THE ASSOCIATION BETWEEN VERBAL FLUENCY DEFICITS, DEPRESSION, AND QUALITY OF SLEEP AMONG ALZHEIMER’S DISEASE PATIENTS

BY

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Student’s Declaration

I, the undersigned, declare that this is my original work and has not been submitted to any other college, institution, or university other than the United States International University-Africa in Nairobi for academic credit.

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This thesis has been presented for examination with my approval as the appointed supervisor.

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Abstract

Alzheimer’s disease represents a growing health challenge in the elderly population across the globe. The disease is associated with negative implications on survival, cost-of-care, and quality-of-life (World Alzheimer Report, 2015). Of the various debilitating cognitive symptoms associated with Alzheimer’s disease, language deficits are associated with much greater clinical relevance because of its interference with aspects such as self-care, recreational activities, employment, and behavior. One of the early signs of aphasia in Alzheimer’s disease is impairment in verbal fluency (Ferris & Farlow, 2013). Because of the widespread trend of bilingualism in today’s scenario (Ansaldo & Saidi, 2014), it is essential to study language deficits within the framework of the bilingual brain. Therefore, in this study, second language deficits as measured by verbal fluency tasks were assessed in Alzheimer’s disease patients as compared to healthy elderly individuals. Additionally, the study evaluated the relationship between second language deficits and depression and sleep disturbances in Alzheimer’s disease patients relative to healthy older individuals. No correlation was found between depression and sleep and verbal fluency tasks in Alzheimer’s patients. However, the rate of depression was significantly higher in Alzheimer’s patients. In healthy individuals, there was a significant effect for habitual sleep efficiency on semantic task scores, \( F(1, 6) = 6.069, p = .049 \). Furthermore, semantic task score was significantly higher than phonemic task score in healthy individuals, \( t(9) = 3.939, p = .003 \), suggesting an increased cognitive load associated with the phonemic task.
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Dedication

This work is dedicated to all the brave souls fighting Alzheimer’s disease and to their families.
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Chapter One

Introduction

1.0 Introduction

Firstly, this chapter discusses the study background, statement of purpose, and the significance of the study. Additionally, sections pertinent to the study objectives, questions, and hypotheses are presented. From the study design point of view, limitations of the study are acknowledged and the operational definitions of the study variables are discussed.

1.1 Background

Alzheimer’s disease is a type of dementia associated with a decline in various cognitive functions such as memory, language, attention, perception, planning, and other executive abilities (Mayeux & Stern, 2012). When compared to other types of dementia, Alzheimer’s disease is the leading contributor to dementia burden (Ott et al., 1995). The typical age of onset of Alzheimer’s disease is above 60 years. Although young-onset Alzheimer’s disease is prevalent, it is not a common occurrence (World Alzheimer Report, 2015). Factors such as increasing life expectancy and declining fertility rates in many regions of the world contribute to older individuals representing an increasing percentage of the total population (Lunenfeld & Stratton, 2013). In 2015, there were 900 million elderly individuals worldwide. This figure is estimated to increase by 56%, 138%, 185%, and 239% in the high-, upper-middle-, lower-middle-, and low income countries, respectively, between 2015 and 2050 (World Alzheimer Report, 2015).
Alzheimer’s disease is associated with significant negative effects on survival and morbidity. Patients with Alzheimer’s disease may find it difficult to perform everyday activities such as walking, getting out of the bed, and bathing, among other activities. Furthermore, Alzheimer’s disease exacerbates the debilitating effects of various comorbid physical and mental illnesses. Due to the nature of symptoms, Alzheimer’s patients become significantly dependent on caregivers for day-to-day activities (World Alzheimer Report, 2015). This increases the strain on caregivers as they experience poor psychological outcomes (Acosta, Rottbeck, Rodriguez, Ferri, & Prince, 2008). Additionally, cost associated with dementia care is significantly high; it has been found to corroborate with cost of chronic conditions such as cancer, stroke, and cardiovascular diseases (Luengo-Fernandez, Leal, & Grey, 2010).

Of the various functional limitations that manifest during the preclinical and three clinical stages, language impairments represent one of them (Ferris & Farlow, 2013). Language deficits have been found to inversely correlate with psychological wellness and ability to maintain a functional level of communication (Hilari & Byng, 2009). One of the early signs of language impairment seen in Alzheimer’s disease is a progressive decline in verbal fluency (Ferris & Farlow, 2013). As the world population is becoming increasingly bilingual (Ansaldo & Saidi, 2014), understanding language function necessitates a bilingual basis. Bilingualism acts as a protective mechanism in delaying the onset of dementia (Bialystok, Craik, & Freedman, 2007; Gold, 2015). However, second language (L2) decline has been observed in Alzheimer’s disease and has been noted to be an early sign of Alzheimer’s disease (McMurrtay, Saito, & Nakamoto, 2009). Furthermore, L2 impairment has been reported to be a common senile phenomenon (Mendez, Perryman, Ponton, &
Cummings, 1999). In light of these data, the present study examined the extent of L2 decline in Alzheimer’s patients as compared to healthy older individuals. Additionally, in this study, the relationship between L2 deficits, depression, and sleep disturbances were examined, given that depression and sleep negatively interfere with language performance (Harrison & Horne, 1997; Henry & Crawford, 2005; Horne, 1988).

