings with caution when attempting to generalize to the broader populations.

Another limitation of this dissertation is the potential impact of mortality selection and loss to follow-up on the findings for each of the empirical chapters. Mortality selection likely resulted in the retention of healthier and more socioeconomically advantaged individuals (Zajacova & Burgard, 2013) in the WLS cohort over time, as individuals with greater health risks or lower socioeconomic resources may have experienced mortality at higher rates. Similarly, loss to follow-up may have disproportionately excluded individuals experiencing higher levels of depressive symptoms or those with less access to social resources, as these individuals are typically more difficult to retain in longitudinal studies. This could have introduced a bias by underestimating the prevalence or severity of depressive symptoms and their associations with social capital and cognitive functioning. For example, the observed patterns in the U-shaped curve of depression and age or the clustering of depressive symptom subtypes may not fully reflect the experiences of individuals who were no longer included in the dataset (Li et al., 2014; Mirowsky & Ross, 1992; Veltman et al., 2017; Yang, 2007). Future research should consider incorporating techniques such as Heckman-style two-step corrections to account for these selection biases and strengthen the generalizability of findings (J. P. Brown et al., 2024).

Additionally, an overarching limitation of this dissertation stems from the conceptual and methodological challenges in defining and measuring social capital. The lack of a universally accepted definition introduces ambiguity into the selection of measures, which may impact the accuracy of both psychological and tangible social capital constructs (De Silva et al., 2005; Forsman et al., 2012, 2013; Hamano et al., 2010). This limitation is further compounded by relying on available measures in the WLS, which may not capture all aspects of these two dimensions of social capital. If additional data were available, I would like to ask respondents about the depth and quality of their social relationships, such as the frequency and perceived satisfaction of interactions, which may better reflect the psychological dimension of social capital. Similarly, questions regarding the intensity of engagement in social activities or networks, beyond mere participation, could provide a more holistic understanding of the tangible dimension. Including these measures could improve the ability of future studies to explore the association between depression and social capital.

Beyond addressing these limitations, the findings of these studies also raise new questions that could be explored in future research. There is a clear need for studies that integrate qualitative methods to better understand how individuals perceive and experience the benefits of social capital, particularly in relation to depression. Qualitative insights could enrich the quantitative findings of this dissertation by providing deeper understanding of the mechanisms through which social capital influences depressive symptoms and cognitive function. Moreover, intervention-based research could explore practical ways to enhance social capital in older adults, assessing the effectiveness of community-based programs designed to increase social interaction and support networks

(Cohen-Cline et al., 2018). Such studies would not only test the causal effects of social capital on depressive symptoms but also provide evidence-based recommendations for policy makers and public health practitioners aiming to mitigate the burden of depression among aging populations (Ferrari et al., 2013; Funk, 2016; Karp, 1997; Liu et al., 2020). Furthermore, another area for future research would be exploring how subtypes of depression evolve across the life course, beginning in early life and extending through mid and later life. Understanding whether depressive symptom subtypes remain stable or shift as individuals age could provide valuable insights for how depressive symptoms may change over the life course (Ni et al., 2017; Rodgers et al., 2014). This approach could reveal critical periods where social or environmental factors exert stronger influences on specific depression subtypes. Additionally, identifying patterns of stability or change may improve interventions tailored to different life stages. Overall, future research can offer a more comprehensive understanding of the nuanced relationship between social capital and depression using different methodologies and expanding the scope to analyze the entire life course.

CONCLUSION

Despite these limitations, this dissertation advances our understanding of the relationships between social capital, depressive symptoms, and cognitive function in older adults. First, the findings from the empirical studies highlight the role of social capital as a protective factor against depressive symptoms and cognitive decline in later life. Tangible and psychological social capital were found to have an impact on depressive symptoms, emphasizing the protective benefits of enhanced social interactions and perceived social support (Bassett & Moore, 2013; Forsman et al., 2013; Landstedt et

al., 2016; Lu & Peng, 2019). This supports the integration of social capital into models of health promotion and disease prevention among aging populations.

Second, the longitudinal nature of the data used in this dissertation emphasizes the dynamic changes in social capital and their effects on depressive symptoms over time, demonstrating that, generally, decreases in social capital are associated with increases in depressive symptoms and cognitive decline (Bennett et al., 2006; Cao et al., 2015; Cohen-Cline et al., 2018; Forsman et al., 2013; Seeman et al., 2011). This reinforces the need for interventions aimed at maintaining or enhancing social capital in later life.

Third, depression should be treated in research as a heterogenous illness, with different subtypes existing based on the cluster of symptoms. Prior research has shown that depressive symptoms can manifest different in mid- and later life (Fiske et al., 2009; Gallo et al., 2006; Hybels et al., 2013). These distinct patterns of depressive symptoms highlight that depression is not a heterogenous condition but a complex one that may require different interventions depending on the age and symptom profile of individuals (Lee et al., 2012; Ni et al., 2017; Ulbricht et al., 2018; Veltman et al., 2017). Investigating these subtypes longitudinally can provide valuable insights into how depression evolves over time, revealing critical periods where interventions may be most effective.

Finally, while exploring the nuanced relationships between different types of social capital and health outcomes, this research contributes to theoretical developments by demonstrating how variations in social capital dimensions influence mental health differently, offering a more complex understanding of how social environments affect aging adults (Cao et al., 2015; Landstedt et al., 2016; Lu & Peng, 2019; Nakamine et al., 2017). The findings of this dissertation reveal that while social capital positively

influences depressive symptoms, its effects appear generally consistent across demographic groups, with no significant gender differences detected. However, the role of gender in shaping access to and experiences of social capital remains an important area for future research (Rodgers et al., 2014; Ross & Mirowsky, 2006). Overall, this dissertation contributes to both empirical and theoretical knowledge in the sociology of aging and mental health, while also highlighting new directions for understanding how social factors shape mental health trajectories later in life.

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