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Nondysphoric Depressive Symptoms and Cognitive Abilities in Healthy Older Adults

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Nondysphoric Depressive Symptoms and Cognitive Abilities in Healthy Older Adults

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

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ABBREVIATIONS

MDD	Major Depressive Disorder
LDD	Late-life Depression
WAHA	Walnut and Healthy Aging
PHQ-9	Patient Health Questionnaire
SDMT	Symbol Digit Modalities Test
TMTA/TMTB	Trails Making Test A and B
BNT	Boston Naming Test
COWAT	Controlled Word Association Test
RAVLT-II	Rey Auditory Verbal Learning Test II
ROCFT	Rey-Osterrieth Complex Figure Test
CPT-II	Conners' Continuous Performance Test II
RMSEA	Root Mean Square Error of Approximation
CFI	Comparative Fit Index
SRMR	Standardized Root Mean Square Residual
LM Test	Lagarange Multiplier Test

ABSTRACT OF THE THESIS

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Research has shown that depression is associated with poorer cognitive performance and cognitive decline. Cognitive functions such as processing speed, language ability, memory, and executive functions have been found to be affected in older adults with depression. However, there is limited research focused on the effects of minimal or mild depressive symptoms in older adults who do not meet criteria for a depressive disorder. Older adults are more likely than any other population to endorse depressive symptoms in the absence of typical dysphoric symptoms that would qualify an individual for a depressive disorder. Understanding the effects of nondysphoric depressive symptoms on cognitive abilities is valuable in detecting and treating more cases of depression in older adults that may otherwise remain undiagnosed. The current study examines the effects of nondysphoric depressive symptoms on processing speed, language ability, memory, attention, and executive functions in healthy older adults. Results of structural regression modeling analyses indicate that the models in the current study of the effect of nondysphoric symptoms of cognitive abilities are not a good fit for the data and cannot be interpreted as significant results. However, trends in the data may suggest relationships similar to models including both dysphoric and nondysphoric

depression and their effect on cognitive abilities. Limitations of the study, possible reasons for poor model fit, and future research directions are discussed.

CHAPTER ONE

INTRODUCTION

In a consensus statement on mood disorders in late life, a large panel of clinical researchers reported that mood disorders in older adults, including depression, are a significant health care issue with inadequate recognition, diagnosis, and treatment, impacting individuals with the disorder, their caregivers, and the health care system (Charney et al., 2003). Fiske et al. (2009) reported that the prevalence of major depressive disorder (MDD) in community samples over the age of 65 is 1-5%. Looking at the broader picture of depressive symptoms independent of a specific disorder, Blazer (2003) reported that clinically significant depressive symptoms are prevalent in 15% of a community sample of older adults. Late-life depression (LLD) is a distinct type of depression that differs from depression experienced in earlier stages of life. Some differences between LLD and depression at earlier life stages that have been studied include increased risk of biological and social factors leading to depression (Fiske et al., 2009). LLD is also related to physical illness and disability, bereavement, and care-giving, which tend to occur more often in old age (Fiske et al., 2009).

Depression in older adults has been shown to have a distinct impact on several life functions including cognitive, physical, emotional, and social functions that affect an older adult's quality of life (Blazer, 2003). Given the relationship between depression and cognitive dysfunction in older adults, it is important to consider which specific areas of cognition are affected in order to effectively treat or prevent these deficits in the future. Several studies of the impact of depression on older adults have examined specific areas of neuropsychological functioning in a number of different clinical settings and

populations and have found mixed results (Boone et al., 1995; Kramer-Ginsberg et al., 1999; Lichtenberg et al., 1995). Generally, older adults have been shown to have deficits due to depression in executive function, memory, and language, with these areas thought to be affected by a deficit in overall processing speed (Butters et al., 2004; Sheline et al., 2006).

Two of the most current studies examining the effects of late-life depression (LLD) on neuropsychological functioning in older adults found that clinically significant depression was related to worse performance in cognitive domains of language ability, memory, and executive function, but these deficits were mediated by processing speed performance (Butters et al., 2004; Sheline et al., 2006). In the first study by Butters et al. (2004), neuropsychological performance of depressed participants was compared with non-depressed, age- and education-matched participants. Results from this study showed that approximately 60% of depressed older adults showed overall cognitive deficits (Butters et al., 2004). When compared to controls, depressed participants performed worse in all domains of neuropsychological performance, including information processing speed, visuospatial ability, executive function, language, and memory (Butters et al., 2004). Following analysis of each neuropsychological domain, information processing speed was analyzed and found to be a mediator of the relationship between depression and performance in all cognitive domains (Butters et al., 2004).

Basing much of their study on that of Butters et al. (2004), Sheline et al. (2006) focused more on the variability within the depressed population without using a control group. They found similar results as Butters et al. (2004) in that there was evidence of cognitive deficits among the older depressed population, with a significant role of

processing speed in mediating those deficits. However, some important differences arose from the study showing that executive function may also be an important mediator of the relationship between different cognitive domains and depression, and that severity of depression and vascular risk factors significantly influenced one or more cognitive performance domains (Sheline et al., 2006). Results of the study indicated that within the depressed group, a greater number of symptoms was related to more severe cognitive dysfunction (Sheline et al., 2006). This study showed the importance of examining severity of depression and overall health as individual factors influencing cognitive performance among older adults with depression.

