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WISDOM Program as a Treatment for Geriatric Psychopathology

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WISDOM Program as a Treatment for Geriatric Psychopathology

by

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the requirements for the degree
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Each person whose signature appears below certifies that this dissertation in his/her opinion is adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy in Psychology.

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CONTENT

Chapter

TABLES

Tables Page

ABBREVIATIONS

MCI	Mild Cognitive Impairment
AD	Alzheimer's Disease
BMC	Behavioral Medicine Center
FMOC	Family Medicine Outpatient Clinics
GDS	Geriatric Depression Scale
GAS	Geriatric Anxiety Scale
RBANS	The Repeatable Battery for the Assessment of Neuropsychological Status
IM	Immediate Memory
ATT	Attention
DM	Delayed Memory
TS	RBANS Total Scale Score

ABSTRACT OF THE DISSERTATION

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As the population of older adults continues to rise, the prevalence and incidence of psychopathology in this group will increase as well. The most common psychopathologies observed in older adults include depression, anxiety, and mild cognitive impairment. These conditions are often co-occurring; however, few interventions are designed to simultaneously address all three. We examined whether the LLU WISDOM adult partial outpatient program was effective in reducing depression and anxiety, as well as improving cognitive function in a sample of older adults. We hypothesized that compared to a control group receiving usual clinical care, participants in the WISDOM program will demonstrate significantly greater improvement in scores on the Geriatric Depression Scale (GDS), Geriatric Anxiety Scale (GAS), and on the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), after controlling for demographic covariates. The sample consisted of 12 participants (7 control and 5 WISDOM), who were 50.0% female with an average age of 71.9 years. The WISDOM group did not significantly improve on GDS, GAS, RBANS Immediate Memory, Attention, Delayed Memory Index, and Total Scale Index scores (*p*s > .05) from baseline to the end of treatment compared to the control group, controlling for covariates. Overall, both the WISDOM and control groups did not exhibit significant

changes in depression, anxiety, or RBANS scores from the beginning of the treatment program to the end of the program. Clinically, it is important to note that while the WISDOM and control participants did not significantly improve in mood and cognitive function, these participants did not become significantly worse either. Overall, this study elucidates the complexity of the relationship between depression, anxiety, and cognitive function in older adults. It also sheds light on the study design limitations and intricacies inherent to further investigation of the relationships between the effects of treatment on depression, anxiety, and cognitive function.

Keywords: Older adult, geriatric, depression, anxiety, mild cognitive impairment, attention, immediate memory, delayed memory

CHAPTER ONE

INTRODUCTION

Older Adult Psychopathology

The older adult population, age 65 years and older, remains the fastest growing population in the United States. By the year 2050, the number of older adults in the United States is projected to grow to 83.7 million, nearly doubling the number of older adults by 2050 compared to those in 2012 (U.S. Census Bureau, 2017). Psychopathology is not as common in older adults as it is in younger age groups (Byers et al., 2010). However, older adults with mental disorders are less likely to seek mental health care than younger age groups and are more likely to discontinue treatment (Conner et al., 2010; Sirey et al., 2001). Moreover, older adults are less likely to receive specialty mental health care compared to younger age groups (Bogner et al., 2009). Given these factors, there is potential for a large public health crisis related to treating older adults with psychopathology, and a need for further study of effective treatment modalities that address the specific nature of late-life psychopathologies.

Contrary to common belief, suffering from depression and anxiety is not part of the normal aging experience. Typically, community-dwelling older adults without a psychiatric diagnosis report low levels of worry, anxiety, and depression (Beck et al., 1996). Depression and anxiety are not extremely common and are less prevalent in older adults than younger adults. However, depression and anxiety are the most prevalent psychopathologies occurring in an estimated 5% and 12% of the community-dwelling older adult population, respectively (Byers et al., 2010). Comorbid depression and anxiety is also commonly seen in older adults (Almeida et al., 2012). The risk factors for

developing depression in late-life include stressful life events and genetic factors such as the serotonin transporter gene (5HTTLPR), Apolipoprotein E gene (APOE), and the brain-derived neurotrophic factor (BDNF) gene (Naismith et al., 2012; Schoevers et al., 2000). Risk factors for late-life depression also include insomnia, high levels of neuroticism, low levels of social support, bereavement, and cardiovascular disease (Alexopolous et al., 1997; Farmer et al., 2002; Pigeon et al, 2008; Prince et al., 1997). Risk factors for developing anxiety in later life include insomnia, being female, increased number of medical conditions, and psychosocial factors such as level of income and education (Almeida et al., 2012; Byers et al., 2010; El-Gabalaway et al., 2011; Neckelmann et al., 2007).

Clinical Presentation in Older Adults

Compared to younger adults, depression and anxiety present differently in the older adult population. Older adults with depression are more likely to report loss of interest in activities, hopelessness, and helplessness compared to younger adults (Hegeman et al., 2012). They are also more likely to endorse somatic symptoms such as pain, sleep disturbance, and loss of appetite (Hegeman et al., 2012; Hybels et al., 2012). Finally, older adults with depression are more likely to report psychomotor retardation and agitation (Hegeman et al., 2012), and less likely to report symptoms such as anhedonia, guilt or shame, worthlessness, enjoying life as much as others, and suicidal ideation (Gallo et al., 1997; Hegeman et al., 2012; Hybels et al., 2012). Differences in depression symptomology presentation are also apparent amongst different groups of older adults. Older adults with late-onset depression (first episode in old age) are less likely to report a positive family history of depression and are more likely to have greater

cognitive impairment compared to individuals with early-onset depression (Gallagher et al., 2010). On the other hand, older adults with early-onset depression (first episode before old age) are more likely to report thoughts of guilt, feelings that life is not worth living, and being suicidal than older adults with late-onset depression (Gallagher et al., 2010).

Similarly, anxiety presents differently in older adults than younger adults. Older adults with anxiety are more likely to report somatic symptoms such as gastrointestinal discomfort, pain, and fatigue, and are less likely to experience negative emotions (Lenze et al., 2005). Older adults are also more likely to pay attention to more positive information than negative information, experience lower emotional arousal, and explain their somatic symptoms medically (Bryant et al., 2013; Charles & Carstensen, 2008; Mather & Carstensen, 2005). Older adults with anxiety are less likely to endorse words such as worry, excessive, or uncontrollable (Mohlman et al., 2012), and are also less likely to worry about finances or social events compared to younger adults (Powers et al., 1992). In addition, older adults are less likely to recognize symptoms of anxiety and are subsequently less likely to seek treatment than younger adults (Mackenzie et al., 2012; Wetherell et al., 2009). These differences in anxiety symptom presentation between older adults and younger adults, and the reduced likelihood of older adults to recognize their symptoms as anxiety make it difficult for clinicians to distinguish between anxiety and other conditions, render a proper diagnosis, and provide subsequent treatment.

Suicide Risk

Despite a lower prevalence of depression and anxiety in older adults compared to younger age groups, depression and anxiety pose a significant threat to older adults as the

severity of both predict of poorer subjective quality of life and well-being, as well as, decreased life satisfaction (Bourland et al., 2000; De Beurs et al., 1999) and late-life suicide (Turvey et al., 2002). Depression is the most predictive risk factor of suicide in older adults occurring in roughly 80% of completed suicides (Conwell et al., 1996). Anxiety has also been strongly associated with late-life suicide (Oude Voshaar, 2015). Specifically, one in six older adults who committed suicide also suffered from a primary anxiety diagnosis (Oude Voshaar, 2015). Although suicidal ideation and attempts typically decrease with age, suicide remains an extremely pertinent issue related to depression.