1.2 Problem Statement

Language deficits represent an important challenge in Alzheimer’s patients as compared to other cognitive functions because language impairments exert a negative impact on non-cognitive domains such as employment and day-to-day activities (Ferris & Farlow, 2013). Because bilingualism is common in the majority of the world (Ansaldo & Saidi, 2014), language deficits essentially need to be studied in bilingual individuals. Research has shown that bilingualism offers cognitive protection and delays the onset of dementia (Bialystok et al., 2007). However, a report by McMurray et al. (2009) reveals regression to L1 to be an early symptom of dementia in Alzheimer’s disease. Furthermore, there is evidence that L2 function may decline as part of normal aging (Mendez et al., 1999). Therefore, in an attempt to further study L2 deficits, the present study examined the extent of L2 decline in bilingual Alzheimer’s patients as compared to healthy bilingual individuals. Factors typically associated with L2 decline include proficiency in L2 (Hope et al., 2015) and the extent of L2 usage (Pearce, 2005) before L2 deterioration occurs; lower rates of L2 proficiency and infrequent L2 usage increase the risk of L2 attrition. Because of the retrospective significance of these factors, facilitating an interventional change with respect to L2 proficiency and L2 usage may not be possible. Therefore, the present study sought to identify potential modifiable factors that have an association with L2 loss in Alzheimer’s
disease. Depression was one of the two factors under investigation, because it exerts a negative effect on language outcomes related to verbal fluency, retrieval, attention, and concentration (Henry & Crawford, 2005; Norris, Blankenship-Reuter, Snow-Turek, & Finch, 1995). Furthermore, stroke patients experiencing language difficulties show higher rates of depression due to reduced levels of day-to-day communication (Hilari & Byng, 2009). Sleep quality represented another factor for investigation because impairments in sleep quality are associated with impairments in language functions such as verbal fluency, retrieval of words, and the process of semantic memory formation (Harrison & Horne, 1997; Horne, 1998; Wagner et al., 2004). Therefore, the study evaluated the correlation between depression, sleep quality, and verbal fluency deficits in Alzheimer’s patients as compared to healthy elderly individuals.

1.3 Significance of the Study

The study results may provide evidence to improve quality-of-life of Alzheimer’s disease patients experiencing L2 deficits. Language impairments interfere with day-to-day functioning and this can be distressing to the patients (Ferris & Farlow, 2013). In fact, language difficulties are associated with compromised psychological wellness and a decline in physical activity (Hilari & Byng, 2009). The study findings would be of interest to clinical psychologists because depression and sleep disturbances are treatable conditions.

1.4 Objectives of the Study

1. To comparatively assess sleep quality in Alzheimer’s patients and healthy elderly individuals (i.e., control group).
2. To compare the depression scores between the Alzheimer’s group and the control group.

3. To assess the semantic and the phonemic abilities in Alzheimer’s patients as compared to the control participants.

4. To assess if there exist differences in the semantic and the phonemic task scores in the Alzheimer’s group as well as the control group.

5. To determine the relationship between depression and L2 verbal fluency impairments in Alzheimer’s disease patients as compared to control group participants.

6. To determine the relationship between sleep quality and L2 verbal fluency impairments in Alzheimer’s disease patients as compared to control group participants.

1.5 Research Questions

1. Are there differences in sleep quality between Alzheimer’s patients and the healthy elderly individuals?

2. Are there differences in depression scores between the Alzheimer’s group and the control group?

3. Are there differences in performances on the semantic task and the phonemic task in L2 between bilingual Alzheimer’s disease patients and bilingual healthy elderly individuals?

4. Are there differences between the phonemic task scores and the semantic task scores in the Alzheimer’s group and the control group?

5. Is there a correlation between depressive symptoms and L2 deficits in Alzheimer’s disease patients as compared to healthy elderly individuals?
6. Is there a correlation between sleep quality and L2 deficits in Alzheimer’s disease patients as compared to healthy elderly individuals?

1.6 Research Hypothesis

1. Sleep quality is poor in Alzheimer’s disease patients.
2. Alzheimer’s patients tend to score higher on depression scales.
3. Bilingual healthy elderly individuals score higher on verbal fluency tasks relative to bilingual Alzheimer’s disease patients.
4. Depression negatively interferes with verbal fluency in Alzheimer’s disease patients.
5. Sleep disturbances impair verbal fluency in Alzheimer’s disease patients.

