A NOVEL COGNTIVE STRESS TEST FOR THE DETECTION OF EARLY ALZHEIMER'S DISEASE IN AFRICAN AMERICANS

by

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Statement of Original Work

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ABSTRACT

The U.S. population is currently undergoing a major demographic transition, with increasing racial and ethnic diversity of the older adult population. As the growing population of older adults advances in age, memory complaints are projected to increase in prevalence particularly among African Americans and present a challenge to clinicians who must differentiate between normal aging and progressive neurocognitive conditions (Celsis, 2000; Sherwin, 2000). As targeted therapeutic interventions and emerging therapies for AD are much more likely to be effective in the earlier stages of the disease (Loewenstein, Curiel, Duara & Buschke, 2017), early assessment and detection of AD, especially in groups more likely to develop the disorder, such as African Americans, has become increasingly important. As such, the current study examined the performance of African Americans, both cognitively normal and those with amnestic-mild cognitive impairment (aMCI), on a novel cognitive stress test, the Loewenstein-Acevedo Scale of Semantic Interference and Learning (LASSI-L) and found that those with aMCI exhibit more impairment in their initial learning and storage of information and suffer from proactive semantic interference due to their inability to inhibit responses. Additionally, this study found that the LASSI-L serves as a better predictor of diagnostic group classification compared to traditional neuropsychological measures. Taken together these findings suggest that the LASSI-L is a highly promising test for the assessment of mild

cognitive impairment among African American older adults, which will hopefully guide prevention and treatment planning within this underserved population.

Chapter 1: Introduction

The U.S. population is currently undergoing a major demographic transition, with increasing racial and ethnic diversity of the older adult population. Over the next several decades one in every five Americans will be age 65 or older and by 2050, the proportion of minorities will far outnumber non-Hispanic whites (U.S. Census Bureau, 2017). As the growing population of older adults advances in age, memory complaints are projected to increase in prevalence and present a challenge to clinicians who must differentiate between normal aging and progressive neurocognitive conditions, such as Alzheimer's disease (Celsis, 2000; Sherwin, 2000). Alzheimer's disease (AD), the most prevalent neurocognitive disorder, is highest among African Americans who are 64% more likely to develop AD when compared to Caucasians (Steenland, Goldstein, Levey, & Wharton, 2016). Despite this higher prevalence, AD in African Americans has gone largely understudied. Increased understanding of AD in African Americans, specifically regarding measures that effectively provide early detections can provide important insights regarding the characteristics of observed memory deficits as well as which of characteristics is more predictive of AD brain pathology and further progression to full AD. Given the paucity of research in the area, this dissertation study examines effective early detection of Alzheimer's disease in African Americans.

Alzheimer's Disease

Alzheimer's Disease (AD) is a progressive degenerative disease of the brain, beginning in middle age or later life, which is characterized by progressive cognitive decline and brain pathology (Logie, Parra, & Della Sala, 2015; Saykin and Rabin, 2014). While several neuropathological these changes occur in AD, research has primarily

focused on the presence of the deposition of amyloid-beta (Aβ) peptide (plaques) and intraneuronal fibrils composed of abnormal tau proteins (tangles) (Hyman et al., 2012). The typical presentation of AD includes an insidious onset, memory impairment, and a gradually progressive course evolving to include declines in other cognitive functions as well as personality, emotion, and functional abilities (Saykin & Rabin, 2014). Currently, a diagnosis of Alzheimer's dementia is based on meeting the criteria outlined in the following three classification systems: 1) *Diagnostic Statistical Manual of Mental Disorders*, 5th edition (DSM-5; refer to Appendix A), 2) the *International Classification of Diseases*, 10th edition (ICD-10; refer to Appendix B), or 3) the *National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) of the United States and the Alzheimer's Disease and Related Disorders Association* (ADRDA) (NINCDS-ADRDA; refer to Appendix C).

AD was first described in 1906 by German psychiatrist and neuropathologist, Alois Alzheimer (Zilka & Novak, 2006; Cipriani, Dociotti, Picchi, & Conuccelli, 2011). Dr. Alzheimer, whose initial work largely focused on correlating psychiatric symptoms to pathology of the nervous system, examined a woman by the name of Auguste Deter who was suffering from memory loss, disorientation, depression, and hallucinations (Zilka & Novak, 2006; Cipriani, Dociotti, Picchi, & Conuccelli, 2011). After her death at age 55, Dr. Alzheimer examined Auguste's brain using the newly developed Bielschowsky's silver staining method and described what he saw: "in the center of an otherwise almost normal cell, there stands out one or several fibrils due to their characteristic thickness and peculiar impregnability. Numerous small military foci are found in the superior layers. They are determined by the storage of a peculiar substance in the cerebral cortex. All in

all, we have to face a peculiar disease" (as cited by Cipriani, Dociotti, Picchi, & Conuccelli, 2011, p. 277; Alzheimer, 1907). These observations made by Dr. Alzheimer would be later recognized today as the plaques and tangles characteristic in the brains of patients with the disease. Dr. Alzheimer continued to study patients similar to Auguste and the disease was later termed "Alzheimer's disease" by Dr. Emil Kraepelin, Alzheimer's mentor, in the 8th edition of his Handbook of Psychiatry (Cipriani, Dociotti, Picchi, & Conuccelli, 2011; Kraepelin, 1910).

Research investigating AD has continued in the hundred years since it was first described by Dr. Alzheimer. Today two variants of AD are recognized: sporadic and familial (Schoenberg & Duff, 2011). Sporadic AD, which accounts for over 95% of cases, develops after the age of 65 and follows a slow and insidious course which lasts roughly 10 years (Saykin & Rabin, 2014; Schoenberg & Duff, 2011). Sporadic AD is associated with the APOE gene, of which there are three alleles: epsilon 2, 3, and 4 (Saykin and Rabin, 2014). Because over 60% of AD patients are homozygous for APOE ε4, this allele is considered a risk factor for the development of AD (Saykin & Rabin, 2014). The familial variant of AD occurs before the age of 65 and follows a more rapid progression (Schoenberg & Duff, 2011). The familial variant is associated with mutations in three genes (APP, PSEN1, PSEN2) resulting in autosomal dominant AD by upregulating the production of amyloid beta protein (Saykin & Rabin, 2014). While other genome studies have investigated additional genes associated with AD, none has proven to be useful in predicting the development of AD (Saykin & Rabin, 2014).

The AD Continuum

Historically, AD was synonymous with the later dementing stage of disease, however recent technological advances have allowed us to examine more closely the changes in the brain that occur early in the disease (Sperling et al., 2011). Over the last decade, research has demonstrated that biological changes characteristic of AD (i.e. plaques and tangles) can be detected through cerebral spinal fluid (CSF) and imaging (i.e. MRI and PET amyloid scans) decades prior to the stage of dementia (Sperling, Mormino, & Johnson, 2014). As a result, AD is now conceptualized as a continuum, ranging from individuals at risk for further decline (i.e. evidencing biological correlates of AD) to the later dementing stage of the disease (Dubois et al., 2016). Based on these biological correlates (i.e. plaques and tangles), researchers have determined that AD occurs in three phases: the preclinical stage, mild cognitive impairment (MCI), and dementia (Sperling et al., 2011). Although exact transitional periods are difficult to determine, and likely involve some overlap (Sperling et al., 2011), understanding the different phases of AD is important in that it allows for the diagnostic accuracy of patient presentation as well as the identification of potentially optimal opportunities to employ treatments and emerging therapies. In order to provide clarity, the following sections will review the three stages of AD.

Preclinical AD. The preclinical stage of AD represents a new addition to the AD disease model (Dubois et al., 2016). Individuals identified as being in the preclinical stage are those who evidence biomarkers which are signature of the disease, namely Aβ and tau depositions, but whose cognitive functioning is normal on objective neuropsychological measures (Duara et al., 2011). These neuropathological changes

serve as biomarkers for the identification of the disease and can be identified by laboratory tests such as Positron Emission Tomography (PET) amyloid imaging or by assessing the ratios of Aβ and tau present in cerebrospinal fluid (CSF) (Loewenstein et al., 2012). Other biomarkers used include the identification of medial temporal atrophy on magnetic resonance imaging (MRI), regional hypometabolism on PET scans, abnormal functional MRI activation patterns, and the presence of an Apolipoprotein ε4 genotype (Loewenstein et al., 2012; Sperling et al., 2011).

As previously mentioned, individuals in the preclinical stage of AD evidence normal cognitive functioning on objective neuropsychological measures. Despite this, these individuals often report subjective cognitive decline (SCD) or "perceived decline in memory and/or other cognitive abilities relative to their previous level of performance, in the absence of objective neuropsychological deficits" (Rabin, Smart, & Amariglio, 2017; Jessen et al., 2014). Several studies have demonstrated the association between SCD and the accumulation of Aβ, finding that increased reports of subjective memory concerns are associated with increased Aβ and neuritic plaque burden (Sperling, Mormino, & Johnson, 2014; Rabin, Smart, & Amariglio, 2017; Amariglio et al., 2012; Perrotin et al., 2012; Harten et al., 2013; Kryscio et al., 2014). These findings suggest that SCD may be an indicator of preclinical AD and that individuals with SCD may be at increased risk for future pathological decline (Rabin, Smart, & Amariglio, 2017).

Multiple longitudinal studies have demonstrated high progression rates of preclinical AD to later stages of the disease. These studies have consistently demonstrated that individuals who evidence increased Aβ deposits are more likely to experience accelerated cognitive decline compared to those without (Wirth et al., 2013;

Mormino et al., 2014; Lim et al., 2014; Landau et al., 2012). While individuals with abnormal biomarkers do not always progress to MCI, studies have found that progression rates are highest in those with subjective memory difficulties not significant enough to warrant a diagnosis of MCI (38.9%) and those with Aβ and an additional biomarker such as elevated tau (32.7%) (Vos et al., 2013; Loewenstein et al., 2012). Of note, research on preclinical AD progression rates has largely failed to examine racial disparities, particularly in African Americans. As a result, little is known about how progression rates in African Americans may compare to those of other racial groups. A thorough literature review only identified one study conducted by Chen et al. (2017) examining the progression from normal cognition to mild cognitive impairment (MCI) in a diverse sample. This study investigated progression rates among Whites (*N*= 92), African Americans (*N*=78), and Hispanics (*N*= 84) from both clinic (*N*=13) and community (*N*= 241) samples over a 7-year period. Results from this study found progression rates for clinic samples to be 30% per year, whereas the conversion rate for the community sample was 5% per year (Chen et al., 2017). Hispanics had the highest progression rates with no significant difference observed between the progression rates of Whites and African Americans (Chen et al., 2017). Consistent with previous research, older age and SCD were risk factors for progressing from normal cognition to MCI (Chen et al., 2017). While this study examined racial differences in progression rates from normal cognition to MCI, more research is needed to examine factors that may influence progression rates in racially and ethnically diverse populations.

Mild Cognitive Impairment. Individuals who progress from a normal level of cognition enter an intermediate phase of clinically probable AD. This intermediate phase

has been termed "mild cognitive impairment" (MCI) (Albert et al., 2011; Petersen, 2004) and represents the stage of cognitive impairment seen between those with normal cognition and those with dementia (Petersen et al., 1999). In 2011, the National Institute on Aging- Alzheimer's Association (NIA-AA) developed core clinical criteria for the diagnosis of MCI to be utilized by healthcare providers without access to advanced imaging techniques or cerebrospinal fluid analysis (Albert et al., 2011). According to these criteria a diagnosis of MCI is made when there is a) concern regarding a change in cognition b) impairment in one or more cognitive domains c) preservation of independence in functional abilities and d) no evidence of dementia (Albert et al., 2011).

In order to make a diagnosis of MCI there first should "be evidence of concern about a change in cognition, in comparison with the person's previous level" (Albert et al., 2011, p.271). This concern regarding a change in cognition "can be obtained from the patient, from an informant who knows the patient well, or from a skilled clinician observing the patient" (Albert et al., 2011, p. 271). Once it has been established that there is concern regarding a change in cognition, formal cognitive testing should be conducted to determine if there is impairment in one or more cognitive domains (i.e. memory, executive functioning, attention, language, and visuospatial skills[1]) (Albert et al., 2011). Individuals with memory impairment, more specifically in episodic memory (i.e. the ability to learn and retain new information) most commonly progress from MCI to AD dementia (Albert et al., 2011). Impairment is characterized as "lower performance in one or more cognitive domains that is greater than would be expected for the patient's age

[1] For more information regarding cognitive domains interested readers are referred to *Diagnostic and statistical manual of mental disorders* (5th ed., pp.593-595).

and educational background" which is demonstrated by test scores 1 to 1.5 standard deviations below what would be expected for age and education matched peers (Albert et al., 2011, p. 271). If the patient is tested repeatedly, a decline in performance should be evident over time (Albert et al., 2011).

If it has been determined than an individual has impairment in one or more cognitive domains, their level of independence and functional abilities should be assessed to establish that functioning is not so severely impaired that a diagnosis of dementia is warranted (Albert et al., 2011). Those with MCI should demonstrate a preservation of independence in functional abilities such as preparing meals, paying bills, and shopping (Albert et al., 2011). While patients may demonstrate mild problems completing these tasks (i.e., taking more time to complete a task, being less efficient, making more errors) they should be able to complete these tasks with minimal aids or assistance (Albert et al., 2011). Lastly, it is important to note that those with MCI should not meet criteria for dementia as "these cognitive changes should be sufficiently mild that there is no evidence of a significant impairment in social or occupational functioning" (Albert et al., 2011, p. 272).

Individuals in the MCI phase of AD are considered at risk for further decline to AD dementia (Petersen, 2011). Longitudinal studies have shown that the rate of conversion from MCI to dementia over a three-year period ranges from 20% to 53% (Black, 1999; Mckelvey et al., 1999; Wolf et al., 1998) and 100% conversion to AD dementia is seen during a 9.5-year period (Morris et al., 2001). Progression rates for African Americans are largely understudied as the vast majority of research includes exclusively Caucasian participants or fails to separately report progression rate estimates

by racial group (Gao et al., 2014). However, a recent study investigating MCI progression in African Americans found an annual progression rate of 5.9%, which is comparable to rates found in Caucasian samples (Gao et al., 2014). While progression rates are largely understudied in diverse populations, research suggests that the greatest risk factors for progressing to AD dementia is the presence of memory deficits and multiple AD biomarkers (Vos et al., 2013; Loewenstein et al., 2012).

AD Dementia. During the last stage of AD, individuals progress to a state of dementia which is a "clinical syndrome characterized by a loss of previously acquired cognitive functions that adversely affects an individual's ability to complete day to day activities" (Schoenberg & Duff, 2011, p.357). According to the NIA-AA, the diagnosis of AD dementia is made when there are cognitive or behavioral symptoms that: a) interfere with the ability to function at work or at usual activities; b) represent a decline from previous levels of functioning and performing; and c) are not explained by delirium or major psychiatric disorder (McKhann et al., 2011). Cognitive impairment due to dementia can be "detected and diagnosed through a combination of (1) history taking from the patient and a knowledgeable informant and (2) an objective cognitive assessment, either a "bedside" mental status examination or neuropsychological testing" (McKhann et al., 2011, p. 265). This cognitive impairment involves a minimum of two of the following domains (i.e. memory, reasoning or judgement, visuospatial abilities, language functions, changes in personality or behavior) (McKhann et al., 2011). AD dementia involves an insidious onset with symptoms gradually presenting over months to years (McKhann et al., 2011). As mentioned previously, those with AD dementia evidence cognitive impairment in numerous domains. For those with AD dementia, the

initial and most prominent cognitive deficits follow either an Amnestic or Nonamnestic presentation (McKhann et al., 2011). The Amnestic presentation of AD dementia is the most common and includes impairments in learning and recall of newly learning information (McKhann et al., 2011). While memory is the primary deficit, those with an Amnestic presentation must also evidence deficits in at least one other cognitive domain such as attention, executive functioning, visuospatial functioning, or language (McKhann et al., 2011). Nonamnestic presentations of AD dementia are less common and involve primary deficits in language, visuospatial, or executive functioning[2] as opposed to memory (McKhann et al., 2011).