Considering the importance of the relationship between depression and cognitive function in older adults, it is important to determine if there is a certain threshold above which depressive symptoms may be impacting cognitive function, and whether subthreshold or minimal depression has similar effects on cognitive function as that of MDD, dysthymia, or other depressive disorders. This question has received increasing attention and is somewhat controversial due to the varying definitions of subthreshold depression (Meeks et al., 2011). With the current criteria for depression, there is a possibility that many cases of depression in older adults are left unrecognized and untreated. Subthreshold depression prevalence rates range from 10% in community samples to about 45 – 50% in long term care settings, which is at least 2 – 3 times more prevalent than MDD (Meeks et al., 2011). Research suggests that associations exist between subthreshold depression and lower cognitive function, executive function deficits, increased medical burden, disability, decreased social support, and negative life events (Meeks et al., 2011). Several studies have examined the clinical implications of

depressive symptoms on a continuum rather than distinct diagnostic categories (Angst & Merikangas, 2001; Kraemer, Noda, & O'Hara, 2004; Lewinsohn et al., 2000; Widiger & Samuel, 2005). Studies examining the effect of depressive symptoms rather than distinct categories or clusters of symptoms may lead to a better understanding of whether minimal or atypical symptoms have similar effects on important functions such as cognitive abilities.

A major determining factor when diagnosing a depressive disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) is the presence of dysphoric symptoms, such as loss of interest or pleasure in life. However, older adults tend to endorse depressive symptoms associated with decreased daily functioning, such as decreased energy and lack of sleep, and are more likely than any other population to endorse depressive symptoms in the absence of these dysphoric symptoms (Fiske et al., 2009). These cases of depression in older adults would not be considered depressive disorders with the current diagnostic criteria and therefore may not receive adequate treatment or attention. Researchers have debated whether this specific type of symptom endorsement should be considered depression or simply a result of general aging. While some explain this phenomenon as a result of disengagement theory, which highlights the older adult's narrowing of social activity (Carstensen, 1992), others suggest the term "depletion syndrome" and consider it as a subtype of masked depression in older adults (Fogel & Fretwell, 1985). While it may be useful to understand the specific etiology of this phenomenon, it could be equally useful for purposes of recognition, prevention, and treatment to understand the phenomenon's effects on aspects of life, such as cognitive function, regardless of its etiology.

Very little research exists that examines nondysphoric depressive symptoms directly. Most studies include nondysphoric depression as a subset of a particular depressed population being studied. One study found a prevalence rate of 5.6% of older adults in a community setting who were described as having a nondyphoric depression syndrome that was similar to major depression, but did not include symptoms of dysphoria (Gallo et al, 1997). Another study found that endorsement of nondysphoric depressive symptoms was more common in minor or subthreshold depression, with nearly 80% of individuals in the general population reporting at least one depressive symptom not including core symptoms of dysphoria (Broadhead et al., 1990).

Functional impairment in individuals reporting nondysphoric depressive symptoms may be more likely in older adults than other populations. Gallo et al. (1997) found that, compared to non-depressed older adults, nondysphoric depressed older adults were at an increased risk for mortality, impaired activities of daily living, psychological distress, and cognitive impairment. This study suggests that there is a possible relationship between nondysphoric depressive symptoms and functional outcomes in older adults, but more research is needed to understand these relationships. While Gallo et al. (1997) addressed the importance of cognitive function in relationship to depression in older adults, the reported findings of cognitive impairment among nondyshporic older adults used less than stringent definitions of cognitive impairment such as cutoff scores from insensitive cognitive screening tests or subjective observed problems with "memory, judgment, or thinking." Examples such as this show the need for studies that include more sensitive cognitive ability measures and consider of depressive symptoms on a continuum rather than in categories, in order to determine whether a relationship

exists between nondysphoric depression and functional outcomes such as cognitive ability.

The current study is an examination of the effect of nondysphoric symptoms of depression on cognitive abilities in healthy older adults. Specific cognitive abilities measured in the study include processing speed, memory, language, and executive function. Additionally, this study will examine the relative, continuous effects of increasing depressive symptoms on cognitive function, considering that impairment from depressive symptoms may range in severity outside of established categorical depressive disorders. This study is also an attempt to control for common risk factors of depression in old age, specifically overall physical and mental health, by focusing on a community sample screened for illnesses common among older adults. Given the increasing focus on the effects of depression in older adults, understanding the effect of nondysphoric depressive symptoms on cognitive functioning in individuals within a healthy population is important in addressing and treating these symptoms to ensure better quality of life and cognitive functioning for all individuals in late life. It is expected that the results of this study will show a relationship between increased nondysphoric depressive symptoms and decreased cognitive function. Specifically, it is expected that as the number of reported nondysphoric depressive symptoms increase, cognitive performance in the areas of processing speed, memory, language, and executive function will decrease.