1.7 Limitations of the Study

Because the present study was correlational in nature, a causative link connecting depression or sleep problems with L2 deficits cannot be inferred. Due to the inherent bias associated with convenience sampling and small sample size used, generalizability of the study results may be limited. Because self-report measures are used for the assessment of sleep quality and depression in healthy individuals, there can be a potential for discrepancy between actual and perceived degrees of depression and sleep quality. Additionally, because of the use of a caregiver-based report of bilingual proficiency, there is a potential for discrepancy between the actual language proficiency and the subjective report.

1.8 Definition of Terms

**Bilingualism:** refers to the use of two languages by an individual (American Speech-Language-Hearing Association, 2004). Bilingual speakers may have a minimum proficiency
in the two languages, an intermediate level of proficiency, or an advanced level of proficiency. The range includes ability to communicate orally alone to a native speaker-like proficiency (National Association for Language Development in the Curriculum, 2011).

**First language (L1):** refers to the language an individual was first exposed to as a child. Another term for first language is mother tongue (National Association for Language Development in the Curriculum, 2011).

**Second language (L2):** refers to the language that is acquired after L1. If a child was exposed to both L1 and L2 from the time of birth and had equal opportunities to use both languages, the bilingual experience is said to be simultaneous. On the other hand, if the L2 is learned after the L1 is learned, the bilingual experience is referred to as sequential bilingualism (American Speech-Language-Hearing Association, 2004).

**Verbal fluency:** corresponds to performance on the phonemic and semantic tasks. Phonemic fluency corresponds to the ability to retrieve words that begin with a particular letter (for e.g., F, A, or E). On the other hand, semantic fluency corresponds to the ability to retrieve words that are represented as clusters (e.g., category of animals) (Salvatierra, Roselli, Acevedo, & Duara, 2007).

1.9 Chapter Summary

Alzheimer’s disease is a growing healthcare challenge globally and is associated with negative effects on survival and morbidity (World Alzheimer Report, 2015). In addition, it significantly increases the strain on caregiving, and as a result, caregivers experience poor psychological outcomes (Acosta et al., 2008). Alzheimer’s disease is characterized by various symptoms related to cognition and behavior that significantly impair one’s level of
functioning (Ferris & Farlow, 2013). Among these debilitating symptoms, language impairments are one of the important challenges faced by Alzheimer’s patients, because it limits their everyday activities (Hilari & Byng, 2009). Although Alzheimer’s disease is characterized by progressive deterioration, which includes gradual language function decline, any attempt to improve the quality-of-life of these patients may be beneficial. Research shows language impairments to be linked to depression and sleep disturbances (Harrison & Horne, 1997; Henry & Crawford, 2005; Horne, 1998). Therefore, the current study examined the association between verbal fluency deficits in Alzheimer’s patients, depression, and sleep disturbances.
Chapter 2

Literature Review

2.0 Introduction

This section presents the global and regional prevalence data of dementia together with its implications on quality-of-life, cost of healthcare, mortality, and caregiving burden. Subsequently, pathogenic mechanisms responsible for Alzheimer’s disease and the clinical features are discussed. As seen with any type of dementia, Alzheimer’s disease leads to a decline in various cognitive functions that lead to behavioral disturbances (Mayeux & Stern, 2012). One of the cognitive disturbances associated with Alzheimer’s disease is the language impairment (Ferris & Farlow, 2013). Reduced language abilities significantly impair ability to communicate effectively and thus the quality-of-life is affected negatively (Hilari & Byng, 2009). Therefore, the nature of language difficulties seen in Alzheimer’s disease is discussed. Bilingualism serves as an important reference point for understanding language outcomes because bilingual individuals form a substantial proportion of the world population today (Ansaldo & Saidi, 2014). Therefore, a discussion of the neural basis of bilingualism and the protective effect of bilingualism against dementia is presented. Subsequently, language deficits in Alzheimer’s disease and in normal aging are discussed. Evidence shows sleep disturbances and depression to negatively impact language performance (Harrison & Horne, 1997; Henry & Crawford, 2005; Horne, 1998; Norris, Blankenship-Reuter, Snow-Turek, & Finch, 1995; Wagner et al., 2004). Therefore, a theoretical framework for the assessment of correlation between sleep quality, depression, and language outcomes in Alzheimer’s disease patients as compared to healthy elderly individuals is discussed.
2.1 Epidemiology of Dementia

Dementia is an important global health challenge. Typically, the age of onset for dementia is 60 years and above. Young-onset dementia is not so frequent a phenomenon as it accounts for only 2% to 8% of all cases. The incidence of dementia is 3.9 individuals per 1000 person-years between 60 and 64 years of age, and it drastically increases to 104.8 per 1000 person-years above 90 years of age. It has been estimated that nearly 46.8 million people were affected by dementia in 2015. These figures are projected to double every 20 years with 74.7 million and 131.5 million people being estimated to be affected by 2030 and 2050, respectively. Based on the above data, it can be seen that the incidence of dementia is increasing worldwide (World Alzheimer Report, 2015).