Prevalence studies indicate that the rate of AD dementia in the U.S. is estimated to be least 4.7 million (Herbert, Weuve, Scherr, & Evans, 2013; Alzheimer's Association, 2017). Furthermore, studies indicate that the risk of developing AD seems to be highest among African Americans who are 64% more likely to develop AD when compared to Caucasian Americans (Steenland, Goldstein, Levey & Wharton, 2016). Several studies have examined the incidence and prevalence of AD dementia by race (Hebert et al., 2010; Tang et al., 2001; Katz et al., 2012; Kukull et al., 2002; Fitzpatrick et al., 2004; Plassmann et al., 2011) including a meta-analysis conducted by Steenland et al. (2016), which found that the estimated AD prevalence rates for those ages 65-90 years to be 5.5% for Whites and 8.6% for African Americans. Similar results were observed in a review conducted by Mehta and Yeo (2017) who examined the prevalence and incidence rates of all types of dementia diagnosis among different racial and ethnic groups and

[2] Expanded information regarding AD deficits in language, visuospatial and executive functioning can be found below in the Neurocognitive Symptoms of AD section.

found higher dementia prevalence rates for African Americans, ranging from 7.2% to 20.9%, with an average annual incidence rate of 2.6%. Taken together, the higher incidence and prevalence of AD dementia rates in African Americans likely reflects a combination of biological, psychological, and socioeconomic factors.

Symptoms of AD

Given that AD is the most prevalent of the dementia syndromes, identifying contributing signs and symptoms of the disease has become increasingly important. Research has identified several neuropathological signs, neuropsychological symptoms, and neurocognitive symptoms characteristic of AD (Schoenberg & Duff, 2011).

Neuropathology of AD. Since AD was first described by Dr. Alois Alzheimer in 1906, research has continued to investigate the neuropathology of the disease (Zilka & Novak, 2006; Cipriani, Dociotti, Picchi, & Conuccelli, 2011). Definitive diagnosis of AD can only be made at autopsy because the brain of an individual with AD does not show any gross anatomical alterations that can be identified diagnostically. Thus, a histological examination must be conducted to observe microscopic evidence of the disease (Perl, 2010). There are three pathognomonic changes which can be detected in the brain of someone with AD: amyloid-beta (Aβ) peptide deposits or "plaques", neurofibrillary "tangles" composed of tau proteins, and brain atrophy (Raskin, Cummings, Hardy, Schuh, & Dean, 2015). Other changes that occur include "synaptic loss, neuronal loss, gliosis, degenerative changes in white matter, granulovacuolar degeneration, cerebral amyloid angiopathy, and other protein aggregates" (Raskin, Cummings, Hardy, Schuh, & Dean, 2015).

Currently, the amyloid hypothesis is the dominant model of AD neuropathology

and is based on the discovery that the amyloid precursor protein (APP) gene on chromosome 21 leads to the development of typical Alzheimer neuropathology secondary to the production of too much Aβ (Selkoe & Hardy, 2016). According to this hypothesis, changes in Aβ metabolism, which may result from genetic mutations, results in a relative increase in Aβ (Raskin, Cummings, Hardy, Schuh, & Dean, 2015). This increase in Aβ results in the formation of plaques, which results in changes in synaptic function and local inflammatory responses (Raskin, Cummings, Hardy, Schuh, & Dean, 2015). This inflammation results in synaptic loss, neuritic dystrophy and over time oxidative stress along with altered neuronal ionic homeostasis and other biochemical changes (Raskin, Cummings, Hardy, Schuh, & Dean, 2015). Following these events, tau protein is hyperphosphorylated leading to intraneuronal neurofibrillary tangles (Raskin, Cummings, Hardy, Schuh, & Dean, 2015). This cascade results in widespread synaptic and neuronal dysfunction, as well as, cell death which then leads to extensive Aβ and tau pathology resulting in progressive dementia (Raskin, Cummings, Hardy, Schuh, & Dean, 2015).

In short, the progression of AD is typically characterized by buildup of amyloid plaques followed by the development of neurofibrillary tangles. In the early stages of AD, early accumulation of abnormal brain amyloid can be detected in several brain areas (e.g., precuneus, posterior cingulate, anterior cingulate and frontal, temporal, parietal cortical regions). These amyloid deposits, which can be indicators of early fibrillary formation in cognitively intact individuals, are detectable 20 years or more before the emergence of any significant neuropsychological deficits (Loewenstein et al., 2017). Neurofibrillary tangles, which emerge later in the disease course, have been found to appear in the pyramidal cells of the neocortex, hippocampus, amygdala, and brainstem

(Zec, 1993). Because AD damage often occurs first in the temporal lobe and associated structures, deficits in memory and higher-order cognitive functioning are typically noticed early on (Salmon & Bondi, 2009; Zec, 1993). As the disease progresses other brain areas are affected (e.g. prefrontal and parietal), with motor and sensory cortical areas usually remaining intact (Perl, 2010).

Neuropsychiatric Symptoms of AD. Neuropsychiatric symptoms (NPS), defined as noncognitive behavioral and psychiatric symptoms including disturbances of mood, perception, and behavior, are also associated with neurodegenerative diseases (Ismail et al., 2016). NPS, which are common in MCI and dementia, have been associated with poorer outcomes, increased caregiver burden, increased functional impairment, higher rates of institutionalization, poorer quality of life, higher burden of neuropathological markers of dementia, and accelerated progression to severe dementia or death (Ismail et al., 2016; Lyketsos et al., 2011; Fischer, Ismail, & Schweizer, 2012; Balestreri, Grossberg, & Grossberg, 2000; Karttunen et al., 2011; Peters et al., 2015; Zubenko et al., 1991). Several studies have identified four different types of NPS: hyperactivity (i.e., aggression, disinhibition, irritability, aberrant motor behavior and euphoria), psychosis (i.e., delusion, hallucination and sleep disorder), affective (i.e., depression and anxiety) and apathy (i.e., apathy and appetite disorder) (Zhao et al., 2016; Aalten et al., 2007; Cheng et al., 2012). Studies investigating the prevalence rates of NPS have offered mixed results, likely due to differences in study settings, population demographics, evaluation methods, and severity of cognitive impairment (Zhao et al., 2016; Fuh, 2006; Mega et al., 1996; Teri et al., 1988). In an effort to produce more precise estimates of NPS prevalence in AD, Zhao et al. (2016) conducted a meta-analysis and found that the most frequently

reported NPS in those with AD was apathy 49%, followed by depression 42%, aggression 40%, anxiety 39%, and sleep disorder 39%. Because NPS commonly occurs in neurodegenerative disease such as AD and other dementias, early recognition and intervention may aid in improving the prognosis of the patient (Zhao et al., 2016).

Neurocognitive Symptoms of AD. Due to pathological changes in the brain, which interrupt neural networks, individuals with AD evidence several cognitive deficits. In fact, deficits in episodic memory, or the ability to learn and retain new information, is considered the clinical hallmark of AD (Weintraub, Wicklund, & Salmon, 2012). Research indicates that deficits in episodic memory stem from an individual's inability to properly consolidate and store new information (Broe et al., 2003). What little information is consolidated is quickly forgotten and there is rarely an improvement over the amount of information an individual can learn across numerous trials (Weintraub, Wicklund, & Salmon, 2012). As a result, on measures of immediate and delayed memory, individuals with AD evidence impaired performance, with delayed memory typically being most impaired (Harciarek & Jodzio, 2005). When given tasks involving recognition memory where individuals are given memory cues, individuals with AD evidence impaired performance often producing both false positive (i.e., endorsing a stimulus as being present when it was not) and false negative errors (i.e., rejecting a stimulus when it was present) (Weintraub, Wicklund, & Salmon, 2012).

In addition to memory impairment, individuals with AD may also present with deficits in other cognitive domains such as language, visuospatial, or executive functioning. In those with deficits in language functioning, the individual experiences difficulty with word-finding, confrontation naming (i.e. the ability to name a viewed

stimulus, verbal comprehension, and semantic verbal fluency (i.e. categories) (McKhann et al., 2011;Harciarek & Jodzio, 2005;Weintraub, Wicklund, & Salmon, 2012; Rascovsky, Salmon, Hansen, Thal, & Galasko, 2007).

The deficits in visuospatial functioning in those with AD include impaired spatial cognition (i.e. knowledge about environment), object agnosia (i.e. inability to recognize objects), impaired face recognition, simultanagnosia (i.e. inability to perceive more than one object at a time), alexia (i.e. inability to read), and constructional apraxia (i.e. inability to build, assemble, or draw objects) (Parasuraman, Greenwood, & Alexander, 2000; Thompson, Stopford, Snowden, & Neary, 2005; McKhann et al., 2011).

Deficits in executive functioning include impaired attention (e.g. divided attention), reasoning, decision making, judgment (e.g. poor understanding of safety risks), and problem solving (e.g. difficulty planning complex or sequential activities) (McKhann et al., 2011; Perry & Hodges, 1999).

Even with these deficit areas, global deficits in AD are not typically manifested until the later stages of the disease when individuals increasingly are affected by agnosia (i.e. inability to interpret sensory information), apraxia (i.e. inability to perform purposeful motor actions), and aphasia (i.e. loss of ability to understand or express speech) (Schoenberg & Scott, 2011).

AD in African Americans

Research investigating knowledge and beliefs about AD between racial groups indicates that African Americans have more racial constrained beliefs about the disease (Dilworth-Anderson, Gibson, & Burket, 2013; Jett, 2006; Mahoney et al., 2005). For example, studies indicate that at the initial stages of memory loss, family members report

difficulty distinguishing memory loss from personality or normal aging (i.e. viewing their older relative as just "slipping") (Dilworth-Anderson, Gibson, & Burket, 2013; Jett, 2006). When family members observe memory loss, it is often attributed to other health conditions (e.g. diabetes, neurosyphilis) or emotional distress (e.g. depression, stress) (Potter, Roberto, Brossoie, & Blieszner) rather than a dementing illness. Moreover, family members often times report being unsure at which point memory loss becomes severe enough to indicate dementia (Potter, Roberto, Brossoie, & Blieszner, 2017; Cahill, Pierce, Werner, Darley, & Bobersky, 2015). Additionally, research shows that African Americans are significantly more likely than Caucasian Americans to perceive memory loss and dementia as a normal part of aging and are thus more likely to accept changes rather than viewing them as problematic (Mahoney, Cloutterbuck, Neary, & Zhan, 2005; Potter et al., 2017). While some studies attribute these "misconceptions" regarding AD symptoms to disparities in education, income, and access to information among African Americans, research controlling for these variables still find these racial constrained beliefs prevalent among African Americans (Dilworth-Anderson & Gibson, 2002; Lee et al., 2012; Connell et al., 2009; Mahoney et al., 2005).

Research demonstrates that in the instances where memory loss is viewed as problematic, affected individuals and their family members are more likely to seek help from other family members, friends, or trusted allies such as the church rather than health care providers due to historic discrimination, intergenerational traumatization and current experiences of discrimination (Mahoney et al., 2005; Dilworth-Anderson, Gibson, & Burket, 2013; Jett, 2006). Mistrust in healthcare providers and the health care system amongst African Americans is often attributed to "the unique combination of racism,

slavery and segregation"[3] which has been exacerbated by historical ethical violations, such as the Tuskegee syphilis experiment (Kennedy, Mathis, & Woods, 2007, p. 57.; Boulware, Cooper, Ratner, LaVeist, & Powe, 2003). In fact, study conducted by Green and colleagues (1997) found that Fifty-two percent of African Americans were aware of the Tuskegee Study and that Twenty-two percent of these individuals reported that because of the study they would be less likely to participate in research themselves. Exacerbating these factors and reinforcing this narrative are continued concerns about interpersonal and technical competence of health care providers, as well as, expectations of racism and experimentation during routine health care (Jacobs, Rolle, Ferrans, Whitaker, & Warnecke, 2006). Unfortunately these expectations of discrimination, are often reinforced by microaggressions (i.e. "brief and commonplace daily verbal, behavioral, or environmental indignities, whether intention or unintentional, that communicate hostile, derogatory, or negative racial slights") (Sue, Capodilupo, Torino, Bucceri, Holder, Nadal, & Esquilin, 2007) such as African Americans having memory concerns dismissed and memory problems attributed to drinking habits (Boulware, et al., 2003; Mahoney et al., 2005). Furthermore, given the institutionalized racism embedded within the United States physicians perceive African Americans more negatively on a number of barriers that affect health care (Van Ryn, & Burke, 200), and African Americans, particularly those who endorse high perceptions of racism and classism, report less satisfaction with health care as well as less treatment adherence (Glover, Sims, & Winters, 2017, Cuffee, Hargraves, Rosal, Briesacher, Schoenthaler, Person,... &

[3] Interested readers are directed to Kennedy and colleagues (2007) review *African Americans and Their Distrust of the Health Care System: Healthcare for Diverse Populations.*

Allison, 2013; Sims, Diez-Roux, Gebreab, Brenner, Dubbert, Wyatt,... & Taylor, 2016; Hausmann, Hannon, Kresevic, Hanusa, Kwoh, & Ibrahim, 2011). Given that majority of African Americans relate their experiences of discrimination to race/ethnicity, and roughly two thirds of graduating physicians are Caucasian, it is not surprising that African Americans report more discrimination and distrust in physicians than any other racial or ethnic group (Banks, Kohn-Wood,& Spencer, 2006; Mickelson &Williams, 1999; Castillo-Page, 2010; Hausmann, et al., 2011; Sims, et al., 2016; Cuffee, et al., 2013; Jacobs, et al., Glover, et al., 2017). As a result of these factors, conducting medical and psychological research within the African American community, including that on AD, faces a number of barriers which unfortunately adversely affect the research body (Hamel, Penner, Albrecht, Heath, Gwede, & Eggly, 2016). This is particularly problematic as the rate of AD in African Americans is higher than that of other group and projected to increase as the baby boomers enter late life (Mehta & Yeo, 2017; Colby & Ortman, 2017).

Risk and Protective Factors. Given that the rate of AD in African Americans is higher than that of other groups, and a projected increase in this population expected over the next few decades, understanding factors that protect or contribute to AD in African Americans has become increasingly important (Colby & Ortman, 2017). Research examining the higher incidence rates of AD in African Americans points to several risk factors, including those in the biological, health, and psychological domains.

Biological Risk Factors. One of the most established biological risk factors for AD is the prevalence of an Apolipoprotein ε4 (APOE ε4) genotype (Schoenberg & Scott, 2011). While rates of this genotype has been found to be higher in African Americans

than in Whites, research has failed to demonstrate a consistent relationship between APOE ε4 prevalence, AD, and cognitive decline in African Americans (Barnes & Bennett, 2014; Logue et al., 2011; Evans et al., 2003; Reitz et al., 2013). A likely reason for the inconsistency among research findings to date rests in the fact that, for research described above, African Americans are generally underrepresented in AD research, most of which involves non-Hispanic Whites (Shin, & Doraiswamy, 2016). Recently, one of the largest genome studies involving African Americans was conducted and confirmed that the APOE ε4 allele, along with the ABCA7 gene, is related to increased risk of Alzheimer's disease among African Americans (Reitz et al., 2013).

Health Risk Factors. A number of health conditions more prevalent in the African American population such as diabetes, hypertension, and obesity have been identified as capable of increasing the risk of developing AD (Barnes & Bennett, 2014; Steenland et al., 2016). These health risk conditions occur more often in African American populations compared to Whites and are likely the result of environmental, biological, and socioeconomic factors (Barnes & Bennett, 2014; Steenland et al., 2016). Specifically, African Americans are at least 50% more likely to have diabetes than Whites (Signorello et al., 2007; Carter & Pugh, 1996; Centers for Disease Control and Prevention, 2003; Mokdad et al., 2003; Harris et al., 1998; Cowie, Harris, Silverman, Johnson, & Rust, 1993; & Harris et al., 1990), 51% more likely to be obese (Center for Disease Control and Prevention, 2009), and 13% more likely to have hypertension then their White peers (Murray et al., 2018; Nwankwo, Yoon, Burt, & Gu, 2013). One explanation for this link may be that dementia in African Americans is most often of mixed pathology, often involving vascular factors which increase risk of further cognitive

decline (Barnes & Bennett, 2014; Steenland et al., 2016). However, more data is needed to investigate the relationship between these conditions and the neuropathology present in AD, particularly in the African American population.