Older adults who have suicidal thoughts are more likely to commit suicide successfully than younger age groups (Conwell & Thompson, 2008; Fremouw et al., 1990; McIntosh et al., 1994). The ratio for suicide attempts to completed suicides in older adults is two to four attempts for every completed suicide which is much lower than in younger age groups (Conwell & Thompson, 2008; Mościcki, 1997). A few possible explanations for why suicide attempts are more fatal in older adults is that many live alone and do not have people who can check in on them often, they are more frail, and use more lethal means of suicide than younger age groups (Conwell & Thompson, 2008; Conwell et al., 2002).

Quality of Life and Associated Health Risks

Depression and anxiety have been found to have a bi-directional relationship with disability. Turner and Noh (1988) discovered that individuals with a physical disability are at a significantly elevated risk for developing depression symptomology. Depression is also predictive of the development of disability in older adults (Penninx et al., 1999).

Specifically, depression increased the risk of disability in activities of daily living by 67% and mobility disability by 73% over a six-year period (Penninx et al., 1999). Proposed explanations of depression significantly increasing the risk of subsequent disability include decreased physical activity, less social contact with relatives, and being less likely to seek medical treatment for depression (Penninx et al., 1999; Sullivan, 1990).

Anxiety has also been directly associated with increased risk for disability (Brenes et al., 2008; De Beurs et al., 1999) regardless of having an anxiety diagnosis. Furthermore, El-Gabalawy et al. (2013) discovered that older adults with an increasing number of chronically painful conditions such as back pain, migraines, and arthritis were at higher risk for comorbid anxiety and poor self-rated health. Finally, comorbid depression and anxiety have been found to significantly increase the risk of disability in older adults (Brenes et al., 2008).

Depression and anxiety also have a significant association with medical illness. The most commonly reported medical illness associated with both depression and anxiety is cardiovascular disease. Frasure-Smith et al. (1993) found that depression was a significant predictor of six-month mortality in individuals who were discharged from the hospital after a myocardial infarction even after controlling for left ventricular dysfunction (Frasure-Smith et al., 1993). In fact, older adults with depression post myocardial infarction had a four times greater risk of dying (26% vs. 7%) within four months after discharge from the hospital compared to older adults who were not depressed (Romanelli et al., 2002). Older adult patients with depression were also more likely to have had a prior myocardial infarction compared to patients who were not depressed (Romanelli et al., 2002).

Forty five percent of chronic heart failure outpatients are also diagnosed with anxiety (Friedmann et al., 2006). Co-morbid depression and anxiety is also commonly seen in patients with chronic obstructive pulmonary disease and having both predict worse quality of life (Di Marco et al., 2006). Specifically, depression and anxiety have been found in 18.8% and 28.2% of COPD patients, respectively, compared to 6.1% and 5.3% of healthy older adults (Di Marco et al., 2006).

Cognitive Impairment

Mild cognitive impairment (MCI) is another prevalent problem in older adults occurring in roughly 19% with prevalence rates further increasing with age (Lopez et al., 2003). MCI remains a clinically significant issue affecting older adults as individuals with MCI are more likely to progress to dementia and often report depressive symptoms (Modrego & Ferrández, 2004; Lyketsos et al., 2002). The clinical criteria for diagnosing MCI include concerns regarding a change in cognition, impairment in one or more cognitive domains, preservation of independence in functional abilities, and the absence of dementia (Albert et al., 2011). Essentially, individuals with MCI are characterized as having clinically significant cognitive impairments while continuing to function independently without significant disability in their daily functioning (Petersen et al., 1997). Generally, MCI is seen as a transitory state between normal age-related cognitive changes and dementia. More specifically, researchers contend that MCI is commonly the precursor to Alzheimer's disease (AD) (Buratti et al., 2015; Morris et al. 2001). The results of a recent study indicate that 26% of the participants with MCI progressed to AD (Buratti et al., 2015). However, not all individuals diagnosed with MCI progress to AD or another type of dementia. In a study investigating MCI reversion, 16% of individuals

diagnosed with MCI reverted back to normal or near-normal cognitive functioning (Koepsell & Monsell, 2012).

In order to explain the potential causes of MCI, researchers have predominantly focused on the neuropathological and neurobiological changes consistent with AD (Morris et al., 2001; Price & Morris, 1999; Troncoso et al., 1996). The neuropathology consistent with AD and thought to be responsible for the cognitive changes associated with MCI include the build-up of amyloid plaques, neurofibrillary tangles, hippocampal atrophy, and entorhinal atrophy (Price & Morris, 1999; Troncoso et al., 1996). However, these neuropathological and neurobiological changes are not as severe in individuals with MCI compared to those with dementia, presumably because MCI is considered an earlier, less severe stage of AD that precedes dementia. Apart from AD, cardiovascular disease, stroke, and depression have been identified as alternate potential causes of MCI (Gunstad et al., 2005; Kimura et al., 2000; Modrego & Ferrández, 2004).

Cognitive Impairment and Depression

In recent years, researchers have begun to examine the complex relationship between depression and MCI, as comorbid depression and MCI are commonly observed with prevalence rates varying between 3% and 63% (Lyketsos et al., 2002; Panza et al., 2010). Panza et al. (2010) theorized that this wide range of prevalence rates is due to investigators utilizing different diagnostic criteria, diagnostic assessments, and definitions of both depression and MCI. Some symptoms of depression and cognitive impairment, such as depressed mood, irritability, and poor concentration overlap, making it difficult for researchers and clinicians to distinguish between the conditions (Panza et al., 2010). Nevertheless, comorbid depression and MCI have been associated with negative

outcomes. Late-onset depression, in particular, has been found to be a significant risk factor for cognitive impairment in the elderly, and has been associated with increased risk of progression from MCI to dementia (Barnes et al., 2004; Modrego & Ferrández, 2004). Specifically, older adults with comorbid depression and MCI are at twice the risk for developing AD compared to MCI patients without depression (Modrego & Ferrández, 2004).

In addition, individuals with comorbid MCI and depression experience greater cognitive deficits compared to individuals with MCI alone (Johnson et al., 2013). Those with comorbid MCI and depression experience greater impairment on immediate and delayed memory tasks compared to individuals with MCI alone (Johnson et al., 2013). Older adults with comorbid depression and MCI experience impairment in retrieving information that they had just learned and encoding information compared to healthy controls leading to significant impairments in memory (Hudon et al., 2008). Additionally, a significant relationship between depression and impairment in attention has been established (Porter et al., 2003). One study investigating remission of depression and impairment in attention found that impairment in attention improved in patients who experienced a remission in depression (Huang, 2009). Conversely, impairment in attention was found to continue after remission of depression which was defined as patients no longer meeting depressive episode criteria for a period of three months (Paelecke-Habermann et al., 2005).