The estimated increase in dementia burden is attributable to various factors such as increased life expectancy, reduced fertility rates, negative lifestyle choices, and urbanization (Harper, 2012; Lunenfeld & Stratton, 2013; Mayeux & Stern, 2012). Life expectancy is increasing globally and in 2015, there were 900 million elderly individuals worldwide. In fact, the magnitude of increase in older people is estimated to raise by 56% in high income countries (as classified by the World Bank in 2015), as compared to 138%, 185%, and 239% in the upper middle-income, lower-middle income, and low income countries, respectively during the period of 2015 to 2050 (World Alzheimer Report, 2015). Apart from increased life expectancy, reduced fertility rates in many parts of the world are attributable to the increasing proportion of older individuals (Lunenfeld & Stratton, World Alzheimer Report, 2015). Lifestyle factors such as increased dietary sodium intake, high fat and sugar, physical inactivity, and smoking underscore the negative impact of chronic diseases in older individuals, including dementia (Mayeux & Stern, 2012; Notkola et al., 1998; World

One of the implications of increasing dementia burden lies on the cost-of-care. In a study conducted in the UK, it was found that the cost for dementia care inclusive of health and social care costs was comparable with that of the cumulative cost of cancer, cardiovascular diseases, and stroke (Luengo-Fernandez et al., 2010). Another study conducted in Sweden found the per year cost of dementia to equate to the cost of depression, stroke, alcohol use disorder, and osteoporosis (Wimo, Johanson & Jonsson, 2007). The 10/66 Dementia Research Group involving participants from Latin America, India, and China found dementia costs to exceed that of depression, hypertension, diabetes, and heart disease in all countries except India (Sousa et al., 2010). These findings point to the significant cost burden associated with dementia.

Although the burden of dementia is growing globally, low and middle income countries may particularly face the challenge of not providing adequate care to its increasing number of elderly individuals because of economic constraints. In addition, many low and middle income countries have a superimposed burden due to increased prevalence of infectious diseases, diseases related to mother and child health, and chronic illnesses such as hypertension, diabetes mellitus, and cardiovascular diseases. About 58% of people with dementia are from the low and middle income countries and this figure is estimated to increase to 63% and 68% between 2030 and 2050 in these areas. This discussion points to the particularly challenging scenario in the under-developed and developing nations, which include Kenya (World Alzheimer Report, 2015).
In addition to cost consequences, dementia is associated with increased mortality and significant quality-of-life impairment. There is a wealth of evidence to suggest that dementia adversely interacts with other co-existing physical and mental health disorders, and increases the dependence on caregivers with a corresponding increase in the cost of care. Individuals with dementia may require assistance for everyday activities such as getting in and out of bed, bathing, eating, dressing, and emptying bowel and bladder (World Alzheimer Report, 2015). It has been reported that there are negative implications of dementia on caregiver’s wellness as well; psychological burden on caregivers as assessed by a 22-item Zarit Burden interview was significantly higher in caregivers of dementia patients (Acosta et al., 2008). Based on the above discussion, it can be concluded that dementia is associated with negative effects on survival, quality-of-life, cost, and healthcare resources.

2.2 Alzheimer’s Disease as the Leading Type of Dementia

Of all the identifiable causes of dementia, Alzheimer’s disease is a common type accounting for nearly three-fourth of all cases (World Health Organization, 2017). In the Rotterdam study that involved screening of 7,528 individuals aged 55 to 106 years for dementia, 474 positive cases were reported (Ott et al., 1995). In this study, Alzheimer’s disease was identified to be the cause of dementia in 72% of the cases. Other forms of dementia, namely, vascular dementia, dementia due to Parkinson’s disease, and other types of dementia occurred in 16%, 6%, and 5% of the study individuals, respectively. Additionally, the investigators of this study reported that the prevalence of Alzheimer’s disease increased significantly in the study population with aging, while the proportion of vascular dementia, dementia due to Parkinson’s disease, and other dementias decreased with
aging (Ott et al., 1995). In the following section, the pathogenic mechanisms responsible for Alzheimer’s disease will be discussed.

2.3 Neuronal and Molecular Basis of Alzheimer’s Disease

The neuronal basis of Alzheimer’s disease is linked to three specific factors, namely, amyloid plaques, neurofibrillary tangles, and gradual loss of neuronal connectivity (Serrano-Pozo, Frosch, Masliah, & Hyman, 2011). An explanation of the mechanisms underlying these pathogenic factors is presented herein.

