THE IMPACT OF SOCIAL CAPITAL ON DEPRESSION AND TREATMENT-SEEKING BEHAVIOR IN BLACK/AFRICAN AMERICAN AND WHITE/CAUCASIAN ADULTS AGED 50 AND OVER

AN ABSTRACT

SUBMITTED ON THE FOURTH DAY OF MAY 2022

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IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

OF THE SCHOOL OF MEDICINE

OF TULANE UNIVERSITY

FOR THE DEGREE

OF

DOCTOR OF PHILOSOPHY

BY

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Abstract

Background: Depression in older adults is a public health issue. There is a growing population of older adults in the United States; furthermore, the proportion of the population of older adults who identify as a racial minority are growing at a faster rate than non-minority identifying older adults.

Methods: A secondary quantitative data analysis was conducted using data from the Health and Retirement Study (HRS) 2016. Six thousand two hundred and forty-five individuals who self-identified as either Black/African American or White/Caucasian and were over the age of 50, completed measurements of hand grip strength and answered and submitted the questionnaire on psychosocial measurements. Both the measurements of hand grip strength and the psychosocial measurements were included in this study. Independent t-tests were used to determine if measurements of social capital varied by race for individuals with and without depressive symptoms and multivariate logistic regression was adopted to predict medication usage to treat depression. Analysis was also undertaken to determine if measurements of grip strength and/or asymmetrical grip strength should be used as a non-self-reported measurement of depressive symptoms.

Results: Possession of social capital did vary between Black/African Americans and White/Caucasian older adults with and without depressive symptoms. Black/African Americans had higher scores on three of the four social capital measures (both structural social capital measures, and one functional measure (close ties)) compared to White/Caucasians. The same pattern was observed when restricting the sample to only
individuals with sub-threshold and/or major depression. There was also a significant impact of social capital on medication use, varied by gender. The likelihood of taking medication decreases as social capital scores increased, and the decrease in likelihood of taking medication was more for females than males. Additionally, maximal hand grip strength was confirmed to be a potential indicator of depressive symptoms.

**Conclusions:** Social capital impacts risk factors for depression in Black/African American and White/Caucasian older adults and their decision to take medication to treat depressive symptoms. Future targeted interventions using social capital as a measure to impact treatment for depression needs to be race and gender specific.
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List of Abbreviations

AHEAD  Cohort, born before 1924
ANOVA  Analysis of variance
ADL    Activity of daily living
CDC    Centers for Disease Control and Prevention
CES-D  Center for Epidemiologic Studies Depression Scale
CODA   Children of Depression cohort, born 1924 to 1930
CST    Corticospinal system or tracts
DI     Dorsal interossei
DIP    Distal interphalangeal
DSM-5  Diagnostic and Statistical Manual of Mental Disorders
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<th>Description</th>
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<tr>
<td>EBB</td>
<td>Early Baby Boomer cohort, born 1948 to 1953</td>
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<tr>
<td>ECT</td>
<td>Electroconvulsive therapy</td>
</tr>
<tr>
<td>EDC</td>
<td>Extensor digitorum communis</td>
</tr>
<tr>
<td>EDM</td>
<td>Extensor digiti minimi muscles</td>
</tr>
<tr>
<td>EIP</td>
<td>Extensor indicis proprius</td>
</tr>
<tr>
<td>FDP</td>
<td>Flexor digitorum profundus</td>
</tr>
<tr>
<td>FDS</td>
<td>Flexor digitorum superficialis</td>
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<tr>
<td>fMRI</td>
<td>Functional magnetic resonance imaging</td>
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<tr>
<td>FTF</td>
<td>Survey conducted face-to-face</td>
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<tr>
<td>GED</td>
<td>General Educational Development</td>
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<tr>
<td>HIV</td>
<td>Human immunodeficiency virus</td>
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<tr>
<td>HRS</td>
<td>The Health and Retirement Study</td>
</tr>
<tr>
<td>HS</td>
<td>High School</td>
</tr>
<tr>
<td>IADL</td>
<td>Instrumental Activities of Daily Living</td>
</tr>
<tr>
<td>IP</td>
<td>Interphalangeal joints</td>
</tr>
<tr>
<td>ISR</td>
<td>Institute for Social Research at the University of Michigan</td>
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<tr>
<td>LBB</td>
<td>Late Baby Boomer (LBB) cohort, born 1960 to 1965</td>
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<td>LBQ</td>
<td>Leave-Behind Questionnaire</td>
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<tr>
<td>LH</td>
<td>Left-hemisphere of the brain</td>
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<tr>
<td>MAOIs</td>
<td>Monoamine oxidase inhibitors</td>
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<td>MBB</td>
<td>Mid Baby Boomer cohort, born 1954 to 1959</td>
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<tr>
<td>MDD</td>
<td>Major Depressive Disorder</td>
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<tr>
<td>MP</td>
<td>Metacarpophalangeal joint</td>
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<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>MSA</td>
<td>U.S. Metropolitan Statistical Area</td>
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<td>NIA</td>
<td>National Institute of Aging</td>
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ORL  Oblique retinacular ligament
PD   Parkinson’s Disease
PI   Palmar interossei
PIP  Proximal interphalangeal
PRD  Previous Diagnosis
RH   Right-hemisphere of the brain
rTMS Repetitive transcranial magnetic stimulation
SD   Subthreshold or Subsyndromal Depression
SES  Socioeconomic status
SSA  Social Security Administration
SSRIs Selective serotonin reuptake inhibitors
SNRI s Serotonin and norepinephrine reuptake inhibitors
WB   War Baby cohort, born 1942 to 1947

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Appendix A  Biopsychosocial Model for Depression

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Chapter 1

Introduction

Statement of the Problem

Depression, a condition characterized as a persistent sad, anxious, or empty feeling, or a feeling of hopelessness and pessimism, is not a normal part of aging (U.S. Surgeon General, 1999). However, the Centers for Disease Control and Prevention (CDC) estimates that 7 million older adults age 65 and older in the United States (U.S.) are affected by depression (CDC Healthy Aging Program, 2021). Chronic untreated depression in older adults could potentially complicate chronic conditions such as heart disease, diabetes, and stroke; it is also associated with higher health care costs and higher mortality rates due to suicide and heart disease (Frederick et al., 2007; Katon et al., 2003; Snowden et al., 2008; Unützer et al., 1997). In 2010, Greenberg et al. (2015) estimated that the economic burden of depressive disorders in the United States was $210.5 billion per year, with 45%-47% due to direct medical costs (e.g., medical services, pharmacy costs) which are shared by individuals, employers (if the individuals are employed) and society, and the remaining cost attributed to workplace absence and productivity.

In 2020, approximately 38% of the population in the U.S. identified as a member of a racial or ethnic minority group (Jones et al., 2021). Between 2016 and 2030, the population of individuals over the age of 65 who identify as a racial or ethnic minority is projected to increase by 89% compared to a 39% projected increase of White/Caucasian older adults; the population of Black/African American adults over the age of 65 is
projected to increase by 73% (Administration for Community Living, 2017). Thus, racial and ethnic minority groups of older adults 65+ are some of the fastest growing older adult sub-populations in the country. Despite advancements in public health infrastructure, medical research, and preventative healthcare practices increasing life expectancy and quality of life in later years for most Americans, minority older adults still experience a disproportionate burden of preventable disease, death, and disability in comparison to non-minority residents of the United States (CDC, 2010).

Previous studies have produced conflicting results on the racial disparity of depression in older adults. According to a review of over twenty years of published literature on late life depression in older African Americans by Picket, Bazeleais, & Bruce (2013), some studies have found no significant racial differences between African American older adults and Caucasian older adults in depression. On the other hand, some studies have found Caucasians to have higher rates of depression than African Americans (Picket, Bazeleais, & Bruce, 2013). However, the National Institute of Minority Health and Health Disparities (2021) has found that adult African Americans are 20% less likely to report serious psychological distress than adult Whites are and are more likely to suffer from debilitating symptoms. The burden of disability for underdiagnosed or untreated depression is with the minority population (Bailey, Mokonogho, & Kumar, 2019). Regardless of whether African American older adults experience a higher prevalence of depression in comparison to Caucasian older adults, what is significant is that in the next ten years, minority populations are projected to represent 1 in 4 older adults, with over 7.5 million adults over the age of 65, identifying as African American (Mui, Burnette, & Chen, 2002; U.S Census Population Projections, 2017). Further research into factors that
may decrease the burden of depression on the minority older adult population and increase healthcare utilization for treatment purposes is therefore prudent.

The CDC notes that adults can be successfully identified for depression with routine, systematic screening. The methods used to identify depression in adults often rely on a variety of screening tools which require the individual to be able to portray their feelings accurately and honestly (CDC, 2009). Some of the screening tools used to diagnose clinical depression are self-report instruments (Biggs, Wylie, & Ziegler, 1978; Knesevich et al., 1977; Ng, How, & Ng, 2016; Radloff, 1977). However, an initial screening test that does not rely on respondent’s self-reporting could potentially allow physicians and other healthcare providers to screen for mental health issues such as depression and conduct further tests and alter their care as necessary. Having a “lab test” for depression could also provide a physical measurement that patients could rely on; they would be able to have a biomarker where they can see results of improvement. Use of a “lab test” as a component of a depression diagnosis would be particularly impactful for minority older adults since recent studies have shown that African Americans are less likely to report psychological symptoms, less likely to seek and receive adequate mental health treatment, and more likely to delay treatment until symptoms are debilitating (Bailey, Mokonogho, & Kumar, 2019; Lawson, 2002; Nelson, Shahid, Cardemil, 2020).

There is a dearth of research examining non-invasive, objective tests not reliant on self-reporting that could potentially indicate depression in older adults. Hand grip strength has been proposed as a physical biomarker for older adults and it is a potential explanator of morbidity and mortality (Bohannon, 2019). Researchers have proposed grip strength be regularly included as part of a physical exam of a patient because it has been
shown to be a predictor of not only functional limitations and disability in later life but also musculoskeletal disease such as osteoporosis, sleep problems and diabetes, and even periodontal disease (Aravindakshan, Hakeem, & Sabbah, 2020; Bohannon, 2019; Rantanen et. al., 1999). Moreover, current neuropsychological theories of emotion, particularly depression, suggest that functional motor asymmetries (i.e., failure to demonstrate asymmetric grip strength) are observed in depressed young boys (Emerson et al., 2001). To be sure, little is known regarding the impact of depression on the anatomy and physiology of the brain and its relationship with functional motor asymmetries in older adults. Although functional motor asymmetries have been observed as a biomarker of depression in younger male children, further study is needed before it can act as a biomarker for depression in all individuals. If the findings of this analysis are similar to the results of other studies on asymmetric grip strength and depression, then the measurement of functional motor asymmetry has the potential to be a physical biomarker of depression and act as a simple, and non-invasive objective test of depression in older adults.

Having an objective test in diagnosing depression in older adults is just one factor in determining why individuals may or may not seek treatment for depressive symptoms. An abundance of research has been conducted examining disparities in healthcare-seeking behavior by factors such as age, race, gender, and socioeconomic/health insurance status for depressive symptoms (Cardemil, Nelson, & Keefe, 2015; Crystal et.al., 2003; Harman, Edlund, & Fortney, 2004; Hinton et. al., 2006). A review of the literature suggests that Black/African Americans suffer from lower rates of treatment for depression than Whites, that older men are significantly less likely to be referred to
treatment and acknowledge depressive symptoms as an ailment in comparison to older women. In addition, individuals without insurance are less likely to initiate treatment for depressive symptoms (Harman, Edlund, & Fortney, 2004; Hinton et. al., 2006; Simpson et. al., 2007). While predisposing variables such as age, gender, and race impact observed disparity in diagnosis and treatment for depressive symptoms, they cannot be modified. However, there are several factors that can potentially be modified, including social behavior that could reduce disparity in healthcare utilization for depression. Extensive studies have established a significant correlation between social determinants (e.g., social capital, socioeconomic conditions, cultural and social norms etc.) and depression (Assari, 2014; Fisher & Baum, 2010; Schaan, 2013; Shittu et al., 2014). To date, there is limited research on how the intersections of race and gender affect health-seeking behavior of minority older adults with both subthreshold and major depressive disorder. Understanding the prevalence of depression in minority older adults and the impact that social determinants of health have on help-seeking behavior will inform future strategies for targeted interventions. Further research is needed to not only determine if there is an objective measure of depression but also how healthcare utilization of different groups is impacted in different ways.

**Purpose Statement**

This study proposes to investigate the potential mitigating impact that social capital has on prevalence of depressive symptoms and treatment of depressive symptoms and the potential association between asymmetrical hand grip strength and depression in older adults.
Study Aims

Among Black/African American and White Caucasian individuals aged 50 and over, the aims of this study are:

Aim 1: Explore social capital possession differences between Black and White older adults with and without depressive symptoms.

a) Determine if social capital serves as a protective measure against depression.
   a. Hypothesis: social capital measures will serve as a buffer, or protective factor against depression and will vary by race and/or gender.

b) Assess how social capital may influence the decision to seek treatment for depression.
   a. Hypothesis: social capital will influence individuals in seeking out treatment for depressive symptoms and will vary by race and/or gender.

Aim 2: Examine the relationship between grip strength asymmetry in depressed older adults.

a) Determine if grip strength asymmetry is associated with depression in older adults.
   a. Hypothesis: there will be a negative association between the difference in grip strength between hands and depression. Smaller differences in grip strength between hands will be associated with higher levels of depression.
b) Assess whether medication mediates the impact of grip strength asymmetry as an indicator of depression.

a. Hypothesis: there will be a significant interaction between grip strength asymmetry and depression diagnosis and medication (e.g., depression group with medication will be different than no depression group with no medication).
Chapter 2

Review of the Literature

Major Depressive Disorder and Subthreshold Depression

The Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-5) is a gold standard manual that defines and classifies mental disorders in the U.S. (Khoury, Langer, & Pagnini, 2014). A diagnosis of Major Depressive Disorder (MDD) according to the standards set by DSM-5 include experiencing five or more of the following symptoms persistently in the past two weeks: feeling depressed, loss of interest/pleasure, weight loss or gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, feeling worthless or excessive/inappropriate guilt, decreased concentration, or thoughts of death/suicide (Grant et al. 2015). The DSM-5 has also been amended from a previous version, the DSM-IV, and a diagnosis of MDD also includes several specifiers that exclude a history of manic episodes, excludes individuals whose symptoms are more aligned with other psychotic disorders and excludes individuals whose symptoms may be attributable to the physiological effects of medication or other substances (Hasin et al, 2018; MDCalc, 2022).

There is no consensus in defining subthreshold or subsyndromal depression (SD) with symptomatic cut-offs between SD and MDD often being arbitrary in nature (Karsten et al., 2011). An example of one operationalized definition that has been proposed using the DSM-5 is: individuals with two or more of the diagnostic criteria for MDD (Cuijpers & Smit, 2004; Juruena, 2012). Judd et al. (1994) found that individuals with depression
are symptomatic 60% of the time and the majority of that time is spent in a SD state and not an MDD state and there is a consensus in research that the incidence of MDD in individuals with SD is higher than in individuals without SD (Carrellas, Biederman, & Uchida, 2017; Cuijpers & Smit, 2004). SD is associated with functional impairment and early intervention efforts might assist in preventing progression to MDD (Carrellas, Biederman, & Uchida, 2017; Hybels, Pieper, & Blazer, 2009). The following literature review of risk factors for depression does not distinguish between MDD and SD, this is due to the fact that the classification systems currently used in psychiatry are based on subjective descriptions of symptoms, and thus distinguishing between the two is a limitation in both psychiatry and research (Juruena, 2012).

The Prevalence of Depression

Major Depressive Disorder and Subthreshold Depression is a growing public health concern in the United States. There is an estimated 7 million older adults age 65 and older in the United States who are affected by depression (CDC Healthy Aging Program, 2021). Worldwide, an estimated 3.8% of the population, or approximately 280 million people have depression (Institute of Health Metrics and Evaluation, 2019). Depression is a leading cause of disability and chronic untreated depression could potentially complicate other conditions such as heart disease, diabetes, and stroke; it is also associated with increased mortality rates due to suicide and heart disease (Frederick et al., 2007; Katon et al., 2003; Snowden et al., 2008; Unützer et al., 1997; World Health Organization, 2021). Women are more likely to be affected by depression than men and residents in countries in the Middle East and North Africa suffer from some of the
highest depression rates in the world (Ferrari et al., 2013; World Health Organization, 2021).

In the United States, data from the National Health Interview Survey show that among adults, individuals aged 18-29 experienced the highest percentage of depressive symptoms at 21%, followed by those aged 45–64 (18.4%) and 65 and over (18.4%), and lastly, by those aged 30–44 (16.8%) (Villarrorel & Terlizzi, 2020). Weinberger et al. (2017) conducted an analysis with data from the National Survey on Drug Use and Health and concluded that the prevalence of depression in not only significantly increasing over time, but that increases in depression prevalence are happening for both the youngest and the oldest age groups.

Previous studies have examined the prevalence of depression based on racial groups by primarily categorizing race into one of the five racial categories designated by the U.S. Census. The U.S. Office of Management and Budget mandates that the U.S. Census Bureau collect race data for a minimum of five groups: White, Black or African American, American Indian or Alaska Native, Asian, and Native Hawaiian or Other Pacific Islander (U.S. Census Bureau, 2021). Studies have shown that Asian adults are the least likely to receive a depression diagnosis from a healthcare provider of the five racial categories (Kalibatseva & Leong, 2011; Shao, Ritchie, & Bailey, 2016; Villarrorel & Terlizzi, 2020).

However, review of the literature shows contradictory results when examining racial and/or ethnic disparities in the prevalence of depressive symptoms (Bailey, Mokonogho, & Kumar, 2019; Riolo, Nguyen, Greden, & King, 2005). Early studies showed that Black/African American adults experienced higher rates of depression than
White/Caucasian adults (Neighbors et al. 1983; Somervell et al. 1989; Warheit, Holzer, & Arey, 1975). On the other hand, more recent research shows that the racial disparity in depression may not exist, or that White/Caucasian adults may have higher rates of major depressive disorder than Black/African American adults (Riolo, Nguyen, Greden, & King, 2005; Williams et al., 2007; Wyman et al. 2020). The analysis of these data sets all mention that there are also many barriers in diagnosing depressive disorder, that Black/African Americans and other racial minorities may be underdiagnosed, and that the true prevalence of depressive symptoms within that community is underreported in data collection (Riolo, Nguyen, Greden, & King, 2005; Sohail, Bailey, & Richie, 2014; Wyman et al. 2020). The purpose of this analysis is to examine the prevalence of depression in White/Caucasian and Black/African American older adults.

**Treatment for Depression**

Aim 2b of this study is to assess whether a pharmacological treatment mediates the impact of grip strength asymmetry as an indicator of depression. A brief review about the potential pathways in which medication used to treat depression may impact grip strength can be found in Chapter 5. However, there are multiple known and effective treatment options to treat depressive symptoms (World Health Organization, 2021). Treatment options for depression fall into one of four categories: antidepressants, mood stabilizers, nonchemical therapies and psychosocial therapy (Duval, Lebowitz, & Macker, 2006).
Antidepressants

Selective serotonin reuptake inhibitors (SSRIs) are the most widely prescribed type of antidepressant. They generally cause fewer side effects and an overdose of SSRIs is less likely to be serious. Some examples of bands of SSRIs include: Prozac®, Paxil®, Zoloft® (Mayo Clinic, 2019; National Health Service, 2021). Serotonin is a neurotransmitter that affects mood, energy level, appetite, and sleep and SSRIs work by increasing the available levels of the neurotransmitter serotonin in the brain (Mental Health America, 2020). Neurons communicate via neurotransmitters and a transporter molecule recycles unused transmitter and returns it back to the pre-synaptic cell. An SSRI binds to SERT (serotonin transporter) and blocks activity, allowing for more serotonin to remain in the brain in the spaces between neurons (Swanson, 2013).

Serotonin and norepinephrine reuptake inhibitors (SNRIs) work in the same way that SSRIs work, and some common brands include: Cymbalta® or Pristiq®. Norepinephrine is a neurotransmitter that affects energy level, focus, and attention and the norepinephrine transporter (NET) is located in the plasma membrane of noradrenergic neurons (Zhao, 2004). SNRIs work by blocking the reabsorption of serotonin and norepinephrine into the nerve cells, thus increasing the levels of active neurotransmitters in the brain (Swanson, 2013).