Psychological Risk Factors. While psychological factors have been shown to increase the risk associated with cognitive decline and progression to AD, few studies to date have examined these factors in African Americans. For example, both depression and chronic stress have been linked to higher rates of AD and since African Americans report higher incidence of depression and stress, these psychological factors may play a larger role in AD for this population (Turner, Capuano, Wilson & Barnes, 2015; Machado et al., 2014; Zannas et al., 2015). Social issues such as racial discrimination have also been linked to decreased psychological well-being and higher rates of depression and stress in African Americans, as well as, health care satisfaction and treatment adherence (Hudson, Neighbors, Geronimus, & Jackson, 2015; Glover, Sims, & Winters, 2017; Sims, et al., 2016). As such, more studies are needed to examine psychological and sociological factors that negatively impact African Americans and the extent to which these factors further contribute the higher incidence of AD seen in this population.

Protective Factors. In addition to the aforementioned risk factors, a number of protective factors have been identified within African American communities focused on spirituality, religious involvement and family support. Churches have long been viewed as trusted organizations within African American communities, and as a result, many African American families report relying on their churches for support and as a source of information during times of need (Taylor, Chatters, Woodward, & Brown, 2013;

Mahoney et al., 2005). Similarly, African American families report a preference to rely on trusted and understanding family members and close friends for help rather than seeking outside help, with some individuals' insisting on complete reliance on family due to views of familial responsibility (Potter, et al., 2017).

Studies demonstrate that religious and spiritual involvement along with strong family support serves as a protective lifestyle factor for individuals experiencing cognitive decline (Agli Bailly, & Ferrand, 2014). Religious attendance and social activities (i.e. singing, praying, attending sermons, studying scripture, socializing) have been found to benefit cognitive health by promoting active and engaging lifestyles which require various cognitive exercises (Hill, 2008; Agli et al., 2014; McNamara, 2002). These cognitive exercises strengthen frontal circuits in the brain, train episodic memory, improve introspection and attention which may prevent or delay cognitive decline (Hill, 2008; Agli et al., 2014; McNamara, 2002; Koenig, 2012). Further, religious and social involvement provide outlets for psychological stressors, reduce anxiety, reduce depression, and provide a greater sense of meaning and life purpose (Hill, 2008). Reduced psychological stress protects against elevated blood cortisol levels which may otherwise result in hippocampal atrophy and subsequent memory loss (Hill, 2008; Conrad, 2008; Csernansky et al., 2006; Sapolsky, 2000).

For those diagnosed with AD, personal faith, prayer, church connections, and family support enabled individuals to keep a positive attitude as they came to terms with living with the disease (Agli et al., 2014). Further, individuals who put their lives in the hands of a third party, namely God, reportedly feel more confident and secure, felt relieved from worrying about an uncertain future, and adapted better their diagnosis

(Stuckey, 2003; Beuscher & Grando, 2009). African Americans providing care to a family member diagnosed with AD are also likely to benefit from religious involvement and additional family support. Research has shown that African American caregivers exhibit higher levels of religiosity compared to their Caucasian counterparts as a response to caregiving strains (Dilworth-Anderson, Williams, Gibson, 2002; Wykle & Segall, 1991) and that this religiosity along with additional family support lead to less caregiver burden and stress and more positive appraisals of caregiving (Wilks, Spurlock, Brown, Teegen, & Geiger, 2018; Napoles et al., 2010).

AD Diagnostic Methods

Traditionally, AD is diagnosed during the later stages of the disease when there is evidence of impairment in memory and at least one additional cognitive domain other than memory, which interfere with activities of daily living (Dubois et al., 2007). While corroborating biomarker evidence may indicate brain pathology early in AD, imaging and laboratory assessments are both costly and offer limited diagnostic clarity since known biomarkers have also been found across a broad clinical spectrum including cognitively normal individuals (McKhann et al., 2011). Thus, in order to gain diagnostic clarity, individuals are often referred for a neuropsychological evaluation to assess cognitive functioning. Because deficits in memory, and more specifically episodic memory, are the hallmark feature of AD, the evaluation of memory performance is essential to determine if AD related impairments are present.

Traditional neuropsychological measures used to assess memory disorders were originally developed to identify advanced memory impairments seen in dementia and are based on paradigms that have gone relatively unchanged for over 60 years (Brooks &

Loewenstein, 2010). Several studies have found that these traditional measures lack the sensitivity needed to detect earlier stages of AD, as cognitive changes occurring during this period are more subtle (Rentz et al, 2013; Pettigrew et al., 2015). In fact, research has demonstrated that individuals in the preclinical stage of AD, who evidence abnormal amyloid and tau deposition, score in the normal range on these traditional measures (Rentz et al, 2013; Pettigrew et al., 2015). Because neuropathological changes are present up to 20 years or more before observable deficits are present, those individuals at risk of further decline may go undetected by traditional measures. Efforts to mitigate this issue have examined the sensitivity of composite scores comprised of several traditional measures used together; however, it has been shown that his method is also insensitive to subtle changes in memory (Loewenstein et al., 2017).

In addition to their lack of sensitivity, traditional measures also fail to account for realistic environmental challenges and individual differences (Loewenstein et al., 2017). Specifically, the administration of traditional measures occurs under optimal conditions including a quiet environment and minimized distractions. Unfortunately, this pristine testing environment does not translate well to the demands in the real-world environment in which people are required to use multiple cognitive resources, multitask, and manage a wide array of stimuli simultaneously (Loewenstein et al., 2017). As such individuals in optimal testing environments are not required to utilize as many cognitive resources and therefore typically perform better than they would in the real-world. This is due to the fact that these optimal environments allow individuals to employ cognitive reserve and compensatory strategies that may mask underlying neuropsychological deficits (Stern, 2009).

In regard to individual differences, traditional measures have largely underrepresented minority individuals in their normative samples. Previous normative studies have included only a small number of African American participants with diverse ages and educational levels and have generally not excluded participants with neurologic disease or those who develop dementia after a short follow up (Schneider et al., 2015; Lucas et al., 2005; Dotson, Kitner-Triolo, Evans, & Zonderman, 2008; Holtzer et al., 2008). In addition to insufficient normative data, research has demonstrated racial disparities in testing performance. Research has found that African Americans, along with other minority groups, typically score lower than Whites on traditional measures of verbal and nonverbal abilities despite equivalent education and socioeconomic level which further reduces specificity of cognitive impairment (Schneider et al., 2015; Mayeux et al., 2011; Snitz et al., 2009; Cerhan et al., 1998). While these studies have utilized covariance or matching procedures to equate racial groups based on years of education prior to examining test performance, other studies argue that matching based on years of education likely does not address performance discrepancies between racial groups as the quality of education may not be comparable (Manly et al., 1998; Kaufman et al., 1997; Loewenstein et al., 1994; Whitfield & Baker-Thomas, 1999). Research has demonstrated that African Americans have reading skills significantly below their self-reported education levels (Albert & Teresi, 1999; Baker et al., 1996). This discrepancy is likely due in part to the history of segregation of schools in the United States (Manly et al., 1998). Many older African Americans attended segregated schools which received inferior funding, had lower quality teachers, had higher ratios of students to teachers, and lacked sufficient teaching resources (Hanushek, 1989; Hedges et al., 1994; O'Neill,

1990). In addition, African American children were often employed, which reduced their school attendance during the year (Margo, 1985). To address this issue, researchers have adjusted for quality of education as measured by reading ability rather than years of education and have found that the effect of race on test performance was no longer significant (Manly et al, 2002).

Traditional Memory Paradigms. Traditional memory paradigms are based on the notion that rapid rate of forgetting and impaired delayed recall is one of the most sensitive indicators of AD and can best predict progression to dementia in cognitively normal individuals (Loewenstein et al., 2004; Ashford et al., 1989; Locasio et al., 1995; Troster et al., 1993; Welsh et al., 1991; Masur et al., 1994). More recently, it has been recognized that deficits in initial learning may play a larger role demonstrating that attentional resources and learning strategies may impact memory processes (Greenaway et al., 2006; Schneider, Boyle, Arvanitakis, Bienias & Bennet, 2007; Loewenstein et al., 2017; Loewenstein et al., 2003). One of the most common memory paradigms utilized to assess both traditional and more recent indicators of AD, is list learning, which includes the presentation of stimuli to be remembered over several learning trials. These assessments examine different aspects of memory such as storage and consolidation, immediate and delayed memory, and recognition of target stimuli (Loewenstein, Curiel, Duara, & Buschke, 2017). Neuropsychological assessments based on this traditional memory paradigm include the Rey Auditory Verbal Learning Test (Schmidt, 1996), the Hopkins Verbal Learning Test-Revised (Brandt & Benedict, 2001), the Buschke Selective Reminding Test (Buschke & Fuld, 1974), the California Verbal Learning Test-Second Edition (Delis, Kramer, Kaplan, & Ober, 2000) and the Consortium to Establish a

Registry for Alzheimer's Disease List-Learning Test (Morris et al., 1989). Other commonly used traditional memory paradigms include the examination of immediate and delayed memory for story passages as seen on the Wechsler Memory Scale Fourth Edition (WMS-IV) Logical Memory subtest, paired associate learning as seen on the WMS-IV Verbal Paired Associates subtest, and retention of simple or complex geometric designs as seen on the Brief Visual Memory Test-Revised (BVMT-R) and the Rey Complex Figure Test (RCFT) (Wechsler, 2009; Benedict, 1997; Meyers & Myers, 1995; Loewenstein, Curiel, Duara, & Buschke, 2017).

While traditional memory paradigms rely on passive encoding through the presentation of stimuli to-be-remembered over several learning trials, newer paradigms have employed an active encoding approach. These more active paradigms, termed controlled learning paradigms, avoid the limitations of traditional list learning measures by providing the examinee with a cue that the to-be-remembered information should be organized by, and by doing so, increase the depth of processing and encoding of the information presented (Loewenstein et al., 2017; Buschke, Sliwinski, Kuslansky, & Lipton, 1995; Buschke, Sliwinski, Kuslansky, & Lipton; 1997; Thomson & Tulving, 1970). Not only does controlled learning ensure proper processing and encoding but the cues used may allow individuals to access information during retrieval (Loewenstein, Curiel, Duara, & Buschke, 2018). Studies have shown that individuals with AD are unable to properly use these category cues and thus will still demonstrate impaired performance (Adam et al., 2007; Grober & Buschke, 1987; Grober Buschke, Crystal, Bang, & Dresner, 1988).

In addition, individuals with AD have been found to be susceptible to semantic interference, or the ability to deal with competing stimuli within a semantic category, on a number of measures (Loewenstein et al., 2003; Loewenstein et al., 2004; Ebert & Anderson, 2009; Cushman et al., 1988; Davis et al., 2002). Semantic interference can be further differentiated into proactive and retroactive semantic interference. Proactive semantic interference (PSI) occurs when old semantic learning interferes with the learning of new semantic information (Loewenstein et al., 2017). This is demonstrated by list learning measures in which the learning of a first semantic category (Animals) repeated over multiple trials interferes with the learning of the same semantic category on a second list. For example, if a person is unable to recall a newly presented word to-be-remembered such as "Dog" because they previously learned and remember the word "Cat," proactive interference has occurred. Retroactive semantic interference (RSI) occurs when newly learned semantic information interferes with previously learned semantic information (Loewenstein et al., 2017). RSI is demonstrated by list learning measures when the recall of the first category of semantic stimuli (Animals) is difficult due to interference of the second list of semantic stimuli. For example, if a person is unable to recall the word "Tiger" because more recently they were given the word "Lion" to remember, retroactive interference has occurred.

While traditional memory paradigms have examined PSI and RSI, they have several limitations that reduce their sensitivity to identifying the earliest stages of AD (i.e. preclinical AD and MCI) (Crocco, Curiel, Acevedo, Czaja, & Loewenstein, 2014). While some traditional measures may include competing to-be-remembered lists, controlled learning is not emphasized and there are insufficient numbers of semantically

to-be-remembered stimuli which does not allow for the appropriate examination of PSI and RSI (Loewenstein, et al., 2017). Uncontrolled learning in these paradigms thus does not account for individual attentional resources or learning strategies (Loewenstein et al., 2018). Furthermore, traditional measures lack multiple trials of the second semantically related list which prevents the examination of an individual's ability to recover from proactive semantic interference (Loewenstein et al., 2017). Recovery from PSI is valuable in that it represents strong initial learning and memory.

Taken together PSI, RSI, and recovery from PSI enable us to not only compare an individual to a demographically related normative group, but also to their own initial learning and retrieval abilities (Loewenstein et al., 2017). Thus, novel measures that adequately examine controlled learning, PSI, RSI, and recovery from PSI, may demonstrate enhanced sensitivity to the earlier stages of AD which may also prove valuable for those racial groups not traditionally represented in normative data.

Novel Memory Paradigm. A novel paradigm, the Loewenstein-Acevedo Scales of Semantic Interference and Learning (LASSI-L), was developed to address the aforementioned limitations commonly found in traditional memory paradigms (Curiel et al., 2013). The LASSI-L instructs a person to remember a list of 15 common words that are organized around three semantic categories (i.e. fruits, musical instruments, articles of clothing), with each category consisting of five target words. After reading the list of 15 words, the examinee is asked to recall the words. This free recall is followed by a cued recall in which the person is presented with each category cue and asked to recall the words belonging to that category. The person is then presented with the 15 words from the original list (List A) for a second time and again asked to recall the items belonging to

each category. Then, a second semantically related list (List B) is presented in the same manner in which the first list (List A) was administered. Following the presentation of List B, the person is asked to free recall List B words, assessing for semantic PSI. Free recall of list B is followed by a cued recall. List B words are presented for a second time, followed by a second cued recall trial to assess for recovery from PSI. To assess for RSI, the person is then asked to freely recall the original List A words. This is followed by a cued recall of List A. After a 20-minute delay the person is asked to freely recall words from both Lists A and B.

The LASSI-L demonstrates several strengths over traditional memory paradigms (Loewenstein et al., 2017). First, the LASSI-L explicitly identifies the semantic categories which learning should be organized before target words are presented. This explicit identification decreases the impact that attentional resources and learning strategies may have on memory. Second, the LASSI-L provides a second list of words in which each word is semantically related to a target on the first list. Third, multiple exposures to both List A and List B increase encoding by increasing the depth of initial processing of to-be-remembered information. Lastly, the LASSI-L provides the evaluation of PSI and RSI as well as a unique measure of recovery from PSI.

LASSI-L Clinical Findings. Validation studies of the LASSI-L have demonstrated high test-retest reliability as well as high concurrent and discriminant validity (Loewenstein & Acevedo, 2005; Curiel et al., 2013). Several studies have demonstrated the LASSI-L's ability to differentiate between cognitively normal (CN) individuals and those ranging in severity of impairment (Curiel et al., 2013; Loewenstein et al., 2016). Diagnostic classification studies with the LASSI-L have found that it

demonstrated high levels of sensitivity and specificity with an overall correct classification rate of 90%, which is significantly higher than classification rates obtained by traditional neuropsychological assessment measures (Curiel et al., 2013; Crocco, Curiel, Acevedo, Czaja, & Loewenstein, 2014). Similar results were obtained for a validation study of the LASSI-L among Spaniards (Matias-Guiu et al., 2016). In regard to severity of impairment, Crocco and colleagues (2014) found that amnestic MCI (aMCI) patients evidenced higher PSI and RSI effects than CN individuals. These PSI and RSI effects are due to the LASSI-L's high degree of shared semantic cueing, which elicits significant numbers of semantic intrusions, particularly for impaired individuals. Loewenstein and colleagues (2016) examined the LASSI-L in individuals ranging in degree of cognitive impairment and found that deficits on the LASSI-L were observed in 89% of those with MCI, 47% of those with preclinical MCI, 33% with subjective memory complaints, and 13% of those classified as normal.