Researchers have linked the co-occurrence of depression and MCI to shared cardiovascular risk factors (Alexopoulos et al., 1997; Frasure-Smith et al., 1993; Vermeer et al., 2003). Alexopoulos et al. (1997) hypothesize that cerebrovascular disease increases

the risk for, precipitates, or maintains depression. Hypertension and stroke have also been significantly associated with late-life depression (Frasure-Smith et al., 1993; Romanelli et al., 2002; Taylor et al., 2004). Additionally, diabetes, cardiac disease, and hypertension have been significantly associated with cerebral white matter lesions which have been identified as white matter hyperintensities in neuroimaging studies using MRIs (Gunstad et al., 2005; Krishnan et al., 1997; Taylor et al., 2003). These shared cardiovascular risk factors lead to a change in cerebral blood flow which is associated with white matter hyperintensities and results in the subsequent manifestation of depressive and cognitive impairment symptoms (Coffey et al., 1989; Gunstad et al., 2005). However, Barnes et al. (2006) discovered that depressive symptoms increase the risk for MCI independent of potential cardiovascular disease.

An alternate explanation of the association between depression and MCI is that depressive symptoms may cause hippocampal atrophy and decreased left hippocampal volume (Sheline et al., 1996; Steffens et al., 2002). It is hypothesized that individuals with depression are more likely to experience a hypersecretion of glucocorticoids, which are adrenal steroids released during stress (Sheline et al., 1996). A hypersecretion of glucocorticoids can be neurotoxic and lead to atrophy of the hippocampus which is important for learning and memory (Sheline et al., 1996). Conversely, it can be argued that individuals may develop depression as a reaction to cognitive decline (Chen et al., 1999; Panza et al., 2010). Alternatively, the development of late-life depressive symptoms may demonstrate an underlying neuropathological condition such as AD or other dementias that explains the onset of depressive symptoms, rather than depressive

symptoms serving as a predictor for subsequent development of cognitive impairment (Chen et al., 1999; Li et al., 2011).

Given the relationship between depression and MCI, it is important to investigate the outcomes of treating depression and MCI in older adults. One study found that cognitive impairment was significantly improved after antidepressant treatment of depression in older adults; however, cognitive functioning did not completely return to normal levels (Butters et al., 2000). In another study investigating treatment of depression and cognitive functioning using an antidepressant, Lyketsos et al. (2003) found a significant reduction in depressive symptoms in participants with comorbid MCI and depression, but not a significant improvement in cognition. Finally, in a study investigating post-stroke depression and MCI, it was discovered that the remission of depressive symptoms in individuals with MCI resulted in significant improvement in cognitive functioning compared to individuals whose depressive symptoms were not reduced (Kimura et al., 2000).

Since not all participants in the previous studies responded to pharmacological treatment, nonpharmacological treatments of comorbid MCI and depression must be considered. Alexopoulus et al. (2003) discovered that problem solving therapy targeting executive dysfunction in depressed older adults significantly improved their depressive symptoms compared to older adults with executive dysfunction treated with supportive therapy; moreover, the individuals in the problem-solving therapy treatment group experienced less disability compared to their counterparts. Problem solving therapy focused toward the caregiver of an individual with AD has also significantly reduced depressive symptoms for the individuals with AD and their caregivers, as well (Teri et

al., 1997). The research on improving cognitive functioning and depressive symptoms remains mixed; however, researchers have demonstrated that pharmacological treatment of depression in older adults can result in significant improvements in depressive symptoms as well as cognitive functioning. Nonpharmacological treatment targeting cognitive symptoms also has been shown to significantly reduce depressive symptoms and disability.

Cognitive Impairment and Anxiety

Just as depression has been associated with MCI, late-life anxiety has been found to be a significant predictor of cognitive decline (Sinoff & Werner, 2003). However, late-life anxiety is not as commonly associated with MCI compared to depression. It is suggested that anxiety may predict development of depression which has been associated with subsequent development of MCI and progression to dementia (Modrego & Ferrández, 2004; Sinoff & Werner, 2003). In addition, it is difficult to separate memory loss from anxiety (Sinoff & Werner, 2003). Comorbid depression and anxiety have been discovered as a significant risk factor for subsequent MCI (DeLuca et al., 2005). In one study, researchers discovered that there was accelerated memory decline in individuals with comorbid depression and anxiety compared to those without comorbid depression and anxiety (DeLuca et al., 2005). Despite some literature indicating a relationship between anxiety and cognitive decline, more research needs to be conducted to further elucidate this relationship.

Current Study

It is apparent that the population of older adults will continue to rise and, consequently, that cases of psychopathology in this group will increase as well (U.S.

Census Bureau, 2017). The most common psychopathologies in older adults include depression, anxiety, and MCI (Byers et al., 2010; Lopez et al., 2003). These psychopathologies are complex and present uniquely in older adults making it difficult for clinicians to diagnose and treat these conditions. In addition, depression, anxiety, and MCI are commonly comorbid, adding to the difficulty and complexity of treatment (Almeida et al., 2012; DeLuca et al., 2005). These conditions are associated with negative consequences that affect the quality of life and longevity of older adults. With these concerns in mind, it is imperative to find a treatment modality that specifically targets depression, anxiety, and MCI simultaneously.

The WISDOM program is an intensive, comprehensive, and multidisciplinary treatment program offered at Loma Linda University Behavioral Medicine Center (LLU BMC) that focuses on reducing geriatric depression, anxiety, and improving cognitive function. The program is 8 weeks in duration and utilizes a multi-pronged approach to treatment of both mood and cognitive symptoms that includes reminiscence therapy, problem solving therapy, cognitive behavioral therapy, behavioral health education, memory fitness, cognitive training, and enhanced coordination of care. This study will investigate the effectiveness of the WISDOM program in treating depression, anxiety, and cognitive impairment in community-dwelling older adults.

Aims and Hypotheses

The primary aim of this study was to determine whether the WISDOM program is an effective treatment program in reducing geriatric depressive and anxiety symptoms compared to a clinical comparison group (control). We hypothesized that compared to the control group, participants in the WISDOM program will have significantly greater

improvement (i.e., reduction) in scores on the Geriatric Depression Scale (GDS) and Geriatric Anxiety Scale (GAS) than the control group.

The second aim of this study was to determine whether the WISDOM program is an effective treatment program in improving cognitive functioning, as memory fitness and cognitive training are components of the treatment program. We hypothesized that compared to the control group, participants in the WISDOM program will have significantly greater improvement in scores on the RBANS Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores from the beginning to end of treatment.

The final aim of this study was to determine whether there is a relationship between depression severity and cognition. We hypothesized that there would be a negative relationship between depression severity and cognition such that higher scores on the GDS will significantly predict lower scores on the RBANS-A Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores.

CHAPTER TWO

METHODS

Participants

Subjects were recruited from the Loma Linda University Behavioral Medicine Center WISDOM adult partial program (LLU WISDOM). A clinical comparison group receiving standard of care treatment was recruited from the Loma Linda University Department of Family Medicine Outpatient Clinics (LLU FMOC). Standard of care treatment included treatment of depression and anxiety, through medication management and follow-up with a primary care physician, but no interventions for cognitive function. Subjects were required to fall within the age range of 60-89, be native English speakers (English is primary language), and screen positive for depression on the Patient Health Questionnaire-9 (> 4, 5 – 27), GDS-Short Form (> 5, 6 –15), or GDS-Long Form (> 9, 10 – 30). Individuals who were excluded from this study were individuals who indicated they were taking cognition-enhancing drugs, including Aricept, Exelon, Razadyne, or Namenda, and/or were diagnosed with dementia.