Three other classifications of antidepressants are: atypical antidepressants, such as Remeron® or Wellbutrin®; tricyclic antidepressants, such as Tofranil® or Pamelor®; and monoamine oxidase inhibitors (MAOIs), such as Nardil® or Parnate® (Mayo Clinic, 2019). Medications in these three categories are less likely to be prescribed to treat depression for a variety of reasons; they may cause more side effects than SSRIs or
SNRIs, or they may be more restrictive in their use. As an example, MAOIs require a strict diet because of potentially dangerous and deadly interactions with certain foods and other medications (Mayo Clinic, 2019).

*Mood Stabilizers, Nonchemical Therapies, and Psychosocial Therapies*

This analysis focuses exclusively on the use of medication to treat depressive symptoms in older adults. However, there are many other treatment options available to mitigate the impact of depression. Mood stabilizers such as lithium salts or antiepileptics help regulate mood and prevent from fluctuation to extreme highs (mania) and extreme lows (depression) (Young, 2004). In comparison, antidepressants are used with the intended purpose to lift mood. Several examples of nonchemical therapy options used to treat depression are repetitive transcranial magnetic stimulation (rTMS) a noninvasive procedure that uses magnetic fields to stimulate nerve cells in the brain by delivering repetitive magnetic pulses in order to improve symptoms of depression, electroconvulsive therapy (ECT) where the brain is stimulated while the patient is under anesthesia, light therapy, or insomnia treatments (Duval, Lebowitz, & Macker, 2006; Gebara et al., 2018; Mayo Clinic, 2018; Mayo Clinic, 2022). Various forms of psychotherapy or cognitive behavioral therapy, used either solely or in combination with medication have been shown to be just as effective as antidepressants and are used more commonly to treat depressive symptoms in comparison to mood stabilizers or more invasive nonchemical options such as ECT (DeRubeis, Siegle, & Hollon, 2008; Tay, Subramaniam, & Oei, 2019).
Risk and Protective Factors for Depression

Similar to many mental health conditions, depression is a multifactorial disease with many unknown causes and risk factors (Gonda et al., 2018; Shadrina, Bondarenko & Slominsky, 2018). However, studies have shown that many lay individuals believe that depressive symptoms are primarily social and cultural in origin (e.g., stress/conflict in relationships, early life experience, changes in life circumstances, and stress) (Fu & Parahoo, 2009; Harris & Brown, 1996; Lauber et al., 2003). This chapter focuses on how social behavioral factors impact depression diagnosis and the decision to seek treatment in Black/African American and White/Caucasian older adults; however, a brief review of both the risk and protective factors of depressive disorder is necessary to understand the scope of issues that combined, potentially lead to depressive symptoms. The following review of literature is organized using categorizations first proposed in George Engel’s 1977 Biopsychosocial Model to explain illness: biological, psychological, and social (Engel, 1977). The Biopsychosocial Model is commonly used as a guide in understanding depression in research and Figure 1 is an adapted model that incorporates the variables of interest for this study; a more comprehensive summary of findings from previous studies can be found in Appendix A.
Figure 1. Biopsychosocial Model for Depression

*Boxes and arrows in red include variables and pathways of interest for this study

**Risk and Protective Factors for Depression in Older Adults**

George Engel’s 1977 Biopsychosocial Model to explain illness includes biological, psychological/cognitive, and social risk factors (Engel, 1977). Adapted from the works of Avasthi (2016); Barton et. al (2017); Engel (1977); and Seligman & Maier (1967); some examples of biological risk factors for depression include: traumatic brain injury, stress, physical disability, chronic pain, sleep disorder, drug affects, acute illness, and neurotransmitter dysfunction. While examples of psychological risk factors for depression include: assumptions and beliefs, anxiety disorders, trauma, addiction, explanatory styles, disposition and learned helplessness, these examples do not constitute an exhaustive list, but rather, some of the most common biological and psychological risk factors for depression found in recent studies. Although specific biological risk factors are beyond the purview of this study, the point is that some of these biological risk
factors may impact hand grip strength, itself a potential diagnostic tool for depression.

The potential link between depression and grip strength are discussed in Chapter 5.

Older adults are at a specific developmental stage in their lives where both physical and cognitive declines in health are an expected part of aging but are also increased risk factors for depression (Vink, Aartsen, Schoevers, 2008). Although the prevalence of physical and cognitive risk factors increases with age, age may also act as a protective measure for some of the social risk factors of depression. Social risk factors such as a decreased social network or low socioeconomic status, are adverse social conditions that are associated with poor health outcomes (Alderwick & Gottlieb, 2019). Childhood trauma, acute traumatic events, chronic stress, and relationships are just a few of the many social risk factors for developing depression in adulthood (Hammen et al., 2009; Mandelli, Petrelli, & Serretti, 2015; Santini et al., 2015; Sattler et al., 2006). However, older adults may be more prepared to cope with encountering frequent loss, as loss of loved ones is more expected at later stages of life; thus, they may have an easier time accepting death (Schum et al., 2005; Vink, Aartsen, Schoevers, 2008).

**Theoretical Framework for Treatment-Seeking Behavior**

The primary theoretical framework for this study is a modified version of Ronald M. Andersen’s 1968 Healthcare Utilization Model for determining use of healthcare services (Andersen, 1968). A review of the literature shows that this model has been used as the theoretical background in studies investigating the use of health services for a variety of diseases (Babitsch, Gohl, & von Lengerke, T, 2012). Andersen’s (1995) Revised Behavioral Model for Healthcare Utilization can be found in Appendix C. The model examines different factors that influence the decision to seek healthcare services.
The three categorizations examined in this model include: Predisposing, Enabling, and Need.

**Predisposing Factors**

Examples of predisposing factors that may influence the decision to seek healthcare services include demographic factors such as age, gender, marital status, education, and race (Babitsch, Gohl, & von Lengerke, 2012). For this study, predisposing variables of analysis included age, race, gender, education level, comorbidities, and previous diagnosis of depression.

The sample population for this analysis is limited to individuals, aged 50 and over. The application of Andersen’s Healthcare Utilization Model (1995) to treat depression in older adults is of interest due to the fact that as individuals age, their decision-making process changes as well (Andersen, 1995). In both hypothetical laboratory tasks and decision-making in the real world, older adults are less likely than younger adults to conduct exhaustive information searches prior to making a decision (Chen, Ma, Pethtel, 2011; Löckenhoff, 2018; Löckenhoff et al., 2016). In addition, older adults are more likely to make a decision based on their prior experience (Löckenhoff, 2018). Subsequently, when presented with the choice to treat depressive symptoms with medication, the decision-making process for older adults may be more likely to be impacted based on their own previous experience with treating depression with medication, and they also may be more amenable to social factors impacting their decision-making process since they are less likely to exhaustively review sources of information (Mata & Nunes, 2010). For the purpose of this study, age was categorized as 50-64, 65-79, and 80+, although this is an arbitrary age cutoff in categorizing “older
adults,” these age categories were selected due to the potential life changing events that happen at age 65. Although retirement ages vary widely, at the age of 65 for these individuals, they become eligible for Medicare, eliminating a potential barrier to accessing treatment for depressive symptoms if they didn’t previously have health insurance. Financially, there are several other additional benefits for those between the ages of 65 and 80. At the age of 65, individuals have a higher standard deduction amount if they or their spouse is 65 or older and full social security benefits can be claimed at age 66 if the individual was born between 1943-1954, with full benefits claimed at a later age if the individual was born later than 1954 (Internal Revenue Service, 2022; Social Security, 2022). Although the financial benefits of turning 65 may be minimal and have little to no impact on the health and wellbeing of the individual, the age of 65 is a commonly used cutoff in the United States when determining age eligibility for benefits. An additional cutoff was made at 80 years of age as there is evidence that those over the age of 80 experience significant functional decline and have difficulty in completing activities of daily living (ADL) (e.g., dressing, eating) and instrumental activities of daily living (IADL) (e.g., grocery shopping, cooking). Difficulty in completing any of the aforementioned tasks is associated with increased depressive symptomology and decreased social support (Hajek et al., 2022).

In addition, gender and race are also treated as predisposing factors for individuals when making healthcare-related decisions. In a study that reviewed the decision-making process of men and women with serious health conditions, such as heart or lung disease, researchers found that most men preferred doctor-controlled decisions while women preferred to retain considerable responsibility in making their own
healthcare decisions (Perkins et al., 2019). Previous research has also shown that there are racial and ethnic disparities in overall communication about treatment options, with minority-identifying individuals often receiving less information from their doctors about treatment decision-making (Ashton et al., 2003; Lin & Kressin, 2015). Black/African American individuals have been shown to prefer a shared healthcare decision making process as much as White/Caucasian but have reported being responsible for initiating conversations with their doctors about their care (Peek et al., 2011). In addition, disparity between the use of mental health services between Black/African American and White/Caucasian older adults may also be due to deeper systemic divides; older Black/African American adults have been shown to be more likely to internalize mental health stigma and have a less positive attitude toward accessing mental health services (Conner et al., 2010). Thus, social factors may impact older individuals of different genders and/or races in varying ways and this study aims to examine the potential differences.

Education level has been shown to be a strong predictor of desire for involvement in healthcare decision making with those with lower education levels being less confident and knowledgeable about their condition and treatment options (Jayakumar & Bozic, 2020; Thompson, Pitts & Schwankovsky, 1993). In addition, education level has also been used as a measurement of socioeconomic status (SES), and low SES has been found to be associated with higher levels of depression (Freeman et al., 2016). Thus, for the purpose of this study to distinguish between low education and high education and by using education-level as a representative of low and high SES, participants were categorized into HS/GED or HS/GED+, with HS/GED representing “low.” Earnings
outcomes by educational attainment in the US estimate that a college graduate will earn approximately $800,000 more over their lifetime in comparison to an individual who graduated from HS or obtained their GED and did not graduate from college (Daly & Bengali, 2014).

Chronic general health conditions were also determined to be a predisposing factor of depressive disorder. The prevalence of depression is substantially higher for individuals diagnosed as having comorbid depression, or depression in addition to a chronic health condition such as diabetes or heart disease (Puyat et al., 2017). Seven chronic conditions are included for this analysis and they include: high blood pressure, diabetes, cancer of any kind excluding skin, lung disease, heart condition, stroke, and arthritis. Of the 6,244 individual who answered some or part of the questions related to chronic conditions, 60.2% had high blood pressure, 25.1% had diabetes, 15.3% had cancer (not including skin), 10.2% had lung disease, 23.5% had a heart condition, 6.5% had a stroke, and 57% had arthritis. It should be noted that these questions were phrased to the respondent as “has a doctor ever told you that...”; thus, for a chronic condition such as cancer, it is not possible to determine whether the individual currently has cancer or had cancer in the past. Of the individuals in the sample population, 15% had never been diagnosed with any of the seven chronic conditions listed previously, 24.3% had one of these conditions, and 60.2% had been diagnosed with two or more chronic conditions. Previous studies have shown that individuals with chronic health conditions may be more prone to depression, potentially due to the symptoms caused by these chronic conditions (Puyat, 2017; Ramasubbu, 2012). Moreover, individuals with depressive symptoms have been shown to have a preference for clinician-directed
decision-making when discussing treatment preferences to treat other conditions (Moise et al., 2017)

**Enabling Factors and Need Factors**

Enabling factors may include income, health insurance, and having a regular source of care such as a family doctor or primary care physician and need factors may include evaluated or self-reported health status (Babitsch, Gohl, & von Lengerke, 2012). One of the most common reasons for individuals to not seek treatment for depressive symptoms was that they claimed that could not afford the cost (Chekroud et al., 2018). Thus, the enabling factor for this analysis was operationalized by having or not having health insurance. In the U.S., health insurance is provided by a mix of public and private, for-profit and nonprofit insurers and the Patient Protection and Affordable Care Act was signed into law by President Barrack Obama in 2010 (Tikkanen et al., 2020). The individuals included in the sample population for this analysis were all over the age of 50 and the data was collected in 2016, so not only were these individuals eligible to access health insurance through the Affordable Care Act, but in the U.S., for individuals over the age of 65, the federal government funds Medicare; thus, it is unlikely that the individuals in the sample population for this analysis did not have access to health insurance (Tikkanen et al., 2020).

Need factors in Andersen’s (1995) Revised Behavioral Model for Healthcare Utilization may include evaluated and self-reported health status. For instance, if an individual has a compound fracture, their pain levels and the fact that they have a broken bone that is breaking through their skin, may lead an individual to evaluate their need for healthcare services as very high; they need to seek medical attention. However, many
methods that are used to diagnose depression rely on a variety of screening tools which require the individual to be able to accurately and honestly portray their feelings (CDC, 2009). There is a real need to have an objective measure or some biomarker that indicates depressive symptomology. This study proposes the use of asymmetrical hand grip strength to be used as a biomarker for depression and the effectiveness of treatment for depression. A review of how depression affects the brain and how this can impact handgrip strength can be found in Chapter 5.

**Social Factors**

Previous studies have shown that many lay individuals believe that depressive symptoms are primarily social and cultural in origin (e.g., stress/conflict in relationships, early life experience, changes in life circumstances, and stress) (Fu & Parahoo, 2009; Harris & Brown, 1996; Lauber et al., 2003). One criticism of Andersen’s original 1968 Healthcare Utilization Model is that it does not take into account culture and social interaction, and the impact that this has on determining use of health care services but Andersen claims that predisposing factors may include social factors or health beliefs (Andersen, 1995; Guendelman, 1991; Portes, Kyle, Eaton, 1992). Categorizing a social factor should not be considered exclusively a predisposing factor. This paper acknowledges that social factors are multidimensional constructs, and proposes using measurements of social capital, a social factor that focuses on reciprocal social relationships between individuals and/or groups of people that has positive benefits, as a social factor that impacts predisposing, enabling, and need factors that impact treatment-seeking decisions of older adults with depression (Harpham, Grant, & Thomas, 2002; Onyx & Bullen, 2000; Xu, 2013). Both structural social capital, membership in groups,
and functional social capital, quality of relationships, should also be considered an enabling and need factor because social capital can influence healthcare decision making at different stages. Thus, this study proposes adapting Andersen’s Healthcare Utilization Model to include social capital pathways that impact predisposing, enabling and need factors (Figure 2) (Andersen, 1995). The history of the use of social capital measurements in research can be found in the following sections of this chapter. An analysis on how measurements of social capital vary by predisposing factors such as gender and race and how it has a potentially negative impact on healthcare utilization can be found in Chapter 4.

In summary, health care providers need to effectively design interventions to not only reduce the prevalence of depression but also treat individuals with depressive symptoms effectively. Analysis confirming that predisposing factors have an impact on healthcare utilization can be found in Chapter 3. Determining a biomarker that increases perceived need by an individual with depressive symptoms and emphasizing to health care providers the need to understand how social influences such as structural or functional social capital affect why individuals may choose to engage in health services-seeking behavior and how these behaviors may vary by gender and/or race has the potential to increase access to care.
Figure 2. Healthcare Utilization Model to Treat Depression in Individuals 50 and Over

* Variables and pathways of analysis in this study are in red.

Adapted from Andersen’s 1995 Healthcare Utilization Model

Thus, the primary interest of this study is in how social capital can potentially serve as a protective factor for depressive symptoms and how it can influence individuals in their treatment decision making for those who are over the age of 50. While there are many biological and psychological risk factors for depression, the primary aim of this study is to focus on how a social factor, social capital, can impact depressive symptoms in Black/African American and White/Caucasian older adults. Subsequently, the following is a summary of how social capital is used in research and how it impacts depression.
The Concept of Social Capital in Research

For the purpose of this paper, social capital is defined as the “resources accessed by individuals as a result of their membership of a network or group” (Kawachi & Berkman, 2014). Measurable indicators of social capital in this study include measurements of social networks, where information could potentially be exchanged (structural member and structural volunteer), as well as measurements of level of trust (social support and close ties), which could potentially indicate a higher value in the exchange of ideas and information (Kawachi & Berkman, 2014; Rouxel et al., 2015). For instance, advice and recommendations exchanged between individuals who consider themselves “close” may be more valued and more likely to be taken under consideration than information exchanged between individuals who are merely acquaintances. Regardless, measurements of a social network as well as the strength/trustworthiness of the network are considered when operationalizing social capital in this study. Nevertheless, one of the main difficulties in determining measurable outcomes of a social theory such as social capital is the fact that there is no universal definition for the concept of “social capital” (Conrad, 2007). Most social scientists will agree that the term is a multidimensional construct that focuses on reciprocal social relationships between individuals and/or groups of people that has positive benefits (Harpham, Grant, & Thomas, 2002; Onyx & Bullen, 2000; Xu, 2013).

Throughout the 1990s, when the concept of social capital became a popular export from academic theory to everyday language, it lost much of its original meaning by acting as the social cure-all theory to a wide variety of applications (Portes, 1998). Despite originating from a wide range of perspectives, James Farr’s (2004) *Social*
Capital: A Conceptual History, first credits the use of the term “social capital” to Karl Marx in 1867. However, the Marxist notion of the term “social capital” does not appear to be a measurement of the strength of social connections as the term is used in research today (Farr, 2004; Kawachi, Subramanian, & Kim, 2008). Classical social theorist Emile Durkheim’s work on the link between social disintegration and suicide is also often cited in order to demonstrate a link between social capital as a protective factor in population health (Farr, 2004; Kushner & Sterk, 2005). Kawachi et al. (1997) credits Durkheim with demonstrating that social cohesion is related to the health of the population by demonstrating that suicide rates were higher in less cohesive populations (Kushner & Sterk, 2005). However, additional analysis indicates that Durkheim’s data reveals that suicide rates are actually highest amongst those who are the most socially integrated, disputing his claim that social integration is a protective factor for population health (Kushner, 1995; Kushner & Sterk, 2005. This has caused researchers today to have difficulties with explaining the definitive aspects of the concept and has also hindered progress in the overall field of social capital, especially with regards to issues related to minority health, aging, and depression (Conrad, 2007; Harpham, Grant, & Thomas, 2002; Kawachi, Subramanian, & Kim, 2008).

Defining Social Capital

Many researchers agree with the claims of Putnam (1993), that there are five principal characteristics of the concept of social capital: community networks, civic engagement and participation, a sense of belonging, reciprocity, and trust. Nevertheless, a solid definition of the concept of social capital eludes most researchers which has led to
difficulties in differentiating the concept from other measurements of social factors such as social support and social networks, all of which have unique defining qualities.

For the purpose of this study, one of the main components that differentiates social capital from other social concepts is the idea that the availability of social capital allows individuals to understand the type of relationship that they are engaging in and allows them to exchange ideas, resources, information, and services (Ramlagan, Peltzer, & Phaswana-Mafuya, 2013). It has also been theorized that engaging in a relationship as a form of social capital appeals to an empathetic human nature (Robison et al., 2012). In comparison to other forms of social support or social networking, every individual and group unit that engages in social capital expects to benefit in some way from the relationship; that is, the acts of support and exchange of ideas are in some way reciprocal in nature.

Past Measurements of Social Capital

Multiple studies have used quantitative measures of social capital and many of these studies use proxy measures in order to establish levels of social capital (Carrillo Álvarez & Riera Romaní, 2017; Harper & Kelly, 2003; Harpham, Grant, & Thomas, 2002). For instance, indicators of trust and civic norms from the World Values Survey have been used to determine the strength of association or relationships in order to predict how a factor such as trust can be associated with potential market and economic growth (Knack & Keefer, 1997). Another example uses data from the English Longitudinal Study of Ageing to determine if social capital is a determinant of oral health in older adults (Rouxel et al., 2015). Other survey data has also been used to collect information such as levels of trust in institutions and levels of participation within a community.
(Narayan & Pritchett, 1997). Even studying the availability and access to communication devices has allowed researchers to approximate the density of social networks and the potential availability of social capital within a community (Temple & Johnson, 1998).

Comparative studies on social capital have used instruments such as the General Social Survey and have examined social capital in terms of civic involvement, group membership, and scales of confidence in the local government, combining data from both academic and commercial sources (Putnam, 1993, 1995). Comparisons have also been made between different immigrant and/or minority groups. These studies have shown that certain minorities/immigrants may exhibit higher levels of social capital based on location and the type of community that they join. For instance, individuals of Chinese descent may be well-off in San Francisco, California in comparison to individuals of Mexican descent in San Diego, California, because of the already invested and established minority community in the area. Chinese individuals located in San Francisco may have increased access or referrals to important services due to other individuals within the community contributing to increasing the availability of social capital, ensuring the establishment of a more stable, successful, and cohesive community group (The World Bank, 2011). Thus, when studying social capital and its impact on health issues such as depression, it is important to note that social capital affects individuals of different age, gender, and race in disparate ways. Research into the distinct ways in which social capital influences individuals is necessary in order to develop the most appropriate and effective targeted health interventions in the future.
The Availability of Social Capital in the United States

Researchers claim that there has been a decline in community engagement in America, and thus, a decline in the availability of social capital within the older adult population (Putnam, 1995). There have been marked declines in the number of group memberships, volunteer participation, and attendance at religious functions (Costa & Kahn, 2001; John & Morris, 2004; Lyons & Fabiansson, 1998). Researchers theorize that this decreased involvement of individuals within a community has resulted in a decreased amount of social capital (Cannuscio, Block, & Kawachi, 2003). Thus, an elderly individual may not have access to a social network which would include a safety net such as an individual looking in on them, reminding them to take their medication, or taking them to appointments, due to the number of social connections within the community eroding.