Amyloid precursor protein (APP) is a cell-membrane protein that has the potential to become pathogenic beta-amyloid peptide (Rodgers, 2008). APP undergoes enzymatic processing by alpha-secretase, beta-secretase, and gamma-secretase. Based on the enzymes involved, APP enters one of the two specific cellular biochemical pathways. When alpha-secretase is involved, APP is transformed to aAPPα, a neuron-friendly component. On the other hand, when beta-secretase and gamma-secretase are involved, toxic beta-amyloid peptides are formed. As the number of beta-amyloid peptides increases, these peptides clump to form insoluble masses called protofibrils and fibrils. These moieties then combine with other cellular components to form insoluble plaques called amyloid plaques—a characteristic neuronal component implicated in the pathogenesis of Alzheimer’s disease (Serrano-Pozo et al., 2011; Rodger, 2008).

The second characteristic feature of Alzheimer’s disease is neurofibrillary tangles. In healthy functioning neurons, microtubules are involved in the cellular transport of nutrients and transport of neurotransmitters from the cell body to the axon terminal. Tau is a protein molecule that attaches to a specific number of phosphate molecules and the phosphate-bound
tau provides structural stability to microtubules. In Alzheimer’s disease, tau gets attached to more than the optimal number of phosphate groups and this leads the tau to detach from microtubules and consequently, microtubules become dysfunctional. Finally, the internal transport system becomes disrupted and the neuronal communication processes are deregulated (Serrano-Pozo et al., 2011).

The third characteristic feature is a progressive decline in the synaptic connections (Kimura et al., 2010). Plaques and tangles have been identified to be important precursors of synaptic loss (Rodgers, 2008). Additionally, there is neuronal death and the associated brain regions undergo atrophy (Gomez-Isla et al., 1996). As Alzheimer’s disease progresses into the final stage, there is widespread loss of neurons and the brain atrophies significantly (Rodgers, 2008).

Factors related to urbanization such as unhealthy diet and air pollution contribute to the accumulation of pathogenic substrates discussed above. A study conducted in Finland reported high levels of blood cholesterol in middle-life to increase the risk of developing Alzheimer’s disease by nearly three times (Notkola et al., 1998). Unhealthy dietary patterns including high-fat and high-cholesterol consumption increase the rate of production and accumulation of amyloid proteins (Morris, 2004). In addition, data from animal studies show high cholesterol to accelerate neuronal loss and other pathogenic brain changes seen in Alzheimer’s disease (Greenwood & Winocur, 1996; Refolo et al., 2000). Furthermore, in a recent study conducted in the US, air pollution was linked to increased risk of developing Alzheimer’s disease (Cacciottolo et al., 2017). The study reported that women exposed to higher than the safe levels of particulate air matter were at two-fold increased risk of developing dementia. Particulate matter increases the rate of generation of amyloid peptides
and augments other mechanisms responsible for Alzheimer’s disease (Cacciottolo et al., 2017).

In the next section, overt manifestations of the aforementioned neuronal changes will be discussed. The course of Alzheimer’s disease is as follows: the preclinical stage, the mild stage, the moderate stage, and the severe stage.

2.4 Preclinical and Clinical Stages of Alzheimer’s Disease

The definitive starting point for the development of Alzheimer’s disease is yet to be scientifically established. It remains unclear as to what differentiates normal aging brain changes from the changes observed in Alzheimer’s disease. However, the neural mechanisms and corresponding physical and mental symptoms observed once the disease sets in have been well-studied (Rodgers, 2008). Symptoms in Alzheimer’s disease follow the same stages in all individuals, namely: mild stage, moderate stage, and the severe stage (Ferris & Farlow, 2013). Nevertheless, survival varies with age at diagnosis; in those aged 80 years and above at diagnosis, survival is about 3 to 4 years. In younger individuals, the time to death from disease onset may extend up to 10 years. Apart from age, factors such as gender, presence of comorbid conditions, and extent of cognitive decline determine the survival duration (Rodgers, 2008). Men are at higher mortality risk than women; this is linked to the increased prevalence of comorbid conditions like arrhythmia, cancer, and chronic obstructive pulmonary disease in men (Gambassi et al., 1999). In the Hui et al. study (2003), in those with the most rapid decline in cognitive function relative to baseline, the mortality rate increased by nearly eight-fold compared to those patients whose cognition declined the least relative to baseline value. In summary, age at disease onset, sex, severity of cognitive
impairment, and presence of comorbidity affect the survival of Alzheimer’s disease individuals.