CHAPTER TWO

METHOD

Participants

Participants for this study were part of a larger inter-departmental study being conducted by the Department of Nutrition in the School of Public Health at Loma Linda University that is focused on examining the effects of daily walnut consumption on cognitive abilities in healthy older adults. This larger study, called the Walnut and Healthy Aging (WAHA) study, is a longitudinal randomized controlled trial with strict inclusion, exclusion, and participation criteria. All participants in the current study are subject to these criteria related to the WAHA study.

Participants in the current study were required to be aged 60-80 years, and have a Mini Mental Status Examination (MMSE) score ≥ 25. In order to ensure participants were in good health, exclusion criteria for participants in both the WAHA and the current study included: (1) morbid obesity [Body Mass Index (BMI) ≥ 40 kg/m^2]; (2) uncontrolled diabetes (HbA1c > 8%); (3) uncontrolled hypertension (on-treatment blood pressure is $\geq$ 150/100 mm Hg); (4) any prior cerebrovascular accident (CVA), craneo-encaphalic trauma (TCE), transient ischemic attack (TIA), or stroke; (5) any relevant psychiatric illness, such as Major Depressive Disorder, Bipolar Disorder, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, or Schizophrenia; (6) any advanced cognitive deterioration, such as dementia, Alzheimer's Disease, Lewy body dementia, vascular dementia, or frontotemporal lobar degeneration; (7) any neurodegenerative diseases, such as Parkinson's Disease, Multiple Sclerosis, Amyotrophic Lateral Sclerosis, Huntington's Disease, or epilepsy; (8) any chronic illness expected to shorten survival,

such as heart disease (II/IV degree), advanced hepatic diseases, hematological diseases, cancer in the last five years, or chronic alcoholism; (9) any chronic liver disease, such as cirrhosis, portal hypertension, or in chronic viral hepatitis; (10) kidney failure (GFR < 30 mL/min); (11) any chronic blood disease (such as severe anemia, leukemia, melodysplasia, thrombocytosis, etc.); (14) drug addiction; (15) bereavement in the first year of loss; (16) allergy or intolerance to walnuts; (17) customary use of fish oil (> 500 mg/d) or flaxseed oil supplements.

Additional inclusion criteria specific to the current study and not related to the WAHA study included any endorsement of depressive symptoms not related to dysphoria or no reported depressive symptoms at all on the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001). PHQ-9 items #1 and #2 were considered "dysphoric" symptoms, which included: 1. "Little interest or pleasure in doing things"; and 2. "Feeling down, depressed, or hopeless." PHQ-9 items #3 – 9 were considered "non-dysphoric" symptoms, which included: 3. "Trouble falling asleep or staying awake"; 4. "Feeling tired or having little energy"; 5. "Poor appetite or overeating"; 6. "Feeling bad about yourself – or that you are a failure or have let yourself or your family down"; 7. "Trouble concentrating on things, such as reading the newspaper or watching television"; 8. "Moving or speaking so slowly that other people could have noticed. Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual"; and 9. "Thoughts that you would be better off dead, or hurting yourself."

Of the 366 participants enrolled in the WAHA study, 95 participants (25.9%) endorsed dysphoric symptoms of depression and were not included in the current study, leaving a total of 271 participants in the analyses. The mean depression score of the

sample was 1.17 (*SD* = 1.32) and had a range of 0 to 8, with 108 participants (39.7%) reporting no depressive symptoms (i.e., a depression score of 0). Participants' in the current study were 75.7% Caucasian (9.9% Hispanic) and had a mean age of 69.7 years (*SD* = 3.87). Participants reported a mean of 15.77 (*SD* = 2.35) years of total education. See Table 1 for complete participant demographic characteristics.

Table 1. *Demographic characteristics and PHQ-9 scores of sample*

Characteristic	*N* (%)	Mean (SD)
Gender		
Male	99 (36.4)	
Female	173 (63.6)	
Age		69.66 (3.87)
60 – 65	41 (15.1)	
66 – 70	122 (44.8)	
71 – 75	86 (31.6)	
76 – 80	23 (8.5)	
Ethnicity		
Caucasian	206 (75.7)	
Hispanic	27 (9.9)	
Asian	19 (7.0)	
African American	11 (4.0)	
Other	5 (1.8)	
Missing	4 (1.5)	
Education		15.77 (2.35)
Under 12 yrs	3 (1.1)	
12 – 16 yrs	177 (65.1)	
16 – 20 yrs	92 (33.8)	
PHQ – 9 Score		1.17 (1.32)
0	108 (39.7)	
1	74 (27.2)	
2	49 (18.0)	
3	26 (9.6)	
4 – 8	14 (5.1)	

Measures and Procedures

The WAHA study was approved by Loma Linda University's Institutional Review Board. Participants for the WAHA study were screened in a phone interview for inclusion/exclusion criteria through the Nutrition Department at Loma Linda University. Eligible phone-screened participants were then invited to the Loma Linda University Campus to attend a group information meeting about the study. Those participants interested in enrolling in the study after the group information meeting were invited to a personal interview where the participant signed and was provided a copy of an informed consent document, completed the MMSE, and provided a detailed medical history to the Nutrition Department staff. Following final determination of eligibility, participants were randomized to either the daily walnut consumption group or the non-walnut control group and contacted to schedule their baseline blood, diet, and neuropsychological tests.