Participants from the LLU WISDOM underwent a screening evaluation by the lead psychiatrist and WISDOM program clinical staff prior to beginning treatment as part of their regular clinical examination. LLU FMOC patients were patients of Family Medicine physicians who had screened positive for depression on either the Geriatric Depression Scale (GDS), GDS – Short Form (GDS-SF), or Patient Health Questionnaire (PHQ-9) which were administered as part of their usual clinical examination. Subjects received $10 in compensation for their time during their follow-up appointment.

Procedure

At the initial visit, participants completed the informed consent documents, demographics questionnaire, and were administered the RBANS Form A by examiners. During the follow-up visit 8-9 weeks later, participants were administered the RBANS Form B by examiners. Additionally, FMOC participants completed measures of depression (GDS) and anxiety (GAS) symptoms at both baseline and follow-up visits. GDS and GAS scores for LLU WISDOM program participants, which had been administered by the WISDOM program clinical staff at the beginning and end of treatment, were obtained from medical charts to reduce repeat testing effects.

Measures

Demographic Variables

Participants reported their age, gender, ethnicity, handedness, level of education, level of income, employment status, marital status, and current prescription medications.

Depression Severity

Depression severity was assessed using the Geriatric Depression Scale (GDS; Yesavage et al., 1982). The GDS is a 30-item questionnaire on which participants were asked to respond "*yes*" or "*no*" to questions about the last week: "*Do you feel that your life is empty?*", "*Do you feel happy most of the time?*", and "*Do you often feel downhearted and blue?*". The purpose of the GDS is not to diagnose participants with depression. Rather, it is used as a screening tool to assess for depressive symptomology and severity. Each item on the GDS was given a score of 0 or 1 with total scores ranging from 0 to 30. Scores in the range from 0 to 9 indicated normal functioning, 10 to 19 indicated mild depression, and 20 to 30 indicated severe depression. The GDS has been

tested for reliability and validity and has shown to be a reliable and valid measure of geriatric depression with a reported test-retest reliability coefficient of 0.94 and a Cronbach's alpha coefficient of 0.94 (Yesavage et al., 1982).

Anxiety Severity

Anxiety severity was assessed using the Geriatric Anxiety Scale (GAS; Segal et al., 2010). The GAS is a 30-item self-report questionnaire that measures somatic, cognitive, and affective anxiety symptom domains in older adults (Segal et al., 2010). The GAS includes items such as "*my heart raced or beat strongly*", "*I felt like things were not real or like I was outside myself*", and "*I felt restless, keyed up, or on edge*". Participants responded to these items by indicating over the last week how often they have experienced each symptom (0, *not at all* to 3, *all of the time*). A total score for the GAS was calculated by summing items 1 through 25. Items 26 through 30 measure common areas of concern reported by geriatric patients with anxiety, but were not added to the GAS total score (Segal et al., 2010). Scores on the GAS ranged from 0 (indicating no anxiety) to 75 (indicating severe anxiety). The GAS has been shown to be a reliable instrument for assessing anxiety symptomology in a community-dwelling sample of older adults and a clinical sample of older adults as the reported Cronbach's alpha for these population samples were 0.93 and 0.93, respectively (Segal et al., 2010).

Cognitive Function

Cognitive function was assessed using the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph et al., 1998). The RBANS is a neuropsychological assessment that takes between 30-40 minutes to administer and provides a measure of cognitive function across five cognitive domains: Immediate

Memory, Visuospatial/Constructional Ability, Attention, Language, and Delayed Memory. There are twelve subtests that constitute the five cognitive domains. These subtests include List Learning, Story Memory, Figure Copy, Line Orientation, Digit Span, Symbol Digit Coding, Picture Naming, Semantic Fluency, List Recall, List Recognition, Story Recall, and Figure Recall. Raw scores of each subtest within each domain were added together and the sums were converted to domain scaled scores based on age-adjusted norms. In addition to scaled scores on specific domains, a total score was calculated. The total score provides a measure of participant's overall cognitive function. The RBANS was designed to be repeatable and minimize practice effects. Form A was administered during baseline assessment and Form B was administered during the follow-up assessment. The RBANS has been validated on individuals with probable Alzheimer's disease (AD), probable Huntington's disease, and normal individuals (Randolph et al., 1998). Specifically, the RBANS has demonstrated good sensitivity (90%) and specificity (90%) in detecting and characterizing dementia (Randolph et al., 1998).

Statistical Analyses

Cross-Sectional Analyses

A series of stepwise linear regression analyses using SPSS 19 were conducted to examine whether higher GDS scores would significantly predict poorer performance on the RBANS-A Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores. The independent variable included depression severity measured by participants' scores on the GDS. The dependent variables included the Immediate Memory Index, a composite age-adjusted score based on performance on the List Learning and Story

Memory subtests, Attention Index, a composite age-adjusted score based on performance on the Coding and Digit Span subtests, Delayed Memory Index, a composite age-adjusted score based on performance on the List Recall, List Recognition, Story Memory, and Figure Recall subtests, and Total Scale Index, a composite age-adjusted score based on performance across all subtests. Education was included in the regression models as a covariate.

Longitudinal Analyses

A series of independent *t*-tests using SPSS 19 were conducted to determine group differences between the WISDOM participants and the control participants on baseline GDS scores, GAS scores, and RBANS Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores.

A series of univariate analysis of variance tests (ANCOVAs) using SPSS 19 were conducted to test hypotheses that participants in the WISDOM program would show significantly greater declines in scores compared to the control group on the GDS and GAS, and improved scores on the RBANS Immediate Memory, Attention, Delayed Memory Index, and Total Scale Index scores from the beginning of the treatment program to the end of the program. Change scores were calculated by subtracting individuals' baseline scores from the corresponding follow-up scores on variables of interest. The independent variable included group (WISDOM and control). Education and baseline GDS, GAS, RBANS-A Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores were included as covariates.

CHAPTER THREE

RESULTS

Cross-Sectional Analyses

The cross-sectional sample consisted of 17 participants (11 control and 6 WISDOM). The sample was 64.7% female and the average age was 71.5 years. With regard to level of income, the sample was bimodal between $0 - $25,999 (41.2%) and more than $75, 000 (41.2%). Most participants endorsed having some college education (29.4%). The ethnicities of participants were 82.4% White/Caucasian and 17.6% Hispanic/Latino (See Table 1 for additional demographic category information). 76.5% of the sample were taking antidepressant medications; none of the participants were taking cognition enhancing medications.

Table 1

Demographics for Cross-Sectional Analyses

Total *N* = 17	Total *N* (%)
Gender	
Females	11 (64.70%)
Males	6 (35.30%)
Ethnicity	
Hispanic or Latino	3 (17.60%)
Non-Hispanic White/Caucasian	14 (82.40%)
Education	
Less than high school	1 (5.90%)
High school diploma	2 (11.80%)
Some college	5 (29.40%)
AA degree	4 (23.50%)
BA degree	1 (5.90%)
Post-graduate degree	4 (23.50%)
Family Income Level	
$0 - $25,999	7 (41.20%)
$26,000 - $51,999	1 (5.90%)
$52,000 - $74,999	2 (11.80%)
More than $75,000	7 (41.20%)
Age (*SD*)	71.53 (4.72)

Table 2

Variable Characteristics for Cross-Sectional Analyses

Measure	Mean (*SD*)	Min	Max
GDS	18.41 (6.65)	5.00	29.00
GAS	25.71 (13.95)	3.00	53.00
RBANS A Immediate Memory Index	85.00 (18.38)	49.00	114.00
RBANS A Attention Index	91.24 (19.30)	53.00	118.00
RBANS A Delayed Memory Index	92.53 (15.10)	64.00	117.00
RBANS A Total Scale Index	86.41 (16.16)	61.00	110.00

Note. GDS = Geriatric Depression Scale. GAS = Geriatric Anxiety Scale.