The decline in community engagement may play a role in declining social capital but the shift in living arrangements of minority older adults is another factor that may impact the availability of social capital (Cannuscio, Block, & Kawachi, 2003; Bubolz, 2001). Historically, many families may have found themselves residing in multigenerational households due to necessity and/or cultural traditions; older adults would rely on younger family members to assist them as needed (Keene & Batson, 2010; Mindel, 1979). However, as older adults strive to maintain an independent and active lifestyle away from younger family members, whether by choice, or necessity, their ties to the community and their social connections outside of their family become essential in maintaining a sense of independence and autonomy as well as acting as a protective factor in developing depression.
**Social Capital and Minority Health**

Availability of networks, friendships, and groups where social capital can be accessed is especially important as researchers have begun to study the impact of how a social function, such as social capital, may have an impact on overall physical and mental health. Unsurprisingly, studies have shown that the presence of higher amounts of social capital available to individuals within groups is associated with higher levels of positive health outcomes in older adults (Kawachi, 1999; Nyqvist et al., 2014). Thus, social capital has the potential to be an essential mechanism in reducing the prevalence of depression within the ever-expanding minority older adult population.

Moreover, research has also shown that decreased social connections and decreased involvement in community activities may lead to depression, a negative mental health outcome that may lead to physical impairment among the elderly (Katon, 2003). This is important to note because minority older adults experience a disproportionate burden of preventable disease, death, and disability in comparison to non-minority older residents of the United States (CDC, 2010). Current research demonstrates that individuals who suffer from serious illnesses have an increased rate of survival and decreased amounts of time devoted to recovery based on their social networks (Michael et al., 1999; Southwell et al., 2020; Sweet et al., 2018). Thus, the importance of the social network or even just the connectedness that elderly individuals have with family members, other individuals, and the rest of their community becomes essential in maintaining positive health outcomes, particularly with managing depression (Cao et al., 2015; Forsman et al., 2012).
Increasing levels of social capital in order to promote health is of particular interest because factors that impact or determine health outcomes can be divided into non-modifiable factors, such as genetics, sex, and age, and potentially modifiable factors, such as lifestyle, culture, and environmental conditions (Speller, 2007). Of these various domains, it is estimated that behavioral patterns have up to a 40% impact as a health determinant for mortality (Speller, 2007). Although Dahlgren & Whitehead (1991) identify many potentially modifiable factors, it could be argued that interventions that target improving social and community networks have the greatest chance of success for the least amount of cost (Rund et al., 1994).

Minority older adults are a vulnerable population with regards to morbidity and mortality rates of aging-related diseases due to the fact that there are many potentially modifiable social determinants of health that cause observable health disparities between minority older adults and non-minority individuals (Dong et al., 2012; Silveira & Ebrahim, 1998; Wallace, 2014). At a time where there is the possibility of completely effective and curative treatments, many older adults who identify as part of the minority population may have difficulties with accessing treatment options and preventative measures, due to cultural, financial, or personal barriers (Dunlop et al., 2002; Kim et al., 2012; Rhee et al., 2019). If minority older adults are destined for a poorer health status in comparison to their non-minority counterparts, a study on social capital and minority older adults may increase the success of potential targeted interventions, and may play an essential role in minorities being able to overcome health disparities and improving their overall health status by increasing social capital (Dominguez & Arford, 2010).
Many previous studies recognize that minorities are a vulnerable population and research has been done on specific groups within the African American population, such as low-income mothers or rural residents at risk for contracting human immunodeficiency virus (HIV), women’s use of mammography as a diagnostic tool, and combating poverty. Findings have concluded that the availability of support and local community resources should be taken into consideration when assessing the potential for positive outcomes (Cené et al., 2011; Dean et al., 2014; Dominguez & Watkins, 2003; Warren, Thompson, & Saegert, 2001). Few of them have focused on the potential impact that social capital may have on the mental health of the elderly African American population in the US. If older African American adults are destined for poorer health in comparison to their Caucasian counterparts, the study of the availability of social capital may help us understand how minorities could overcome health disparities and improve their overall mental health status.

**The Different Levels of Social Capital**

The lack of universal measurements of social capital makes it difficult to establish comparisons within and across data sets (Lochner, Kawachi, & Kennedy, 1999). It makes it difficult for investigators to assess how much social capital is available within a group or community, and how much social capital an individual may be able to access, which in turn makes it difficult to inform targeted social capital interventions to combat illness (De Silva et al., 2005). Although most researchers agree that social capital can be assessed by measuring amounts of trust, strength of networks, and transactions of reciprocity that facilitates coordination and cooperation for the mutual benefit of both parties involved,
measurement instruments often vary by study (Ferlander, 2007; Kawachi, 1999; Putnam, 1995; Xu, 2013).

Previous studies do acknowledge that there are many different types or levels of social capital being scrutinized that may have an impact on the health outcomes of older adults. On an individual level, bonding, bridging, and linking social capital have been established and measured in some form. Studying bonding social capital examines the relationship that exists between individuals who share a social identity, implying a certain degree of trust (Szreter & Woolcock, 2004; Xu, 2013). Bridging social capital examines the links and connections between distinct groups, with the implication that the trust level is not as strong as the one that exists with bonding social capital (Norstrand & Xu, 2012). Research on linking social capital examines the relationships between individuals and groups that appear to be in some way unequal, such as the relationship between a corporation and an individual employee (Ferlander, 2007). There has also been a distinction between cognitive social capital, which is based on mutual trust, and structural social capital, which is facilitated by social networks (Xu, 2013). Both cognitive and structural forms of social capital are usually found to be connected, and often reinforce the strengths of one another (Uphoff & Wijayaratna, 2000). Acknowledging the different levels of social capital is particularly important when working with minority populations due to individuals already identifying with a unique group. Not only may levels of bonding social capital be higher, but access to forms of bridging social capital may be more limited due to minority status.

A further differentiation can be made between structural and functional social capital. Structural social capital emphasizes the behavior of the individual and is
primarily operationalized as participation in social activities and volunteering (Rouzel et al., 2015). Structural social capital is thought to be shaped by institutions and policies, with a review of recent cohort studies showing an inverse relationship between social participation (structural social capital) and mortality (Nyqvist et al., 2014; Uphoff, 2000). Thus, the greater the social network and the greater the amount of social participation, the lower the risk of mortality (Nyqvist et al., 2014). Functional social capital is believed to shape behavior and is strongly related to mental health outcomes (De Silva et al., 2005). It measures the relational aspect of social interactions, and the quality of these relationships (Rouzel et al., 2015). Previous studies have used measurements of social support and the quality of support available to individual as measures of functional social capital. Differentiating between types of social capital is important in research because different types of social capital may impact health outcomes in different ways (Harpham, Grant, & Thomas, 2002). In the case at hand, the aim is to determine if social capital differs by race and/or gender in individuals with depression, and if social capital differs by race and/or gender in individuals with depression who decide to take medication for depressive symptoms. Thus, social capital is operationalized in this study as structural social capital (social activities and volunteering) and functional social capital (social support and close ties).
Chapter 3

Disparity and Predisposing Factors in Depression and Treatment-Seeking Behavior of Older Adults

The Study Sample

The Health and Retirement Study (HRS) is a longitudinal panel study primarily sponsored by the National Institute of Aging (NIA), with additional funding from the Social Security Administration (SSA) and administered by the Institute for Social Research (ISR) at the University of Michigan. The majority of HRS data is publicly available and free to use by any researcher or analyst. First conducted in 1992, it has been fielded every two years since that date and was launched to provide data on individuals over the age of 50 in the U.S. (Sonnega et al., 2014). Further information on the HRS survey design and methodology can be found at: https://hrs.isr.umich.edu/documentation/survey-design. The 2016 wave consists of seven cohorts:

- The initial HRS cohort of adults, born 1931 to 1941. This cohort was first interviewed in 1992 and subsequently every two years.

- AHEAD cohort, born before 1924, initially a separate study (The Study of Assets and Health Dynamics Among the Oldest Old). This cohort was first interviewed in 1993 and subsequently in 1995, 1998, and subsequently every two years.

- Children of Depression (CODA) cohort, born 1924 to 1930. This cohort was first interviewed in 1998 and subsequently every two years.
• War Baby (WB) cohort, born 1942 to 1947. This cohort was also first interviewed in 1998 and subsequently every two years.

• Early Baby Boomer (EBB) cohort, born 1948 to 1953. This cohort was first interviewed in 2004.

• Mid Baby Boomer (MBB) cohort, born 1954 to 1959. This cohort was first interviewed in 2010.

• Late Baby Boomer (LBB) cohort, born 1960 to 1965. This cohort was first interviewed in 2016.

Participants for this study were recruited from eighty-four U.S. Metropolitan Statistical Areas (MSA) and non-MSA counties (MSA- a high population density at its core and close economic ties throughout the area (e.g., Tampa/St. Petersburg or Dallas-Ft. Worth). There is an oversampling of Blacks and Floridians and subsequently, sample weights at the individual and household levels are provided by HRS and based on U.S. Census data (Heeringa & Connor, 1995).

Baseline interviews were typically conducted face-to-face (FTF), with follow-up interviews in subsequent years conducted by telephone from 1994 until 2004. In 2004, HRS added new features for data collection: measurements of physical health (blood pressure, lung function, gait, balance, and grip strength) which were introduced into the study which required an enhanced FTF interview that could not be conducted over the phone and a Leave-Behind Questionnaire (LBQ) or electronic questionnaire focused on psychosocial data collection (Clarke et. al., 2008; Fisher & Ryan 2018). Thus, in 2006, the survey mode changed so that half of the sample received an enhanced FTF interview and the other half of the sample received a telephone interview or alternate interview
method. This resulted in all participants receiving an enhanced FTF interview every 4 years, beginning with the 2006/2008 waves. In total, 43,478 individuals have participated in the Health and Retirement Study through the 2016 wave (Fisher & Ryan 2018). Data from all waves can be accessed by the public on the HRS website (https://hrs.isr.umich.edu/).

**The Sample Population**

For the purpose of this study, data was analyzed using the results of the 2016 HRS. In the 2016 wave, 20,912 individuals completed at least a portion of the HRS. However, only 6,245 individuals met the inclusion criteria for this analysis. Inclusion for the present analyses was based on the following criteria: 1) Individuals self-identified as either Black/African-American or White/Caucasian; 2) were at least 50 years old, 3); completed the LBQ or electronic questionnaire on psychosocial measurements; 4) were single hand dominant (either left-dominant, right-dominant, but not both); 5) gave full effort on hand grip trials; and 6) completed all four hand grip trials (two measurements for each hand).

**Demographic Characteristics of the Sample Population**

More than half of the individuals in the present sample were females ($n = 3582$, 57.4%) (Table 1). Most individuals included in the sample were White/Caucasian ($n = 4746$, 76.0%). Additionally, most participants were right-hand dominant ($n = 5642$, 90.3%).

The average age of individuals in the present sample was 66.85 years old ($sd = 11.04$), with the youngest individual being 50 years old, and the oldest individual being a
102-year-old. Individuals’ ages were categorized into three groups: 1) 50 to 64-year-olds; 2) 65 to 79-year-olds; and 3) 80-year-olds or older. There were 3035 individuals in the 50 to 64-year-old group (48.6%), 2228 in the 65 to 79-year-old group (35.7%), and 982 in the 80 or older group (15.7%).

Respondents also provided information regarding their highest level of education. The highest proportion of respondents reported they had a GED or high school degree \( (n = 3035, 48.6\%) \). Fewer participants reported having a Master’s degree \( (n = 502, 8.0\%) \) or professional degree \( (n = 205, 3.3\%) \), a four-year college degree \( (n = 918, 14.7\%) \), a two-year degree \( (n = 402, 6.4\%) \), or no degree \( (n = 911, 14.6\%) \). 4.4% of respondents did not know or had at least some college education \( (n = 272) \). However, for all statistical analysis that include education level, participants were distinguished as being a GED/High School graduate or less or by having anything more than a GED/High School level of education. The number of respondents who had a GED/High School or less was 3946 (63.2%) and 2027 (32.5%) had achieved more than a high school degree or equivalency.

**Table 1.**

_Demographic Characteristics of Sample from HRS 2016 (n=6245)_

<table>
<thead>
<tr>
<th></th>
<th>Frequency (n)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>2663</td>
<td>42.6</td>
</tr>
<tr>
<td>Female</td>
<td>3582</td>
<td>57.4</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White/Caucasian</td>
<td>4746</td>
<td>76.0</td>
</tr>
<tr>
<td>Black/African-American</td>
<td>1499</td>
<td>24.0</td>
</tr>
<tr>
<td><strong>Handedness</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left-hand dominant</td>
<td>603</td>
<td>9.7</td>
</tr>
<tr>
<td>Right-hand dominant</td>
<td>5642</td>
<td>90.3</td>
</tr>
</tbody>
</table>
Determining Depression Statistics in the Sample Population

HRS has used several different tools as measurements of depression over different waves. In the 2016 wave, a shortened version of the CES-D, comprised of eight questions, was used to determine depression in the study sample. First designed by Radloff in 1977, the original CES-D comprises 20 items designed to assess the level of depressive symptomology. A copy of the original questions can be found in Appendix B. HRS uses an eight-question shortened form of the CES-D, with respondents answering in a simple yes/no/don’t know/refused format which has been found to be a reliable measurement of depression (Turvey, Wallace & Herzog, 1999; Van de Velde, Levecque & Bracke, 2009). The eight items from the original CES-D included in the shortened form include: feeling depressed, feeling everything was an effort, sleep was restless, feeling happy, feeling lonely, enjoying life, feeling sad, feeling as if couldn’t get going. The two questions related to happiness and enjoying life were reverse scored in the analysis. This means that if the respondent first replied “yes” they felt depressed, and then “no” they weren’t enjoying life, each response would be assigned an arbitrary value of “one.” In this model depression can be analyzed on a scale of zero to eight, with a

<table>
<thead>
<tr>
<th>Education</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No degree</td>
<td>911</td>
<td>14.6</td>
</tr>
<tr>
<td>GED/High School</td>
<td>3035</td>
<td>48.6</td>
</tr>
<tr>
<td>Two-year degree</td>
<td>402</td>
<td>6.4</td>
</tr>
<tr>
<td>Four-year degree</td>
<td>918</td>
<td>14.7</td>
</tr>
<tr>
<td>Master’s degree</td>
<td>502</td>
<td>8.0</td>
</tr>
<tr>
<td>Professional Degree</td>
<td>205</td>
<td>3.3</td>
</tr>
<tr>
<td>Unknown/some college</td>
<td>272</td>
<td>4.4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Age (Category)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>50 – 64 years old</td>
<td>3035</td>
<td>48.6</td>
</tr>
<tr>
<td>65 – 79 years old</td>
<td>2228</td>
<td>35.7</td>
</tr>
<tr>
<td>80 years or older</td>
<td>982</td>
<td>15.7</td>
</tr>
</tbody>
</table>
zero-score meaning no depressive symptoms and a score of eight indicative of the highest-level depressive symptoms. A score greater than or equal to 3 has a sensitivity of 0.71 and a specificity of 0.79 in predicting depressive disorder (Xiang et al., 2018). Respondents were classified as having sub-threshold depression if they had a score of three or four. They were classified as having major depression with a score of five or higher (Xiang et al., 2018). The following represents a summary of depression variables of the study population.

**Depression Variables.** Individuals in the sample had depression scores ranging from zero to eight \( (m = 1.42, sd = 1.95) \). Scores ranging from zero to two were considered “no depression”, scores ranging from three to four were “sub-threshold depression”, and scores of five or higher were coded as “major depression”.

Most individuals in the sample did not have depression \( (n = 4952, 79.3\%) \). Fewer individuals had sub-threshold depression \( (n = 674, 10.8\%) \) or major depression \( (n = 602, 9.7\%) \). Moreover, most individuals in the sample did not have a previous depression diagnosis \( (n = 3942, 63.1\%) \). Fewer participants had a previous depression diagnosis \( (n = 1170, 18.7\%) \) or previous diagnoses were unknown \( (n = 1133, 18.1\%) \). Frequencies and percentages of individuals with major, sub-threshold and no depression across each demographic variable (e.g., gender, race, age, education level, comorbidity) can be found in Table 2. Of note, there were 271 individuals who claimed their education level as Unknown/Some College, since it is unable to be determined if these individuals graduated High School or obtained a GED, they were excluded from subsequent analysis.
Table 2.

Count and Frequency of Major, Sub-threshold and no Depression across Demographic Variables (n=6245)

<table>
<thead>
<tr>
<th></th>
<th>Major Depression</th>
<th>Subthreshold Depression</th>
<th>No Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td><strong>Overall</strong></td>
<td>602</td>
<td>9.7</td>
<td>674</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>180</td>
<td>29.9</td>
<td>277</td>
</tr>
<tr>
<td>Female</td>
<td>422</td>
<td>70.1</td>
<td>397</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>418</td>
<td>69.4</td>
<td>475</td>
</tr>
<tr>
<td>Black</td>
<td>184</td>
<td>30.6</td>
<td>199</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50-64</td>
<td>360</td>
<td>59.8</td>
<td>346</td>
</tr>
<tr>
<td>65-79</td>
<td>167</td>
<td>27.7</td>
<td>217</td>
</tr>
<tr>
<td>80+</td>
<td>75</td>
<td>12.4</td>
<td>111</td>
</tr>
<tr>
<td><strong>Previous Diagnosis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>282</td>
<td>62.7</td>
<td>208</td>
</tr>
<tr>
<td>No</td>
<td>168</td>
<td>37.3</td>
<td>313</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GED/HS</td>
<td>408</td>
<td>67.8</td>
<td>478</td>
</tr>
<tr>
<td>GED/HS +</td>
<td>156</td>
<td>25.9</td>
<td>157</td>
</tr>
<tr>
<td><strong>Comorbidity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>45</td>
<td>7.5</td>
<td>67</td>
</tr>
<tr>
<td>1</td>
<td>106</td>
<td>17.6</td>
<td>124</td>
</tr>
<tr>
<td>2+</td>
<td>447</td>
<td>74.2</td>
<td>479</td>
</tr>
</tbody>
</table>

Additionally, most individuals in the sample did not take drugs regularly for anxiety or depression (n = 3889, 62.3%). However, more than half of individuals with major depression took drugs regularly (n = 300, 57.4%) and approximately 35.7% of individuals with sub-threshold depression took drugs regularly (n = 204).

**Background Analysis**

Although previous studies have noted that predisposing factors of MDD can impact a depression diagnosis, a review of the literature has produced conflicting results
regarding whether there is a relation between race and depression and if the prevalence of depression is higher in individuals who identify as Black/African American in comparison to individuals who identify as White/Caucasian (Assari & Lankarani, 2016; Cole & Dendukuri, 2003). Prior research has found that of those diagnosed with depression, Black/African American individuals are less likely to use antidepressants (variable- “medication”) than White/Caucasian individuals (Gonzales et al., 2008). Although not the primary focus of this particular study, a background analysis was conducted with the study population to address the follow questions:

1) Are race, gender, age, education level, comorbidities and previous diagnosis predisposing factors that impact levels of depression in this sample population?

2) Are there depression differences between White/Caucasian and Black/African American individuals?

3) Are there differences in the use of medication (antidepressants) between White/Caucasian and Black/African American individuals?

4) Does gender and/or race impact use of medication?

5) Does insurance status impact use of medication?