Prior to the participants' baseline neuropsychological testing and daily walnut consumption, they completed several psychological questionnaires including a depression inventory checklist. The participant was then administered a battery of neuropsychological tests by a trained graduate student from the Department of Psychology in the School of Behavioral Health at Loma Linda University. Participants were provided with an abbreviated report of their performance on the neuropsychological testing six to eight weeks following testing.

The neuropsychological battery was intended to measure the cognitive domains of processing speed, language, memory, executive function, and attention/working memory and included the following tests: Symbol Digits Modality Test (SMDT; Smith, 1982), Trailmaking Test A and B (TMTA/TMTB; Reitan, 1958), Boston Naming Test (BNT;

Kaplan, Goodglass, & Weintraub, 2001), Controlled Oral Word Association Test (COWAT; Benton, Hamsher, & Sivan, 1994), Rey Auditory Verbal Learning Test II (RAVLT-II; Schmidt 1996), Rey-Osterrieth Complex Figure Test (ROCFT; Osterrieth, 1994), Stroop Color and Word Test (Golden, 1978), Digit Span (Wechsler, 1997), and Conners' Continuous Performance Test – Second Edition (CPT-II; Conners, 2004).

Standardized scores were used from all tests for the study analyses. Only scores from the "Animals" portion of the COWAT, "Interference" scores from the Stroop Color and Word test, delayed recall scores from the RAVLT-II and ROCFT, and "Detectability" scores from the CPT-II were used. Baseline results of testing from the WAHA study were used in the current study and did not include any testing results after regular walnut consumption regimens were given to the participants.

Statistical Analyses

A structural regression model was analyzed using the two-step modeling strategy in order to test the hypothesis that increased nondysphoric depressive symptoms would negatively impact cognitive function in older adults (see Figure 1). An additional structural regression model with processing speed as a mediator of the relationship between nondysphoric depressive symptoms and all other cognitive domains was also tested (see Figure 2). The first step was to test the fit of the measurement portion of the model to the data, which in this study consisted of the five domains of cognitive function and the two measures associated with each of those domains. The second step of this strategy was then to test the fit of the full structural regression model to the data, which

included nondysphoric depressive symptoms as a predictor of the five cognitive domains. See Table 2 for all variables and correlations included in the analyses.