A series of stepwise linear regressions were conducted to test the hypotheses that higher scores on the GDS would significantly predict poorer performance on the

RBANS-A Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores, controlling for education. Results indicated that scores on the GDS did not significantly predict RBANS-A Immediate Memory ($R^2 = 0.29$, $p > .05$), Attention ($R^2 = 0.49$, $p > .05$), Delayed Memory ($R^2 = 0.29$, $p > .05$), and Total Scale Index scores ($R^2 = 0.33$, $p > .05$) (See Table 3 for unstandardized regression coefficients and confidence intervals).

Table 3

Results of Stepwise Linear Regressions Predicting RBANS-A Domain Index Scores from Depression Severity

Depression Severity	*b*	95% CI	β	*p*-value	R^2
Immediate Memory Index	-0.11	[-1.45, 1.23]	-0.04	0.87	0.29
Attention Index	0.54	[-0.65, 1.73]	0.19	0.34	0.49
Delayed Memory Index	-0.30	[-0.14, 0.80]	-0.13	0.58	0.29
RBANS-A Total Scale Index	0.08	[-1.50, 1.22]	0.03	0.88	0.33

As an exploratory analysis, a series of stepwise linear regressions were conducted to test whether higher scores on the GAS would significantly predict poorer performance on the RBANS-A Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores, controlling for education. Results indicated that scores on the GAS did not significantly predict RBANS-A Immediate Memory ($R^2 = 0.32$, $p > .05$), Attention ($R^2 = 0.47$, $p > .05$), Delayed Memory ($R^2 = 0.33$, $p > .05$), and Total Scale Index scores ($R^2 = 0.33$, $p > .05$) (See Table 4 for unstandardized regression coefficients and confidence intervals).

Table 4

Results of Stepwise Linear Regressions Predicting RBANS-A Domain Index Scores from Anxiety Severity

Anxiety Severity	*b*	95% CI	β	*p*-value	R^2
Immediate Memory Index	-0.25	[-0.87, 0.38]	-0.19	0.41	0.32
Attention Index	0.17	[-0.41, 0.75]	0.13	0.53	0.47
Delayed Memory Index	-0.25	[-0.76, 0.25]	-0.24	0.30	0.33
Total Scale Index	0.01	[-0.53, 0.56]	0.01	0.96	0.33

Longitudinal Analyses

The longitudinal sample consisted of 12 participants (7 control and 5 WISDOM). The sample was 50.0% female with an average age of 71.9 years. The majority of the sample endorsed an income level of more than $75, 000 (41.7%). Most participants endorsed having some college education (33.3%). The sample participants were all Non-Hispanic White/Caucasian. The two groups did not differ on demographics or medications (See Table 5); 75.0% of the sample were taking antidepressant medications; no one was taking cognition enhancing medications.

Table 5

Demographics for Longitudinal Analyses

Total *N* = 12	Control	WISDOM	Statistic	*p*-value
	N = 7	N = 5		
Gender			$\chi^2 = 3.09$	0.08
Male	2 (28.60%)	1 (20.00%)		
Female	5 (71.40%)	4 (80.00%)		
Ethnicity			-	-
Non-Hispanic White/Caucasian	7 (100.00%)	5 (100.00%)		
Education			$\chi^2 = 8.91$	0.06
High school diploma	1 (14.30%)	0 (0.00%)		
Some college	3 (42.90%)	1 (20.00%)		
AA degree	3 (42.90%)	0 (0.00%)		
BA degree	0 (0.00%)	1 (20.00%)		
Post-graduate degree	0 (0.00%)	3 (60.00%)		
Family Income Level			$\chi^2 = 3.98$	0.26
$0 - $25,999	3 (42.90%)	1 (20.00%)		
$26,000 - $51,999	0 (0.00%)	1 (20.00%)		
$52,000 - $74,999	2 (28.60%)	0 (0.00%)		
More than $75,000	2 (28.60%)	3 (60.00%)		
Age *(SD)*	71.43 (4.89)	72.60 (2.88)	$t = -0.48$	0.65
Time *(SD)*	9.39 (0.71)	8.34 (1.47)	$t = 1.48$	0.20
Antidepressants			$\chi^2 = 2.86$	0.09
Yes	4 (57.10%)	5 (100.00%)		
No	3 (42.90%)	0 (0.00%)		
Anxiolytics			$\chi^2 = 3.36$	0.07
Yes	0 (0.00%)	2 (40.00%)		
No	7 (100.00%)	3 (60.00%)		
Cog Enhancing Medications			-	-
Yes	0 (0.00%)	0 (0.00%)		
No	0 (0.00%)	0 (0.00%)		
Hypertension Medications			$\chi^2 = 3.36$	0.07
Yes	7 (100.00%)	3 (60.00%)		
No	2 (28.60%)	2 (40.00%)		
Other Medications			$\chi^2 = 1.03$	0.31
Yes	6 (85.70%)	3 (60.00%)		
No	1 (14.30%)	2 (40.00%)		

Note. Time = Weeks elapsed between baseline assessment and follow-up assessment.

Prior to running the main analysis, a series of independent sample t-tests were conducted using SPSS 19 to test the mean differences between the WISDOM group and the control group in baseline GDS, GAS, and RBANS Immediate Memory, Attention, Delayed Memory, Total Scale Index scores. Results of the independent samples t-tests indicated significant baseline differences between the WISDOM and the control group such that the WISDOM group had a greater mean RBANS-A Total Scale Index score ($t = -2.61$, $p < .05$) than the control group at baseline.

Table 6

Results of Independent Samples T-Test Comparing Baseline Scores between Group (Control and WISDOM)

Measure		Mean (*SD*)	Min	Max	*t*-value	*p*-value
GDS1					0.50	0.63
	Total	18.17 (7.46)	5.00	29.00		
	Control	19.14 (7.20)				
	WISDOM	16.80 (8.44)				
GAS1					0.16	0.88
	Total	23.25 (14.82)	3.00	53.00		
	Control	23.86 (15.16)				
	WISDOM	22.40 (16.04)				
RBANS A Immediate Memory Index					-1.79	0.11
	Total	86.75 (16.43)	57.00	109.00		
	Control	80.29 (15.59)				
	WISDOM	95.80 (14.24)				
RBANS A Attention Index					-2.05	0.07
	Total	95.25 (17.42)	64.00	118.00		
	Control	87.57 (18.53)				
	WISDOM	106.00 (8.49)				
RBANS A Delayed Memory Index					-1.70	0.12
	Total	93.42 (13.42)	68.00	113.00		
	Control	88.57 (14.41)				
	WISDOM	100.20 (9.23)				
RBANS A Total Scale Index					-2.61	0.03*
	Total	87.33 (15.59)	63.00	110.00		
	Control	79.29 (12.50)				
	WISDOM	98.60 (12.74)				

Note. Total Sample $N = 12$. GDS1 = Geriatric Depression Scale total score at baseline (Time 1). GAS1 = Geriatric Anxiety Scale total score at baseline (Time 1). *denotes significance at $p < .05$.