Methods

To answer the first question, a multiple linear regression was calculated to predict continuous depression symptom scores to determine if the predisposing factors of race, gender, previous diagnosis, age, comorbidity, and education impact levels of depression. Previous studies have already noted that race, gender and previous diagnosis do impact depression levels and this analysis was done just to confirm that this was also true in the
study sample (Assari & Lankarani, 2016; Cole & Dendukuri, 2003). Three separate analyses were conducted to assess questions two through four. First, a chi-square test of independence was performed to examine the relationship between race and depression. Although a review of the literature has produced conflicting data, it was predicted Black/African American individuals in the sample would have higher rates of depression than White/Caucasians. Then, a chi-square test of independence was conducted to determine whether there were differences between White/Caucasian and Black/African American individuals (among those with depressive symptoms) in terms of their medication usage. Finally, the interaction between gender and race in terms of medication usage was assessed by examining a three-way contingency table. Separate Chi-square analyses were conducted to determine whether significant differences in medication use across races was observed in males and/or females. All statistical analysis for this study was conducted using IBM® SPSS® Statistical Package Version 26 and sample weights provided by the HRS and based on U.S. Census data were considered for all subsequent analysis to account for the oversampling of Black/African American individuals in the sample population (Heeringa & Connor, 1995). For all subsequent analysis presented in this study, the reference group for race is Black/African American, for gender is female, for age is 50-64, and for education is high school graduate/GED or less.

Results

In order to confirm that the findings in previous studies that race, gender, age, education level, comorbidities and previous diagnosis can impact depression, a continuous depression symptom score (ranging from zero to eight) was calculated as the dependent variable, based on the following categorical predictor/independent variables:
Previous depression diagnosis, gender, race, age, comorbidity, and education. Previous depression diagnosis was coded as 0 = no and 1 = yes. Gender was coded as 0 = females, 1 = males. Race was coded as 0 = Black/African American, 1 = White/Caucasian. Age was coded as 0 = 50-64, 1 = 65-79, and 2 = 80+. Comorbidity was analyzed by categorizing chronic conditions as 0, 1, or 2 + and education was coded as HS/GED = 0, and HS/GED+ = 1. An F-test of overall significance was run in order to determine if a regression model including gender, race, previous diagnosis, age, education, and comorbidity is a better fit than a model that predicts depression but contains none of the predisposing risk factors for depression. R-squared or $R^2$ is also reported in order to determine the response variable variation that is explained by the regression model. A significance value of $p \leq 0.05$ was set as the level of statistical significance for all analysis.

A significant regression equation was found ($F(6,5075) = 157.824$, with an $R^2 = .157$). This means that the combined effect of these three variables accounted for approximately 15.7% of the variability in depression symptom scores. Individuals’ predicted depression symptom score was equal to $.994 + 1.55(\text{Previous depression diagnosis}) - 0.132(\text{Gender}) - 0.421(\text{Race}) - .06(\text{Age Group}) + .289(\text{Comorbidity}) - .095(\text{Education})$. Predicted depression scores increased by 1.55 for individuals with a previous diagnosis of depression. Depression scores for those with a previous diagnosis ($m = 2.59$, $sd = 1.90$) were significantly higher than those with no previous diagnosis, $p = .001$. There was a significant difference in education group. Those with only a HS or GED had higher depression scores ($m = 1.53$, $sd = 1.99$) than those with more than a HS degree ($m = 1.12$, $sd = 1.77$), $p < .001$. There was a significant relationship between
comorbidity and depression scores, $p < .001$. As depression scores increased, comorbidity increased. There was no significant difference on depression scores due to age groups.

All of the independent variables were significant predictors of depression symptom scores with the exception of age (Table 3).

**Table 3.**

*Multiple Regression of Previous Diagnosis, Gender, Race, Age, Comorbidity, and Education on Depression Scores*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>.994</td>
<td>.082</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Previous Diagnosis</td>
<td>1.55</td>
<td>.06</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.132</td>
<td>.05</td>
<td>0.014</td>
</tr>
<tr>
<td>Race</td>
<td>-0.421</td>
<td>.06</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Age Group</td>
<td>-0.060</td>
<td>.036</td>
<td>.682</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>.289</td>
<td>.037</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Education</td>
<td>-0.095</td>
<td>.28</td>
<td>.001</td>
</tr>
</tbody>
</table>

*B*- Unstandardized beta. The value represents the slope of the line between the predictor variable and the dependent variable.

*SE*- Standard error of the regression. Represents the average distance that the observed values fall from regression line.

*P*- Statistical significance value set at .05.

Furthermore, a chi-square test of independence was performed to examine the relation between race and depression. The proportion of individuals with depression differed by race, $\chi^2 (2) = 31.976, p < .001$. In the present sample, rates of sub-threshold and major depression were higher for Black/African American individuals than White/Caucasian individuals: 81.1% of White/Caucasian individuals in the sample had no depression, while 74.4% of Black/African-American individuals in the sample had no depression; 10.0% of White/Caucasian individuals had sub-threshold depression, while 13.3% of Black/African-American individuals in the sample had sub-threshold
depression; and 8.8% of White/Caucasian individuals in the sample had major depression, while 12.3% of Black/African American individuals in the sample had major depression. Table 4 displays the count and percentage of individuals in each depression category for each race.

**Table 4.**

_Cross-Tabulation of Race and Depression_

<table>
<thead>
<tr>
<th>Race</th>
<th>White/Caucasian</th>
<th>Black/African American</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Depression</td>
<td>3840 (81.1%)</td>
<td>1112 (74.4%)</td>
</tr>
<tr>
<td>Sub-Threshold Depression</td>
<td>475 (10.0%)</td>
<td>199 (13.3%)</td>
</tr>
<tr>
<td>Major Depression</td>
<td>418 (8.8%)</td>
<td>184 (12.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>4733</td>
<td>1495</td>
</tr>
</tbody>
</table>

There were 1276 individuals in the sample with sub-threshold or major depression. Of those individuals, it was tested whether the rates of medication use differed by race. The null hypothesis predicted no difference in medication usage rates by race. The alternative hypothesis predicted that White/Caucasian individuals would have higher rates of medication. A chi-square test of independence was performed to examine the relation between rates of medication use and race (for individuals with sub-threshold or major depression). The proportion of medication use in individuals with sub-threshold or major depression did indeed differ by race, $X^2 (3) = 11.346, p = .010$. Approximately 48.9% of White/Caucasians in the sample with sub-threshold or major depression took medication, whereas approximately 40% of Black/African Americans in the sample with sub-threshold or major depression took medication. Table 5 displays the count and percentage of individuals who used and did not use medication across each race.
Table 5.

Cross-Tabulation of Race and Medication Use for Depressed Individuals

<table>
<thead>
<tr>
<th>Medication Use</th>
<th>White/ Caucasian</th>
<th>Black/ African American</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>371 (48.9%)</td>
<td>133 (39.6%)</td>
</tr>
<tr>
<td>No</td>
<td>385 (50.8%)</td>
<td>202 (60.1%)</td>
</tr>
<tr>
<td>n/a</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>758</td>
<td>336</td>
</tr>
</tbody>
</table>

Of the 1094 individuals who had either major or sub-threshold depression, there was no significant difference between White/Caucasian males who took medication and Black/African males, $X^2 (3) = 5.202, p = .158$. However, White/Caucasian males took medication 8.0% more often than Black/African American males ($43.1 - 35.1 = 8.0$). The difference between medication use in females across race was significant, $X^2 (1) = 6.151, p = .013$. White/Caucasian females took medication 10.0% more often than Black/African American females ($52.1 - 42.1 = 10.0$). Table 6 displays the three-way contingency table examining medication use, gender, and race.

Table 6.

3-way Contingency Table of Gender, Medication Use and Race of Depressed Individuals

<table>
<thead>
<tr>
<th>Gender</th>
<th>Medication Use</th>
<th>White/ Caucasian</th>
<th>Black/ African American</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>Yes</td>
<td>109 (43.1%)</td>
<td>40 (35.1%)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>144 (56.9%)</td>
<td>74 (64.9%)</td>
</tr>
<tr>
<td>Females</td>
<td>Yes</td>
<td>262 (52.1%)</td>
<td>93 (42.1%)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>241 (47.9%)</td>
<td>128 (57.9%)</td>
</tr>
<tr>
<td>Total</td>
<td>Yes</td>
<td>371 (49.1%)</td>
<td>133 (39.7%)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>385 (50.9%)</td>
<td>202 (60.3%)</td>
</tr>
</tbody>
</table>
Conclusion

In summary, for this sample population, race, gender, previous diagnosis, education, and comorbidity were confirmed to be predisposing factors in a depression diagnosis. In addition, the proportion of individuals with depression differed by race, $X^2 (2) = 31.976, p < .001$. Rates of sub-threshold and major depression were higher for Black/African American individuals than White/Caucasian individuals. Also, the proportion of medication use in individuals with sub-threshold or major depression did indeed differ by race, $X^2 (3) = 11.346, p = .010$. Although there was no significant difference between White/Caucasian males who took medication and Black/African males, $X^2 (3) = 5.202, p = .158$. There was a significant difference between medication use in females across race, $X^2 (1) = 6.151, p = .013$. 
Chapter 4

Social Capital and Depression

Study Aims

Social capital can potentially serve as a protective factor for depressive symptoms in older adults and may influence their decision to seek treatment; however, use of social capital may differ by race and/or gender (Irwin et al., 2008; Murayama et al., 2015). Thus, if social capital is going to be used in a targeted public health intervention to reduce depression in older adults, it is important to understand how different subgroups within the population are impacted by social capital, and, if social capital is found to reduce depressive symptoms, to determine what type of social capital is most effective.

The aim of this portion of the analysis is to explore social capital possession differences between Black/African American and White/Caucasian older adults with and without depressive symptoms. Furthermore, this analysis will be able to:

a) Determine if social capital serves as a protective measure against depression
   a. Hypothesis: social capital measures will serve as a buffer, or protective factor against depression and will vary by race and/or gender.

b) Assess how social capital may influence the decision to seek treatment for depression
   a. Hypothesis: social capital will influence individuals in seeking out treatment for depressive symptoms and will vary by race and/or gender.
Social Capital Variables

The HRS does not specifically measure social capital as a variable in the psychosocial component of the LBQ. However, there is information pertaining to participants’ family status, including their marital status, spousal living arrangements, number of children, family and friends they are close with, and ratings of those close ties. These measures served as the bases for participants’ social capital variable(s). For the purpose of this study social capital was measured in four different ways; structural social capital was operationalized as measures of social participation (structural member) and volunteering and functional social capital was operationalized using data related to close ties and perceived social support (Rouxel et al., 2015). Thus, there are two measures of structural social capital, including structural member and structural volunteer and two measures of functional social capital, including close ties and social support included in this study.

Structural Social Capital

Structural Member Social Capital

Data for determining structural member social capital was determined from the original 20 questions the LBQ asked about social participation and social engagement. Of the original 20 items covered in this section of the LBQ, there were five items that could allow us to reasonably assume that at least one other individual was involved in the activity from which the participant could potentially gain some social capital. For example, questions that did not infer interaction with other individuals such as: how often do you walk for 20 minutes or more, were not included in the calculation for structural
member social capital. The five items that were included in the calculation for structural member social capital were:

1) Do activities with grandchildren, nieces/nephews, or neighborhood children?

2) Attend an educational or training course?

3) Go to a sport, social, or other club?

4) Attend meetings of non-religious organizations, such as political, community, or other interest groups?

5) Participate in a local community arts group such as a choir, dance, photography, theatre, or music group?

Respondents were able to specify how often they engaged in these activities, with the options being daily, several times a week, once a week, several times a month, at least once a month, not in the last month, or never/not relevant. If they responded that they engaged in any of these activities at least once a month or more, then this was included in calculating their structural member score of social capital (0 = no, 1 = yes). A Structural membership score was calculated as the sum of the five structural social capital variables, with a five indicating a high level of structural member social capital.

Approximately 36.5% of individuals participated in activities with their grandchildren (n = 2282) and 32.8% did not (n = 2047). Approximately 30.7% of individuals did not respond to this item (n = 1916). Most individuals did not participate in educational events (n = 3743, 59.9%), non-religious activities (n = 3567, 57.1%), nor community arts programs (n = 3897, 62.4%). Almost half of individuals did not
participate in clubs \(n = 2820, 45.2\%\). *Structural membership* scores varied from zero to five \(m = 1.27, sd = 1.13\).

*Structural Volunteer*

Calculation of *structural volunteerism* consisted of two questions from the original 20 asked on the LBQ about social participation and social engagement. Respondents were asked how often they:

1) Do volunteer work with children or young people?

2) Do any other volunteer or charity work?

Possible responses included: daily, several times a week, once a week, several times a month, at least once a month, not in the last month, or never/not relevant. If they responded that they engaged in any of these activities at least once a month or more, then this was included in calculating their structural member score of social capital with a yes/no response \(0 = \text{no}, 1 = \text{yes}\). Most individuals did not volunteer with youth \(n = 3711, 59.4\%\), and approximately half of individuals in the sample did not volunteer with charity \(n = 3129, 50.1\%\). *Structural volunteer* scores varied from zero to two \(m = 0.42, sd = 0.66\). For a frequencies and percentages of responses to structural social capital measures, see Table 7.
Table 7.

**Structural Social Capital Measures**

<table>
<thead>
<tr>
<th>Structural Social Capital</th>
<th>Frequency (n)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Structural Membership</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activities with Grandchildren</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>2282</td>
<td>36.5</td>
</tr>
<tr>
<td>No</td>
<td>2047</td>
<td>32.8</td>
</tr>
<tr>
<td>Unknown</td>
<td>1916</td>
<td>30.7</td>
</tr>
<tr>
<td>Educational Events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>570</td>
<td>9.1</td>
</tr>
<tr>
<td>No</td>
<td>3743</td>
<td>59.9</td>
</tr>
<tr>
<td>Unknown</td>
<td>1932</td>
<td>30.9</td>
</tr>
<tr>
<td>Clubs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1514</td>
<td>24.2</td>
</tr>
<tr>
<td>No</td>
<td>2820</td>
<td>45.2</td>
</tr>
<tr>
<td>Unknown</td>
<td>1911</td>
<td>30.6</td>
</tr>
<tr>
<td>Non-Religious Activities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>764</td>
<td>12.2</td>
</tr>
<tr>
<td>No</td>
<td>3567</td>
<td>57.1</td>
</tr>
<tr>
<td>Unknown</td>
<td>1914</td>
<td>30.6</td>
</tr>
<tr>
<td>Community Arts Programs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>446</td>
<td>7.1</td>
</tr>
<tr>
<td>No</td>
<td>3897</td>
<td>62.4</td>
</tr>
<tr>
<td>Unknown</td>
<td>1902</td>
<td>30.5</td>
</tr>
<tr>
<td><strong>Structural Volunteer</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volunteer with youth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>624</td>
<td>10.0</td>
</tr>
<tr>
<td>No</td>
<td>3711</td>
<td>59.4</td>
</tr>
<tr>
<td>Unknown</td>
<td>1910</td>
<td>30.6</td>
</tr>
<tr>
<td>Volunteer with charity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1204</td>
<td>19.3</td>
</tr>
<tr>
<td>No</td>
<td>3129</td>
<td>50.1</td>
</tr>
<tr>
<td>Unknown</td>
<td>1912</td>
<td>30.6</td>
</tr>
</tbody>
</table>

**Functional Social Capital**

Functional social capital shapes behavior and measures the relational aspects of social interactions and the quality of relationships (De Silva et al., 2005; Rouxel et al., 2015). Previous studies have used measurements of social support and the quality of support available to an individual as measures of functional social capital. Subsequently, functional social capital was measured in two ways, close ties and social support. Close
ties measures included the number of children, family and friends that individuals self-reported that they are close with, as well as whether they live with their spouse (0 = no, 1 = yes). A Close Ties score was calculated as the sum of those four items. Social support was measured by asking individuals to rate how much they could rely on their spouse, children, family, and friends in an emergency (0 = not at all, 1 = a little, 2 = some, 3 = a lot). A Social Support score was calculated as the sum of those ratings across all four items, with a higher number equating to a higher level of social support.

Most individuals were either married (n = 3434, 55.0%) or partnered (n = 404, 6.5%). 38.5% of participants listed their marital status as “other” (n = 2405). Approximately 39.4% of individuals in the sample lived with their spouse (n = 2461), and 23.8% (n = 1485) did not. There were data missing from 36.8% of the sample regarding spousal living arrangements (n = 2299).

On average, respondents had approximately 2.51 children (sd = 2.73), 3.82 family members (sd = 5.14) with closer relationships, and 4.32 friends (sd = 5.91) with close relationships. A subsequent measure of “close ties” was calculated as the sum of the number of children, family, friends, and whether respondents live with a spouse. Close ties scores ranged from zero to 173 (m = 10.05 sd = 9.71).

Individuals were also asked about their relationships with their 1) spouses, 2) children, 3) family, and 4) friends. Specifically, they were asked whether they could rely on their spouse, children, family and friends in an emergency. Responses ranged from 0 (not at all) to 3 (a lot). Additionally, a “social support” score was calculated as the sum of the scores for spouse, child, family, and friends. Social Support scores ranged from zero
to 12 \( (m = 7.60 \; sd = 2.69) \). Table 8 displays response frequencies for measures of social support items.

Table 8.

*Measures of Social Support*

<table>
<thead>
<tr>
<th>Rely on in an Emergency</th>
<th>Frequency (n)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Spouse</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not at all</td>
<td>63</td>
<td>1.0</td>
</tr>
<tr>
<td>A little</td>
<td>139</td>
<td>2.2</td>
</tr>
<tr>
<td>Some</td>
<td>347</td>
<td>5.6</td>
</tr>
<tr>
<td>A lot</td>
<td>2314</td>
<td>37.1</td>
</tr>
<tr>
<td>No Response</td>
<td>3382</td>
<td>54.2</td>
</tr>
<tr>
<td><strong>Child(ren)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not at all</td>
<td>166</td>
<td>2.7</td>
</tr>
<tr>
<td>A little</td>
<td>369</td>
<td>5.9</td>
</tr>
<tr>
<td>Some</td>
<td>905</td>
<td>14.5</td>
</tr>
<tr>
<td>A lot</td>
<td>2371</td>
<td>38.0</td>
</tr>
<tr>
<td>No Response</td>
<td>2434</td>
<td>39.0</td>
</tr>
<tr>
<td><strong>Family</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not at all</td>
<td>476</td>
<td>7.6</td>
</tr>
<tr>
<td>A little</td>
<td>7.8</td>
<td>11.8</td>
</tr>
<tr>
<td>Some</td>
<td>1149</td>
<td>18.4</td>
</tr>
<tr>
<td>A lot</td>
<td>1675</td>
<td>26.8</td>
</tr>
<tr>
<td>No Response</td>
<td>2207</td>
<td>35.3</td>
</tr>
<tr>
<td><strong>Friends</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not at all</td>
<td>192</td>
<td>3.1</td>
</tr>
<tr>
<td>A little</td>
<td>756</td>
<td>12.1</td>
</tr>
<tr>
<td>Some</td>
<td>1476</td>
<td>23.6</td>
</tr>
<tr>
<td>A lot</td>
<td>1494</td>
<td>23.9</td>
</tr>
<tr>
<td>No Response</td>
<td>2327</td>
<td>37.3</td>
</tr>
</tbody>
</table>

**Methods and Results**

Aim 1: Explore social capital possession differences between Black/African American and White/Caucasian older adults with and without depressive symptoms.

In order to determine if there were social capital possession differences between Black/African American and White/Caucasian older adults with and without depressive symptoms.
symptoms, first it was determined if types of social capital differed between Black/African American and White/Caucasian adults over the age of 50.

Social capital was categorized as structural social capital or functional social capital. There were two composite variables calculated to measure structural social capital: 1) member structural, and 2) volunteer structural. There were two composite variables calculated to measure functional social capital: 1) close ties, and 2) social support. A series of four independent t-tests were conducted, treating each of the social capital variables (two structural and two functional) as the dependent variables and race as the independent variable with two levels: White/Caucasian and Black/African American. The alpha level was set to .05 for significance. Additionally, Cohen’s d effect size was calculated to measure the magnitude of difference between White/Caucasians and Black/African Americans in standard deviation units. Cohen’s d, or standardized mean difference is used to measure effect size, while the p-value can tell whether there is an effect, Cohen’s d indicates the practical significance (Lakens, 2013). Cohen suggested that a Cohen’s d = .2 be considered a small effect size, while a Cohen’s d = .5 represents a medium effects size and a Coehn’s d = .8 represents a large effect size (Cohen, 1988).

There were significant differences in White/Caucasians and Black/African Americans across all four variables measuring social capital (See Table 9 for specific values). Black/African Americans had significantly higher scores on the member structural and volunteer structural measures than White/Caucasians. However, the magnitude of each of these effects was small (Cohen, 1988). Additionally, Black/African Americans had significantly higher scores on the close ties measure than White/Caucasians, and Black/African Americans had significantly lower scores on the
social support measure than White/Caucasians. Again, the magnitude of each of these effects was small (Cohen, 1988).