The LASSI-L has also been shown to correlate with biomarker evidence and brain structural changes associated with AD. In regard to biomarker evidence, the LASSI-L has been shown to correlate to amyloid depositions (Loewenstein et al., 2017). Specifically, deficits on initial learning of List A on the LASSI-L was found to significantly correlate with amyloid depositions in the anterior cingulate (-.49) and frontal lobes (-.44) (Loewenstein et al., 2017). When looking at different diagnostic groups (i.e. subjective memory complaints, preclinical MCI, and MCI), all evidenced deficits in recovery from PSI which was associated with increased amyloid deposition throughout the entire brain (rs= -.60), precuneus (rs= -.62), posterior cingulate (rs= -.50), and anterior cingulate (rs=

-.48) (Loewenstein et al., 2017). Taken together, these results indicate that the LASSI-L is sensitive to subtle cognitive impairments and increasing amyloid load.

Studies investigating the LASSI-L and its association to volumetric loss in AD prone brain areas has found that preclinical MCI individuals evidenced greater LASSI-L deficits particularly with regards to failure to recover from PSI and delayed recall. These deficits were associated with increased dilation of the inferior lateral ventricle and decreased MRI volumes in the hippocampus, precuneus, superior parietal region, and other AD prone areas (Crocco et al., 2018). Similar results have been observed in individuals with aMCI. Specifically, aMCI patients who demonstrated failure to recover from PSI evidenced reduced volumes in the hippocampus (rs=0.49); precuneus (rs = 0.50); rostral middle frontal lobules (rs = 0.54); inferior temporal lobules (rs = 0.49); superior parietal lobules (rs = 0.47); temporal pole (rs = 0.44); and increased dilatation of the inferior lateral ventricle (rs = −0.49) (Loewenstein et al., 2017). Taken together these results demonstrate that performance on the LASSI-L and more specifically observed frPSI is uniquely and strongly related to volumetric loss in AD prone brain areas.

Clinical Relevance. With the growing number of diverse older adults and rates of AD expected to increase dramatically over the next few decades, a variety of initiatives have pushed for an earlier detection of AD in order to provide better treatment (Albert et al., 2011; Sperling et al., 2011; Dubois et al., 2016). Theoretically, early detection would allow for earlier treatment or interventions with disease modifying therapies before the onset of dementia (Dubois et al, 2016). While no such intervention or therapy currently exists, early interventions may benefit patients by stopping or significantly slowing the progression of AD or by increasing the time spent in the mild stages of the disease

(Dubois et al., 2016). An intervention of this ability would dramatically reduce health care costs. Projections estimate that an intervention that delayed the onset of AD dementia by 5 years would result in a 57% reduction in the number of patients affected which would reduce the costs of Medicare from $627 to $344 billion dollars (Sperling et al., 2011). This delay would also result in prolonged functional independence and greater quality of life for patients and their families. Patients and their families would then be able to better plan and prepare for the future by having the opportunity to make living, care, financial and legal arrangements while they still have preserved insight (Antoine & Pasquier, 2013; Holt, 2011; Mattsson, Brax, & Zetterberg, 2010; Dubois et al., 2016).

Early detection would also allow those in healthcare to better serve patients. Physicians would have the opportunity to offer therapies that address symptoms such as anxiety or impaired sleep while also monitoring prescribed medications that could inadvertently exacerbate dementia (Dubois et al., 2016). With the clear benefits of early detection and promise of novel disease modifying pharmacological interventions on the horizon, it is increasingly important to develop diagnostic tools capable of identifying AD in the earlier stages.

Chapter 2: Purpose and Specific Aims

The purpose of this dissertation study is to extend the body of research on effective early detection of AD in African Americans. While the LASSI-L has demonstrated effectiveness above and beyond traditional measures at differentiating between normal individuals and those ranging in severity of impairment, these studies have largely consisted of White and Hispanic individuals. Therefore, this study will examine the performance of African Americans, both cognitively normal and those with amnestic-mild cognitive impairment, on the LASSI-L. Further, this study will assess if the LASSI-L serves as a better predictor of diagnostic group classification and MRI volumetric reductions in AD prone areas in African Americans compared to traditional neuropsychological measures.

This dissertation was designed to fulfill three specific aims, which, along with the related research questions, are detailed below.

Specific Aim 1: Explore Whether There are Differences in Performance on the LASSI-L Between aMCI and Cognitively Normal African American Older Adults.

Research Question 1a. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures reflecting initial learning and storage of information?

Research Question 1b. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures reflecting

proactive semantic interference (PSI) and failure to recover from proactive semantic interference (frPSI)?

Research Question 1c. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures reflecting retroactive semantic interference (RSI)?

Research Question 1d. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures reflecting delayed recall?

Research Question 1e. After controlling for covariates, are there differences on LASSI-L measures by diagnostic group in African American older adults.

Specific Aim 2: To Determine if Performance on the LASSI-L Serves as a Better Predator of Diagnostic Group Classification Compared to Other Neuropsychological Tests in African American Older Adults.

Research Question 2a. What is the relationship between scores obtained on the LASSI-L and diagnostic group classification in African American Older Adults?

Research Question 2b. What is the relationship between scores on traditional neuropsychological measures and diagnostic group classification in African American Older Adults?

Specific Aim 3: To Explore How Neuropsychological Measures are Related to Volumetric Reductions in AD Prone Regions in African American Older Adults.

Research Question 3a. Are LASSI-L measures of PSI and frPSI related to MRI Volumetric Reductions in left hemisphere AD prone regions?

Research Question 3b. Are Traditional Neuropsychological Measures related to MRI Volumetric Reductions in left hemisphere AD prone regions?

Chapter 3: Methods

Participants and Procedure

This dissertation study examined 44 (28 male, 16 female) independent community dwelling African Americans aged 60-years-old or older (mean age=64.5 years, SD= 4.45), with the vast majority having a high school education (mean=12.4; SD=1.66). Participant data was selected from an NIH-funded study at the University of Miami School of Medicine, which was designed to measure the longitudinal trajectories of decline in PreMCI participants. Participants in this NIH-funded study were recruited from the University of Miami's Center on Aging/CREATE Center as well as the Memory Disorder Clinic. Interested individuals were prescreened for eligibility through an extensive clinical interview, which included the Neuropsychiatric Inventory (NPI), Mini-Mental State Examination (MMSE) (Folstein et al., 1975), and Clinical Dementia Rating Scale (CDR) (Morris, 1993). Participants and their informants signed Informed Consent forms. Eligible participants, who were 60 years of age or older and did not meet DSM-5 criteria for Major Neurocognitive Disorder, active Major Depressive Disorder, active Substance Use disorder in the last 6 months, or any other neuropsychiatric diagnosis, were subsequently administered a standard neuropsychological battery. Measures selected for this study, which are described below, took approximately 45 minutes to complete and included the Hopkins Verbal Learning Test -Revised (Benedict et al., 1998), National Alzheimer's Coordinating Center (NACC) delayed paragraph recall (Beekly et al., 2007), Category Fluency (Lucas et al., 1998), the Block Design subtest from the Wechsler Adult Intelligence Scale, Fourth-Edition (Wechsler, 2008), and Trail Making Test (Parts A and B) (Reitan, 1958). The Loewenstein-Acevedo Scale for

Semantic Interference and Learning (LASSI-L) was also administered but was not used for diagnostic determination. Participants received a stipend of fifty dollars for completing these assessments.

Diagnostic determination was based on the independent clinical interview and performance on the neuropsychological tests. Participants were diagnosed as cognitively normal (CN) if: a) there was no subjective memory complaints by the participant and/or collateral informant; b) no evidence by clinical evaluation or history of memory or other cognitive decline ; c) Global Clinical Dementia Rating Scale of 0; d) the neuropsychological battery was deemed normal and generally no measures in the neuropsychological battery fell 1 standard deviation or more below normal limit relative to age and education normed data. Participants were diagnosed with Amnestic-MCI (aMCI) if: a) there was subjective memory complaint by the participant and/or collateral informant; b) Global Clinical Dementia Rating Scale of 0.5; c) no impairment in social and/or occupational function; d) neuropsychological testing confirmation of memory impairment as evidenced by performance at or below 1.5 standard deviations expected for age and education adjusted normative data on the HVLT-R delayed recall or NACC delayed paragraph recall.

After completing neuropsychological testing, interested and eligible participants also received MRI scans (n=29). Participants signed separate informed consent forms for this portion of the study. MRI scans were performed with the 3T Siemens Trio scanner at the Applebaum Diagnostic Imaging Center, University of Miami Health System, with assistance of the staff MRI technologists. Total imaging time for each participant was estimated to be 45 minutes. Participants received an additional fifty-dollar stipend for

undergoing MRI scans. Psychometrists scoring the cognitive/neuropsychological and functional evaluations as well as those individuals providing MRI analyses were blind to participant diagnosis.

For the purpose of this dissertation all participants self-identifying as African American on a demographic form were selected. Participants were excluded from the present study's dataset if they reported a race or ethnicity other than African American (e.g. Haitian, Cuban, Hispanic)(n=396) and if they did not complete the full neuropsychological battery (n=5).

Measures

The following measures will be described below: Neuropsychiatric Inventory (NPI), Mini-Mental State Examination (MMSE), Clinical Dementia Rating Scale (CDR), Hopkins Verbal Learning Test-Revised (HVLT-R), National Alzheimer's Coordinating Center (NACC) Delayed Paragraph Recall, Category Naming Fluency, Wechsler Adult Intelligence Scale, Fourth-Edition (WAIS-IV) - Block Design subtest, Trail Making Test, and the Loewenstein-Acevedo Scale for Semantic Interference and Learning (LASSI-L).

Neuropsychiatric Inventory (NPI). The Neuropsychiatry Inventory was developed to assess a wide range of behavior problems common in individuals with dementia. Ten distinct behavior domains are assessed: delusions, hallucinations, dysphoria, anxiety, euphoria, agitation/aggression, apathy, irritability/lability, disinhibition, and aberrant motor behavior. Scripted questions are asked to an informant, ideally a daily caregiver, about the individual's behavior in the past month. Each section has screening questions; if the behavior has occurred, more detailed questioning assesses its frequency on a 4-point scale and severity on a 3-point scale. An updated version of

the tests also assesses for sleep and appetite/eating disorders (Cummings, 1997). This revision also introduced a 6-point caregiver distress scale which ranges from 0 (no distress) to 5 (very severe distress), which were added to each domain. The suggested administration time for the original scale was anywhere from 7 to 10 minutes, although that number is dependent on the informant and how much information they provide.

The NPI has produced high interrater reliability and internal consistency (Cummings, Mega, Gray, et al., 1994). Test-retest reliability by a second interviewer within three weeks was generally adequate, with the lowest correlations for irritability/lability. Neuropsychological findings suggest that all behavior problems assessed by the NPI were greater in AD patients compared to age-matched control subjects, of which, the most common was apathy, which was exhibited by 72% of patients. The NPI has been used successfully to differentiate the behavioral symptoms of AD and PD (Aarsland et al., 2001), it has also been used to assess psychiatric symptoms in many subcortical, neurodegenerative disorders (Litvan, Cummings, & Mega, 1998).

Mini-Mental State Examination (MMSE). The MMSE is a cognitive screener widely used for dementia (Milne et al., 2008). The test assesses a restricted set of cognitive functions simply and quickly, as a result the standardized administration only takes about 5 to 10 minutes. A perfect score on the MMSE is 30 points. Points are obtained from several domains including: working memory (serial 7s and spelling "world" backwards); language and praxis (naming, following commands, and construction); orientation; memory (delayed recall of three items); and attention span (immediate recall of three items) (Banos & Franklin, 2002).

MMSE scores decrease with age and increase with education (Tombaugh & McIntyre, 1992). Less educated individuals tend to make errors on the first serial subtraction, spelling backwards, repeating phrases, writing, naming the season, and copying (Jones & Gallo, 2002). Cultural and educational limitations need to be considered as they may lower scores below the cut-off of no cognitive impairment of 24. African Americans and Hispanics are more likely than European Americans to have been erroneously identified as demented (Espino et al., 2001). Test-retest reliability over 24 hours for nondemented psychiatric inpatients was high (r = .89, same examiner; r = .83, different examiner) (Folstein, Folstein, & McHugh, 1975). Four-week test-retest reliability for dementia patients was nearly perfect (r = .99) (McCaffrey, Duff, and Westervelt, 2000). The MMSE is most effective in distinguishing patients with moderate or severe deficits from control subjects (Tombaugh & McIntyre, 1992). It is not as effective at differentiating between mildly demented patients from normal subjects (Knight, 1992).

Clinical Dementia Rating Scale (CDR). The CDR compares AD patients with healthy controls in six categories of cognitive functioning (i.e. memory, orientation, judgement and problem solving, community affairs, home and hobbies, and personal care) (Berg et al., 1988). The instrument is administered via a semi-structured interview to both the participant and an informant (e.g., relative, caregiver). The score is calculated algorithmically and given on a 5-point scale of impairment (0 = no impairment, 0.5 = questionable, 1 = mild, 2 = moderate, 3 = severe) (Morris, 1993). There are several factors that contribute to the utility of the CDR: 1) the six categories used for rating dementia severity are directly linked to validated clinical diagnostic criteria (Morris,

Mckeel, Fulling, Torack, & Berg, 1988); 2) it has high inter-rater reliability for both physicians (Burke et al., 1988) and nonphysicians (McCulla et al., 1989); and 3) an expanded and more quantitative version of the scale can be obtained by summing the ratings in each of the six categories to provide an overall sum score (Berg et al., 1988).

Hopkins Verbal Learning Test -Revised (HVLT-R). The HVLT-R is a list learning task comprised of 12 words, four in each of three semantic categories for three learning trials. Following a 20 to 25-minute delay patients are asked to recall as many words as they are capable of. Immediately after the delayed recall a 24-word yes/no recognition trial is administered containing all 12 target words plus six semantically related words and six unrelated ones. Scores include one for each learning trial, a total acquisition score, a learning measure, delayed free recall, percent retention, and delayed recognition. Recognition scores are calculated for true positives, false positives, a discrimination index, and a measure of the recognition trial response bias.

A test-retest interval of one year for middle-aged adults produced a moderate total recall reliability correlation (r = .49) while delayed recall reliability was significant but lower (r = .36) (Woods et al, 2005). Several variables (i.e. percent retained, learning, intrusions, and repetitions) indicated lower reliability. Validity studies demonstrated the comparability of HVLT-R recall and recognition measures to memory measures form other tests, particularly verbal memory tests (Lacritz et al., 2001; Shapiro et al., 1999). Neuropsychologically, patients with AD exhibit a learning deficit on the HVLT-R (Hogervorst et al., 2002). Further, they are more likely to say “yes” to semantically related foils on the recognition trial (Hogervorst et al., 2002).

National Alzheimer's Coordinating Center (NACC) Delayed Paragraph Recall. NACC delayed paragraph recall requires the subject to recall a story read aloud by the examiner, both immediately and after a 20-minute delay. Scoring allows several acceptable responses for each item recalled. Participants can gain points by paraphrasing, but a verbatim score can also be obtained from allocating a point for each item recalled exactly as delivered in the story. The verbatim score was intended to serve as potentially more sensitive than the paraphrase score in detecting very early memory decline (Craft et al., 2000). The reliability coefficient of the immediate condition for the normative sample by age group ranged from adequate to high (.77-.88) while delayed recall was high (.80-.90) (WMS-IV Technical Manual, 2008). The test-retest reliability of the paragraph recall was adequate for both the immediate and delayed conditions (.70-.79) (WMS-IV Technical Manual, 2008). Delayed paragraph recall is also sensitive to AD. Participants with AD scored significantly lower than matched controls on the delayed condition, with this difference producing a large effect size (2.20).

Category Naming Fluency. In this task, individuals are simply asked to name as many animals, fruits, and vegetables that they can think of, without being given any other cues or restrictions. Individuals with disorders such as those affecting the temporal lobe have demonstrated category deficits. Temporally-based disorders such as Alzheimer's Disease, demonstrate this deficit, which can be attributed to a breakdown in sematic knowledge about different categories. Normative data for the Category Naming test is further stratified by age, sex, and education. Contemporary practitioners favor the use of normative groups established by Heaton in 2004 (Strauss, Sherman, & Spreen, 2006).

Additional normative data has also been created for Spanish speakers living in the United States (Acevedo et al., 2000).