Additionally, a series of univariate ANCOVAs using SPSS 19 were conducted to test differences between the WISDOM and control group in follow-up GDS, GAS, and RBANS B Immediate Memory, Attention, Delayed Memory, Total Scale Index scores.

Results of the univariate ANCOVAs indicated that there were not any significant differences between the WISDOM and the control group at follow-up in GDS, GAS, and RBANS B Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores, after controlling for education and baseline scores.

Table 7

Results of Univariate ANCOVAs Predicting Differences between Group (Control and WISDOM) in Follow-up

Measures		Mean (SD)	Min	Max	*F*	*p*-value
GDS2			6.00	28.00	0.62	0.46
	Total	17.50 (6.20)				
	Control	18.00 (5.86)				
	WISDOM	16.80 (7.29)				
GAS2			2.00	64.00	1.37	0.28
	Total	24.17 (15.39)				
	Control	27.57 (17.97)				
	WISDOM	19.40 (10.88)				
RBANS B Immediate Memory Index			49.00	112.00	0.65	0.45
	Total	88.33 (19.51)				
	Control	78.43 (17.70)				
	WISDOM	102.20 (12.85)				
RBANS B Attention Index			75.00	115.00	0.15	0.71
	Total	99.33 (14.02)				
	Control	93.29 (15.04)				
	WISDOM	107.80 (6.91)				
RBANS B Delayed Memory Index			64.00	117.00	0.20	0.67
	Total	92.25 (18.46)				
	Control	88.14 (16.87)				
	WISDOM	98.00 (20.98)				
RBANS B Total Scale Index			63.00	112.00	0.91	0.37
	Total	88.17 (14.41)				
	Control	80.43 (11.73)				
	WISDOM	99.00 (10.63)				

Note. GDS2 = Geriatric Depression Scale total score at follow-up (Time 2). GAS2 = Geriatric Anxiety Scale total score at follow-up (Time 2). Education and baseline scores were included as covariates.

A series of univariate ANCOVAs using SPSS 19 were conducted to test the hypotheses that participation in the WISDOM program would significantly reduce scores

on the GDS and GAS as well as improve scores on the RBANS Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores from the beginning of the treatment program to the end of the program, controlling for education and baseline scores. Results of the univariate ANCOVAs indicated that the WISDOM participants did not significantly improve in GDS (F = 0.62, p > .05), GAS (F = 1.68 , p > .05), RBANS Immediate Memory (F = 0.65, p > .05), Attention (F = 0.15, p >.05), Delayed Memory Index (F = 0.20, p > .05), and Total Scale Index scores (F = 0.91, p > .05) from baseline to the end of treatment compared to the control group, controlling for education and baseline scores. With four outliers removed from the analysis, there was a significant difference between WISDOM participants and control participants such that the WISDOM participants had significantly worse Delayed Memory Index scores (F = 13.40, p <.05), from baseline to the end of treatment compared to the control group, after controlling for education and baseline Delayed Memory Index scores. Outliers were removed if participant scores were two standard deviations from the mean Delayed Memory Index Score.

Table 8

Results of Univariate ANCOVAs Predicting Change Scores from Group (Control and WISDOM)

Measure		Mean (*SD*)	Min	Max	F	*p*-value
GDS			-9.00	11.00	0.62	0.46
	Total	-0.67 (5.03)				
	Control	-1.14 (2.67)				
	WISDOM	0.00 (7.62)				
GAS			-21.00	12.00	1.37	0.28
	Total	0.92 (9.22)				
	Control	3.71 (6.02)				
	WISDOM	-3.00 (12.10)				
Immediate Memory Index			-14.00	19.00	0.65	0.45
	Total	1.58 (10.66)				
	Control	-1.86 (11.77)				
	WISDOM	6.40 (7.40)				
Attention Index			-6.00	12.00	0.15	0.71
	Total	4.08 (7.10)				
	Control	5.71 (6.42)				
	WISDOM	1.80 (8.11)				
Delayed Memory Index			-29.00	10.00	0.20	0.67
	Total	-1.17 (9.91)				
	Control	-0.43 (3.87)				
	WISDOM	-2.20 (15.66)				
RBANS Total Scale Index			-8.00	13.00	0.91	0.37
	Total	0.83 (6.66)				
	Control	1.14 (6.99)				
	WISDOM	0.40 (6.95)				

Note. GDS = Geriatric Depression Scale. GAS = Geriatric Anxiety Scale. Education and baseline scores were included as covariates.

Finally, a series of pairwise comparison *t*-tests were conducted using SPSS 19 to determine whether there was significant improvement on GDS, GAS, RBANS Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores over time regardless of group. Results indicated that there were no significant improvements on GDS, GAS, RBANS Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores over 8-9 weeks.

Table 9

Results of Total Sample Pairwise T-Test Comparisons between Subtest and Domain Index Scores

Measures	Mean	*SD*	95% CI	*t*	*p*
GDS2 – GDS1	-0.67	5.03	[-3.87, 2.53]	-0.46	0.66
GAS2 – GAS1	0.92	9.22	[-4.94, 6.77]	0.34	0.74
IM Index B – IM Index A	1.58	10.66	[-5.19, 8.35]	0.52	0.62
ATT Index B – ATT Index A	4.08	7.10	[-0.43, 8.60]	1.99	0.07
DM B Index – DM A Index	-1.17	9.91	[-7.46, 5.13]	-0.41	0.69
TS Index B – TS Index A	0.83	6.66	[-3.40, 5.06]	0.43	0.67

Note. Mean = Mean difference. GDS = Geriatric Depression Scale. GAS = Geriatric Anxiety Scale. IM = Immediate Memory. ATT = Attention. DM = Delayed Memory. TS = RBANS Total Scale.

CHAPTER FOUR

DISCUSSION

Cross-Sectional Analyses

Overall, greater endorsement of depressive as well as anxiety symptoms did not significantly predict poorer cognitive function on the RBANS-A Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores. Contrary to the current study, previous literature indicates that depressive symptoms have had a strong relationship with poorer cognitive function in older adults (Kimura et al., 2000). There is also support for anxiety being associated with poorer cognitive function in older adults; however, there is less evidence in support of anxiety symptoms being associated with poorer cognitive function compared to depressive symptoms (Sinoff & Werner, 2003). The relationship between anxiety symptoms and cognitive function seems to be more complex than that of depressive symptoms and cognitive function. Previous researchers argue that the development of anxiety symptoms in older adults actually leads to the onset of depressive symptoms and subsequent decline in cognitive function (Modrego & Ferrández, 2004; Sinoff & Werner, 2003). Moreover, the simultaneous effects of depressive and anxiety symptoms on cognitive function was not investigated in the present study and merits future investigation. Since the GAS measures separate domains of anxiety (e.g., somatic, cognitive, affective; Segal et al., 2010), more in depth investigation of how these separate domains may relate to the decline in cognitive function is also warranted.