Table 9.

Means (and Standard Deviations) of Social Capital Variables by Race

<table>
<thead>
<tr>
<th>Social Capital Measures</th>
<th>White/ Caucasian</th>
<th>Black/ African American</th>
<th>t</th>
<th>p</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural Social Capital</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Member structural</td>
<td>1.24 (1.10)</td>
<td>1.39 (1.22)</td>
<td>-3.14</td>
<td>&lt; .001</td>
<td>0.13</td>
</tr>
<tr>
<td>Volunteer structural</td>
<td>0.40 (0.63)</td>
<td>0.52 (0.76)</td>
<td>-4.30</td>
<td>&lt; .001</td>
<td>0.17</td>
</tr>
<tr>
<td>Functional Social Capital</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Close Ties</td>
<td>9.70 (9.02)</td>
<td>11.57 (12.15)</td>
<td>-4.14</td>
<td>&lt; .001</td>
<td>0.17</td>
</tr>
<tr>
<td>Social Support</td>
<td>7.69 (2.68)</td>
<td>7.21 (2.69)</td>
<td>4.64</td>
<td>&lt; .001</td>
<td>0.18</td>
</tr>
</tbody>
</table>

*p- Statistical significance value set at .05
Significant p-values are bolded

Then the data was analyzed to determine what types of social capital possession differences there were between White/Caucasian and Black/African American older adults with depressive symptoms. For this analysis, the same measures of Structural and Functional social capital that were assessed in the previously were assessed in this analysis. However, in this analysis the sample was restricted to those with either major or sub-threshold depression ($n = 1276$). A series of four independent t-tests were conducted, treating each of the social capital variables (two structural and two functional) as the dependent variables and race as the independent variable with two levels: White/Caucasian and Black/African American. The alpha level was set to .05 for significance. Additionally, Cohen’s d effect size was calculated as a measure of the magnitude of difference between White/Caucasians and Black/African Americans in standard deviation units.
For the structural social capital measures, there were (marginally) significant differences between White/Caucasians and Black/African Americans on member structural scores, $t = -1.92, p = .056$, and volunteer structural scores, $t = -1.94, p = .054$ (Table 10). The magnitudes of the effects were small ($Member structural$ Cohen’s $d = 0.16$; $Volunteer structural$ Cohen’s $d = 0.18$). This means that, on average, Black/African Americans scores on the member structural measure were only 0.16 standard deviations higher than White/Caucasians, and 0.18 standard deviations higher on the volunteer structural measure.

Table 10.

Means (and Standard Deviations) of Social Capital Variables for Individuals with Depression by Race

<table>
<thead>
<tr>
<th>Social Capital Measures</th>
<th>White/Caucasian</th>
<th>Black/African American</th>
<th>t</th>
<th>p</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Structural Social Capital</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Member structural</td>
<td>0.93 (1.01)</td>
<td>1.10 (1.10)</td>
<td>-1.92</td>
<td>.056</td>
<td>0.16</td>
</tr>
<tr>
<td>Volunteer structural</td>
<td>0.26 (0.54)</td>
<td>0.37 (0.68)</td>
<td>-1.94</td>
<td>.054</td>
<td>0.18</td>
</tr>
<tr>
<td><strong>Functional Social Capital</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Close Ties</td>
<td>7.66 (6.67)</td>
<td>11.56 (15.42)</td>
<td>-3.39</td>
<td>.001</td>
<td>0.33</td>
</tr>
<tr>
<td>Social Support</td>
<td>6.42 (2.87)</td>
<td>6.32 (2.75)</td>
<td>0.43</td>
<td>.669</td>
<td>n/a</td>
</tr>
</tbody>
</table>

$p$: Statistical significance value set at .05
Significant $p$-values are bolded

For the functional social capital measures, there was a significant difference between White/Caucasians and Black/African Americans on the close ties measure, $t = -3.39, p < .001$ (Table 10). On average, Black/African Americans scores were 0.33 standard deviations higher than White/Caucasians. There was no statistical difference between races on the social support measure, $p = .67$. 
Aim 1a: Determine if social capital serves as a protective measure against depression

In order to determine if social capital serves as a protective factor against depression, the first area of interest is to determine if social capital measures significantly predicted depression scores. Specifically, it was predicted that the social capital measures would serve as a buffer, or protective factor against depression. This would be supported if, controlling for the non-modifiable variables of gender, race, previous diagnosis, age, education level, and comorbidity, there was a decrease in depression symptoms for each social capital measure. The null hypothesis associated with each of the social capital measures would be that there is no significant change in depression symptoms based on each individual social capital measure.

To test this, a multiple linear regression was calculated and included previous diagnosis, gender, race, age, education level, comorbidity and the four social capital measures as predictors of depression symptom scores. An F-test of overall significance found a significant regression equation \( F(10,4044) = 96.375 \), with an \( R^2 = .192 \). This means that the combined effect of these ten variables accounted for approximately 19.2% of the variability in depression symptom scores. Individuals’ predicted depression symptom scores was equal to 2.24 + 1.29 (Previous depression diagnosis) – 0.17 (Gender) – 0.35 (Race) – 0.1 (Age) – 0.06 (Education) + 0.247 (Comorbidity) – 0.12 (Structural Member score) – 0.10 (Structural Volunteer score) – 0.001 (Close Ties) – 0.13 (Social Support). The three demographic variables (previous diagnosis, gender, and race) were all significant predictors of depression symptom scores, \( p \)’s < .05 (See Table 11).

Of the social capital variables, both structural social capital measures were significant indicators of depression symptoms. Controlling for all other variables in the
model (previous diagnosis, gender, education, age, comorbidity, and race), for every unit increase in \textit{structural member} scores, depression symptom scores decreased by 0.13 units. Similarly, for every unit increase in \textit{structural volunteer} scores, depression symptoms decreased by 0.10 units. Concerning the functional social capital measures, \textit{social support} was a significant indicator of depression symptoms, \( p < .001 \), but not \textit{close ties}, \( p = .719 \). For every unit increase in social support scores, depression symptoms decreased by 0.13 units.

\textbf{Table 11.}

\textit{Multiple Regression of Previous Diagnosis, Gender, Race, Age, Education, Comorbidity and Social Capital on Depression Scores}

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>2.24</td>
<td>0.12</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Previous Diagnosis</td>
<td>1.30</td>
<td>0.07</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.17</td>
<td>0.05</td>
<td>0.002</td>
</tr>
<tr>
<td>Race</td>
<td>-0.35</td>
<td>0.07</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Age</td>
<td>-0.10</td>
<td>0.038</td>
<td>0.009</td>
</tr>
<tr>
<td>Education</td>
<td>-.060</td>
<td>0.031</td>
<td>0.055</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>.247</td>
<td>0.040</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Structural Member</td>
<td>-0.132</td>
<td>0.026</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Structural Volunteer</td>
<td>-0.10</td>
<td>0.04</td>
<td>0.025</td>
</tr>
<tr>
<td>Close Ties</td>
<td>-0.001</td>
<td>0.003</td>
<td>0.719</td>
</tr>
<tr>
<td>Social Support</td>
<td>-0.13</td>
<td>0.01</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

\( B \)- Unstandardized beta. The value represents the slope of the line between the predictor variable and the dependent variable.

\( SE \)- Standard error of the regression. Represents the average distance that the observed values fall from regression line.

\( P \)- Statistical significance value set at .05.

From the 34 variables and interactions terms, a significant regression equation was found (\( F(34,4020) = 30.672 \)), with an \( R^2 = .206 \) (Table 12). This means that the combined effect of all the variables and their interactions accounted for approximately
20.6% of the variability in depression symptom scores, including the interactions in the model slightly increased $R^2$.

Gender, previous diagnosis, and comorbidity were significant predictors of depression, all $p$’s < .05. For the social capital measures, the only significant predictor was social support, $p = .01$. Controlling for all other variables in the model, as social support scores decrease, depression scores increase. There were, however, significant interactions between the other social capital measures and demographic variables.

There was a significant interaction between previous diagnosis and structural member such that the protective factor of structural member (i.e., as structural member increases, depression decreases) was most effective for participants with no previous diagnosis of depression, $p = .01$. There was also a significant interaction between previous diagnosis and social support, $p < .001$. The protective buffer of social support on depression (i.e., lower depression scores with those who scored higher on social support) was more prevalent in individuals with no previous diagnosis.
Table 12.

*Multiple Regression (including Interaction Terms) of Race, Gender, Previous Diagnosis, Age, Education, Multimorbidity and Social Capital on Depression Scores*

<table>
<thead>
<tr>
<th>Predictors</th>
<th>B</th>
<th>SE</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>1.90</td>
<td>0.29</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Race</td>
<td>-0.27</td>
<td>0.21</td>
<td>.197</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.47</td>
<td>0.17</td>
<td>.004</td>
</tr>
<tr>
<td>Previous Diagnosis (PRD)</td>
<td>2.43</td>
<td>0.19</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Age Group</td>
<td>-0.20</td>
<td>0.12</td>
<td>.090</td>
</tr>
<tr>
<td>Education</td>
<td>-0.12</td>
<td>0.10</td>
<td>.218</td>
</tr>
<tr>
<td><strong>Comorbidity</strong></td>
<td>0.39</td>
<td>0.13</td>
<td>.002</td>
</tr>
<tr>
<td><strong>Social Capital Measures</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Structural Member</td>
<td>-0.08</td>
<td>0.09</td>
<td>.367</td>
</tr>
<tr>
<td>Structural Volunteer</td>
<td>0.18</td>
<td>0.14</td>
<td>.206</td>
</tr>
<tr>
<td>Close Ties</td>
<td>-0.004</td>
<td>0.009</td>
<td>.688</td>
</tr>
<tr>
<td><strong>Social Support</strong></td>
<td>-0.09</td>
<td>0.04</td>
<td>.010</td>
</tr>
<tr>
<td><strong>Interactions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race x Structural Member</td>
<td>-0.05</td>
<td>0.07</td>
<td>.461</td>
</tr>
<tr>
<td>Race x Structural Volunteer</td>
<td>0.14</td>
<td>0.11</td>
<td>.214</td>
</tr>
<tr>
<td>Race x Close Ties</td>
<td>-0.01</td>
<td>0.01</td>
<td>.061</td>
</tr>
<tr>
<td>Race x Social Support</td>
<td>0.01</td>
<td>0.03</td>
<td>.731</td>
</tr>
<tr>
<td>Gender x Structural Member</td>
<td>0.010</td>
<td>0.05</td>
<td>.063</td>
</tr>
<tr>
<td>Gender x Structural Volunteer</td>
<td>-0.01</td>
<td>0.09</td>
<td>.934</td>
</tr>
<tr>
<td>Gender x Close Ties</td>
<td>0.000</td>
<td>0.01</td>
<td>.964</td>
</tr>
<tr>
<td>Gender x Social Support</td>
<td>0.02</td>
<td>0.02</td>
<td>.255</td>
</tr>
<tr>
<td><strong>PRD x Structural Member</strong></td>
<td>-0.16</td>
<td>0.07</td>
<td>.013</td>
</tr>
<tr>
<td><strong>PRD x Structural Volunteer</strong></td>
<td>-0.07</td>
<td>0.11</td>
<td>.528</td>
</tr>
<tr>
<td><strong>PRD x Close Ties</strong></td>
<td>-0.01</td>
<td>0.01</td>
<td>.383</td>
</tr>
<tr>
<td><strong>PRD x Social Support</strong></td>
<td>-0.12</td>
<td>0.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Age Group x Structural Member</td>
<td>0.02</td>
<td>0.04</td>
<td>.637</td>
</tr>
<tr>
<td>Age Group x Structural Volunteer</td>
<td>0.01</td>
<td>0.02</td>
<td>.631</td>
</tr>
<tr>
<td>Age Group x Close Ties</td>
<td>-0.09</td>
<td>0.07</td>
<td>.199</td>
</tr>
<tr>
<td>Age Group x Social Support</td>
<td>0.01</td>
<td>0.004</td>
<td>.161</td>
</tr>
<tr>
<td>Education x Structural Member</td>
<td>-0.003</td>
<td>0.04</td>
<td>.936</td>
</tr>
<tr>
<td>Education x Structural Volunteer</td>
<td>0.010</td>
<td>0.05</td>
<td>.841</td>
</tr>
<tr>
<td>Education x Close Ties</td>
<td>-0.001</td>
<td>0.01</td>
<td>.810</td>
</tr>
<tr>
<td>Education x Social Support</td>
<td>0.008</td>
<td>0.01</td>
<td>.478</td>
</tr>
<tr>
<td>Comorbidity x Structural Member</td>
<td>-0.02</td>
<td>0.04</td>
<td>.541</td>
</tr>
<tr>
<td>Comorbidity x Structural Volunteer</td>
<td>-0.04</td>
<td>0.06</td>
<td>.585</td>
</tr>
<tr>
<td>Comorbidity x Close Ties</td>
<td>0.01</td>
<td>0.01</td>
<td>.304</td>
</tr>
<tr>
<td>Comorbidity x Social Support</td>
<td>-0.02</td>
<td>0.02</td>
<td>.140</td>
</tr>
</tbody>
</table>

*Note. Significant effects and interactions in bold*
There were no significant interactions between any of the social capital variables and gender, \( p \)'s > .05. Therefore, the prediction that social capital would impact White/Caucasian women more than the other groups was not supported. There was one slightly significant, negative interaction between race and close ties, \( p = .061 \). For every unit increase in close ties, Black/African Americans’ depression symptoms decreased by 0.01 units, whereas White/Caucasians’ depression symptoms decreased by 0.03 units.

Aim 1b: Assess how social capital may influence the decision to seek treatment (medication) for depression.

One of the enabling factors in the decision to use healthcare services, in this case, using medication to treat depressive symptoms, is health insurance. Individuals often cite concerns about the cost and/or lack of health insurance as one of the main barriers to seeking needed care (Baicker et al., 2018; Rowan, McAlpine, & Blewitt, 2013). Subsequently, the enabling factor of medical insurance was also included in this analysis as an independent variable, in addition to the predisposing factors of gender and race.

First, a chi-square test of independence was conducted to determine whether the proportion of individuals who used medication varied by insurance status and by race. Most individuals had insurance (\( n = 3867, 61.9\% \)). However, there was no difference in medication usage for individuals with and without insurance, and no significance in the proportion of individuals with medication and insurance across races, \( X^2 (1) = 0.328, p = .567 \).
### Table 13.

*Contingency Table of Counts and Frequencies of Race, Medication Use, and Insurance Status*

<table>
<thead>
<tr>
<th>Insurance</th>
<th>Medication Use</th>
<th>White/Caucasian</th>
<th>Black/African American</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Yes</td>
<td>610 (22.9%)</td>
<td>178 (23.0%)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>2058 (77.1%)</td>
<td>597 (77.0%)</td>
</tr>
<tr>
<td>No</td>
<td>Yes</td>
<td>307 (26.5%)</td>
<td>44 (10.4%)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>852 (73.5%)</td>
<td>381 (89.6%)</td>
</tr>
</tbody>
</table>

Furthermore, there was similar medication use for individuals with and without insurance across race and gender (Figure 3). Approximately 20% of Black/African American males with insurance used medication, compared to 17% of Black/African American females, 27% of White/Caucasian Males and 25% of White/Caucasian Females. For individuals without insurance, White/Caucasian males had the highest percentage of medication users (34%), followed by Black/African American females (15%), White/Caucasian females (13%) and Black/African American males (5%) (see striped bars in Figure 3).

![Figure 3. Medication use with and without insurance by race and gender](image-url)
A multivariate logistic regression was conducted to predict medication use (yes or no), treating insurance, race, gender, previous diagnosis, age group, education, comorbidities, the structural social capital measures (Structural Member and Structural Volunteer), and the functional capital measures (Close Ties and Social Support) as independent variables (Table 14). The logistic regression model was statistically significant, $\chi^2(11) = 947.781$, $p < .001$. The model explained approximately 37.8% of the variance in medication use ($R^2 = 0.378$) and correctly classified 62.5% of cases.

Insurance was a significant predictor in the model, $p = .033$. Individuals with insurance were 1.40 times more likely to take medication than those who did not have insurance. Race was also a significant predictor in the model, $p = .025$. White/Caucasian individuals were 1.38 times more likely to take medication than Black/African American individuals. Gender was also a significant predictor, $p = .004$. Males were approximately 27% less likely to take medication than females. Previous diagnosis was the strongest predictor in the model, $p < .001$. Individuals with a previous diagnosis were 15.18 times more likely to take medication than those who did not have a previous diagnosis. Education and multimorbidity were not significant predictors in the model.

For the social capital measures, structural member ($p = .02$) and structural volunteer ($p = .002$) were significant predictors in the model. The likelihood of medication use decreased by 0.89 times for every unit increase in structural member. The likelihood of medication use also decreased by 0.75 times for every unit increase in structural volunteer score. Close ties and social support were not significant predictors in the model.
Table 14.

Multivariate Logistic Regression of Predisposing Factors and Social Capital

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE</th>
<th>P</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-2.16</td>
<td>0.28</td>
<td>&lt;.001</td>
<td>0.12</td>
</tr>
<tr>
<td>Insurance</td>
<td>0.33</td>
<td>0.16</td>
<td>.033</td>
<td>1.40</td>
</tr>
<tr>
<td>Race</td>
<td>0.32</td>
<td>0.14</td>
<td>.025</td>
<td>1.38</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.31</td>
<td>0.11</td>
<td>.004</td>
<td>0.73</td>
</tr>
<tr>
<td>Previous Diagnosis</td>
<td>2.72</td>
<td>0.11</td>
<td>&lt; .001</td>
<td>15.18</td>
</tr>
<tr>
<td>Age Group</td>
<td>-0.29</td>
<td>0.10</td>
<td>.003</td>
<td>0.75</td>
</tr>
<tr>
<td>Education</td>
<td>0.01</td>
<td>0.12</td>
<td>.903</td>
<td>--</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>-0.03</td>
<td>0.09</td>
<td>.743</td>
<td>--</td>
</tr>
<tr>
<td>Structural Member</td>
<td>-0.12</td>
<td>0.05</td>
<td>.020</td>
<td>0.89</td>
</tr>
<tr>
<td>Structural Volunteer</td>
<td>-0.30</td>
<td>0.09</td>
<td>.002</td>
<td>0.75</td>
</tr>
<tr>
<td>Close Ties</td>
<td>-0.004</td>
<td>0.01</td>
<td>.488</td>
<td>--</td>
</tr>
<tr>
<td>Social Support</td>
<td>-0.004</td>
<td>0.02</td>
<td>.859</td>
<td>--</td>
</tr>
</tbody>
</table>

*Note. Significant effects are in bold

B- Unstandardized beta. The value represents the slope of the line between the predictor variable and the dependent variable.

SE- Standard error of the regression. Represents the average distance that the observed values fall from regression line.

P- Statistical significance value set at .05.

However, one of the main interests of this study is to determine whether social capital impacts as an enabling factor in healthcare utilization, in this study operationalized as insurance status and whether this varies by race and gender. Thus, a multivariate logistic regression was conducted to predict medication use (yes or no), treating insurance, race, gender, the structural social capital measures (Structural Member and Structural Volunteer), functional capital measures (Close Ties and Social Support) as independent variables. Moreover, all two-way interactions between gender and social capital measures and race and social capital measures, as well as three-way interactions between gender, race and social capital, were included in the model. In this way, the impact of social capital on medication use, controlling for insurance (a covariate in the
model) was assessed. Additionally, the inclusion of interaction terms in the model will determine whether the potential effect of social capital was modified by gender, race, or both.

The logistic regression model was statistically significant, $X^2 (19) = 144.132, p < .001$. The model explained approximately 6% of the variance in medication use and correctly classified 78.6% of cases. Gender was a significant predictor in the model, $p = .017$. Males were approximately half as likely to take medication as females (Odds Ratio = 0.53). Social support was also a significant predictor of medication use, $p < .001$. The likelihood of medication use decreased by 0.844 times for every 0.17 unit increase in social support scores. In other words, increased social support decreases the likelihood of medication use.

Furthermore, there were several significant interactions. There was a significant interaction between structural volunteer scores and gender, $p = .003$. The likelihood of taking medication decreased more for females than males with increased levels of structural volunteer. Figure 4 displays the average proportion of medication users for males and females with low, medium and high scores on the structural volunteer score (low = 0, medium = 1, high = 2).
Figure 4. *Average proportion of medication users for males and females by structural volunteer scores*

There was also a significant interaction between social support and race, \( p = .003 \).