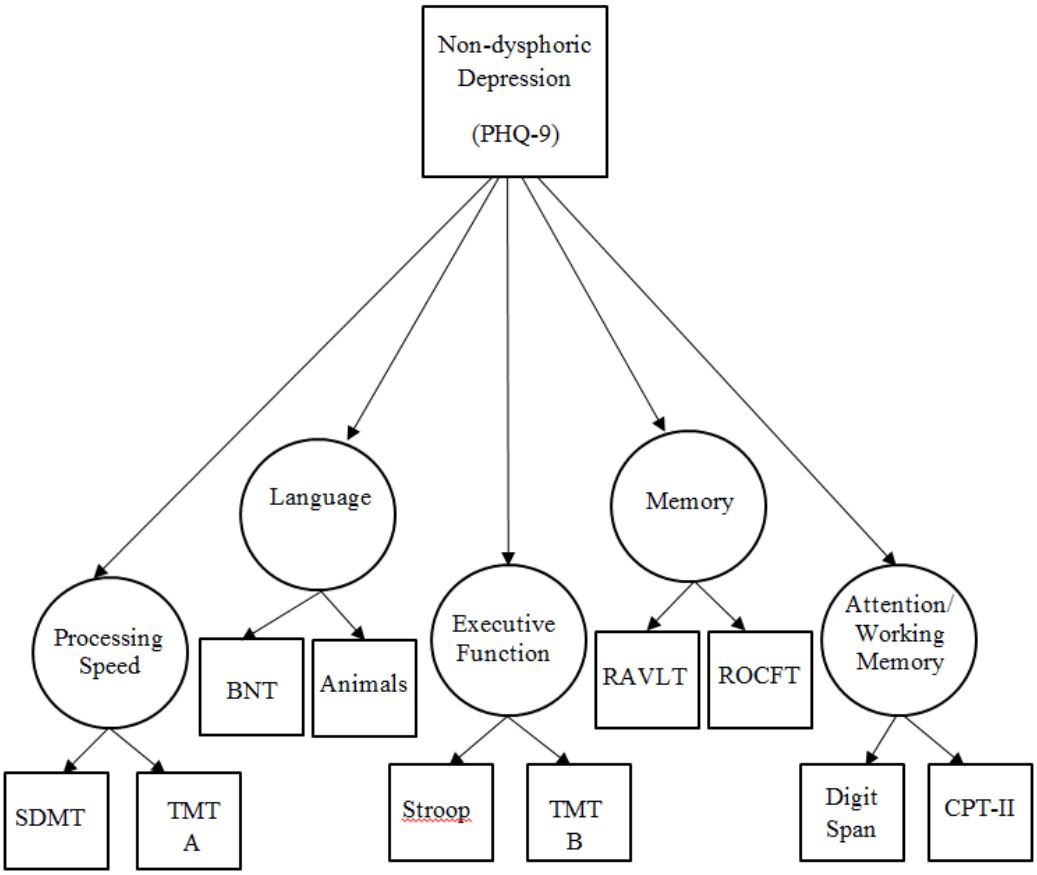

Figure 1. Hypothesized model (Model 1) of the effect of nondysphoric depression on cognitive abilities in healthy older adults.

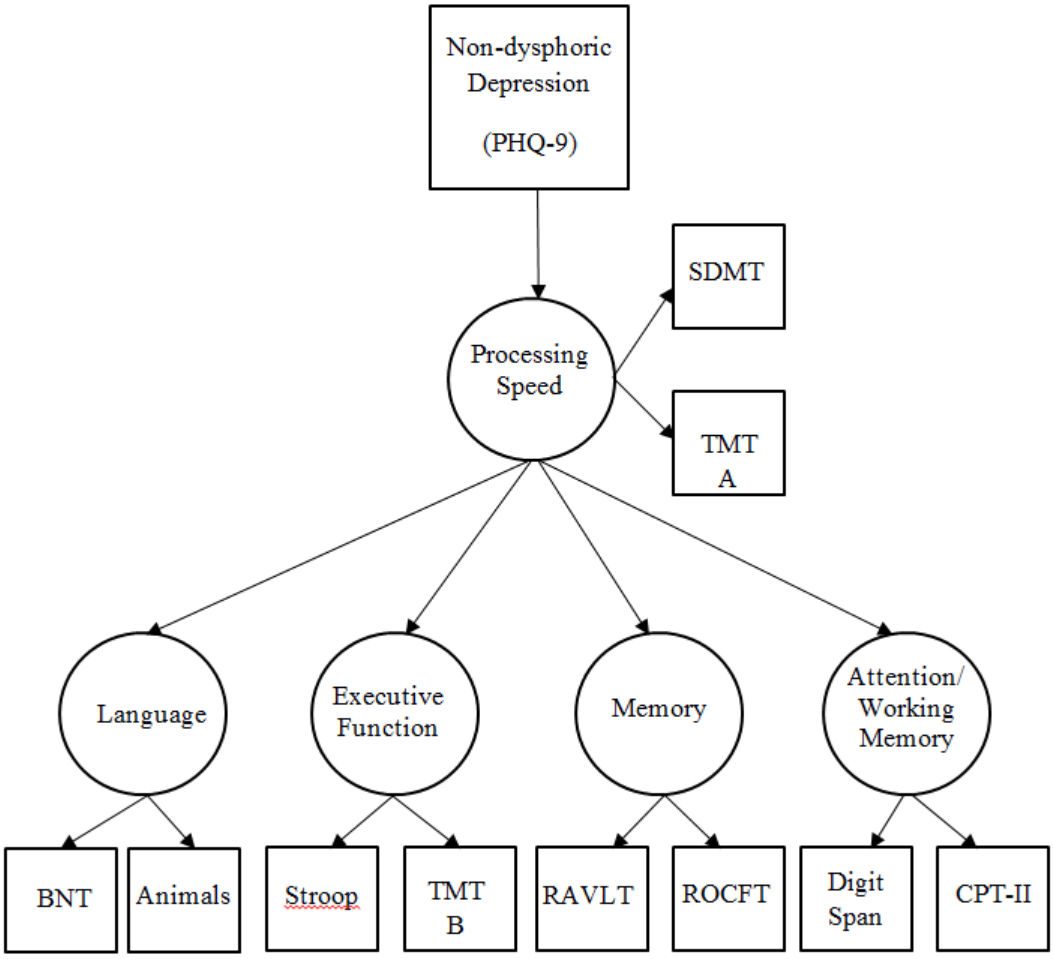

Figure 2. Hypothesized model (Model 2) of the effect of nondysphoric depression on cognitive abilities in healthy older adults with Processing Speed as a mediator of the relationship between depression and cognitive abilities.