Test-retest correlations tend to be high, usually higher than .70 for semantic fluency with short (e.g., one week) as well as long (e.g., five years) intervals (Basso et al., 1999; Ross, 2003; Levine et al., 2004). Practice effects can be observed after short retest intervals. Wilson et al. (2000) showed that fluency for the same category shows a small but consistent increase across 20 administrations over a span of four weeks. This increase was observed in normal participants as well as those who had sustained head injuries. Validity studies looking at correlations between different semantic category tasks (e.g., animals, vegetables) are moderately high (.66-.71; Riva et al., 1999); however, the values are not satisfactorily high to establish equivalency among forms.

Block Design Subtest from the Wechsler Adult Intelligence Scale, Fourth-Edition. In Block Design, the individual is presented with red and white blocks: two, four, or nine, depending on the item they are working on. Each block has two white sides, two red sides, and two half-red half-white sides with the colors divided along the diagonal. The participant is required to use the blocks to produce replicas of a model design presented by the examiner within a given amount of time. Block Design items are presented in order of increasing difficulty. On the sample item and the first four items, the model design is presented both as a construction made by the examiner and a design pictured in the test stimulus book. For the next ten items, the model design is presented only as a picture in the test booklet. The sample item and items 1 and 2 use two blocks; items 3 to 10 use four blocks; and items 11 through 14 use nine blocks. The WAIS-IV has a "basal" starting level at item 5 for examinees aged 16 to 90. If the examinee does

not obtain a perfect score on either item 5 or 6, the preceding items are administered in reverse order until the examined obtains a perfect score on two consecutive items.

The technical manual reports split-half reliability coefficients for 13 age groups: these coefficients are all at or above .80 (PyschoCorp, 2008b). Test retest reliability of the WAIS-IV Block Design for 298 subjects retested over intervals of eight to 82 days was .80 overall. Test-retest data show a notable improvement from first testing to second testing, suggesting a significant practice effect. Neuropsychologically, Block Design is generally recognized as the best Weschler scale measure of visuospatial organization. Scores tend to be lower in the presence of any kind of brain impairment, indicating that test performance is affected by multiple factors. Specifically, for patients with AD, Block Design scores correctly classified 91% of AD patients (Weintraub, Wicklund, & Salmon, 2012). Block Design has also proven to be a useful predictor of the disease as a relatively low Block Design score in the early stages, when the diagnosis is still in question, may herald the onset of the disease (Arnaiz et al., 2001). The test is also one of the most useful neuropsychological tests for predicting which patients will deteriorate the most rapidly (Small et al., 1997).

Trail Making Test (Parts A and B). The Trail Making Test measures cognitive flexibility, sequencing ability, and visual-motor speed. The Trail Making Test (parts A and B) are a subtest from the Army Individual Test (1944) used as measures of attention, scanning, visual-motor tracking, divided attention, and set-shifting abilities. Trails A is a measure of visual scanning and motor speed. In Trails A, the participant is given a page with a set of numbered circles scatters about the page and is asked to draw a line between consecutive numbers. Trails B is a more specific measure of executive functioning as it

requires reasoning ability other higher-order processes (Golden, Espe-Pfeifer, & Wachsler-Feider, 2000; Kortte, Horner, & Windham, 2002). In Trails B, the participant is given a sheet with randomly distributed circled numbers and circled letters and asked to draw a line connecting A-1, B-2, C-3, and so forth in a sequencing pattern. Scores are based on total time to complete task, and the number of errors made. Cut-off scores were used in the original interpretation of the test (Reitan & Wolfson, 1985), but contemporary practitioners favor the sensitive of the use of *t*-scores based normative groups established by Heaton in 2004 (Strauss, Sherman, & Spreen, 2006). Test-retest reliability has been shown to vary depending on age and population studied. A study looking at 384 normal adults aged between 15 and 83 years who were retested about 11 months after the initial test session showed adequate reliability for Part A (.79) and high for Part B (.89). Similar findings were reported by Levine et al. (2004) for mostly Caucasian, well-educated male subjects (.70 for A and B). Mitrushina and Satz (1991) examined test-retest reliability in older adults after a 1-year period and found coefficients that were low for part A (.53-.64) and higher for part B (.67-.72). Interrater reliability has been reported as .94 for Part A and .90 for Part B (Fals-Stewart, 1991). As for sensitivity, the Trail Making Test is sensitive to dementing disorders such as AD (Chen et al., 2000); however, the task does not distinguish adequately among dementing disorders (Barr et al., 1992).

Loewenstein-Acevedo Scale for Semantic Interference and Learning (LASSI-L). The LASSI-L instructs a person to remember a list of 15 common words that are fruits, musical instruments or articles of clothing (five words per category). The person is asked to read the words for the target list out loud as each is presented individually at 4-second intervals. In the unlikely event that the person cannot correctly read the word, the

word is read by the examiner and the person is asked to repeat the word. After the person has read all 15 words, they are asked to recall the words. After free recall has ended, they are presented with each category cue (e.g., clothing) and asked to recall the words that belonged to that category. Participants are then presented with target stimuli for a second learning trail with subsequent cued recall to strengthen the acquisition and recall of the List A targets. The exposure to the semantically related list (i.e., List B) is then conducted in the same manner as exposure to List A. List B consists of 15 words different from List A, 5 of which belong to each of the three categories used in List A (i.e., fruits, musical instruments, articles of clothing).

Following the presentation of the List B words, the person is asked to free recall the List B words, assessing proactive interference effects. Then, each category cue is given, and they are asked to recall each of the List B words that belonged to each of the categories. List B words are presented again, followed by a second category-cued recall trial. Finally, to assess retroactive interference they are asked to free recall the original list A words. Free-recall and cued recall scores for List A and List B targets are then obtained after a 30-minute delay. Primary measures for this project are the second cued recall score, and first cued recall score for list B. Test-retest reliabilities were high and the accuracy of classification of aMCI patients versus elderly subjects exceeded 90% (see Crocco et al., 2013; Curiel et al., 2013).

Chapter 4: Results

The main objective of this study was to examine the performance of African Americans, both cognitively normal and those with amnestic-mild cognitive impairment, on a novel cognitive stress test, the Loewenstein-Acevedo Scale of Semantic Interference and Learning (LASSI-L) Each of the study's three specific aims and their associated results are presented below:

Specific Aim 1: Explore Whether There are Differences in Performance on the LASSI-L Between aMCI and Cognitively Normal African American Older Adults.

Assumption analyses included boxplots of all dependent variables to assess for outliers, and interpretation of the Shapiro-Wilk's test to determine normality (as $n < 50$). There were no outliers in the data, as assessed by inspection of boxplots. Data was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). For each dependent variable there was homogeneity of variances, as assessed by Levene's test for equality of variances with the exception of LASSI-L List A Free Recall 1 Intrusions, p= .001; LASSI-L List A Cued Recall 1 Intrusions, p= .000; LASSI-L List A Cued Recall 2 Intrusions, p=.000; LASSI-L List B Cued Recall 1 intrusions, p= .021. For the instances in which Levene's test for equality of variance was violated, a series of non-parametric Mann Whitney U tests of ranks was performed. Because results were the same for parametric and nonparametric analysis they were still interpreted.

Research Question 1a. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures regarding initial learning and storage of information?

A one-way analysis of variance (ANOVA) was conducted to evaluate the research question of whether there were statistically significant differences in African American older adult cognitive status when examined in terms of LASSI-L measures regarding initial learning and storage of information. A criterion for significance of $p < .05$ was used. When examining initial learning and storage of information, cognitively normal (CN) individuals were found to recall significantly more words than those with aMCI (Table 4-1). For instance, on the LASSI-L List A Free Recall 1, CN individuals recalled significantly more words (M= 8.37) compared to aMCI individuals (M= 6.25), $F(1,42)$= 8.36, p= .006. Similar results were seen on the LASSI-L List A Cued Recall 1 where CN individuals recalled significantly more words (M=9.79) compared to aMCI individuals (M=7.70), $F(1,42)$= 8.34, p= . 006) and on the LASSI-L List A Cued Recall 2, where CN individuals recalled significantly more words (M=12.70) compared to aMCI individuals (M=10.25), $F(1,42)$= 25.94, p= .000. Furthermore, on the LASSI-L List A Cued Recall 1, CN individuals made significantly fewer intrusions (M=0.25) compared to aMCI individuals (M=1.05) $F(1,42)$= 8.27, p=.006. Similar results were seen on the LASSI-L List A Cued Recall 2, where CN individuals made significantly fewer intrusions (M=0.12) compared to aMCI individuals (M=.95) $F(1,42)$=11.309, p= .002. The number of intrusions made during the initial free recall on the LASSI-L List A Free Recall 1 did not significantly differ between CN (M=.17) and aMCI (M=.45) individuals $F(1,42)$=8.27, p=.09.

Table 4-1. ANOVA Summary Table for LASSI-L Initial Learning and Storage

	SS	*df*	*MS*	*F*	*p*
LASSI-L List A Free Recall 1					
Between Groups	49.261	1	49.261	8.364	.006*
Within Groups	247.375	42	5.890		
Total	296.636	43			
LASSI-L List A Cued Recall 1					
Between Groups	47.728	1	47.728	8.347	.006*
Within Groups	240.158	42	5.718		
Total	287.886	43			
LASSI-L List A Cued Recall 2					
Between Groups	65.928	1	65.928	25.949	.000*
Within Groups	106.708	42	2.541		
Total	172.636	43			
LASSI-L List A Free Recall 1 Intrusions					
Between Groups	.876	1	.876	2.994	.091
Within Groups	12.283	42	.292		
Total	13.159	43			
LASSI-L List A Cued Recall 1 Intrusions					
Between Groups	6.982	1	6.982	8.272	.006*
Within Groups	35.450	42	.844		
Total	42.432	43			
LASSI-L List A Cued Recall 2 Intrusions					
Between Groups	7.425	1	7.425	11.309	.002*
Within Groups	27.575	42	.657		
Total	35.000	43			

Table Notes. *$p<.05$

Research Question 1b. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures reflecting proactive semantic interference (PSI) and failure to recover from proactive semantic interference (frPSI)?

A one-way analysis of variance was conducted to evaluate the research question of whether there were statistically significant differences in African American older adult cognitive status when examined in terms of LASSI-L measures reflecting proactive semantic interference (PSI) and failure to recover from proactive semantic interference (frPSI). A criterion for significance of $p < .05$ was used. On LASSI-L measures reflecting PSI, (Table 4-2), results demonstrated higher scores for those who were CN compared to those with aMCI. For instance, on the LASSI-L List B Free Recall those who were CN freely recalled more words (M= 6.45*)* than those with aMCI (M= 4.85). This difference was statistically significant, $F(1,42) = 5.85$, p= .020. On LASSI-L List B Cued Recall 1 those who were CN recalled more words when cued (M= 7.16) than those with aMCI (M= 5.00). This difference was statistically significant, $F(1,42)$= 8.424, p= .006.Intrusions on LASSI-L List B Cued Recall 1 were also statistically significant $F(1,42) = 25.78$, p= .000 with CN individuals making fewer intrusions (M= 3.24) than those with aMCI (M= 7.07) $F(1,42) = 25.78$, p= .000. The number of intrusions made during free recall on the LASSI-L List B Free Recall did not significantly differ between CN (M=1.67) and aMCI (M=2.20) individuals $F(1,42)$=.85, p= .36.

Similar results were observed for the LASSI-L measure reflecting frPSI, LASSI-L List B Cued Recall 2, those were cognitively normal recalled more words when cued (M=

10.79) than those with aMCI (*M*= 8.25). This difference was statistically significant *F*(1,42)= 13.472, *p*= .001. The number of intrusions made during List B Cued Recall 2 did not significantly differ between CN (*M*=2.5) and aMCI (*M*=3.65) individuals *F*(1,42)=3.64, *p*= .06.

Table 4-2. ANOVA Summary Table for LASSI-L PSI and frPSI measures

	SS	*df*	*MS*	*F*	*p*
LASSI-L List B Free Recall 1					
Between Groups	28.219	1	28.219	5.853	.020*
Within Groups	202.508	42	4.822		
Total	230.727	43			
LASSI-L List B Cued Recall 1					
Between Groups	51.212	1	51.212	8.424	.006*
Within Groups	255.333	42	6.079		
Total	306.545	43			
LASSI-L List B Cued Recall 2					
Between Groups	70.473	1	70.473	13.472	.001*
Within Groups	219.708	42	5.231		
Total	290.182	43			
LASSI-L List B Free Recall 1 Intrusions					
Between Groups	3.103	1	3.103	.854	.361
Within Groups	152.533	42	3.632		
Total	155.636	43			
LASSI-L List B Cued Recall 1 Intrusions					
Between Groups	119.401	1	119.401	25.782	.000*
Within Groups	194.508	42	4.631		
Total	313.909	43			
LASSI-L List B Cued Recall 2 Intrusions					
Between Groups	14.427	1	23.201	3.965	.063
Within Groups	166.550	42	7.303		
Total	180.977	43			

Table Notes. *$p<.05$

Research Question 1c. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures reflecting retroactive semantic interference (RSI)?

A one-way analysis of variance was conducted to evaluate the research question of whether there were statistically significant differences in African American older adult cognitive status when examined in terms of LASSI-L measures reflecting retroactive semantic interference (RSI). A criterion for significance of $p < .05$ was used. On LASSI-L measures reflecting retroactive interference (Table 4-3.), results demonstrated higher scores for CN individuals compared to those with aMCI. For instance, on the LASSI-L List A Free Recall 2 short delay those who were CN freely recalled more words (M=6.45) than those with aMCI (M=4.38), $F(1,42) = 4.65$, p= .037. The number of intrusions made during LASSI-L List A Free Recall 2 short delay did not significantly differ between CN (M=2.29) and aMCI (M=3.75) individuals $F(1,42)=3.18$, p= .08. The number of words recalled during LASSI-L List A Cued Recall 1 after delay did not significantly differ between CN (M=7.46) and aMCI (M=6.50) individuals $F(1,42)=1.36$, p= .25. Similarly, the number of intrusions made during LASSI-L List A Cued Recall 1 after delay did not significantly differ between CN (M=3.63) and aMCI (M=4.7) individuals $F(1,42)=1.31$, p= .26.

Table 4-3. ANOVA Summary Table for LASSI-L RSI measures

	SS	*df*	*MS*	*F*	*p*
LASSI-L List A Free Recall 2 Short Delay					
Between Groups	28.219	1	28.219	4.652	.037*
Within Groups	252.758	42	6.066		
Total	282.977	43			
LASSI-L List A Cued Recall 1 After Delay					
Between Groups	10.019	1	10.09	1.362	.250
Within Groups	308.958	42	7.356		
Total	318.977	43			
LASSI-L List A Free Recall 2 Short Delay Intrusions					
Between Groups	23.201	1	23.201	3.177	.082
Within Groups	306.708	42	7.303		
Total	329.909	43			
LASSI-L List A Cued Recall 1 After Delay Intrusions					
Between Groups	12.607	1	12.607	1.305	.260
Within Groups	405.825	42	9.662		
Total	418.432	43			

Table Notes. *$p<.05$

Research Question 1d. When cognitive status is grouped by amnestic-MCI and cognitively normal are there statistically significant differences in African American older adults cognitive status when examined in terms of LASSI-L measures regarding delayed recall?

A one-way analysis of variance was conducted to evaluate the research question of whether there were statistically significant differences in African American older adult cognitive status when examined in terms of LASSI-L delayed recall (Table 4-4). A criterion for significance of $p < .05$ was used. On the LASSI-L Delayed Free Recall, CN individuals freely recalled more words (M=17.75) than those with aMCI (M=9.70), $F(1,42)= 21.53$, $p= .000$. The number of intrusions made on LASSI-L Delayed Recall did not significantly differ between CN (M=17.75) and aMCI (M=9.70) individuals $F(1,42)=.77$, $p=.38$.

Table 4-4. ANOVA Summary Table for LASSI-L Delayed Recall

	SS	*df*	*MS*	*F*	*p*
LASSI-L Delayed Free Recall					
Between Groups	706.936	1	706.936	21.536	.000*
Within Groups	1378.700	42	32.826		
Total	2085.636	43			
LASSI-L Delayed Free Recall Intrusions					
Between Groups	5.603	1	5.603	.774	.384
Within Groups	304.033	42	7.239		
Total	309.636	43			

Table Notes. *$p<.05$

Research Question 1e. After controlling for covariates, are there differences on LASSI-L measures by diagnostic group in African American older adults?