2.4.0 Preclinical stages associated with Alzheimer’s disease

Alzheimer’s disease has its onset in the entorhinal region, which is in close proximity to the hippocampus. As neurons degenerate in this region, the process spreads to the hippocampus. The hippocampus is involved in learning and consolidation of memory; therefore, learning and memory deficits may arise. Additionally, ventricles, the fluid filled compartments within the brain, enlarge as the disease progresses. It has been observed that these changes occur about 10 to 20 years before the symptoms of Alzheimer’s disease become overt (Rodgers, 2008).

One of the early symptoms associated with Alzheimer’s disease before it becomes clinically detected is mild cognitive impairment (MCI; Snowdon, 1996). MCI consists of different types, but the one most frequently associated with Alzheimer’s disease is of the amnestic type (Dubois et al., 2007). Amnestic MCI is characterized by significant memory deficits that cannot be explained by normal aging, while other symptoms of Alzheimer’s disease are absent. It has been found that nearly 8 of the 10 people with amnestic MCI develop Alzheimer’s disease within six years (Rosenberg & Lyketsos, 2008). The causative mechanisms corresponding to Alzheimer’s disease facilitated through amnestic MCI remain unclear. Evidence shows a role of genetic factors in intertwining MCI and Alzheimer’s disease (Fleisher et al, 2007). Furthermore, it has been found that different brain regions are activated during specific tasks in healthy individuals as compared to those with MCI (Rosenberg & Lyketsos, 2008). People with MCI have greater movement difficulties as
compared to healthy individuals and the difficulty is lower than that observed in Alzheimer’s disease. Individuals with MCI and significant motor disturbances are twice more likely to develop Alzheimer’s disease than those with good movement control in the lower body parts. There is speculation that before Alzheimer’s disease becomes clinically detectable, dysfunctional changes accumulate in the brain regions associated with motor coordination. If this hypothesis is true, in individuals with amnestic MCI and motor disturbances, especially of the lower extremities, a risk prediction of those who would progress into Alzheimer’s disease could be made (Rodgers, 2008).

2.4.1 The clinical stages of Alzheimer’s disease

As Alzheimer’s disease progresses, there is an increased accumulation of beta-amyloid plaques, neurofibrillary tangles, and increased synaptic loss, as discussed previously. Additionally, the cortex becomes progressively atrophied. Memory decline becomes more significant and other cognitive functions deteriorate as well (Rodgers, 2008). It is at this mild stage that Alzheimer’s disease becomes clinically diagnosable and the associated features are listed below (Ferris & Farlow, 2013):

1. Memory deficits
2. Disturbances in short-term memory
3. Loss of interest in pleasurable activities that were previously enjoyable
4. An impaired ability to recall names of day-to-day objects
5. Repeating queries

An individual in the mild stage may be apparently healthy except for the cognitive function decline, which is generally regarded as a normal aging process (Rodgers, 2008).
However, if detected early, the patient’s functional level can be preserved for a longer duration. Additionally, cost associated with care for functional Alzheimer’s disease patients is less when compared to Alzheimer’s disease patients who need institutional care (Leifer, 2003).

During the moderate stage, there is widespread cortical atrophy and the processes affected include language, reasoning, sensory perception, and conscious thinking. Symptoms may include (Ferris & Farlow, 2013):

1. Increased memory loss
2. Reduced attentional ability
3. Impairment in organizing thoughts and logical thinking
4. Language deficits
5. Impaired basic daily activities
6. Impairment in impulse control (e.g., disrobing at inappropriate times, and use of vulgar words)
7. Inability to learn new things or to respond to novel situations

In the severe stage, Alzheimer’s disease affects most brain regions with continued atrophy and increased ventricular volume. At this stage, patients do not recognize their loved ones and lose their communicative ability completely. They become entirely dependent on caregivers for support. Symptoms include (Ferris & Farlow, 2013; Rodgers, 2008):

1. Significant reduction in weight
2. Seizures
3. Skin infections
4. Difficulty in swallowing
5. Altered sleep routine
6. Bladder and bowel incontinence

From the above discussion, it can be seen that there is a fundamental progressive cognitive function loss from the time of disease onset even at the stage of amnestic MCI. Of the various debilitating symptoms associated with Alzheimer’s disease, language deficits pose significant challenge because of its impact on everyday interactions. Therefore, in the next section, the nature of language impairments experienced by Alzheimer’s disease patients from the time of disease onset will be discussed.