Table 2. *Intercorrelations and Standard Deviations for Variables of Interest*

Var.	*SD*	1	2	3	4	5	6	7	8	9	10
1. SDMT	.89	1.0									
2. TMT A	.69	-.43	1.0								
3. BNT	.97	.11	-.18	1.0							
4. COWAT (Animals)	1.11	.19	-.14	.32	1.0						
5. Stroop	7.29	.11	-.11	-.04	-.12	1.0					
6. TMT B	.89	-.41	.41	-.19	-.12	-.11	1.0				
7. RAVLT	1.38	.29	-.18	.11	.17	.08	-.19	1.0			
8. ROCFT	.98	.23	-.19	.31	.12	-.01	-.21	.14	1.0		
9. Digit Span	2.79	.19	-.11	.23	.24	.06	-.34	.16	.13	1.0	
10. CPT-II (Detect.)	9.74	-.04	.03	-.03	-.10	.06	.01	-.07	-.08	-.07	1.0

Fit indices that were used to indicate the fit of the model included Model χ^2, Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and Standardized Root Mean Square Residual (SRMR). Generally, a lower and non-

significant value of Model χ^2 indicates a better fit of the model. Values of RMSEA lower than .05 indicate close model fit, values between .05 and .08 indicate reasonable fit, and above .10 indicate poor model fit. Additionally, the upper limit of the 90% Confidence Interval of RMSEA must not exceed .10. CFI requires a value greater than .90 to indicate a reasonably good fit for the model. Values of SRMR less than .10 indicate a good fit for the model. Standardized residuals were examined for values greater than |.10| (Kline, 2010).

Adjustments of the model were made using theory, research, logic, and modification indices to find the most suitable model for the data. Model χ^2 was also important in determining the best-fitting model. The Lagrange Multiplier (LM) was used to estimate Model χ^2, with a larger estimated χ^2 value indicating a need to expand or add a path to the model to improve the fit. All data were analyzed for outliers and violations of assumptions prior to analysis. All models converged and were admissible. Analyses were performed using the structural equation modeling software EQS (Bentler, 1989). Parameters were estimated using the maximum likelihood estimation.

CHAPTER THREE

RESULTS

The first step of statistical analyses testing the fit of the proposed measurement model for Model 1, including all five cognitive domains and their respective measurement variables indicated that it was not a good fit for the data, χ^2 (25) = 68.14, p < .001; CFI = .848; SRMR = .067; RMSEA = .08, 90% CI [0.06, 0.10]. Several standardized residuals of Model 1 were also greater than |.10|, indicating poor fit. Using the suggested changes indicated by the LM Test of the measurement model, as well as relevant theory, previous research, and logic to improve model fit, the cognitive domain of Executive Function and its related variables were removed from the model and the remaining model was tested for goodness of fit (Model 1a). Analysis of the resulting measurement model indicated an improved and overall good fit for the data, χ^2 (14) = 20.14, p > .1; CFI = .967; SRMR = .034; RMSEA = .040, 90% CI [0.00, 0.08]. No standardized residuals of Model 1a were greater than |.10|.

The hypothesized structural components of Models 1 and 2 were then tested using the underlying measurement structure of Model 1a (i.e., without the Executive Function factor). Results indicated that Model 1 was a poor fit for the data, χ^2 (24) = 119.31, p < .001; CFI = .508; SRMR = .123; RMSEA = .121, 90% CI [0.10, 0.14]. Several standardized residuals were greater than |.10|. Model 2 demonstrated an improved fit compared to Model 1, but an overall poor fit to the data, χ^2 (24) = 55.75, p < .001; CFI = .836; SRMR = .058; RMSEA = .070, 90% CI [0.05, 0.09]. Several standardized residuals of Model 2 were greater than |.10|; however, there were fewer compared to Model 1.Using the LM Test and supporting theory, research, and logic, an improvement

of model fit was suggested by adding a path with the cognitive domain of Attention/Working Memory predicting the cognitive domain of Language. However, after testing this respecified model, no improvement in fit was found, and thus the previous model was retained as the final model in the interests of parsimony. See Figure 3 for a diagram of the final model that fit the data best and corresponding path coefficients.

Results of the best-fitting model indicate a relatively poor fit for the data in this study. Considering the overall poor fit, path coefficients may be still useful to examine, but should be interpreted with caution. Results of the final model suggest that nondysphoric symptoms of depression may negatively impact cognitive performance in processing speed tasks, which may be positively related to performance in all other cognitive areas of Language, Memory, and Attention/Working Memory.