While increased social support decreases the likelihood of medication use, higher levels of social support were associated with a lower likelihood of medication use for Black/African Americans compared to White/Caucasians (Figure 5).

Figure 5. *Proportion of medication users by race and social support scores*
Finally, there was a significant three-way interaction between social support, gender, and race on predicted medication use. Again, for all individuals, as social support increased, the likelihood of medication usage decreased. However, White/Caucasian females had higher likelihood of medication use than the other groups, particularly when social support scores were higher (Figure 6). Beta coefficients, standard errors, significance levels, and odds ratios for all predictors are displayed in Table 11.

Figure 6. Proportion of medication users by race, gender, and social support
Table 15.

*Multivariate Logistic Regression (including Interactions Terms) of Insurance, Race, Gender, and Social Capital on Medication Use*

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE</th>
<th>P</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-0.11</td>
<td>0.31</td>
<td>.735</td>
<td>0.900</td>
</tr>
<tr>
<td>Insurance</td>
<td>-0.14</td>
<td>0.09</td>
<td>.138</td>
<td>0.870</td>
</tr>
<tr>
<td>Race</td>
<td>-0.23</td>
<td>0.32</td>
<td>.475</td>
<td>0.797</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.64</td>
<td>0.27</td>
<td>.017</td>
<td>0.530</td>
</tr>
<tr>
<td>Structural Member</td>
<td>0.06</td>
<td>0.12</td>
<td>.612</td>
<td>1.063</td>
</tr>
<tr>
<td>Structural Volunteer</td>
<td>-0.44</td>
<td>0.22</td>
<td>.043</td>
<td>0.646</td>
</tr>
<tr>
<td>Close Ties</td>
<td>0.00</td>
<td>0.01</td>
<td>.868</td>
<td>1.002</td>
</tr>
<tr>
<td>Social Support</td>
<td>-0.17</td>
<td>0.05</td>
<td>&lt; .001</td>
<td>0.844</td>
</tr>
<tr>
<td>Structural Member * Gender</td>
<td>-0.28</td>
<td>0.20</td>
<td>.162</td>
<td>0.760</td>
</tr>
<tr>
<td>Structural Volunteer * Gender</td>
<td>0.98</td>
<td>0.33</td>
<td>.003</td>
<td>2.669</td>
</tr>
<tr>
<td>Close Ties * Gender</td>
<td>-0.03</td>
<td>0.03</td>
<td>.298</td>
<td>0.974</td>
</tr>
<tr>
<td>Social Support * Gender</td>
<td>0.10</td>
<td>0.06</td>
<td>.086</td>
<td>1.108</td>
</tr>
<tr>
<td>Structural Member * Race</td>
<td>-0.21</td>
<td>0.13</td>
<td>.117</td>
<td>0.813</td>
</tr>
<tr>
<td>Structural Volunteer * Race</td>
<td>0.20</td>
<td>0.24</td>
<td>.398</td>
<td>1.223</td>
</tr>
<tr>
<td>Close Ties * Race</td>
<td>-0.01</td>
<td>0.01</td>
<td>.324</td>
<td>0.987</td>
</tr>
<tr>
<td>Social Support * Race</td>
<td>0.14</td>
<td>0.05</td>
<td>.003</td>
<td>1.154</td>
</tr>
<tr>
<td>Structural Member * Gender * Race</td>
<td>0.23</td>
<td>0.22</td>
<td>.288</td>
<td>1.264</td>
</tr>
<tr>
<td>Structural Volunteer * Gender * Race</td>
<td>-1.09</td>
<td>0.38</td>
<td>.004</td>
<td>0.336</td>
</tr>
<tr>
<td>Close Ties * Gender * Race</td>
<td>0.04</td>
<td>0.03</td>
<td>.175</td>
<td>1.039</td>
</tr>
<tr>
<td>Social Support * Gender * Race</td>
<td>-0.12</td>
<td>0.06</td>
<td>.033</td>
<td>0.885</td>
</tr>
</tbody>
</table>

*Significant predictors and interactions bolded

B- Unstandardized beta. The value represents the slope of the line between the predictor variable and the dependent variable.

SE- Standard error of the regression. Represents the average distance that the observed values fall from regression line.

P- Statistical significance value set at .05.

**Discussion**

There was variability between Black/African American and White/Caucasian older adults with regard to social capital possession. Black/African Americans had higher scores on three of the four social capital measures (both structural social capital...
measures, and one functional measure (close ties)) compared to White/Caucasians. The same pattern was observed when restricting the sample to only individuals with sub-threshold and/or major depression. One possible explanation for differences in social capital possession by race is that faith-based organizations are a significant institution within the African American community in the United States and an important source of social capital (Holt et al., 2014; Yeary et al., 2012). Another possible reason why Black/African American older adults had higher social capital scores may be due to how social capital was calculated for the purpose of this analysis. In this study, social capital was calculated as a summation of the number of individuals they felt close to (close ties), the number of individuals they felt they could rely on in an emergency (social support) and the number of activities they participated in (structural member and structural volunteer). Previous studies recognize cultural and racial differences in kin support and extended family networks within the White/Caucasian and Black/African American communities (Johnson & Barer, 1990; Sarkisian & Gerstel, 2004). For instance, Black/African American older adults have been found to have more active support networks due to an expanded network of fictive kin (Johnson & Barer, 1990). Fictive kin, are relationships based on religious rituals or close friendship ties rather than by blood or marriage; thus, an expanded network of fictive kin equates to higher levels of social capital for the Black community (Ebaugh & Curry, 2000). Based on past research, it is not surprising that higher levels of social capital equate to lower levels of depression (Cao et al., 2015; De Silva et al., 2005; Fujiwara & Kawachi, 2008). As structural social capital levels increased, depression scores decreased. The same inverse relationship was also observed for social support but not close ties.
The present study also investigated whether social capital impacted who takes medication to treat depressive symptoms. There were significant impacts of structural volunteer social capital on medication use, varied by gender. The likelihood of taking medication to treat depressive symptoms decreased as structural volunteer scores increased, and this decrease was more significant for females than males. Males were found to be approximately half as likely to take medication as females (Odds Ratio = 0.53). Men have been found to be less likely to be referred to depressive treatment and the disparity in gender in taking medication for MDD or SD may be due to issues such as: traditional masculine values, the stigma of mental health issues, and the different ways men express depression (Addis, 2008; Cochran & Rabinowitz, 2003; Hinton et al., 2006). Studies have shown that men are often reluctant to engage in help-seeking behavior due to their views that traditional masculine norms include being strong and successful while also avoiding emotional experiences which are linked to femininity (Staiger et al., 2020). Studies have also shown that men fear taking psychiatric medications, and would rather endure depressive symptoms than take medication (Lynch, Long, & Moorhead, 2018; Rice et al., 2020). One of the main components to men being reluctant to treat psychiatric issues such as depression is that stigma, prejudice, and discrimination associated with mental health conditions have a negative influence on intentions towards seeking treatment (Conner et al., 2010; Harris et al., 2020).

Additionally, race mediated the impact of social support on medication use. Higher levels of social support were associated with a lower likelihood of medication use for Black/African Americans compared to White/Caucasians. Black/African American older adults have been found to be more likely to be influenced by stigma and prejudice
associated with having a mental health condition and are less likely to be accepting of seeking mental health treatment in comparison to White/Caucasian older adults (Conner et al., 2010; McGregor et al., 2020). Furthermore, White/Caucasian females had a higher likelihood of medication use than the other groups, particularly when social support scores were higher. In this study, social support was operationalized by asking participants about the number of individuals that they felt they could rely on. Although prior research has consistently proclaimed that there is a positive relationship between levels of social support and health, there is some evidence that not only can individuals close to a depressed individuals act as a source of social support, but they can also suffer from the effects of stigmatization (Birtel, Wood, & Kempa, 2017; Mickelson, 2001). Subsequently, not only can large amounts of social capital (functional social capital and structural social capital) act as a protective factor in developing depression but it can also potentially act as a hindrance to individuals seeking treatment for depression due to the stigma and prejudice associated with mental health conditions.

Limitations

There are several limitations to this study. First, although social capital has been measured as a summation of social participation and relationships in previous studies, all of this data is self-reported which may be different from actualized data (Harper & Kelly, 2003; Harpham, Grant, & Thomas, 2002; Tucker, Welk, & Beyler, 2011). Thus, the interpretation of a “close relationship” may differ across respondents. A challenge encountered when conducting a secondary data analysis on social capital is that it is difficult to assess quality versus quantity when it comes to relationships. Although higher levels of social participation and greater numbers of relationships were equated with
higher levels of social capital, realistically, a few high-quality relationships may be just as effective in decreasing the risk of depression in older adults as a high level of social capital as measured by this study (House, Landis, & Umberson, 1988).

Second, depression was assessed by a single measure, the CES-D 8 short form. While this instrument has been found to be an accurate measure in predicting depressive disorder, it does not include any follow-up measurements and fails to take into account recent events (Xiang et al., 2018). The data was collected in the U.S. in 2016 and some of the major news events of that year have been found to increase depressive symptoms within the population. Studies on the 2016 presidential election, the Flint water crisis in Michigan, and the Pulse nightclub shooting in Orlando, Florida are just several events in 2016 where researchers have observed higher levels of depression related to these specific events (Ezell & Chase, 2021; Felix et al., 2021; Yan et al., 2021).

Moreover, additional factors, such as socioeconomic status (SES), physical location, and cultural context, often associated with levels of social capital were not considered (Asadi-Lari et al., 2016; Kaasa, 2019; Ziersch et al., 2009). Overall socioeconomic status, and factors related to SES such as home ownership, have been linked to the availability of social capital (Almedom & Glandon, 2008; Manturuk, Lindblad, & Quercia, 2010). Higher SES neighborhoods have higher levels of social capital (Almedom & Glandon, 2008). Higher levels of social participation, cohesion, and social networks, all components of social capital, have also been reported for individuals who reside in rural areas in comparison to urban areas (Ziersch et al., 2009). In addition, the cultural context, or cultural beliefs of the individuals in this study were not included in this analysis. Although all of the participants in this study identified as either
Black/African American or White/Caucasian, their cultural background was not considered. For instance, recent immigrants are less likely to live in neighborhoods with strong social ties (Yong et al., 2020). There are numerous external factors that other studies have linked to the availability of social capital in the community that were not considered for the models of analysis in this study that may have an addition impact in not only the prevalence of depressive symptoms, but also the willingness of individuals to treat depression.

Finally, treatment for MDD and SD was solely assessed by medication usage and excluded other forms of the treatment such as psychotherapy or repetitive transcranial magnetic stimulation (rTMS) therapy. Black/African Americans have been found to prefer counseling to pharmacotherapy to treat depression (Givens et al., 1999)

**Conclusion**

The analysis shows that social capital impacts predisposing risk factors for depression in Black/African American and White/Caucasian older adults. However, based on the results of this study, any targeted interventions using social capital as a measure to impact treatment for depression need to be race and gender-specific.
Chapter 5

Grip Strength and Diagnosing Depression

Study Aims

Hand grip strength may potentially serve as a biometric marker for depressive symptoms in older adults and may influence their decision to seek treatment if their depression diagnosis was based on a “lab test” instead of primarily being self-initiated and self-reported. Researchers have proposed grip strength be regularly included as part of a physical exam of a patient because it has been shown to be a predictor of not only functional limitations and disability in later life but also musculoskeletal disease such as osteoporosis, sleep problems and diabetes, and even periodontal disease (Aravindakshan, Hakeem, & Sabbah, 2020; Bohannon, 2019; Rantanen et. al., 1999). Current neuropsychological theories of emotion, particularly depression, suggest that functional motor asymmetries (i.e., failure to demonstrate asymmetric grip strength) are observed in depressed young boys but little is known about the impact of depression on the anatomy and physiology of the brain and its relationship with functional motor asymmetries in older adults (Emerson et al., 2001). Thus, if the measurement of functional motor asymmetries has the potential to be a physical biomarker of depression and act as a simple, non-invasive objective test of depression in older adults, it is important to determine if the same results observed in young children are also applicable to depressed individuals who are older. The aim of this portion of the analysis is to explore whether maximal hand grip strength and/or asymmetric hand grip strength are good indicators of
depressive symptoms in older adults and whether measurements of grip strength change after seeking treatment for depressive symptoms. Furthermore, this analysis will be able to:

Examine the relationship between grip strength asymmetry in depressed older adults.

a) Determine if grip strength asymmetry is associated with depression in older adults.

   a. Hypothesis: there will be a negative association between the difference in grip strength between hands and depression. Smaller differences in grip strength between hands will be associated with higher levels of depression.

b) Assess whether medication mediates the impact of grip strength asymmetry as an indicator of depression.

   a. Hypothesis: there will be a significant interaction between grip strength asymmetry and depression diagnosis and medication (e.g., depression group with medication will be different from no depression group with no medication).

Review of the Literature

Grip Strength as a Predictor for Health

Handgrip strength has been proposed as a non-invasive biomarker for health in older adults that is easily applicable and cost-effective (Bohannon, 2019; Neidenbach et al., 2019). Measurements of grip strength have been shown to be a predictor of not only functional limitations and disability in later life but also musculoskeletal disease such as
osteoporosis, sleep problems and diabetes, and even periodontal disease (Aravindakshan, Hakeem, & Sabbah, 2020; Bohannon, 2019; Rantanen et. al., 1999). For example, low
grip strength has been shown to be associated with cardiometabolic disease and has been
used to identify high-risk individuals of morbidity and mortality (Celis-Morales, 2017;
Peterson et al., 2019). In hospitalized patients, weakening grip strength has been found to
be an indicator of longer hospital stays, higher rehospitalization rates, and increased rates
of postoperative complications for surgical patients (Norman et al., 2001). Particularly in
older adults, the loss of hand grip strength is associated with increased functional
limitations which equates to the loss of independence (Bohannon, 2008; Norman et al.,
2011).

Grip strength has even been associated with mental health issues; low grip
strength has been independently associated with dementia and incidence of Alzheimer’s
Disease (Buchman et al., 2007; Cui et al., 2021; Shin et al., 2012). Previous studies have
also shown that grip strength is associated with incidence of depression (Kim, Choe, &
Chae, 2009; Marques et al., 2020; McDowell, Gordon, & Herring, 2018; Watson & Ring,
2008). Many of these studies noted a significant but weak association between grip
strength and depression. However, current neuropsychological theories of emotion,
particularly depression, suggest that functional motor asymmetries (i.e., failure to
demonstrate asymmetric grip strength) are observed in depressed young boys (Emerson
et al., 2001). Normally, individuals who claim to be single-hand dominant have a slight
difference in maximal handgrip strength between their dominant and non-dominant hand,
typically ranging between 0% and 30% (Crosby & Wehbé, 1994). Previous research has
shown that handgrip strength asymmetry is associated not only with functional
limitations, but also with lower cognitive function (Collins et al., 2020; McGrath et al., 2020; McGrath et al., 2021). Thus, asymmetrical grip strength may be a more powerful indicator of depression in lieu of a sole measure of hand grip strength. There is a lack of research examining functional motor asymmetry in older adults, and how the treatment of depressive symptoms (taking medication) may have an impact in restoring asymmetric grip strength.

**Neuroanatomy of Depression and Grip Strength**

The neural basis of depression is a complex issue and is incompletely understood (Pandya et al., 2012). This review is not meant to be an exhaustive study of how depression impacts the brain, but rather a review of research that indicate three parts and/or systems of the brain may be impacted by depressive symptoms and also have an impact in controlling hand grip strength and they include: the cerebellum, basal ganglia, and the corticospinal system.

The cerebellum has a well-recognized role in motor function (Koziol, 2014). It is known as the “little brain” and is located at the back of the head below the temporal and occipital lobes above the brainstem (Johns Hopkins, 2022). It accounts for approximately 10% of the brain’s volume and contains over 50% of the total number of neurons in the brain (Knierim, 2020). The cerebellum is involved in the maintenance of balance and posture, coordination of voluntary movements, motor learning, and cognitive function; the cerebellum is responsible for modifying the motor commands to make movements more adaptive and accurate (Jimsheleishvili & Dididze, 2020; Knierim, 2020). Functional neuroimaging studies have shown that cerebellar gray and white matter volume have a significant positive relationship with grip strength in older adults (Koppelmans, 2015).
stroke victims, in patients who have a good recovery there are clear changes in the activation of the cerebellar hemisphere opposite the injured side. This suggests that there is a link between cerebellar activation and recovery from hand weakness due to stroke, adding further evidence that the cerebellum plays a role in controlling hand grip strength (Small et al., 2002).

Basal ganglia are a component of the cerebrum that are a collection of masses of gray matter that lie deep in the brain and surround the thalamus (Hall, 2011). It refers to a group of subcortical nuclei that are primarily responsible for motor control, executive functions and behaviors and emotions (Lanciego, Luquin, & Obeso, 2012). The main components of basal ganglia are the striatum (caudate nucleus, putamen, nucleus accumbens, and olfactory tubercle), the globus pallidus, the ventral pallidum, the substantia nigra, and the subthalamic nucleus (Fix, 2008). A review of studies on the underlying role basal ganglia play in grip force control show that basal ganglia activation was present during precision and power gripping tasks (Prodoehl, Corcos, & Vaillancourt, 2009). Additionally, patients with movement disorders such as Parkinson’s Disease (PD) are thought to be affected by basal ganglia functioning. Individuals with PD with lesioning on the basal ganglia have trouble controlling their grip on objects, they may exhibit a higher grip force than necessary to complete a task and impairment in releasing objects (Kopell & Greenberg, 2008).

The corticospinal system or tracts (CST) is the principal motor system for controlling movements, such as handgrip (Martin, 2005). The corticospinal tract originates in several parts of the brain and is a white matter motor pathway that controls movements of the limbs and body (Kolb & Whishaw, 2009). Handgrip is thought to
critically depend on the CST as evidenced by the fact that in comparing healthy subjects with those who have had a stroke, functional magnetic resonance imaging (fMRI) has shown that grip strength is strongly correlated with the integrity of the CST (Schultz et al., 2012). Chronic CST injury caused by stroke leads to finger weakness and overall reduced hand function (Wolbrecht et al., 2018).

In examining how depression impacts the brain, previous studies have shown that depression is associated with less activation of the cerebellum (Konarski et al., 2005). Research has shown that both functional and structural abnormalities are present in individuals with emotional disorders, such as depression (Schutter & Van Honk, 2005). Using fMRI, researchers have found that subjects with major depressive disorder or subjects who have a high genetic risk factor for major depression have been found to exhibit abnormalities in the left cerebellum (Liu et al., 2010). Magnetic resonance imaging (MRI) has also shown the depressive episodes have impacted aspects of the basal ganglia. In those with major depressive disorder, left putamen volumes correlated inversely with length of illness and left globus pallidus volume correlated with the number of prior depressive episodes experienced by the subject (Lacerda et al., 2003). It has also been noted that there is an increased incidence of depression associated with lesions impacting the basal ganglia and that this might be indicative of the neuroanatomy of depression (Ross & Rush, 1981). In stroke patients, patients with left caudate strokes and left caudate lesions had a higher incidence of depression than patients with right basal ganglia lesions (Lafer, Renshaw, & Sachs, 1997). Researchers have also begun to note that there are white matter abnormalities in individuals with major depressive
disorder, implicating CST and CST-related abnormalities in depressive symptoms (Sacchet et al., 2014).

Antidepressants have been found to be related to reduced physical function and inversely associated with grip strength in geriatric patients (Agarwal et al., 2020; Dudzińska-Griszek, Szuster, & Szewieczek, 2017). However, there is evidence that antidepressant use, specifically SSRIs, have a role in the recovery of improved motor function in stroke patients with and without depressive symptoms. Thus, although the exact function is not known, antidepressants have the potential to restore asymmetric hand grip strength in individuals diagnosed with depressive symptoms (Elzib et al., 2019; Pinto et al., 2017).

In addition, the left half of the brain controls the right side of the body and the right half of the brain controls the left side of the body (MacNeilage, Rogers, & Vallortigara, 2009). Depression impacts different sides of the brain in different ways. For an individual with depressive symptoms, the right-hemisphere (RH) of the brain becomes hyperactive and the left-hemisphere (LH) of the brain becomes relatively hypoactive (Hecht, 2010). Thus, measures of strength in the right hand may be impacted differently than measures of strength in the left hand in an individual with depressive symptoms and measurements of asymmetric grip strength may be a more accurate predictor of depressive symptoms rather than just maximal hand grip measures of the dominant hand.