An adjusted analysis of covariance was conducted to control for overall impairment and literacy level and to determine if after controlling for these covariates, if there were differences on LASSI-L measures by diagnostic group in African American older adults. A criterion for significance of $p < .05$ was used. After controlling for covariates (Table 4-5), there was only a significant difference on LASSI-L List A Cued Recall 2 F (1,37) = 11.24, p = .002 and LASSI-L List B Cued Recall 1 Intrusions F(1,37)= 35.70, p =.000 by diagnostic group indicating more impairment for aMCI on these LASSI-L measures compared to the cognitively normal group.

Table 4-5. Test of Between Subjects Effects (ANCOVA)

	Type III SS	*df*	*MS*	*F*	*p*
LASSI-L List B Cued Recall 2 Intrusions					
Corrected Model	18.682	3	6.227	1.487	.234
Intercept	17.133	1	17.133	4.092	.050
WRAT	.424	1	.424	.101	.752
MMSE	7.044	1	7.044	1.682	.203
AA Group	4.869	1	4.869	1.163	.288
Error	154.928	37	4.187		
Total	519.000	41			
Corrected Total	173.610	40			
LASSI-L List B Cued Recall 1 Intrusions					
Corrected Model	153.704	3	51.235	13.281	.000
Intercept	.856	1	.856	.222	.640
WRAT	3.375	1	3.375	.875	.356
MMSE	.111	1	.111	.029	.866
AA Group	137.721	1	137.721	35.700	.000*
Error	142.735	37	3.858		
Total	890.000	41			
Corrected Total	296.439	40			
Intercept					
LASSI-L List A Cued Recall 2					
Corrected Model	64.801	3	21.600	8.306	.000
Intercept	7.901	1	7.901	3.038	.090
WRAT	1.225	1	1.225	.471	.497
MMSE	12.835	1	12.835	4.935	.033
AA Group	29.237	1	29.237	11.242	.002*
Error	96.224	37	2.601		
Total	5851.000	41			
Corrected Total	161.024	40			

Table Notes. *$p<.05$

Specific Aim 2: To Determine if Performance on the LASSI-L Serves as a Better Predator of Diagnostic Group Classification Compared to Other Neuropsychological Tests in African American Older Adults

Before conducting the step-wise logistic regression included in specific aim 2, the sample was evaluated to verify that all of the assumptions of logistic regression (i.e. binary dependent variable, independent observations, multicollinearity, linearity of independent variables to log odds, adequate sample size) were satisfied and all assumptions were met.

Research Question 2a. What is the relationship between scores obtained on the LASSI-L and diagnostic group classification in African American Older Adults?

A step-wise logistic regression was conducted to evaluate the relationship between scores obtained on the LASSI-L and diagnostic group classification in African American older adults. A criterion for significance of $p < .05$ was used. Results (Table 4-6) demonstrated that both the LASSI-L List B Cued Recall 1 intrusions, $b = 1.101$, Wald $\chi^2(1) = 8.04$, $p = .005$, and the LASSI-L Delayed Free Recall $b = -.417$, Wald $\chi^2(1) = 8.105$, $p = .004$ significantly predicted whether an individual would be diagnosed as CN or aMCI. A combined overall sensitivity of 91.7%, a specificity of 85.0%, and an overall classification rate of 88.6% were obtained in distinguishing between individuals who were CN and those with aMCI. These high classification rates were obtained despite the fact that the LASSI-L was the only neuropsychological measure that was not employed as part of the initial diagnostic procedure.

Areas under the receiver operating characteristic curve (ROC) curve on different LASSI-L variables were also examined (Figure 4-1). Results (Table 4-7) showed that the

highest area under the curve was obtained for Cued B1 intrusions with an AUC= .870 (SE=.054); p<.001. A cut off of 3 by Youden's criteria yielded a sensitivity of 83.3% and a specificity of 77.8%. List B Free Recall intrusions has an AUC of .591 (SE)= (.092) which did not reach statistical significance (p=.038).

Research Question 2b. What is the relationship between scores on traditional neuropsychological measures and diagnostic group classification in African American Older Adults?

Since neuropsychological measures such as the HVLT-R, Category Fluency and Trails B were part of the initial diagnostic neuropsychological battery, which was combined with the clinical diagnosis to assign participants to diagnostic groups, using these same measures, particularly the HVLT-R to predict diagnostic group would result in potential tautological or circular reasoning. Nonetheless, a step-wise logistic regression was conducted to evaluate the relationship between scores obtained on traditional neuropsychological measures and diagnostic group classification in African American older adults. A criterion for significance of $p < .05$ was used. Results found that even when entering variables such as HVLT, Category Fluency, or Trails B, these variables did not significantly improve the model. Moreover, while HVLT delayed recall played a large role in determining clinical diagnosis and was individually associated with group membership, this measure did not surpass individual LASSI-L predictors.

Table 4-6. Logistic Regression Predicting Diagnostic Group Based on LASSI-L Measures

	B	SE	Wald	*df*	*p*	Odds Ratio
LASSI-L List B Cued Recall 1 Intrusions	1.101	.388	8.044	1	.005*	3.007
LASSI-L Delayed Free Recall	-.417	.147	8.105	1	.004*	.659
Constant	.496	1.519	.107	1	.744	1.642

Table Notes. *$p<.05$

Table 4-7. Area Under the ROC Curve

	Area	Std. Error[a]	Asymptomatic Sig[b]	Asymptomatic 95% Confidence Interval	
				Lower Bound	Upper Bound
LASSI-L List B Free Recall Intrusions	.591	.092	.308	.411	.770
LASSI-L List B Cued Recall 1	.870	.054	.000*	.764	.977

Table Notes. The test result variable(s): LASSI-L List B Free Recall Intrusions, LASSI-L List B Cued Recall 1 has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased

a. Under the nonparametric assumption
b. Null hypothesis: true area = 0.5

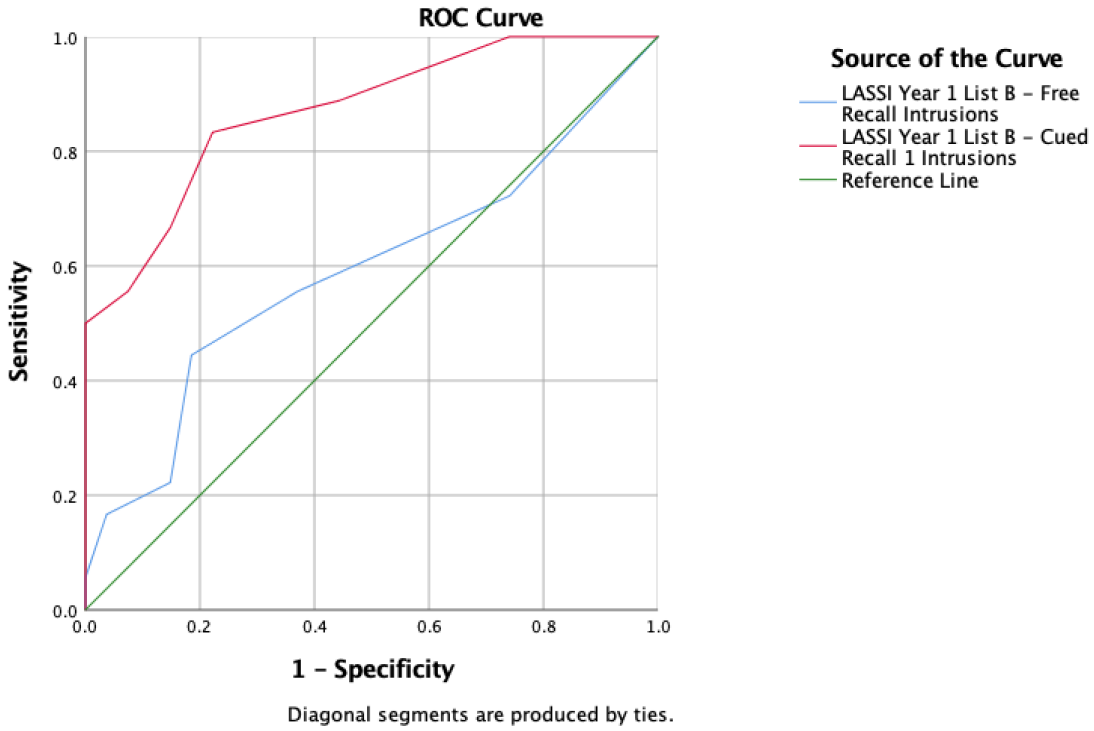

Figure 4-1. Area Under the ROC Curve for LASSI-L variables

Specific Aim 3: To Explore How Neuropsychological Measures are Related to Volumetric Reductions in AD Prone Regions in African American Older Adults?

Before conducting the series of Spearman's rank-order correlation assumption analyses including examination of scatterplots for monotonic relationships between variables were conducted and all assumptions were met.

Research Question 3a. Are LASSI-L measures of PSI and frPSI related to MRI Volumetric Reductions in left hemisphere AD prone regions?

A series of Spearman's rank-order correlations were run to examine if LASSI-L measures were related to MRI Volumetric Reductions in left hemisphere hippocampus, entorhinal cortex, precuneus, temporal lobe (i.e. superior, middle, inferior), parietal (i.e. superior, inferior), and frontal (i.e. superior, rostral orbital). A criterion for significance of $p < .05$ was used. Preliminary results (Table 4-8) demonstrated statistically significant correlations between the left superior frontal region and LASSI-L B1 Cued Recall Intrusions $r_s(27)=-.38$, $p \leq .05$; left rostral orbital frontal and LASSI-L B1 Cued Recall Intrusions $r_s(27)=-.40$, $p \leq .05$; left rostral orbital frontal and LASSI-L B2 Cued Recall Intrusions $r_s(27)=-.44$, $p \leq .05$. However, after the Benjamini and Hochberg (1995) correction on the Spearman Rank Order Correlation Coefficients for each LASSI-L measure (correcting for 10 MRI measures), there were no statistically significant results.

Table 8

Table 4-8. Relationship between LASSI and Left Hemisphere Volumes in Alzheimer's Prone Regions (*N*=29)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14
LASSI-L Cued B1 Recall	—													
LASSI-L Cued B2 Recall	.00**	—												
LASSI-L Cued B1 Intrusions	.098	.06	—											
LASSI-L Cued B2 Intrusions	.023*	.00**	.0**	—										
Left Hippocampus	.32	.82	.19	.80	—									
Left Entorhinal Cortex	.61	.31	.75	.70	.081	—								
Left Precuneus	.96	.67	.96	.61	.043*	.61	—							
Left Superior Temporal	.95	.56	.47	.39	.041*	.28	.06	—						
Left Middle Temporal	.59	.97	.38	.92	.00**	.64	.00**	.00**	—					
Left Inferior Temporal	.64	.76	.10	.28	.047*	.03*	.03*	.045	.00**	—				
Left Superior Parietal	.68	.73	.25	.51	.00**	.15	.00**	.089	.00**	.00**	—			
Left Inferior Parietal	.23	.89	.62	.87	.10	.28	.00**	.00**	.00**	.01**	.02*	—		
Left Superior Frontal	.80	.96	.04*	.54	.001	.02*	.00**	.017*	.00**	.00**	.00**	.00**	—	
Left Rostral Orbital Frontal	.29	.30	.03*	.02*	.02	.42	.019*	.021*	.26	.047*	.00**	.022*	.00**	—

Table Notes. Due to the violation of normality assumptions, non-parametric Spearman Rank Order Correlation Coefficients were performed with two-tailed significance

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

Research Question 3b. Are Traditional Neuropsychological Measures related to MRI Volumetric Reductions in left hemisphere AD prone regions?

A series of Spearman's rank-order correlations were run to examine if traditional neuropsychological measures (i.e. HVLT Total, HVLT delay, Trails B, category fluency) were related to MRI Volumetric Reductions in left hemisphere hippocampus, entorhinal cortex, precuneus, temporal lobe (i.e. superior, middle, inferior), parietal (i.e. superior, inferior), and frontal (i.e. superior, rostral orbital). A criterion for significance of $p < .05$ was used. A criterion for significance of $p < .05$ was used. The Benjamini and Hochberg (1995) correction was employed on the Spearman Rank Order Correlation Coefficients for each traditional measure (correcting for 10 MRI measures), which yielded no statistically significant results (Table 4-9).

Table 9

Table 4-9. Relationship between Traditional Measures and Left Hemisphere Volumes in Alzheimer's Prone Regions (*N*=29)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14
HVLT-R Total	—													
HVLT-R Delay	.00**	—												
Trails B Total	.00**	.00**	—											
Category Fluency	.01**	.028*	.00**	—										
Left Hippocampus	.07	.24	.34	.36	—									
Left Entorhinal Cortex	.17	.41	.45	.40	.082	—								
Left Precuneus	.43	.24	.39	.15	.008**	.03*	—							
Left Superior Temporal	.19	.09	.41	.39	.009**	.11	.03*	—						
Left Middle Temporal	.44	.12	.42	.09	.00**	.44	.00**	.00**	—					
Left Inferior Temporal	.17	.08	.07	.19	.00**	.06	.012*	.059	.00**	—				
Left Superior Parietal	.36	.36	.31	.45	.00**	.11	.00**	.018*	.00**	.00**	—			
Left Inferior Parietal	.41	.41	.45	.18	.012*	.11	.00**	.00**	.00**	.00**	.00**	—		
Left Superior Frontal	.20	.20	.40	.36	.001**	.03*	.00**	.00**	.00**	.00**	.00**	.00**	—	
Left Rostral Orbital Frontal	.23	.23	.36	.36	.00**	.17	.00**	.00**	.017*	.037*	.00**	.00**	.00**	—

Table Notes. *p<.05 (1 tailed), **p<.01(1 tailed)

Chapter 5: Discussion

Given the paucity of research in the area, the purpose of the current dissertation study is to extend the body of research on effective early detection of Alzheimer's disease (AD) in African Americans. There were three primary aims of the current investigation: (1) Explore whether there were differences in performance on the LASSI-L between amnestic-mild cognitively impaired (aMCI) and cognitively normal (CN) African American older adults, (2) to determine if performance on the LASSI-L serves as a better predictor of diagnostic group classification compared to other neuropsychological tests in African American older adults, and (3) Explore how neuropsychological measures are related to volumetric reductions in AD prone regions in African American older adults.

Primary Outcomes

The first aim of the current study was to explore whether there were differences in performance on the LASSI-L between aMCI and CN African American older adults. Specifically, it was predicted that measures sensitive to initial learning and storage, proactive semantic interference (PSI), failure to recover from proactive semantic interference (frPSI) and retroactive semantic interference (RSI) would be more impacted in AA CN versus their aMCI counterparts. Results supported the hypothesis of difference in performance on LASSI-L measures between diagnosis groups, as those who were cognitively normal were better able to learn, encode, and store to-be-remembered information and less susceptible to interference, than those with aMCI. First, with regards to initial learning and storage of information as measured by List A Free and Cued Recall 1 and 2, those who were cognitively normal were able to recall more words and make fewer intrusion errors than those with aMCI. These findings suggest that those who were

CN were better able to learn, encode, and store to-be-remembered information than those with aMCI. Second, after this initial learning and storage of List A, participants were asked to learn a second semantically related list of words (List B). The first presentation of List B assesses for PSI as measured by List B Free and Cued Recall 1. Results found that cognitively normal individuals remembered more words and made fewer intrusion errors. These findings indicate that those with aMCI were more susceptible to the effects of PSI than those who are cognitively normal. Participants were then shown List B again to provide them with the opportunity to recover from these PSI effects. When examining participants' ability or failure to recover from PSI as measured by List B Cued Recall 2, results found that cognitively normal individuals were able to recall more words than those with aMCI. These results indicate that individuals with aMCI are more likely to fail to recover from PSI compared to their cognitively normal counterparts. After the second presentations of List B, participants were asked to recall words from the original list (List A) to assess for RSI. Results indicated that cognitively normal individuals were able to recall more words from the original list during free recall compared to those with aMCI. This finding suggests that those with aMCI were more susceptible to the effects of RSI. Delayed recall of both Lists A and B were assessed after 20-minute delay. Results indicated that cognitively normal individuals were able to recall more words on delayed recall compared to aMCI. Taken together these results supported the hypothesis that African Americans diagnosed with aMCI were more impaired in their ability to learn and remember new information and further, that they were negatively impacted by the effects of PSI and RSI. Furthermore, these results suggest that those with aMCI failed to recover from effects of PSI more so than their CN counterparts.