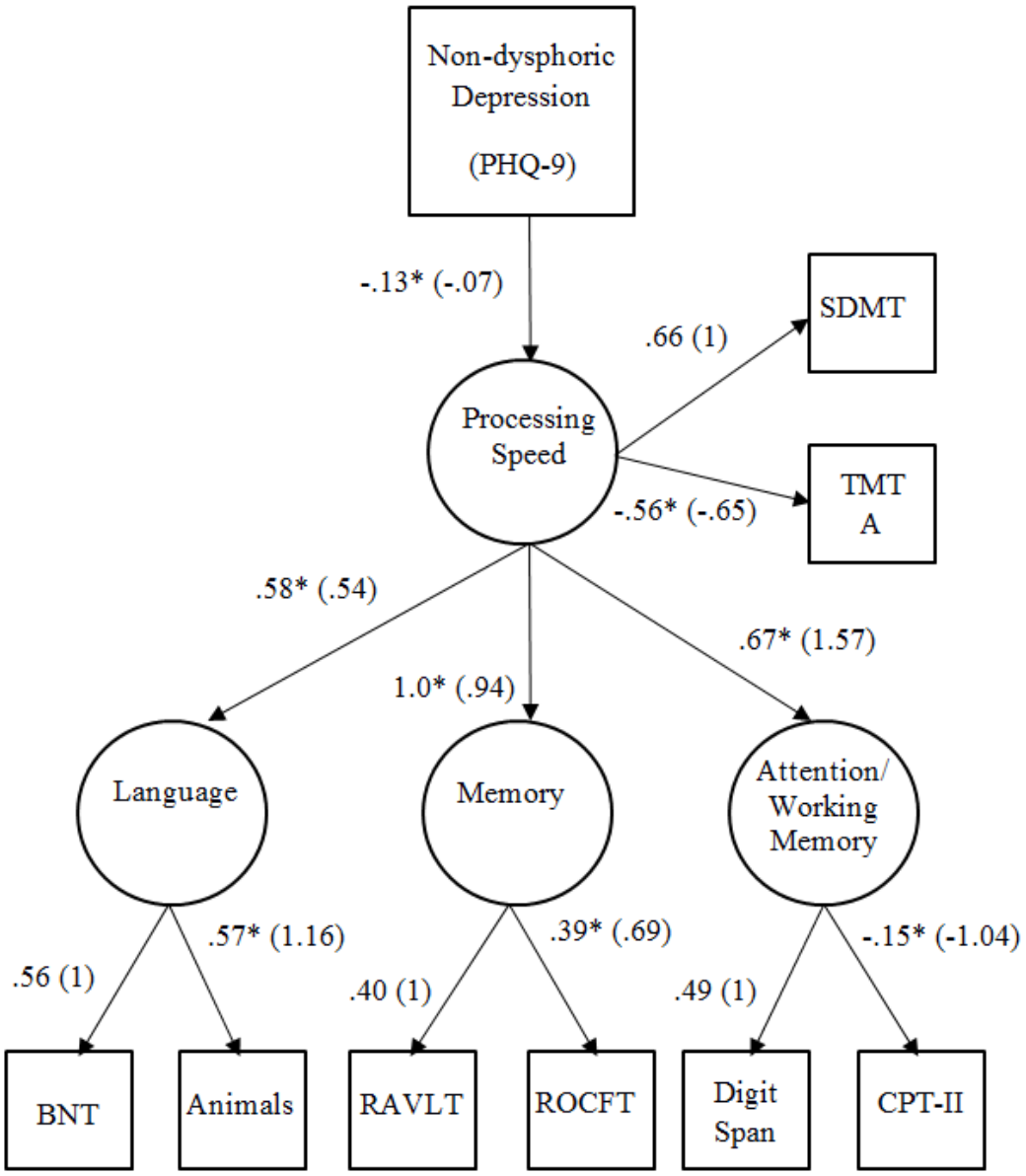

Figure 3. Final path diagram of the structural regression model of the effects of nondysphoric depressive symptoms on cognitive abilities in healthy older adults. Standardized path coefficients are reported with unstandardized coefficients in parentheses. SDMT was used to set the metric for Processing Speed, BNT was used to set the metric for Language, RAVLT was used to set the metric for Memory, and Digit Span was used to set the metric for Attention/Working Memory. An asterisk (*) denotes significance at $\alpha = .05$.

CHAPTER FOUR

DISCUSSION

While the hypothesized models were not an ideal fit for the data in this study, several possible conclusions could be made to inform future research on the relationship between nondysphoric depression and cognitive abilities. First, keeping in mind the lack of fit with the overall model, individual regression coefficients in the final model of this study were similar to those found in the previous studies of Butters et al. (2004) and Sheline et al. (2006). Specifically, the current study found a potential relationship between depressive symptoms and processing speed with a coefficient of ß = -.13, while Butters et al. (2004) found this relationship to have a coefficient of ß = -.16, and Sheline et al. (2006) a coefficient of ß = -.15. Other potential similar relationships between the mediator of processing speed and other cognitive abilities included language, where the current study found a coefficient of ß = .58 compared to Butters et al.'s (2004) ß = .49 and Sheline et al.'s (2006) ß = .53. It may be likely that a better fitting model may substantiate these similarities in relationships and allow for interpretation, which the current study does not allow.

Possible reasons for the lack of fit could be many. Models in the current study were based on previous models of dysphoric depression (Butters et al., 2004; Sheline et al., 2007), but it may simply be that the model does not fit well with nondysphoric depression. A study comparing the two models or the inclusion of dysphoric symptoms into the current sample and model may indicate whether there is a difference between dysphoric and nondysphoric effects of depression. Other possible reasons may be the restricted range from the self-reported PHQ-9 depression scores, considering there were

very few scores above four. While self-reported measures produce an inherent susceptibility of imperfect measurement, those few participants in the current study who reported more nondysphoric symptoms may have not been adequately represented in the overall analysis. Perhaps with a greater range of depressive symptom scores, the model in the current study may have more influence on cognitive function and provide a better fit for the data.