*The Functional Components of the Hand*

Previously, a review of the research on how depression is affecting the brain shows that depression may also affect parts of the brain that control grip strength. Thus,
grip strength may be an indicator of depressive symptoms that could potentially be used to diagnose depression. Functions of the brain have been reviewed and the following is a brief review on how the components of the hand function in order to form a gripping motion. Many components of the human body are involved in maintaining hand grip strength, including the brain, the wrist, muscles in the forearm, and genetics; however, the following focuses primarily on the functional components of the four fingers of the hand, including joints, finger flexion and extension, and grip/grasping, as well as some age-related musculoskeletal changes that could potentially impact the function of the fingers. The bone structure of the hand is not elaborated upon, though it should be noted that diseases such as osteoporosis, often associated with the aging process, may increase the frailty of the bones due to osteoclast-mediated bone resorption and/or inhibition of osteoblast-mediated bone formation, and thus have an impact on hand function if the bone fractures (Khosla et al., 2011).

Joints of the Hand

The hand complex is composed of nineteen bones and nineteen joints distal to the carpals (Kamojima, Miyata, Ota, 2004). Of the five digits that make up the hand complex (four fingers and one thumb), the fingers each have two interphalangeal (IP) joints, the proximal interphalangeal (PIP) and distal interphalangeal (DIP) and each digit has a carpometacarpal joint and a metacarpophalangeal joint (MP) (Levangie & Norkin, 2011).

Both the PIP and DIP joints in the fingers are composed of a head of a phalanx and a base of the phalanx distal to it (Bundhoo & Park, 2005). Each IP can be classified as a uniaxial hinge joint with one degree of freedom, a joint capsule, and it is supported by two collateral ligaments, and smaller versions of a volar/palmar plate (Levangie &
Norkin, 2011). Similar to the MP joint, the proximal joint surface is convex and the distal surface is concave in appearance; the joint surfaces move with respect to one another by simultaneously rolling and gliding in the same direction (Lin et al., 1989). When in operation, the joint is anterior with flexion and posterior with extension (Sharp & Thompson, 2001). However, unlike the MP joints, there is little chance for hyperextension because there is only a small amount of posterior articular surface at the proximal/distal IP joint (Levangie & Norkin, 2011).

The volar plates reinforce the IP joints and provide stability to the joint by limiting hyperextension (Bielefeld & Neumann, 2005). Structurally, the plates are made up of fibrocartilage and blend with the volar capsule portion of the capsule. The fibrocartilage of the plate has the composition to resist both tensile stresses and compressive forces. Along with the collateral ligaments, the volar plates attach to the bases of the phalanges at both the proximal IP and distal IP joints (Hogan & Nunley, 2006). There is an observed pattern of increased range of motion in flexion and extension from the radial to the ulnar side of the hand, this greater range of motion on the more ulnarly located fingers allow for increased grip strength on the ulnar side of the hand (Levangie & Norkin, 2011). This difference in grip strength in different parts of the hand should be taken into account when using grips strength as a biometric marker in individuals of different ages.

In contrast, the MP joints are condylar, biaxial joints and are composed of a convex metacarpal head that is proximal to the concave distal surface of the first phalanx. This allows the joint to roll and glide in the same direction, anteriorly with flexion, and posteriorly with extension (Ayhan & Ayhan, 2020). The volar plates which provide
stability to the MP joint by limiting hyperextension are structurally and functionally the same as the smaller volar plates that are found in the IP joints. The volar plates are lined with hyaline cartilage which enlarges the proximal phalanx’ relatively small articular surface (Sharp & Thompson, 2001). They attach to the base of the proximal phalanx and blend with the MP joint capsule and the deep transverse metacarpal ligament. Superficial to the volar plate, the transverse metacarpal ligament interconnects the MP joints, which tether the heads of the metacarpals of the four fingers (Okafor, Sinkler, Varacallo, 2019). Sagittal bands on each side of the metacarpal head connect each volar plate to the extensor digitorum communis tendon and extensor expansion, they are located dorsal to the deep transverse metacarpal ligament and act to the help stabilize the volar plates over the metacarpal heads. The joint capsule is supported by the radial and ulnar collateral ligaments which are composed of two parts: the collateral ligament proper, and the accessory collateral ligament (Levangie & Norkin, 2011). In flexion, the tension is in the collateral ligaments when in a close-packed position; the MP joints of fingers two through five do not allow for the abduction or adduction of the joints when they are flexed (Sharp & Thompson, 2001). Researchers have observed that the MP joints also experience a relatively small incidence of osteoarthritis (OA) in comparison to the distal IP joints (Fisher et al, 1985).

Mechanisms for Finger Flexion and Extension

The extrinsic muscles of the fingers and thumb have proximal attachments above the wrist and can be divided into flexors and extensors, while the intrinsic muscles of the finger attach distal to the radiocarpal joint (Levangie & Norkin, 2011). The two extrinsic muscles that operate finger flexion are the flexor digitorum profundus (FDP), which
attaches to the skeleton more distally than the second extrinsic muscle, the flexor digitorum superficialis (FDS) because it passes through a split in the FDS tendon (Sharp & Thompson, 2001). For optimal function, the FDS and FDP muscles require stable musculature in the wrist and flexor gliding mechanisms. The components of the gliding mechanisms include: the fibrous retinacular structures which connect the long flexor tendons to the hand, and the bursae and digital tendon sheaths which facilitate the excursion of tendons on the fibrous retinacula friction-free (Levangie & Norkin, 2011). The flexor mechanisms of the four fingers and thumb include the fibro-osseous tunnels which are made up of five annular pulleys and three cruciate pulleys. The shape of the pulleys evenly distributes pressure on the tendon and sheath across the roof of the fibro-osseous tunnels while a finger is in flexion (Levangie & Norkin, 2011).

The extrinsic finger extensors are the extensor digitorum communis (EDC), the extensor indicis proprius (EIP), and the extensor digiti minimi muscles (EDM) (Levangie & Norkin, 2011). The extensor mechanism involves the EDC tendon which is attached by a tendinous slip to the proximal phalanx, through which it extends to the MP joint. The central tendon is attached to the base of the middle phalanx, when tense, it can extend the PIP joint involved in finger extension. In addition, there are lateral bands that are located on both sides of the dorsal midline before they rejoin as one and attach to the distal phalanx. Tension in the lateral bands extends the DIP joint. The extensor hood surrounds the MP joint and receives tendinous fibers from the lumbricales and interossei. Finally, fibers of the oblique retinacular ligament (ORL) attach to the sides of the proximal phalanx and digital tendon sheaths, and continue on to the distal portion of the lateral bands. PIP extension elongates the ORL which creates passive tension that extends the
DIP. DIP extension aids hand opening and DIP flexion creates passive tension that flexes the PIP by elongating the ORL, assisting in finger closure (Sharp & Thompson, 2001).

*Finger Musculature and the Extensor Mechanism*

Some of the muscles involved in transmitting force to otherwise non-contractile extensor mechanisms include the dorsal interossei (DI), the palmar interossei (PI), and the lumbricales. The DI attaches proximally between adjacent metacarpals and attaches distally to the proximal phalanx or to soft tissue. It primarily produces MP abduction but can produce MP flexion as well. They can also produce PIP and DIP extension because they attach to the extensor mechanism. There are four PI muscles that attach proximally to a metacarpal and distally to the proximal phalanx and/or the extensor mechanism. These muscles produce MP adduction and MP flexion, in certain instances. When they have tension in the extensor mechanism, they can also produce PIP and DIP extension. There are four lumbricals that attach proximally to the tendons of the FDP and distally to the extensor mechanism on its radial side. If they are acting alone, they produce MP flexion but they can also produce PIP and DIP extension when they introduce tension into the extensor mechanism. Their attachments transmit force to both the FDP tendon and the extensor mechanism by increasing passive tension in the extensor mechanism and decreasing passive tension in the FDP tendon’s distal portion (Sharp & Thompson, 2001).

*Measuring Hand Grip Strength in the Health and Retirement Study*

In order to determine handedness, participants were simply asked if they had a dominant hand, and if so to indicate if they were either left- or right- hand dominant. In order to determine hand grip strength, measured in kilograms (kg), participants had to be
willing to exert maximal effort in the test. In addition, if the participant reported having had surgery, swelling, inflammation, severe pain or injury in both hands in the past six months, they were excluded from the test.

The protocol used in measuring hand grip strength for the HRS using a Smedley-spring type hand dynamometer is copied from the Protocol provided by Crimmens et al. (2008) in the report on the Documentation of Physical Measures, Anthropometrics, and Blood Pressure in the Health and Retirement Study. The directions were as follows:

- The dynamometer was fit to the respondent’s hand and the respondent practiced once with their dominant hand in a standing position with their arm at their side at a 90-degree angle.

- The respondent was instructed to squeeze the meter as hard as they were able for a couple of seconds and to then let go.

- After the practice measurement, the respondent was instructed to switch to their nondominant hand.

- Two measurements were taken with each hand, alternating hands.

- After each measurement, the interviewer recorded the result and handed the dynamometer back to the respondent.

Special instructions: If the respondent was unable to stand, the measurement was completed with the respondent seated. If the respondent had difficulty holding the
dynamometer, the respondent was allowed to perform the measurement which was conducted with their upper arm resting on a table or other object for support.

Results of the measurement of grip strength were included in this analysis if participants were observed to have exerted maximum effort by the tester, and if they had readings for all four trials (two on the right and two on the left). These readings were then averaged for each hand and then the difference in grip strength was determined by subtracting the right-hand average from the left-hand average and then determining the absolute value of the results so that there were no negative numbers. All measurements were recorded in kilograms.

Results
Maximal Hand Grip Strength and Depression

Normative grip strength data distributed by the Jamar Plus Digital Hand Dynamometer Grip Strength Instruction Manual can be found in Appendix D. For this sample population, the average maximum grip strength in the dominant hand was 30.33 kg (sd = 11.03), with the minimum maximal grip strength as 2 kg and the maximum hand grip strength recorded as 100 kg. It should be noted, that a maximum grip strength of 100 kg is unusual, each individual was required to do two measurements on each hand, and the individual who recorded the 100 kg measurement only recorded a 45 kg measurement for the first grip strength test on their dominant hand. However, this analysis is based on data provided by the Health and Retirement Study and a grip strength of 100 kg is a possibility and thus, the individual was included in the sample population for analysis.

Subsequently, the association between maximal hand grip strength of the dominant hand and depression was analyzed. Previous studies have shown that low
maximal hand grip strength is associated with higher levels of depressive symptoms (Marquez et al., 2021; Smith et al., 2019; Watson & Ring, 2008). Thus, a negative association between grip strength and depression was predicted, such that low grip strength would be associated with higher levels of depression. As a measure of association between grip strength (of the dominant hand) and depression, a correlation analysis was conducted. Specifically, Spearman’s Rho correlation coefficient was calculated. Maximum Grip strength was measured continuously (minimum score = 2.0, maximum score = 100.0), and depression was measured categorically (0 = no depression, 1 = subthreshold depression, and 3 = major depression). In this sample population, there was a small and significant negative correlation between maximum grip strength (with the dominant hand) and depression was observed, \( r = -0.082, p < .001 \). As the maximum grip strength decreases, the likelihood of depression increases.

Aim 2a: Asymmetric Grip Strength and Depression

However, the main purpose of this study was to determine if asymmetric hand grip strength could potentially be a more powerful indicator of depression rather than maximal hand grip strength due to the fact that the association of maximal hand grip strength and depressive symptoms is evident, but often weak. Asymmetric hand grip strength has the potential to be a more sensitive measure of depressive symptoms due to the way that depression impacts different sides of the brain. Thus, a significant, negative association between the difference in grip strength between hands and depression was predicted. Smaller differences in grip strength between hands would be associated with higher levels of depression. The null hypothesis associated with this research question posits no significant relationship between grip difference strength and depression. To
assess this hypothesis, Spearman’s Rho correlation coefficient was calculated. Grip strength difference was measured continuously, and depression measured categorically (0 = no depression, 1 = subthreshold depression, and 3 = major depression).

The average difference in grip strength between hands in this sample population was 3.93 kg ($sd = 3.21$), with a minimum difference of 0 kg and a maximum of 42.25 kg. The average difference in grip strength categorized by depression status can be found in Table 16. There was no significant relationship between the difference in grip strength and depression groups, $r_s = .002, p = .877$. Therefore, the null hypothesis was retained. There was no significant relationship between grip strength difference between hands and depression. Therefore, the answer to the question is no, grip strength difference is not a stronger indicator for depression than max grip strength of the dominant hand.

**Table 16.**

<table>
<thead>
<tr>
<th>Depression Status</th>
<th>Grip Difference Score $M$ ($SD$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No depression</td>
<td>3.92 (3.19)</td>
</tr>
<tr>
<td>Sub-threshold depression</td>
<td>3.88 (3.31)</td>
</tr>
<tr>
<td>Major depression</td>
<td>4.08 (3.21)</td>
</tr>
</tbody>
</table>

Furthermore, a multiple linear regression was calculated, treating depression as the dependent variable, and race, gender, education, age, comorbidity, grip difference, and all possible interactions as independent variables. The overall model was significant, $F (27, 5899) = 9.86, p < .001$ (Table 17). Approximately 4% of the variance was accounted for ($R^2 = 0.043$). However, there were no significant interactions between grip
difference (i.e., asymmetric grip strength) and any of the other variables included in the model. This further suggests that asymmetric grip strength is not a good indicator for depression for any of the demographic categories included in the model.

Table 17.

Multiple Regression (including Interaction Terms) of Race, Gender, Education, Age, Comorbidity and Grip Difference on Depression Scores

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>0.66</td>
<td>0.08</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Race</td>
<td>-0.07</td>
<td>0.03</td>
<td>.037</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.12</td>
<td>0.03</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Education</td>
<td>-0.11</td>
<td>0.03</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Age Group</td>
<td>-0.01</td>
<td>0.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>0.13</td>
<td>0.02</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Grip Difference</td>
<td>-0.02</td>
<td>0.02</td>
<td>.115</td>
</tr>
<tr>
<td>Grip Difference * Age Group</td>
<td>-0.01</td>
<td>0.02</td>
<td>.564</td>
</tr>
<tr>
<td>Grip Difference * Race</td>
<td>0.01</td>
<td>0.02</td>
<td>.511</td>
</tr>
<tr>
<td>Grip Difference * Gender</td>
<td>0.02</td>
<td>0.02</td>
<td>.356</td>
</tr>
<tr>
<td>Grip Difference * Education</td>
<td>0.02</td>
<td>0.02</td>
<td>.154</td>
</tr>
<tr>
<td>Grip Difference * Comorbidity</td>
<td>0.01</td>
<td>0.01</td>
<td>.239</td>
</tr>
<tr>
<td>Grip Difference * Age Group * Race</td>
<td>0.02</td>
<td>0.02</td>
<td>.456</td>
</tr>
<tr>
<td>Grip Difference * Age Group * Gender</td>
<td>0.01</td>
<td>0.01</td>
<td>.191</td>
</tr>
<tr>
<td>Grip Difference * Age Group * education</td>
<td>-0.01</td>
<td>0.01</td>
<td>.458</td>
</tr>
<tr>
<td>Grip Difference * Age Group * Comorbidity</td>
<td>0.002</td>
<td>0.01</td>
<td>.807</td>
</tr>
<tr>
<td>Grip Difference * Race * Gender</td>
<td>-0.001</td>
<td>0.02</td>
<td>.967</td>
</tr>
<tr>
<td>Grip Difference * Race * Education</td>
<td>-0.003</td>
<td>0.01</td>
<td>.790</td>
</tr>
<tr>
<td>Grip Difference * Race * Comorbidity</td>
<td>&lt;0.001</td>
<td>0.01</td>
<td>.956</td>
</tr>
<tr>
<td>Grip Difference * Gender * Education</td>
<td>-0.004</td>
<td>0.01</td>
<td>.763</td>
</tr>
<tr>
<td>Grip Difference * Gender * Comorbidity</td>
<td>-0.004</td>
<td>0.01</td>
<td>.695</td>
</tr>
<tr>
<td>Grip Difference * Education * Comorbidity</td>
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<td>0.01</td>
<td>.162</td>
</tr>
<tr>
<td>Grip Difference * Age Group * Race * Gender</td>
<td>-0.01</td>
<td>0.01</td>
<td>.297</td>
</tr>
<tr>
<td>Grip Difference * Age Group * Race * Education</td>
<td>0.02</td>
<td>0.01</td>
<td>.180</td>
</tr>
<tr>
<td>Grip Difference * Age Group * Race * Comorbidity</td>
<td>-0.01</td>
<td>0.01</td>
<td>.333</td>
</tr>
<tr>
<td>Grip Difference * Race * Gender * Education</td>
<td>-0.01</td>
<td>0.02</td>
<td>.703</td>
</tr>
<tr>
<td>Grip Difference * Race * Gender * Comorbidity</td>
<td>-0.002</td>
<td>0.01</td>
<td>.834</td>
</tr>
<tr>
<td>Grip Difference * Race * Gender * Education * Comorbidity</td>
<td>&lt;0.001</td>
<td>0.001</td>
<td>.613</td>
</tr>
</tbody>
</table>

*Note. Significant variables are in bold.*
B- Unstandardized beta. The value represents the slope of the line between the predictor variable and the dependent variable.

SE- Standard error of the regression. Represents the average distance that the observed values fall from regression line.

P- Statistical significance value set at .05.

Aim 2b: Asymmetric Grip Strength and Medication

In order to assess whether grip strength was impacted by depression and/or medication use, a 3 (depression status: no depression, sub-threshold depression, major depression) x 2 (medication: yes, no) analysis of variance (ANOVA) was conducted. In this analysis, grip strength was treated as the dependent variable. With this analysis, two main effects and an interaction were possible: 1) an effect of depression status on grip strength (i.e., grip strength varies by depression group), 2) an effect of medication use on grip strength, and 3) the interaction between depression group and medication (e.g., major depression group with medication may be different than no depression group with no medication).

There was no significant difference in grip strength difference for those who took medication compared to those who did not, F(1,5013) = 0.063, p = .802, regardless of whether they had depression symptoms or not. There was no interaction between depression group (no depression, sub-threshold depression, and major depression) and medication (Yes/No) in terms of grip strength difference, F(2, 2013) = 1.091, p = .336.

This supports the first prediction, that there would be similar grip strength differences for those who took medication and had no depression symptoms and those who did not take medication and did not have symptoms (See “No Depression” on Figure
However, the second prediction, that those who took medication and had depression symptoms would have higher grip strength than those who did not take medication and had depression symptoms, was not supported. There was no significant difference in grip strength difference, in either the subthreshold or major depression groups, for those who did and did not take medication (Figure 7).

Figure 7. Grip strength difference between hands by depression group

It was also hypothesized that the impact of asymmetric grip strength as an indicator for depression would vary by race and/or gender and would be more likely to impact certain sub-groups within the sample. For example, asymmetric grip strength may be an indicator for depression for men, and not women. Thus, a univariate multiple ANOVA was conducted, treating grip difference as the dependent variable and demographic variables (race, gender, education level, age group, and comorbidity) as independent variables. Additionally, the interaction between depression and medication was also included as an independent variable. All interactions with depression and
medication, and demographic variables were also included in the model. The overall model was significant, $F(117, 5769) = 3.494, p < .001$. There was a significant difference in grip difference by gender, $p = .002$. Males had, on average ($m = 4.60, sd = 2.66$) higher grip difference scores than females ($m = 3.43, sd = 2.63$). There was no significant difference for any of the other demographic variables, all $p$’s > .05.

There was a significant interaction between race, gender, education, depression and medication, $F(4, 5651) = 2.54, p = .038$. Black/African American males, with more than a high school degree, and taking medication, had a greater grip strength difference than those who were not taking medication. However, White/Caucasian males, with more than a high school degree, did not differ on their grip strength difference whether or not they were taking medication. Additionally, White/Caucasian males with less than a high school degree had a greater difference in grip strength if they were not taking medication. However, Black/African American males with less than a high school degree had a greater grip difference if they were taking medication (Figure 8).
Discussion and Limitations

The present study was conducted on a sample based on several inclusion criteria. The sample consisted of Black/African Americans and White/Caucasians who were at least 50 years old, were single-hand dominant, and completed all four grip strength trials (with full effort). Based on these criteria, analyses were conducted on approximately 6,245 individuals. Most of the sample was female, White/Caucasian, and right-handed. Most had at least a high school degree. The preliminary background analysis confirms that there was a small, negative relationship between maximum grip strength (from the dominant hand) and depression. This result is similar to the findings of previous studies, that there is a small negative association between hand grip strength and depressive symptoms (Han et al., 2019; Marquez et al., 2021; Smith et al., 2019; Watson & Ring, 2008).