Given these documented differences on LASSI-L performance occurred within an older African American sample, additional analyses were conducted in order to account for known covariates, such as literacy and global cognition. Specifically, several studies have cited the impact that literacy, as measured by word reading, can have on neuropsychological test performance. For instance, studies show that African Americans obtain significantly lower scores than Caucasians on measures of word list learning and memory, figure memory, abstract reasoning, fluency, and visuospatial skills, but that these racial differences become nonsignificant when adjusting for literacy (Manly, Touradji, Tang, & Stern, 2003). Literacy has been found to be the most influential predictor of cognitive test performance, even after accounting for age, sex, years, of education, and acculturation level and is thus believed to be a better indicator of cognitive reserve (Manly, Byrd, & Touradji, 2004). A longitudinal study examining cognitive decline across racial groups found that older adults with both high and low levels of literacy decline in immediate and delayed memory over time, but that this decline is more rapid for low literacy older adults (Manly, Touradji, Tang, & Stern, 2003).

Because of previous research demonstrating the impact that literacy and cognitive reserve may have on neuropsychological test performance, further analysis was conducted to control for these effects might have on LASSI-L performance. After controlling for these variables using the MMSE and WRAT-4 word reading, cognitively normal and aMCI only differed on their second cued recall of list A and List B cued recall 1 number of intrusions. These results indicate that those with aMCI exhibit more impairment in their initial learning and storage of information and suffer from PSI due to their inability to inhibit responses.

These results are consistent with previous research on Hispanic and non-Hispanic individuals which found that those with aMCI had greater difficulty with initial learning and storage and were more susceptible to PSI compared to those who were cognitively normal (Crocco et al., 2013; Loewenstein et al., 2016). Given that previous studies have demonstrated that PSI is one of the strongest predictors of progression from aMCI to dementia and that cued recall deficits are a more sensitive marker of AD pathology than free recall, the use of the LASSI-L to identify these deficits may prove valuable in identifying those individuals who are at risk of further decline (Curiel et al., 2013; Loewenstein et al., 2016).

Diagnostic Accuracy

The second aim of this study was to determine if performance on the LASSI-L served as a better predictor of diagnostic group classification compared to other neuropsychological tests in African American older adults. The obtained findings indicate that two measures on the LASSI-L (List B Cued Recall 1 intrusions and Delayed Free Recall) significantly predicted whether an individual would be diagnosed as cognitively normal or with aMCI. These measures demonstrated a combined overall sensitivity of 91.7% and a specificity of 85.0%, and a classification rate of 88.6% in distinguishing between individuals who were cognitively normal and those with aMCI.

Since neuropsychological measures such as the HVLT-R, Category Fluency and Trails B were part of the initial diagnostic neuropsychological battery, which was combined with the clinical diagnosis to assign participants to diagnostic groups, using these same measures, particularly the HVLT-R to predict diagnostic group would result in potential tautological or circular reasoning. A strength of the LASSI-L was that it was

completely independent of initial diagnostic formulation. Nonetheless, we conducted post-hoc analyses entering Trails B, LASSI-L and Category Fluency into logistic regression models and only LASSI-L List B Cued Recall 1 intrusions and LASSI-L delayed recall entered into the model. ROC curve analysis demonstrated greatest sensitivity (83.3%) and specificity (77.8%) using LASSI-L List B Cued Recall 1 intrusions when using a cut off of 3.

Taken together, these results indicate that for African American older adults, PSI and delayed recall and measured by the LASSI-L are important diagnostic indicators above and beyond traditional neuropsychological assessments. As such, utilizing the LASSI-L to assess for PSI and delayed recall may provide high diagnostic accuracy for this population earlier in the disease state than measures currently utilized. These results are consistent with previous studies that have demonstrated the LASSI-L's ability to differentiate between cognitively normal individuals and those ranging in severity of impairment in Hispanic and predominately White individuals (Curiel et al., 2013; Loewenstein et al., 2016). Similarly, these results align with previous studies that demonstrate that the LASSI-L evidences higher classification rates than those obtained by other traditional neuropsychological assessment measures among predominately White individuals (Curiel et al., 2013; Crocco, Curiel, Acevedo, Czaja, & Loewenstein, 2014).

Magnetic Resonance Imaging (MRI)

The third aim of this study was to explore how neuropsychological measures are related to volumetric reductions in AD prone regions in African American older adults. After correction for false discovery rates there were no statistically significant results.

This is inconsistent with previous research where the LASSI-L measures related to PSI and frPSI were found to uniquely correlate to volumetric reductions on MRI within medial temporal lobes (e.g. entorhinal cortex) and other AD prone regions (e.g. precuneus, superior frontal and superior parietal regions) (Loewenstein et al., 2017; Crocco et al., 2013; Curiel et al., 2013). There are a number of possible explanations for the lack of significant MRI findings. First, despite including the entire sample (both CN and aMCI), which is consistent with prior studies, the sample size available for MRI scans was modest and did not provide enough statistical power to yield significant results. Secondly, given the population of interest, community based African Americans; the participants may have had underlying conditions other than AD to a greater extent than the samples utilized in previous studies (i.e. Hispanic and predominately White individuals) (Brooks & Loewenstein, 2010). To this point, many participants evidenced cardiovascular risk factors such as hypertension, diabetes, hypercholesterolemia, and history of stroke (Table 5-1). These risk factors, as well as the high rates of past drug use typically seen in African American Baby Boomers (Pope, Wallhagen, & Davis, 2010) may indicate that the memory loss observed in this sample may more accurately be classified as mixed etiology. As such, future studies should seek to recruit a larger sample without major health conditions known to impact cognition. In addition, future studies may benefit from the use of PET amyloid scans and/or tau imaging to determine if other biomarker correlates of AD are be present.

Table 5-1. Cardiovascular Risk Factors.[4]

	aMCI (*n*=16)	Cognitively Normal (21)	X^2 Yate's Correction	*p*
Hypertension	68.8%	52.4%	.45	.51
Diabetes	23.5%	19.0%	.00	1.00
Hypercholestrolemia	31.3%	23.8%	.02	.80
Stroke	11.8%	4.8%	.04	.85

[4] Note. No cases had reported history of CFH or heart attack

Strengths

The current dissertation study has several strengths, including those related to design characteristics. Specifically the current study utilized a detailed, well-established and standardized criteria for the evaluation and diagnosis of both CN and aMCI patients, as well as, expert readings of volumetric magnetic resonance imaging data for participants, and the analyses included false discovery rates to control for false errors (Benjamini & Hochberg, 1995). Additionally, this dissertation study adds to the present literature base by examining LASSI-L. performance in African Americans, which are an underserved and underrepresented population in clinical research. This is significant as prevalence and incidence rates of dementia diagnoses across racial and ethnic groups have found that the rates of dementia among African Americans far outnumber that of other racial and ethnic groups (Mehta & Yeo, 2017). Furthermore, the current study is one of the first to determine the extent to which proactive, retroactive, and failure to recover from proactive semantic interference on a novel cognitive stress test could differentiate between aMCI and cognitively normal African American older adults.

Limitations

While the current study had several strengths, a number of limitations are worth noting. First, one important limitation of the current study is that of the forty-four total participants (24 of these participants were diagnosed as cognitively normal and 20 were diagnosed as having mild cognitive impairment) only 29 of these individuals underwent MRI scans and, as such, normal and MCI participants were combined. Additional participants would have increased the power of statistical tests, allowed for additional covariates such as health factors to be examined, and thus provided higher external

validity. However, recruitment of African American participants is complicated by a number of factors including mistrust of health care providers and researchers, as well as personal and historic discrimination (Mahoney et al., 2005). In fact, many African Americans report being unwilling to participate in research due to historic research instances such as the Tuskegee Study of Untreated Syphilis (Green et al., 1997). Personal discrimination, particularly in the Southern United States where this study was conducted, may also have added to this sense of mistrust in potential participants (Mahoney et al., 2005). Additionally, this study involved the recruitment of participants belonging to the Baby Boom cohort (i.e. those born between mid-1946 and mid-1964) who have been found to be more pessimistic regarding individuals in lower social status or those viewed as less fortunate (Riggs & Turner, 2000; Hogan, Perez, & Bell, 2008). Because this study consists largely of African American participants who were recruited from local churches, the sample is likely skewed and represents unique variance not accounted for by this study.

Second, despite comprehensive screening with the NPI to exclude participants meeting full criteria for a Major Mood Disorder from the study, there is a possibility that some participants may have experienced a sub-syndromal mood disorder that could have affected cognitive performance. We believe that this is unlikely as previous research has shown no evidence between mild mood symptoms and performance on the LASSI-L (Crocco et al., 2018) however future research is needed in order to confirm these findings.

Third, the fact that the LASSI-L's diagnostic accuracy was compared to memory measures used as part of the diagnostic process creates a degree of circularity. Even

though the LASSI-L still compared favorably, future studies would benefit from the comparison of the LASSI-L and neuropsychological measures, which were not used in the diagnostic process.

Finally, it is possible that this community dwelling AA population did not have underlying AD pathology but instead were experiencing cognitive symptoms due to other etiologies. As such, future research should seek to recruit participants with fewer cardiovascular risk factors.

Future Directions

Future studies should continue to investigate the performance of African Americans, with varying severity of impairment, on the LASSI-L. Recruiting a larger sample of participants would help future investigators better evaluate the LASSI-L's diagnostic features and accuracy among this population. Future studies should attempt to recruit a more diverse sample (e.g. different geographic regions, recruitment settings). Due to high levels of health risk factors observed in this sample, future studies should also attempt to recruit participants with family histories of AD, identified with amyloid and tau pathology by PET scan imaging, as well as, focus on individuals with both high and low levels of cardiovascular risk factors so as to better isolate a purely AD pathology. Because literacy has been linked to neuropsychological test performance and rates of cognitive decline over time, future studies should account for literacy levels to examine the effects it may have on performance, particularly in minority populations. Finally, future studies should include the comparison of the LASSI-L's diagnostic accuracy to the diagnostic accuracy rates of other independent neuropsychological measures for African Americans with varying severity of cognitive impairment.

Conclusions

In conclusion, the majority of the results of this dissertation are consistent with the prior literature in that there are differences in performance on the LASSI-L between diagnostic groups and the LASSI-L was able to differentiate between those diagnosed with aMCI and those who are cognitively normal with high-observed specificity and sensitivity. Specifically, the current dissertation found that those with aMCI have greater difficulty with initial learning and storage of information and are more susceptible to PSI and some aspects of frPSI and RSI compared to those who were cognitively normal. However, after controlling for global cognition and literacy, only aspects of PSI and RSI remained predictive. Furthermore, inconsistent with the prior literature, the current study did not find LASSI-L measures related to volumetric reductions in AD prone brain regions. This inconsistency may be due to a modest sample size and/or, given the high rates of cardiovascular risk factors within the sample, the observed memory loss may have been caused by other etiologies. Despite this inconsistency, demographic trends and projected prevalence rates of AD within African Americans (Celsis, 2000; Sherwin, 2000), coupled with the fact that emerging dementias therapies are more effective in the earlier stages, establish an increasing need for early detection of AD in susceptible populations (Loewenstein, Curiel, Duara, & Buschke, 2017). As such, cognitive stress tests such as the Loewenstein-Acevedo Scale of Semantic Interference and Learning (LASSI-L), can provide quick, accurate, and inexpensive diagnostic classification across impairment severity (Loewenstein, Curiel, Duara, & Buschke, 2017) and, as shown in this dissertation, across racial groups. Overall, the current study, in line with previous research, suggests that the LASSI-L holds promise as a diagnostic tool that can be used

by clinicians for identifying mild cognitive impairment among African American older adults.

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Appendix A

Diagnostic Statistical Manual of Mental Disorders 5th Edition Diagnosis of Alzheimer's Disease Critera

Major or Mild Neurocognitive Disorder Due to Alzheimer's Disease

A. The criteria are met for major or mild neurocognitive disorder.
B. There is insidious onset and gradual progression of impairment in one or more cognitive domains (for major neurocognitive disorder, at least two domains must be impaired).
C. Criteria are met for either probable or possible Alzheimer's disease as follows:

For major neurocognitive disorder:

Probable Alzheimer's disease is diagnosed if either of the following is present; otherwise, **possible Alzheimer's disease** should be diagnosed.

(1) Evidence of a causative Alzheimer's disease genetic mutation from family history or genetic testing.
(2) All three of the following are present:
   a) Clear evidence of decline in memory and learning and at least one other cognitive domain (based on detailed history or serial neuropsychological testing).
   b) Steadily progressive, gradual decline in cognition, without extended plateaus.
   c) No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological, mental, or systemic disease or condition likely contributing to cognitive decline).

For mild neurocognitive disorder:

Probable Alzheimer's disease is diagnosed if there is evidence of a causative Alzheimer's disease genetic mutation from either genetic testing or family history.

Possible Alzheimer's disease is diagnosed if there is no evidence of a causative Alzheimer's disease genetic mutation from either genetic testing or family history, and all three of the following are present:

1) Clear evidence of decline in memory and learning.
2) Steadily progressive, gradual decline in cognition, without extended plateaus.
3) No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological or systemic disease or condition likely contributing to cognitive decline).

D. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychological Association.

Appendix B

International Classification of Diseases, 10th Edition Diagnosis of Alzheimer's Disease Critera

F00 Dementia in Alzheimer's disease

The following features are essential for a definite diagnosis:

A. Presence of a dementia as described above.
B. Insidious onset with slow deterioration. While the onset usually seems difficult to pinpoint in time, realization by others that the defects exist may come suddenly. An apparent plateau may occur in the progression.
C. Absence of clinical evidence, or findings from special investigations, to suggest that the mental state may be due to other systemic or brain disease which can induce a dementia (e.g. hypothyroidism, hypercalcaemia, vitamin B12 deficiency, niacin deficiency, neurosyphilis, normal pressure hydrocephalus, or subdural hematoma).
D. Absence of a sudden, apoplectic onset, or of neurological signs of focal damage such as hemiparesis, sensory loss, visual field defects, and incoordination occurring early in the illness (although these phenomena may be superimposed later).

In a certain proportion of cases, the features of Alzheimer's disease and vascular dementia may both be present. In such cases, double diagnosis (and coding) should be made. When the vascular dementia precedes the Alzheimer's disease, it may be impossible to diagnose the latter on clinical grounds.

Includes*:* primary degenerative dementia of the Alzheimer's type

Differential diagnosis. Consider: a depressive disorder (F30-F39); delirium (F05.-); organic amnesic syndrome (F04); other primary dementias, such as in Pick's, Creutzfeldt-Jakob or Huntington's disease (F02.-); secondary dementias associated with a variety of physical diseases, toxic states, etc. (F02.8); mild, moderate or severe mental retardation (F70-F72).

Dementia in Alzheimer's disease may coexist with vascular dementia (to be coded F00.2), as when cerebrovascular episodes (multi-infarct phenomena) are superimposed on a clinical picture and history suggesting Alzheimer's disease. Such episodes may result in sudden exacerbations of the manifestations of dementia. According to postmortem findings, both types may coexist in as many as 10-15% of all dementia cases.

F00.0 Dementia in Alzheimer's disease with early onset

Dementia in Alzheimer's disease beginning before the age of 65. There is relatively rapid deterioration, with marked multiple disorders of the higher cortical functions. Aphasia, agraphia, alexia, and apraxia occur relatively early in the course of the dementia in most cases.

Diagnostic Criteria: As for dementia, described above, with onset before the age of 65 years, and usually with rapid progression of symptoms. Family history of Alzheimer's disease is a contributory but not necessary factor for the diagnosis, as is a family history of Down's syndrome or of lymphoma.