In the cross-sectional sample, a significant relationship between depressive symptoms and poorer cognitive function was not discovered. There are a number of different possible explanations for this. First and foremost, the current sample size did not

provide adequate power to find a significant effect. Apart from inadequate power, our cohort may have been somewhat unique in that participants appeared to endorse a severe level of depression, while also performing well with regard to cognitive function. WISDOM patients, in particular, appeared to be more well-educated and scored above the population mean (>100) on select indices. Another possibility is that the GDS may not have accurately captured the nature of their depressive symptomology. While the GDS has been determined to be a reliable and valid measure for geriatric depressive symptoms (Yesavage et al., 1982), it is limited in response options as participants were forced to answer "*yes*" or "*no*" to statements that they may not have identified "*yes*" or "*no*" with. A broader spectrum of response options such as a Likert-scale response style would have given a more accurate reflection of the severity of participants' depressive symptoms. Clinically, it is important to utilize multiple measures of depressive symptomology (GDS, BDI-2, PHQ-9) to more accurately assess patients and tailor their intervention to their individual needs.

Longitudinal Analyses

The WISDOM group and control group did not exhibit any significant differences in reducing geriatric depression, anxiety, or improving RBANS Immediate Memory, Attention, Delayed Memory, and Total Scale Index scores from the beginning of the treatment program to the end of the program. In fact, across both groups, no significant changes were observed in any of the outcome measures over the 8-9 week period. Problem-solving therapy, a component of the WISDOM treatment program, has been shown to reduce depressive symptoms (Alexopoulus et al., 2003); however, this was not supported by the current study. In addition, the treatment of depressive symptoms has

been associated with an improvement in cognitive function, which was also not supported by this study (Kimura et al., 2000). Again, the dichotomous response format of the GDS may explain why WISDOM participants did not demonstrate a significant reduction in depressive symptoms over time, as more subtle changes may have been better captured with a Likert-scale type format. Another significant limitation was the homogeneity and size of this longitudinal sample. Although similar studies also contain rather small sample sizes due to the inherent logistical and healthcare intricacies of this study design (Scogin & McElreath, 1994), significant findings would have been detected with a larger, more diverse sample.

Clinically, it is important to note that while the WISDOM and control participants did not significantly improve in mood and cognitive function, these participants did not become significantly worse either. The possibility remains that the WISDOM participants may have experienced an initial therapeutic effect and improvement in GDS, GAS, and cognitive function during the beginning weeks of treatment, which may have returned to baseline over time. To account for this, adding an additional assessment time point in the middle of the course of treatment could elucidate whether these patients are experiencing any improvement in mood and cognitive function that may not be captured at the end of treatment. Effort may also have been a factor in the relatively stable performance of WISDOM participants over time. Effort measures were not included in the current study but should be added to future investigations. This could be done utilizing the RBANS Effort Index score (Duff et al., 2011; Silverberg et al., 2007) and/or the RBANS Effort Scale score (Novitski et al., 2012) which would provide further information on whether participants put forth adequate effort and the validity of their cognitive screening scores.

Notably, there was a significant difference regarding baseline RBANS Total Scale Index scores between the control group and WISDOM group such that the WISDOM group had a significantly greater RBANS Total Scale Index mean score. Although significant differences between the two groups were not detected with regard to education and socioeconomic status, it is likely that cognitive reserve associated with higher levels of education and socioeconomic status contributed to the WISDOM participants higher degree of cognitive functioning (Sattler et al., 2012). Essentially, the WISDOM group had less room to improve in cognitive functioning as they had begun the study at a higher baseline level. Premorbid IQ, as well as age and education have been established as predictors of performance on mental status exams such as the MMSE, MoCA, and more expanded neurocognitive screeners including the RBANS (Alves et al., 2013; Christensen & Jorm, 1992; Gontkovsky et al., 2002). A larger sample would allow future investigations to control for factors related to cognitive reserve and premorbid abilities.

While medication was not included in the main analyses of this study, it is important to note that most participants were receiving antidepressant treatment, but neither group demonstrated a significant reduction in GDS, GAS, or improvement in cognitive function during the course of this study. It is possible that these participants initially experienced a therapeutic effect with a significant reduction in mood symptomology and improvement in cognitive function during the initial phases of pharmacological intervention, however, these improvements may have stabilized by the time participants were enrolled in the study. Dose and length of time participants had been on medications were not measured during this study. This adds to the mixed

literature findings of improved depressive symptoms and cognitive function in patients on antidepressant medications (Butters et al., 2000; Lyketsos et al., 2003).

Overall, this study elucidates the complexity of the relationship between depression, anxiety, and cognitive function in older adults. It also sheds light on the study design limitations and intricacies inherent to further investigation of the relationships between the effects of treatment on depression, anxiety, and cognitive function. However, despite these complexities, research in this regard needs to be continued as depression, anxiety, and cognitive function have such strong associations with quality of life and longevity in older adults.

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APPENDIX A

Demographic Information

1. Please indicate your gender: ☐ Female ☐ Male

2. Date of birth: (mm/dd/yyyy) _____/_____/_____

3. Please indicate your handedness:
   a) Right
   b) Left
   c) Ambidextrous

4. Please indicate your race/ethnicity:

   ☐ Asian or Pacific Islander ☐ Black/African American

   ☐ Hispanic or Latino ☐ American Indian/Native American

   ☐ Non-Hispanic White/Caucasian ☐ Other _____

5. Please indicate your marital status:
   a) Married
   b) Remarried
   c) Widowed
   d) Separated
   e) Divorced
   f) Single (never married)

6. Please indicate your highest level of education:
   a) Less than high school
   b) High school diploma or equivalent
   c) Some college, no degree
   d) Associate's Degree
   e) Bachelor's Degree
   f) Post graduate Degree (Master's or Doctoral)

7. Please indicate your total household income:
   a) $0 – $25,999

b) $26,000 – $51,999
c) $52,000 – $74,999
d) More than $75,000

8. Please indicate your employment status:
    a) Employed full-time
    b) Employed part-time
    c) Self-employed
    d) Unemployed

9. Please list the current medications you take:

    a) _____

    b) _____

    c) _____

    d) _____

    e) _____

APPENDIX B

Informed Consent

TITLE: Effectiveness of the WISDOM Program in Improving Geriatric Depression, Anxiety, and Cognitive Function

SPONSOR: Loma Linda University

PRINCIPAL INVESTIGATOR: Grace Lee, Ph.D.
11130 Anderson St.
Loma Linda, CA 92350
(909) 558-8710
GraceLee@llu.edu

Purpose of the Study

The goal of this study is to examine changes in depression and cognitive function in older adults who are receiving either standard primary care treatment or treatment through the WISDOM program.

Why am I Being Asked to Participate?

You are being invited to participate in this study because you are an English-speaking adult between the ages of 60-89, are either enrolled in the WISDOM program or receiving primary care services from Loma Linda University Department of Family Medicine, and reported experiencing depressive symptoms during your last clinic visit with your physician.

What is Involved in My Participation?