Figure 8. Grip strength difference by education, gender, and race
There were also several key findings from the primary research questions. Regarding the first research question, there was no significant relationship between the difference in grip strength and depression. Therefore, grip strength difference was not a stronger indicator of depression than maximum grip strength of the dominant hand. Moreover, grip strength difference was not mediated by medication usage.

However, the findings from this analysis should not eliminate the possibility of asymmetric grip strength being used as an indicator for depression in the future. There were several limitations for this analysis. First, the level of handedness of the participants was not measured. Individuals were only included in the sample if they indicated if they were either left- or right-hand dominant, but not both. However, the strength of the dominant hand varies from individual to individual and while there are some respondents who may be extremely right- or left-handed, most are on a scale of handedness (Prichard, Christman, & Walters, 2020). Subsequently, individuals who claim to be single-hand dominant may have varying natural levels of asymmetric grip strength due to the fact that they use their non-dominant hand frequently to complete tasks. Thus, a measurement of asymmetric grip strength may not be an appropriate biometric marker for depression in individuals who do not exhibit extreme single-handed dominance.

Second, medication usage for the treatment of depressive symptoms was used as the outcome measurement in healthcare utilization. However, the type of medication each respondent took to treat their depressive symptoms was not known. It is highly probable that individuals in the sample were taking a wide variety of antidepressants and potentially even mood stabilizers when asked the question of whether or not they took medication to treat depression. Inclusion of a wide variety of medications that work in
different ways and impact the brain in different ways is not ideal in determining whether
or not asymmetric grip strength is a good indicator for depressive symptoms.

Finally, although single-handed dominance was required to be included in this
analysis, individuals could have been either right- or left-handed. The left and right
halves of the human brain each handle muscles on the opposite side of the body, so the
left side of the brain controls the right side of the body (MacNeilage, Rogers, &
Vallortigara, 2009). However, there is research that has shown that depressive disorders
impact the different sides of the brain in different ways. Depression is associated with an
inter-hemispheric imbalance; right-hemisphere (RH) becomes hyperactive and the left-
hemisphere (LH) becomes relatively hypoactive (Hecht, 2010). Subsequently, maximal
grip strength for a right-hand dominant individual may be impacted differently than
maximal grip strength for a left-hand dominant individual and thus, asymmetric grip
strength may be impacted as well.

Conclusion

There was a significant small, negative correlation between maximum grip
strength (with the dominant hand) and depression, meaning that, as the maximum grip
strength decreases, the likelihood of depression increases. Further research is needed in
order to determine if asymmetric hand grip strength has the potential to be a more
accurate measure of depressive symptoms. The limitations of this secondary data analysis
do not allow for the sample population to include level of handedness or type of
mediation used to treat depressive symptoms, both key factors in determining asymmetric
hand grip strength.
Chapter 6

Conclusions and Future Research Directions

Social Capital, Mental Health, and Healthcare Utilization

Overall, the findings of this study show that levels of social capital did vary by race for those with and without depressive symptomology. Results showed that Black/African Americans actually had higher levels of social capital on three of the four measures used in this analysis (structural member, structural volunteer, and social support). There are several possible reasons why Black/African American older adults had higher levels of social capital in this analysis. Previous studies have shown that there are cultural and racial differences in kin support and extended family networks within the White/Caucasian and Black/African American community where Black/African American older adults have a more active support network due to the fact that they recognize an expanded network of fictive kin (Johnson & Barer, 1990; Sarkisian & Gerstel, 2004). Fictive kin, are relationships based on religious rituals or close friendship ties rather than by blood or marriage; thus, an expanded network of fictive kin equates to higher levels of social capital for the Black/African American community in this sample population due to the fact that social capital was calculated as a summation of the number of individuals they felt close to (close ties), the number of individuals they felt they could rely on in an emergency (social support) and the number of activities they participated in (structural member and structural volunteer (Ebaugh & Curry, 2000).
Social capital also impacted the utilization of medication to treat depressive symptoms. There were significant impacts of structural volunteer social capital on medication use, varied by gender. The likelihood of taking medication decreases as structural volunteer scores increased, and the decrease in likelihood of taking medication was more for females than males. Previous studies have shown that men are less likely to be referred for depressive treatment and the disparity in gender in taking medication for MDD or SD may be due to issues such as: traditional masculine values, the stigma of mental health issues, and the different ways men express depression (Addis, 2008; Cochran & Rabinowitz, 2003; Hinton et al., 2006). One of the main components to men being reluctant to treat psychiatric issues such as depression is that stigma, prejudice, and discrimination associated with mental health conditions have a negative influence on intentions towards seeking treatment (Conner et al., 2010; Harris et al., 2020).

Additionally, race mediated the impact of social support on medication use. Higher levels of social support were associated with a lower likelihood of medication use for Black/African Americans compared to White/Caucasians. Furthermore, White/Caucasian females had higher likelihood of medication use than the other groups, particularly when social support scores were higher. Having a strong social network did not increase the use of healthcare utilization to treat depressive symptoms. Thus, higher levels of social capital may actually have a negative impact on use of mental health services. Higher levels of social capital in the Black/African American community might actually enforce gendered or cultural perceptions of mental health, which may negatively impact desired health outcomes, in this case, treatment of depressive symptoms (Villalonga-Olives, Wind & Kawachi, 2018).
Health, Aging, and Biomarkers for Diagnosis

While the use of asymmetric hand grip strength was proposed as a measurable test for depression and the effectiveness of treatments for depression, it is not a statistically accurate measure at this time. A more thorough discussion into the limitations of this analysis can be found in Chapter 5. But in summary, the level or degree of single-handed dominance could not be analyzed from this secondary data analysis. Rather than classifying individuals as either left- or right- hand dominant, studies have shown that individuals are often inconsistent in their handedness and this inconsistency leads to increased interhemispheric interactions and increased access to process in the right cerebral hemisphere (Prichard, Propper, & Christman, 2013). Emerson et al. (2001) conducted his analysis of asymmetric hand grip strength and depression on young boys who were strongly right-handed, but, in this study, only direction of handedness was considered. In addition, Hecht (2010) notes that depression impacts different sides of the brain in different ways. For an individual with depressive symptoms, the right-hemisphere (RH) of the brain becomes hyperactive and the left-hemisphere (LH) of the brain becomes relatively hypoactive. Handedness, and the differences in cerebral lateralization of emotion in left- vs right- handed individuals may be relevant in studying individuals with depression according to imaging studies (Rachid, 2021). This could potentially impact using asymmetric grip strength as a measurement for depression because the left side of the brain controls the right side of the body and the right side of the brain controls the left side of the body, thus, measurements of asymmetric hand grip strength of depressed individuals may decrease more for individuals who are strongly right-handed in comparison to those who are strongly left-handed.
In addition, the type of medication being used to treat depressive symptoms should be incorporated into any future research. Participants in the 2016 HRS were asked if they took medication for depression and/or anxiety. The most common type of antidepressants prescribed are: selective serotonin reuptake inhibitors (SSRIs) and serotonin and norepinephrine reuptake inhibitors (SNRIs). Common brands of SSRIs include: Prozac®, Paxil®, Zoloft® (Mayo Clinic, 2019; National Health Service, 2021). Common brands of SNRIs include: Cymbalta® or Pristiq®. Serotonin is a neurotransmitter that affects mood, energy level, appetite, and sleep and norepinephrine is a neurotransmitter that affects energy level, focus, and attention (Mental Health America, 2020; Zhao, 2004). Neurons communicate via neurotransmitters and a transporter molecule recycles an unused transmitter and returns it back to the pre-synaptic cell. An SSRI binds to SERT (serotonin transporter) and blocks activity, allowing for more serotonin to remain in the brain in the spaces between neurons. SNRIs work by blocking the reabsorption of serotonin and norepinephrine into the nerve cells, thus increasing the levels of active neurotransmitters in the brain (Swanson, 2013). However, there are also other classifications of antidepressants including: atypical antidepressants, such as Remeron® or Wellbutrin®; tricyclic antidepressants, such as Tofranil® or Pamelor®; and monoamine oxidase inhibitors (MAOIs), such as Nardil® or Parnate® (Mayo Clinic, 2019). Although the exact function is not known, antidepressants, particularly SSRIs, have been shown to have a role in improved motor function of individuals who have had a stroke. Thus, there is the potential that SSRIs could restore asymmetric hand grip strength in individuals diagnosed with depressive symptoms (Elzib et al., 2019; Pinto et al., 2017). Further research on the restoration of grip strength by
other types of antidepressants would be needed before eliminating the possibility of asymmetric grip strength not being an accurate measure of depressive symptoms.

Asymmetric grip strength should not be eliminated as a potential marker of depression despite the results from this analysis. Previous research has shown that handgrip strength asymmetry is associated with functional limitations (Collins et al., 2020). These functional limitations, such as difficulty completing activities of daily living (ADL) (e.g., dressing, eating) and instrumental activities of daily living (IADL) (e.g., grocery shopping, cooking) could potentially lead to depressive symptoms and could also potentially lead to a shrinking social network, and thus smaller amounts of social capital, also potentially leading to an increase in depressive symptomology. The decline of physical ability associated with age could be a potential factor in developing depression. The association between measurements of physical health and mental health should still be considered in future research.

**Revised Healthcare Utilization Model**

![Revised Healthcare Utilization Model](image)

Figure 9. *Revised healthcare utilization model from chapter 2*
The application of Andersen’s Healthcare Utilization Model to treat depression in older adults in this study provided further evidence that is of interest due to the fact that as individuals age, their decision-making process changes as well (Andersen, 1995). In both hypothetical laboratory tasks and decision-making in the real world, older adults are less likely than younger adults to conduct exhaustive information searches prior to making a decision (Chen, Ma, Pethtel, 2011; Löckenhoff, 2018; Löckenhoff et al., 2016). Subsequently, when presented with the choice to treat depressive symptoms with medication, the decision-making process for older adults may make them more amenable to social factors impacting their decision-making process since they are less likely to exhaustively review sources of information (Mata & Nunes, 2010).

One of the main critiques of Andersen’s original 1968 Healthcare Utilization Model is that it does not take into account culture and social interaction, and the impact that this has on determining use of healthcare services (Andersen, 1995; Guendelman, 1991; Portes, Kyle, Eaton, 1992). This analysis shows that a social factor, such as social capital, should not be considered exclusively a predisposing factor when analyzing healthcare decision-making in older adults. Rather, both structural social capital, membership in groups, and functional social capital, quality of relationships should also be considered an enabling and need factor because social capital can influence healthcare decision making at different stages. Analysis in this study has shown how measurements of social capital vary by predisposing factors such as gender and race and how it can have both a positive and negative effect on healthcare utilization (medication) to treat depressive symptoms. Thus, in addition to how social capital varies by predisposing factors such as age, race, gender, previous experience with depression, education level,
and other chronic conditions, social capital also can be classified as both an enabling factor and a need factor as well. Higher levels of social capital might actually enforce gendered or cultural perceptions of mental health, which may negatively impact use of medication to treat depression, subsequently acting as a factor that negatively impacts perceived need of medication (Villalonga-Olives, Wind & Kawachi, 2018).

Although insurance status was considered an enabling factor in this analysis, most of the individuals had insurance and this enabling factor was found to have very little influence on the decision to take medication to treat depression. Furthermore, the need factor analyzed in this paper, grip strength asymmetry was found not to be an indicator of depression in this analysis, perhaps due to reasons listed previously. Thus, the decision-making process for older adults may be more likely to be impacted based on their own previous experience, potentially making them more amenable to social factors impacting their decision-making process since they are less likely to exhaustively review sources of information (Mata & Nunes, 2010).

**Future Research Directions**

One of the limitations of this study is that it is based on a cross-sectional analysis. This study did not follow-up with individuals over time. Although the Health and Retirement Study is intended to be a longitudinal data collection, the interest for this particular analysis relies on some of the strengths of cross-sectional studies in that multiple outcomes and exposures were used in this data analysis. This data is publicly available, and these findings are meant to be preliminary findings to examine the prevalence of social capital and depression in older Black/African American and White/Caucasian older adults. However, the use of cross-sectional data does have a few
weaknesses in that it is difficult to make a causal inference and analysis of this data is susceptible to nonresponse and recall bias (Wang & Cheng, 2020). Nevertheless, these findings on the racial disparities in healthcare decision making to treat depressive symptoms can now be used as a starting point for further in-depth research (Wang & Cheng, 2020).

Moreover, this analysis did not address the issue as to whether minority older adults may actually be underdiagnosed with depressive symptoms. Any future interventions to encourage treatment-seeking behavior to treat depressive symptoms will have to be culturally appropriate to the population that is being targeted.

In addition, a limitation of this analysis is that the sole variable used in measuring treatment for depressive symptoms is medication use. Race may or may not mediate the impact of social capital measures on other treatment options for depressive symptoms, such as the use of nonchemical or psychosocial therapies. Moreover, research shows that Black/African Americans are distrustful of the healthcare system, perhaps due to the historical trauma or everyday racism that these individuals face when interacting with the healthcare system (Bajaj & Stanford, 2021). Racial concordance between the physician and the patient is important to those who identify as Black/African American where life and death issues such as infant mortality rates are often halved when Black/African American newborns are cared for by Black rather than White physicians (Greenwood et al., 2020). This study was unable to take into account the race of the physician who prescribed medication to treat depressive symptoms in this sample. Higher levels of racial concordance between patients and physicians could potentially increase levels of medication use to treat depressive symptoms despite high levels of social capital.
The issues of stigma, prejudice, and discrimination are not addressed in this study. Research has shown that individuals may be reluctant to seek help from mental health professionals due to feelings of embarrassment or the belief that others would have negative reactions if they were to seek mental health help (Barney et al., 2006). Levels of stigma associated with the treatment of depressive symptoms may vary by ethnic and racial identification, with Black/African Americans experiencing higher levels of stigma than White/Caucasians. This helps explain why Black/African Americans with higher amounts of social capital are less likely to take mediation to treat depression.

Additionally, this study only analyzed the impact of social capital on medication use between Black/African American and White/Caucasian older adults to treat depression. The experiences of racial minorities in the United States vary widely and future studies should focus on whether the findings of this study are applicable to other races/ethnicities in order to determine culturally appropriate interventions.
Appendices

APPENDIX A

Biopsychosocial Model of Depression

Adapted from the work of: Avasthi (2016); Barton et. al (2017); Engel (1977); and Seligman & Maier (1967)
The following is a copy of the most current CES-D, originally developed by Renore Sawyer Radloff in 1977 and accessed from the public domain at: https://cesd-r.com/about-cesdr/

<table>
<thead>
<tr>
<th>Below is a list of the ways you might have felt or behaved. Please check the boxes to tell me how often you have felt this way in the past week or so.</th>
</tr>
</thead>
<tbody>
<tr>
<td>My appetite was poor.</td>
</tr>
<tr>
<td>I could not shake off the blues.</td>
</tr>
<tr>
<td>I had trouble keeping my mind on what I was doing.</td>
</tr>
<tr>
<td>I felt depressed.</td>
</tr>
<tr>
<td>My sleep was restless.</td>
</tr>
<tr>
<td>I felt sad.</td>
</tr>
<tr>
<td>I could not get going.</td>
</tr>
<tr>
<td>Nothing made me happy.</td>
</tr>
<tr>
<td>I felt like a bad person.</td>
</tr>
<tr>
<td>I lost interest in my usual activities.</td>
</tr>
<tr>
<td>I slept much more than usual.</td>
</tr>
<tr>
<td>I felt like I was moving too slowly.</td>
</tr>
<tr>
<td>I felt fidgety.</td>
</tr>
<tr>
<td>I wished I were dead.</td>
</tr>
<tr>
<td>I wanted to hurt myself.</td>
</tr>
<tr>
<td>I was tired all the time.</td>
</tr>
<tr>
<td>I did not like myself.</td>
</tr>
<tr>
<td>I lost a lot of weight without trying to.</td>
</tr>
<tr>
<td>I had a lot of trouble getting to sleep.</td>
</tr>
<tr>
<td>I could not focus on the important things.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LAST WEEK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all or Less than</td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
APPENDIX C


### APPENDIX D

**NORMATIVE GRIP STRENGTH DATA:**

<table>
<thead>
<tr>
<th>Age</th>
<th>Hand</th>
<th>Males Mean (lbs)</th>
<th>Males SD</th>
<th>Females Mean (lbs)</th>
<th>Females SD</th>
<th>Males Mean (kg)</th>
<th>Males SD</th>
<th>Females Mean (kg)</th>
<th>Females SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>6-7</td>
<td>R</td>
<td>32.6</td>
<td>4.8</td>
<td>28.6</td>
<td>4.4</td>
<td>14.7</td>
<td>2.2</td>
<td>13.0</td>
<td>2.0</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>30.7</td>
<td>5.4</td>
<td>27.1</td>
<td>4.4</td>
<td>13.9</td>
<td>2.4</td>
<td>12.3</td>
<td>2.0</td>
</tr>
<tr>
<td>8-9</td>
<td>R</td>
<td>41.9</td>
<td>7.4</td>
<td>35.3</td>
<td>8.3</td>
<td>19.0</td>
<td>3.4</td>
<td>15.0</td>
<td>3.8</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>39</td>
<td>9.3</td>
<td>33</td>
<td>8.9</td>
<td>17.7</td>
<td>4.2</td>
<td>15.0</td>
<td>3.1</td>
</tr>
<tr>
<td>10-11</td>
<td>R</td>
<td>53.9</td>
<td>9.7</td>
<td>49.7</td>
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<td>4.4</td>
<td>22.5</td>
<td>3.7</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>48.4</td>
<td>10.8</td>
<td>45.2</td>
<td>6.8</td>
<td>22.0</td>
<td>4.9</td>
<td>20.5</td>
<td>3.1</td>
</tr>
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<td>12-13</td>
<td>R</td>
<td>58.7</td>
<td>15.5</td>
<td>56.8</td>
<td>10.6</td>
<td>26.6</td>
<td>7.0</td>
<td>25.8</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>55.4</td>
<td>16.9</td>
<td>50.9</td>
<td>11.9</td>
<td>25.1</td>
<td>7.7</td>
<td>23.1</td>
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<td>5.6</td>
</tr>
<tr>
<td></td>
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<td>64.4</td>
<td>14.9</td>
<td>49.3</td>
<td>11.9</td>
<td>29.2</td>
<td>8.8</td>
<td>22.4</td>
<td>5.4</td>
</tr>
<tr>
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<td>42.6</td>
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</tr>
<tr>
<td></td>
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<td>19.1</td>
<td>56.9</td>
<td>14</td>
<td>35.6</td>
<td>8.7</td>
<td>25.8</td>
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<tr>
<td>18-19</td>
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<td>71.6</td>
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<td>11.2</td>
<td>32.5</td>
<td>5.6</td>
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<tr>
<td></td>
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<td>27.8</td>
<td>61.7</td>
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<td>42.2</td>
<td>12.6</td>
<td>28.0</td>
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</tr>
<tr>
<td>20-24</td>
<td>R</td>
<td>121</td>
<td>20.6</td>
<td>70.4</td>
<td>14.5</td>
<td>54.9</td>
<td>9.3</td>
<td>31.9</td>
<td>6.6</td>
</tr>
<tr>
<td></td>
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<td>21.8</td>
<td>61</td>
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<td>27.7</td>
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<td>16.2</td>
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<td>22.4</td>
<td>78.7</td>
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<td>35-39</td>
<td>R</td>
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<td>10.9</td>
<td>33.6</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
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<td>68.3</td>
<td>11.7</td>
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Source: Jamar Plus Digital Hand Dynamometer Grip Strength Instruction Manual
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Biography

Katherine (Katie) Mai Kwong graduated from the University of California, San Diego (UCSD) with a Bachelor’s degree in Sociocultural Anthropology and from the University of South Florida, Tampa (USF) with a Master of Public Health degree in Global Health Practice. Prior to joining the Ph.D. program in Interdisciplinary Aging Studies at Tulane University, she conducted international research in the Khwebe Hills, Botswana and Santiago Juan, Belize. Her passion for aging research and working with the older adult population began at USF when she worked as a qualitative researcher on a joint project with USF and a local senior living community. It is there that her interest in social networks and chronic conditions in the older adult population emerged. The culmination of this interest is this dissertation examining the role of social connections and healthcare decision-making amongst minority older adult populations.