Another possible conclusion of this study is the structure of the relationship between nondysphoric depressive symptoms and cognitive abilities is an important consideration, given the limited results of this study. It was hypothesized that in Model 1 there may be a direct impact of nondysphoric depression symptoms on all cognitive domains of Processing Speed, Language, Memory, Executive Function, and Attention/Working Memory based on a previous study by Butters et al. (2004). However, just as Butters et al. (2004) and Sheline et al. (2006) found, analysis of this direct relationship in the current study appeared to be less accurate and a poorer fit for the data than the Model 2 structural relationship with Processing Speed mediating the relationship of depressive symptoms and performance in other cognitive domains. As suggested by the current study and previous studies of Butters et al. (2004) and Sheline et al. (2006), future researchers examining the impact of depression on cognitive functioning should consider the central role that processing speed may have in the other cognitive functions, particularly in older adults. Clinical implications of this central role of processing speed may suggest further research into underlying biological causes of processing speed deficits related to depression in older adults, such as changes in white matter, which have been suggested to disrupt cognitive and emotional control circuits in LLD (Köhler et al.,

2010). Other implications suggest that cognitive training or rehabilitation for older adults with symptoms of depression may focus on improving processing speed, which may lead to an improvement in functioning of other cognitive domains. Biological changes in the brain due to aging may also suggest another possibility in the relationship of depressive symptoms and cognitive abilities in that these depressive may be the early signs or prodrome of dementia or cognitive impairment (Fiske et al., 2009).

Finally, the exclusion of the executive function domain in the analyses of the current study may simply be a result of poorly fitting data due to sampling effects or the fact that the tests used in the current study to measure executive function did not accurately represent this construct. Executive function has historically been a difficult construct to measure, considering that it is a complex function relying on input from other non-executive processes, such as language, memory, and intellectual function, which may result in measurement errors or "impurities" due to deficits in these non-executive processes (Burgess, 1997). In future studies, it may be beneficial to use different or additional measures of executive function than ones used in the current study. It may also be useful to examine executive function as a mediator between depressive symtoms and cognitive abilities, as suggested in Sheline et al. (2006).

This study had potential strengths in establishing a relationship between nondysphoric depression that include using a large, healthy community population of older adults, and analyzing a structural regression model as opposed to the analysis of separate, individual relationships seen in previous studies (Butters et al., 2004; Sheline et al., 2006). Some limitations exist in the potential ability to generalize the results of this specific sample to the population. First, the strict inclusion and exclusion criteria provide

a fairly controlled population, with limited influences of major physical illnesses, disabilities, and mental disorders that are so often comorbid with and influential on depression. However, these criteria also make it difficult to generalize to the larger population where these comorbid factors exist. Also, this study includes a large Caucasian, highly educated, highly religious, and healthier than average population, which may limit the generalizability of the results of this study as well. Loma Linda, California is a highly religious and extraordinarily health-conscious city, which is considered one of five Blue Zone® communities where residents live longer, healthier lives (Buettner, 2005). This sample includes many residents of this community and surrounding areas that may be dissimilar to other communities throughout the country. Further research in other communities and other healthy populations is needed and would strengthen the potential findings of the current study.

In summary, this study failed to find a good fit of data for the proposed models, but shows potential in finding a relationship between nondysphoric depressive symptoms and cognitive function in healthy older adults. It is also possible that the effect of poorer cognitive function in relation to nondysphoric symptoms could be best explained when mediated by processing speed, as suggested in previous research (Butters et al., 2004; Sheline et al., 2007). Future research is needed to test these relationships using other possible structural regression models discussed in this study, keeping in mind the potential central role of processing speed. Evidence from such studies can provide valuable implications in identification and treatment nondysphoric depression and its effects on processing speed and other cognitive abilities in older adults. This research is particularly important because few studies in the past have examined the effect of

depressive symptoms in the absence of dysphoria, which is a relatively common occurrence in the experience of depression in older adults (Fiske et al., 2009). If depressive symptoms are present in older adults, but unrecognized or undiagnosed due to the lack of dysphoric symptoms, cognitive difficulties could exist that may be improved when the depressive symptoms are treated, leading to improvement of older adults' overall quality of life. One study examining the pharmacological treatment of depressed patients showed that as patients' depressive symptoms improved during treatment, the patients' global cognitive function improved (Mandelli et al., 2006). Other research suggests that alternative or supplemental treatments such as physical exercise may improve symptoms other than the core depression symptoms of dysphoria, such as fatigue, poor appetite, and disturbed sleep (McNeil et al., 1991), which may in turn have a positive effect on cognitive performance and other daily functions of older adults. Overall, with more research on nondysphoric symptoms of depression, particularly in older adults, better efforts could be made to improve the quality of life of those suffering from these symptoms.

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