Includes*:* Alzheimer's disease, type 2 presenile dementia, Alzheimer's type

F00.1 Dementia in Alzheimer's disease with late onset

Dementia in Alzheimer's disease where the clinically observable onset is after the age of 65 years and usually in the late 70s or thereafter, with a slow progression, and usually with memory impairment as the principal feature.

Diagnostic guidelines*:* As for dementia, described above, with attention to the presence or absence of features differentiating the disorder from the early-onset subtype (F00.0).

Includes*:* Alzheimer's disease, type 1 senile dementia, Alzheimer's type

World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders*. Geneva, Switzerland: Author.

Appendix C

National Institute of Neurological and Communicative Disorders and Stroke (Nincds) of The United States and The Alzheimer's Disease and Related Disorders Association (Adrda) Diagnosis of Alzheimer's Disease Critera

I. The criteria for the clinical diagnosis of PROBABLE Alzheimer's disease include:

— dementia established by clinical examination and documented by the Mini-Mental Test, Blessed Dementia Scale, or some similar examination, and confirmed by neuropsychological tests;

— deficits in two or more areas of cognition;

— progressive worsening of memory and other cognitive functions;

— no disturbance of consciousness;

— onset between ages 40 and 90, most often after age 65; and

— absence of systemic disorders or other brain diseases that in and of themselves could account for the progressive deficits in memory and cognition.

II. The diagnosis of PROBABLE Alzheimer's disease is supported by:

— progressive deterioration of specific cognitive functions such as language (aphasia), motor skills (apraxia), and perception (agnosia);

— impaired of activities of daily living and altered patterns of behavior;

— family history of similar disorders, particularly if confirmed neuropathologically; and

— laboratory result of:

— normal lumbar puncture as evaluated by standard techniques,

— normal pattern or nonspecific changes in EEG, such as increased slow-wave activity, and

— evidence of cerebral atrophy on CT with progression documented by serial observation.

III. Other clinical features consistent with the diagnosis of PROBABLE Alzheimer's disease, after exclusion of causes of dementia other than Alzheimer's disease, include:

— plateaus in the course of progression of the illness;

— associated symptoms of depression, insomnia, incontinence, delusions, illusions, hallucinations, catastrophic verbal, emotional, or physical outbursts, sexual disorders, and weight loss;

— other neurologic abnormalities in some patients, especially with more advanced disease and including motor signs such as increased muscle tone, myoclonus, or gait disorder;

— seizures in advanced disease; and

— CT normal for age.

IV. Features that make a diagnosis of PROBABLE Alzheimer's disease uncertain or unlikely include:

— sudden, apoplectic onset;

— focal neurologic findings such as hemiparesis, sensory loss, visual field deficits, and incoordination early in the course of the illness; and

— seizures or gait disturbances at the onset or very early in the course of the illness.

V. Clinical diagnosis of POSSIBLE Alzheimer's disease:

— may be made on the basis of the dementia syndrome, in the absence of other neurologic, psychiatric, or systemic disorders sufficient to cause dementia, and in the presence of variations in the onset, in the presentation, or in the clinical course;

— may be made in the presence of a second systemic or brain disorder sufficient to produce dementia, which is not considered to be the cause of the dementia; and

— should be used in research studies when a single, gradually progressive severe cognitive deficit is identified in the absence of other identifiable cause.

VI. Criteria for diagnosis of DEFINITE Alzheimer's disease are:

— the clinical criteria for probable Alzheimer's disease; and

— histopathologic evidence obtained from a biopsy or autopsy.

VII. Classification of Alzheimer's disease for research purposes should specify features that may differentiate subtypes of the disorder, such as:

— familial occurrence;

— onset before age of 65;

— presence of trisomy-21; and

— coexistence of other relevant conditions such as Parkinson's disease.

McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, M. E. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA work group* under the auspices of department of health and human services task force on Alzheimer's disease. *Neurology, 34*(7), 939-944. doi:10.1212/WNL.37.7.939

Appendix D

Loewenstein-Acevedo Scales of Semantic Interference and Learning (LASSI-L)

Code: _____ Date: _____

Loewenstein-Acevedo Scale for Semantic Interference- II Edition (LASSI-II)

TRIAL 1 – LIST A:

Say: **"I am going to show you 15 words, one word at a time. The words will be fruits, musical instruments, or articles of clothing. Each time I show you a word, I want you to read it out loud. Later on, I am going to ask you to tell me, from memory, all of these words, which will be fruits, musical instruments, or articles of clothing.**

Present the first word (i.e., Flute), and say **"This is the first word. Please read this word out loud so that you can remember it later."** For the first word, start timing immediately after you have finished reading the previous sentence. Present all other words without any prompting and wait for Pt to read it out loud. Rate of presentation: One word every 4 seconds. Order of Presentation: 1) Flute; 2) Pear; 3) Sock; 4) Banana; 5) Shirt; 6) Harmonica; 7) Tie; 8) Violin; 9) Strawberry; 10) Piano; 11) Jacket; 12) Mango; 13) Hat; 14) Lime; 15) Guitar

a. If patient has not read the word within 4 seconds or if he/she reads the word incorrectly, the examiner should read the word out loud and ask the patient to repeat the word. For example, if the Pt is unable to read the word "guitar", say **"Guitar. Repeat Guitar"** and wait for Pt to repeat the word before the examiner presents the next word.

b. If patient says that he/she does not know what the word means, say the word followed by its corresponding category. For example, if Pt says that he/she does not know what "guitar" is, say: **"A guitar is a musical instrument. Repeat Guitar."**

"Now I want you to tell me all the words that you just read. Are you ready? Begin."

SET A - RECALL 1 [60 seconds max]		
Stimuli	*Recall Order*	*Intrusions*
1. Banana		
2. Flute		
3. Guitar		
4. Harmonica		
5. Hat		
6. Jacket		
7. Lime		
8. Mango		
9. Pear		
10. Piano		
11. Shirt		
12. Sock		
13. Strawberry		
14. Tie		
15. Violin		
	Free Recall Trial 1-A *Total: _____*	*Intrusions Trial 1-A* *Total: _____*

"Now I want you to tell me all the words from the list that were fruits. Are you ready? Begin."

SET A – CUED RECALL 1 – Fruits [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Banana		
2. Lime		
3. Mango		
4. Pear		
5. Strawberry		
	Cued Recall Trial 1-A ***Total: _____***	***Intrusions Cued Recall Trial 1-A*** ***Total: _____***

"Now I want you to tell me all the words from the list that were musical instruments. Are you ready? Begin."

SET A – CUED RECALL 1 – Musical Instruments [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Flute		
2. Guitar		
3. Harmonica		
4. Piano		
5. Violin		
	Cued Recall Trial 1-A ***Total: _____***	***Intrusions Cued Recall Trial 1-A*** ***Total: _____***

"Now I want you to tell me all the words from the list that were articles of clothing. Are you ready? Begin."

SET A – CUED RECALL 1 – Articles of Clothing [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Hat		
2. Jacket		
3. Shirt		
4. Sock		
5. Tie		
	Cued Recall Trial 1-A ***Total: _____***	***Intrusions Cued Recall Trial 1-A*** ***Total: _____***

SET A, CUED RECALL 1 TOTAL: _____ **INTRUSIONS TOTAL: _____**

TRIAL 2 – LIST A:

Say: **"I am going to show you the 15 words again, one word at a time. As you know, the words are fruits, musical instruments, or articles of clothing. Each time I show you a word, I want you to read it out loud. Later on, I am going to ask you to tell me, from memory, each of these words, which are fruits, musical instruments, or articles of clothing."**

Present the first word (i.e., Flute) and say: **"Please read this word out loud so that you can remember it later."** Present each of the subsequent words without any prompting and wait for Pt to read it out loud. Rate of presentation: One word every 4 seconds.

"Now I want you to tell me all the words from the list that were fruits. Are you ready? Begin."

SET A – CUED RECALL 2 – Fruits [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Banana		
2. Lime		
3. Mango		
4. Pear		
5. Strawberry		
	Cued Recall Trial 2-A ***Total:*** _____	***Intrusions Cued Recall Trial 2-A*** ***Total:*** _____

"Now I want you to tell me all the words from the list that were musical instruments. Are you ready? Begin."

SET A – CUED RECALL 2 – Musical Instruments [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Flute		
2. Guitar		
3. Harmonica		
4. Piano		
5. Violin		
	Cued Recall Trial 2-A ***Total:*** _____	***Intrusions Cued Recall Trial 2-A*** ***Total:*** _____

"Now I want you to tell me all the words from the list that were ~~musical instruments~~ articles of clothing. Are you ready? Begin."

SET A – CUED RECALL 2 – Articles of Clothing [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Hat		
2. Jacket		
3. Shirt		
4. Sock		
5. Tie		
	Cued Recall Trial 2-A ***Total:*** _____	***Intrusions Cued Recall Trial 2-A*** ***Total:*** _____

SET A, CUED RECALL 2 TOTAL: _____ **INTRUSIONS TOTAL:** _____

TRIAL 3 – LIST B:

Say: **"Now I am going to show you a <u>different</u> set of 15 words, one word at a time. The words will be fruits, musical instruments, or articles of clothing. Each time I show you a word, I want you to read it out loud. Later on, I am going to ask you to tell me, from memory, all these <u>new</u> words, which will be fruits, musical instruments, or articles of clothing."**

Present the first word (i.e., Accordion) and say: **"This is the first word of this <u>new</u> set of words. Please read this word out loud so that you can remember it later."** Present each of the subsequent words without any prompting and wait for Pt to read it out loud. Rate of presentation: One word every 4 seconds. Order of Presentation: 1) Accordion; 2) Peach; 3) Shoe; 4) Orange; 5) Pants; 6) Trumpet; 7) Belt; 8) Harp; 9) Grapes; 10) Saxophone 11) Sweater; 12) Pineapple; 13) Gloves; 14) Coconut; 15) Clarinet

a. If Pt has not read the word within 4 seconds or if he/she reads the word incorrectly, the examiner should read the word out loud and ask the patient to repeat the word. For example, if the Pt is unable to read the word "peach", say **"Peach. Repeat Peach"** and wait for Pt to repeat the word before the examiner presents the next word.

b. If Pt says that he/she does not know what the word means, say the word followed by its corresponding category. For example, if Pt says that he/she does not know what "peach" is, say **"A peach is a fruit. Repeat Peach."**

"Now I want you to tell me all the words from the <u>new</u> list that you just read. Are you ready? Begin."

SET B - RECALL 1 *[60 seconds max]*		
Stimuli	***Recall Order***	***Intrusions***
1. Accordion		
2. Belt		
3. Clarinet		
4. Coconut		
5. Grapes		
6. Gloves		
7. Harp		
8. Orange		
9. Pants		
10. Peach		
11. Pineapple		
12. Saxophone		
13. Shoe		
14. Sweater		
15. Trumpet		
	Free Recall Trial 3-B ***Total: _____***	***Intrusions Trial 3-B*** ***Total: _____***

"Now I want you to tell me all the words from the <u>new</u> list that were fruits. Are you ready? Begin."

SET B – CUED RECALL 1 – Fruits [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Coconut		
2. Grapes		
3. Orange		
4. Peach		
5. Pineapple		
	Cued Recall Trial 3-B ***Total:*** _____	***Intrusions Cued Recall Trial 3-B*** ***Total:*** _____

"Now I want you to tell me all the words from the <u>new</u> list that were musical instruments. Are you ready? Begin."

SET B – CUED RECALL 1 – Musical Instruments [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Accordion		
2. Clarinet		
3. Harp		
4. Saxophone		
5. Trumpet		
	Cued Recall Trial 3-B ***Total:*** _____	***Intrusions Cued Recall Trial 3-B*** ***Total:*** _____

"Now I want you to tell me all the words from the <u>new</u> list that are articles of clothing. Are you ready? Begin."

SET B – CUED RECALL 1 – Articles of Clothing [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Belt		
2. Gloves		
3. Pants		
4. Shoe		
4. Sweater		
	Cued Recall Trial 3-B ***Total:*** _____	***Intrusions Cued Recall Trial 3-B*** ***Total:*** _____

SET B, CUED RECALL 1 TOTAL: _____ INTRUSIONS TOTAL: _____

TRIAL 4 – LIST B:

Say: **"I am going to show you again the 15 words that I showed you in the last set of words. I am going to show you one word at a time. The words are fruits, musical instruments, or articles of clothing. Each time I show you a word, I want you to read it out loud. Later on, I am going to ask you to tell me, from memory, all the words from this set of words, which are fruits, musical instruments, or articles of clothing.**

Present the first word (i.e., Accordion) and say **"Please read this word out loud so that you can remember it later."**
Present each of the subsequent words without any prompting and wait for Pt to read it out loud.
Rate of presentation: One word every 4 seconds.

"Now I want you to tell me all the words from the last set of words that were fruits. Are you ready? Begin."

SET B – CUED RECALL 2 – Fruits [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Coconut		
2. Grapes		
3. Orange		
4. Peach		
5. Pineapple		
	Cued Recall Trial 4 – B ***Total:*** _____	***Intrusions Cued Recall Trial 4 – B*** ***Total:*** _____

"Now I want you to tell me all the words from the last set of words that were musical instruments. Are you ready? Begin."

SET B – CUED RECALL 2 – Musical Instruments [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Accordion		
2. Clarinet		
3. Harp		
4. Saxophone		
5. Trumpet		
	Cued Recall Trial 4 – B ***Total:*** _____	***Intrusions Cued Recall Trial 4 – B*** ***Total:*** _____

"Now I want you to tell me all the words from the last set of words that were articles of clothing. Are you ready? Begin."

SET B – CUED RECALL 2 – Articles of Clothing [20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Belt		
2. Gloves		
3. Pants		
4. Shoe		
4. Sweater		
	Cued Recall Trial 4 – B ***Total:*** _____	***Intrusions Cued Recall Trial 4 – B*** ***Total:*** _____

SET B, CUED RECALL 2 TOTAL: _____ **INTRUSIONS TOTAL:** _____

Trial 5 – LIST A RECALL

"Now I want you to think back to the first list of words that I asked you to remember. I want you to tell me all the words in the first set of words. Are you ready? Begin."

SET A - RECALL 2 [Time: 60 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Banana		
2. Strawberry		
3. Flute		
4. Guitar		
5. Harmonica		
6. Hat		
7. Jacket		
8. Lime		
9. Mango		
10. Pear		
11. Piano		
12. Shirt		
13. Sock		
14. Tie		
15. Violin		
	Free Recall Trial 5 – A ***Total:*** _____	***Intrusions Trial 5 – A*** ***Total:*** _____

"Now I want you to tell me all the words from the first set of words that were fruits. Are you ready? Begin."

TRIAL 5 - SET A – CUED RECALL – Fruits [Time: 20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Banana		
2. Lime		
3. Mango		
4. Pear		
5. Strawberry		
	Cued Recall Trial 5 – A ***Total:*** _____	***Intrusions Cued Recall Trial 5 – A*** ***Total:*** _____

"Now I want you to tell me all the words from the first set of words that were musical instruments. Are you ready? Begin."

TRIAL 5 - SET A – CUED RECALL - Musical Instruments [Time: 20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Flute		
2. Guitar		
3. Harmonica		
4. Piano		
5. Violin		
	Cued Recall Trial 5 – A ***Total:*** _____	***Intrusions Cued Recall Trial 5 – A*** ***Total:*** _____

"Now I want you to tell me all of the words from the first set of words that were articles of clothing. Are you ready? Begin."

TRIAL 5 - SET A – CUED RECALL - Articles of Clothing [Time: 20 seconds max]		
Stimuli	***Recall Order***	***Intrusions***
1. Hat		
2. Jacket		
3. Shirt		
4. Sock		
5. Tie		
	Cued Recall Trial 5 – A ***Total:*** _____	***Intrusions Cued Recall Trial 5 – A*** ***Total:*** _____

SET A, CUED RECALL 3 TOTAL: _____ **INTRUSIONS TOTAL:** _____

DELAYED RECALL *[Admin. 20 minutes later – Time 90 seconds max]*

Administration: **"A while ago, I asked you to remember two lists of words. Please tell me all of the words from both lists that you can remember now. Are you ready? Begin."**

1	11	21
2	12	22
3	13	23
4	14	24
5	15	25
6	16	26
7	17	27
8	18	28
9	19	29
10	20	30