2.5 Language Deficits in Alzheimer’s Disease

An initial language impairment associated with Alzheimer’s disease is decline in verbal fluency (Mendez & Cummings, 2003). Assessment of verbal fluency with the help of semantic and phonemic tasks has been found to be useful in the diagnosis of dementia (Azuma et al., 1997; Monasch et al., 1994). The phonemic fluency task requires the individual to retrieve words that begin with a particular letter (e.g., A, F, or E). On the other hand, semantic fluency necessitates the individual to retrieve words that are represented by a category (e.g., animals) (Salvatierra et al., 2007). Decline in verbal fluency is seen as part of normal aging; however, there is a statistically significant decline in semantic and phonemic fluency tasks in Alzheimer’s disease patients as compared to cognitively healthy individuals (Fama et al., 1998). Additionally, in a longitudinal study that assessed verbal fluency over a period of 3 years, individuals with Alzheimer’s disease showed pronounced decline as compared to healthy individuals. Another finding of this study was that the ability to generate
words as a semantic function declined more rapidly as compared to generation of words under the phonemic category in Alzheimer’s disease patients (Salmon, Heindel, & Lange, 1999).

Impaired retrieval processes alone cannot explain the greater decline in the semantic task as compared to the phonemic task because if retrieval difficulties are the only reason, both tasks should be affected to the same extent. Another possible explanation for higher rate of decline in semantic tasks could be that semantic tasks require a higher degree of cognitive processing. However, this has been postulated to be false because in normal individuals, the phonemic task is more difficult than the semantic task. The total number of words produced by cognitively healthy elderly individuals is higher for the semantic category as compared to the phonemic category (Salmon et al., 1999). Therefore, an alternative explanation for the differential decline in the verbal fluency tasks is needed. Individuals with Alzheimer’s disease are more likely than healthy individuals to fail to produce a word in the semantic category in the subsequent years of testing once they fail to produce that word in the first year. This consistency in semantic ability decline leads to the assumption that there is a progressive loss of semantic representation in the brain (Salmon et al., 1999). Furthermore, there is observed a temporal trend in the decline in the category size (number of words produced under each category) in the semantic task (Rohrer et al., 1995). Individuals with Alzheimer’s disease as compared to normal individuals do not statistically differ in the consistency aspect with respect to the phonemic task. This could be because phonemic ability is less dependent on the semantic knowledge and that consistency may be a part of the semantic representation (Salmon et al., 1999).
Semantic knowledge loss in Alzheimer’s disease is linked to progressive cortical atrophy. Evidence from functional imaging techniques show that semantic memory involves lateral and inferior temporal regions of the cerebral cortex (Demonet et al., 1992; Martin et al., 1996), regions that happen to be the site for significant neuronal loss and pathological processes identified in Alzheimer’s disease (Terry & Katzman, 1983). As the pathological sequence involves progressively increasing areas of parietal and temporal lobes, semantic representation deteriorates gradually until semantic fluency is completely lost (Salmon et al., 1999). However, Salmon et al. (1999) caution that the association between cortical atrophy and semantic knowledge loss is from preliminary evidence and note that further research is needed to bolster this finding.

As discussed previously, bilinguals are the majority in the world, and therefore, language studies need to involve bilingual speakers in order to get a better picture. In the following section, the mechanisms of bilingual representation in the brain will be discussed.

2.6 Bilingualism and its Neural Explanations

Present day society is dominated by those who speak more than one language, and Ansaldo and Saidi (2014) refer to bilingualism as a norm rather than an exception. Historically, some countries have always been bilingual, but this trend has become widespread due to globalization. Bilingualism has been associated with improved employment opportunities and this has increased the interest towards acquiring an L2. Additionally, it has been reported that parents select bilingual education (Ansaldo & Saidi, 2014) for their children because bilingualism has been posited to increase certain cognitive abilities, such as increased attentional control and executive functioning (Bialystok, Martin,
& Viswanathan, 2005). Also, bilingualism confers improved intellectual growth, enhanced cognitive flexibility, increased creativity, and openness to experiencing diverse cultures (Ansaldo & Saidi, 2014). Taken together, bilingualism is associated with better economic prospects and improved cognitive, social, and educational outcomes. Therefore, it has become essential to study neuronal basis of language representation from the viewpoint of a bilingual brain because of today’s scenario.

Two specific models that have been proposed to explain the neuronal basis of bilingualism include: the declarative/procedural model and the single network model (Green, 2003; Ullman, 2001). According to the declarative/procedural model proposed by Ullman (2001), distinct brain areas are involved in the processing of L1 and L2. On the contrary, the single network model, posited by Green, involves a single dynamic network, which operates both L1 and L2 (Green, 2003). In his review, Kciuk (2009) mentions that although both the theories have evidence in their support, many studies are in support of the single dynamic model of bilingualism.