You will be asked to complete two evaluations, approximately 8-weeks apart, and each one lasting approximately 1 hour. In each evaluation:

- Research personnel will ask you a brief series of questions regarding your age, ethnicity, level of education, level of income, handedness, and medications.
- You will be asked to complete a written questionnaire regarding the presence of depression and anxiety symptoms
- Research personnel will administer a brief series of tests, involving both oral and written items and lasting approximately 30-45 minutes, that will assess various aspects of cognitive functioning, including but not limited to memory, attention and language.
- Research personnel will access your medical record for information regarding the diagnosis/treatment of dementia, cognitive impairment, mood disorders, and prescribed medications related to mood and/or cognition, including anti-depressants, anti-anxiety medications, and memory-enhancing drugs.

How Will I Benefit?

If you are experiencing symptoms of depression and are not currently receiving mental health services, you will be provided a list of referrals for local outpatient mental health

clinics. Furthermore, this study is expected to benefit society as a whole by advancing scientific knowledge to improve quality of care amongst older adults with depressive symptoms and cognitive decline.

Is This Information Going To Be Confidential?

Upon enrolling in this study, you will be assigned a unique study identification number. All data collected as part of this study will only be identified by this number and not by your name. A key matching your name to your study identification number will be stored in a password-protected file, on a password-protected server, and accessible only to study personnel. Your physician will not have access to your individual study-related data.

What Will You Do With My Study Records?

All study-related forms will be stored in a locked office cabinet in a secure clinic office. Test forms will have no identifying information and will be labeled with a study identification number only. Your data will be stored in a de-identified electronic database that is indexed only by the study identification number. This database will be password-protected and accessible only to study personnel. Additionally, your data will be stored on a computer that is password protected.

What are the Risks Involved?

The risks associated with this study are considered to be minimal. Risks could include potential breach of confidentiality, specifically from the release of sensitive information such as cognitive and psychological test data, and protected health information. However, we will make every effort to maintain confidentiality of all files containing personally-identifying information according to standard research practices outlined in the section above titled "Is This Information Going To Be Confidential?" Another potential risk includes the possibility of experiencing emotional distress or discomfort arising during the assessment process. However, you are invited to skip any questions that you do not feel comfortable answering, and you are welcome to stop the study at any time.

Can I Refuse To Be In This Study?

Participation in this study is completely voluntary. You may refuse to participate in this study and you can also withdraw (stop) at any time. Should you choose to withdraw in the middle of the study, you may also request that your personal study-related data be discarded and removed from study records. Refusal to participate and/or withdrawal from the study will have **no impact** on the clinical care you receive from either the LLU Behavioral Medicine Center or LLU Family Medicine.

Who Can I Call If I Have a Question or Complaint About This Study?

You may call (626) 373-3525 to speak to a study representative. If you have a complaint and want to talk to someone not associated with the study, you may contact the Office of Patient Relations, Loma Linda University Medical Center, Loma Linda, CA 92350. Their phone number is (909) 558-4647.

What Costs Are Involved?

There is no cost to you for participating in this study.

Will I Be Paid To Participate In This Study?
You will receive a $10 Stater Brothers gift card upon completing the second assessment.

SUBJECTS STATEMENT OF CONSENT

- I have read the contents of the consent form and have listened to the verbal explanation given by the investigator.
- My questions regarding this study have been answered to my satisfaction.
- Signing this consent document does not waive my rights nor does it release the investigators, institution, or sponsor from their responsibilities.
- If I have additional questions or concerns, I may call Dr. Grace Lee, one of the Principal Investigators of the study, at (909) 558-8710 during regular business hours. You may also contact Dr. Carolina Osorio at (909) 558-9500 during regular business hours.
- I hereby give voluntary consent to participate in this study.

I understand I will be given a copy of this consent form after signing it.

_____ _____
Signature of Subject Printed Name of Subject

_____
Date

INVESTIGATOR'S STATEMENT

I have reviewed the contents of this consent form with the person signing above. I have explained potential risks and benefits of the study.

_____ _____
Signature of Investigator Printed Name of Investigator

_____
Date

APPENDIX C

Private Health Information Authorization

INSTITUTIONAL REVIEW BOARD
Authorization for Use of
Protected Health Information (PHI)
Per 45 CFR §164.508(b)
RESEARCH PROTECTION PROGRAMS
LOMA LINDA UNIVERSITY | Office of the Vice President of Research Affairs
24887 Taylor Street, Suite 202 Loma Linda, CA 92350
(909) 558-4531 (voice) / (909) 558-0131 (fax)/e-mail: irb@llu.edu

TITLE OF STUDY:	Effectiveness of the WISDOM Program in Improving Geriatric Depression, Anxiety, and Cognitive Function
PRINCIPAL INVESTIGATOR:	Grace Lee, Ph.D.
Others who will use, collect, or share PHI:	Carolina Osorio, M.D. Ecler Jaqua, M.D. Darrell Rice, M.A. Willie Hardeman, M.A. Caleb Barcenas, M.A. Angelica Chakos, M.A.

The study named above may be performed only by using personal information relating to your health. National and international data protection regulations give you the right to control the use of your medical information. Therefore, by signing this form, you specifically authorize your medical information to be used or shared as described below.

The following personal information, considered "Protected Health Information" (PHI) is needed to conduct this study and may include records pertaining to:

Name:_____

Contact Information:_____

_____

- The diagnosis and treatment of dementia and/or cognitive impairment.
- The diagnosis and treatment of mood disorders or other psychological disorders, including depression and anxiety.

- Prescribed medications related to mood and/or cognition, including antidepressants, benzodiazepines, and memory enhancing drugs.

The main reason for sharing this information is to be able to conduct the study as described earlier in the consent form. In addition, it is shared to ensure that the study meets legal, institutional, and accreditation standards.

The individual(s) listed at the top of this page will use this protected health information (PHI) to conduct the present study. It may on occasion during the course of the study also be shared with the Institutional Review Board (IRB) and the Office of Research Affairs of Loma Linda University,

All reasonable efforts will be used to protect the confidentiality of your PHI, which may be shared with others to support this study, to carry out their responsibilities, to conduct public health reporting and to comply with the law as applicable. Those who receive the PHI may share with others if they are required by law.

Subject to any legal limitations, you have the right to access any protected health information created during this study. You may request this information from the Principal Investigator named above, but it will only become available after the study analyses are complete. The authorization expires upon the conclusion of this research study.

You may change your mind about this authorization at any time. If this happens, you must withdraw your permission in writing. Beginning on the date you withdraw your permission, no new personal health information will be used for this study. However, study personnel may continue to use the health information that was provided before you withdrew your permission. If you sign this form and enter the study, but later change your mind and withdraw your permission, you will be removed from the study at that time. To withdraw your permission, please contact the Principal Investigator or study personnel at 1-626-373-3525.

You may refuse to sign this authorization. Refusing to sign will not affect the present or future care you receive at this institution and will not cause any penalty or loss of benefits to which you are entitled. However, if you do not sign this authorization form, you will not be able to take part in this study. You will receive a copy of this signed and dated authorization.

I agree that my personal health information may be used for the study purposes described in this form.

Signature of Patient *or* Patient's Legal Representative	Date

Printed Name of Legal Representative (if any)	Representative's Authority to Act for Patient
Signature of Personnel Obtaining Authorization	Date

APPENDIX C

Private Health Information Authorization

INSTITUTIONAL REVIEW BOARD
Authorization for Use of
Protected Health Information (PHI)
Per 45 CFR §164.508(b)
RESEARCH PROTECTION PROGRAMS
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