2.6.0 Declarative/procedural model

Languages areas in the brain include parts of the frontal lobe, temporal lobe, parietal lobe, and occipital lobe (John, 2000). The frontal lobe is located at the front of the brain and plays an important role in executive functions such as planning, decision making, emotional regulation, and recent memory processing. The temporal lobe is located laterally on the two hemispheres of the brain and plays an important role is auditory signal processing and certain aspects of visual processing, such as identification of faces and perceiving movements. The posterior part the brain is called the occipital lobe and it plays a vital role in the processing of
visual stimuli. The parietal lobe is surrounded by the frontal lobe in the front, occipital lobe at the back, and temporal lobe at the bottom and is associated with the processing of information associated with touch, pain, and pressure (Kalat, 2009, pp. 100-103). Language processing is linked strongly to the left hemisphere of the brain. Furthermore, the basal ganglia, a group of structures at the base of the forebrain is associated with acquisition of grammar and interpreting sounds. Additionally, posterior region of the frontal lobe, also called Broca’s area, is an important structure in expressive language (speech). The junction at the temporal, parietal, and the occipital areas is referred to as Wernicke’s area and is responsible for receptive language (comprehension). An interconnecting network between Broca’s area and Wernicke’s area is called the arcuate fasciculus; this network is responsible for integrating information between the two areas (John, 2000; see Figure 2.1).
According to Ullman (2001), the L1 lexicon is part of declarative memory stored in the temporoparietal area after undergoing processing in the temporal lobe. On the other hand, grammar is stored as part of procedural memory in the left frontal lobe and basal ganglia. Ullman’s theory can be seen as an extension of Paradis’s model of implicit and explicit language acquisition (Paradis, 1994). Implicit learning (acquiring knowledge without being
consciously aware of the process; procedural memory) occurs when an individual is exposed to people who speak a language fluently (e.g., at home). Explicit learning occurs when an individual is introduced to language rules in a formal setting (e.g., school; Paradis, 1994). Based on this, Ullman proposes that L2 grammar is acquired through explicit learning (conscious attempt to acquire knowledge; declarative memory) and therefore, is represented in the temporoparietal regions (interconnecting network of neurons involving temporal and parietal lobes; Ullman, 2001). Evidence in support of this model shows that there is greater activation of the left temporal lobe during L2 usage as compared to L1 usage (Dehaene et al., 1997). Additionally, in a study involving four patients with lesions in their basal ganglia, there was a selective impairment in L1 grammar alone, while L1 lexicon, L2 lexicon, and L2 grammar remained intact (Fabbro & Paradis, 1995).

With the help of the declarative/procedural model, selective recovery of just one language in bilingual individuals following a cerebrovascular incident like stroke can be explained. If L1 and L2 are processed by two distinct regions, loss of functionality in one of the two regions would lead to selective loss of abilities in one language, while the other language ability would be intact. However, the declarative/procedural model does not explain recovery patterns such as alternating antagonistic recovery, wherein use of one language is inversely related to the use of other language, with one of the two languages taking precedence on alternating days (Kciuk, 2009). In alternating antagonism, when an individual speaks one language, use of the other language disappears, and vice versa. This type of recovery contradicts the idea that L1 and L2 are controlled by two different brain regions; L1 and L2 processing must be interconnected for alternating antagonism to be explained.
Therefore, to explain this phenomenon, Green proposed the single dynamic view of bilingual processing, which is discussed below.

2.6.1 Single network model

The single network theory proposes that L1 and L2 share common neural representational network that is dynamic in nature. L2 is acquired with the help of neural representations that are already developed for L1. In order to explain patterns of recovery such as the alternating antagonistic recovery, Green proposed the single network model (Green, 2013). Green’s theory was supported by findings of Sakai, Miura, Narafu, and Muraishi study (2004), where Japanese individuals with no prior training in English were taught formally to convert English verbs from past tense to present tense. Subsequently, they were asked to convert verbs from past tense to present tense in both L1 and L2 (English), as brain activity was evaluated with fMRI. They found the same region to be activated during both L1 and L2 activities. This led to the conclusion that grammar is processed in the same region for both L1 and L2 (Sakai et al., 2004).

Because L1 and L2 share common circuits, Green (2003) proposed a mechanism that governs the L1 and L2 control. In bilingual children, there is an increased ability to selectively attend to a stimulus as compared to monolingual children (Bialystok, 2005). The areas that correspond to executive control and task-switching are at increased activity in bilinguals, and because of these factors, they exercise greater control due to language switching (Bialystok, 2005). This forms the behavioral basis of language control. The neural basis of language control has been linked to the anterior loop, an interconnecting circuit involving the prefrontal cortex, the basal ganglia, and the thalamus (Fabbro, Peru, & Skrap,
The first level of the anterior loop involving prefrontal cortex has been associated with language switching (Kciku, 2009). In a study that involved picture-naming with participants switching languages between pictures, compared to no switching, there was increased activity in the prefrontal cortex (Hernandez, Dapretto, Mazziotta, & Bookheimer, 2001). The second level of anterior loop involving the basal ganglia has been implicated in language switching as well. In a study that involved participants to read word-couples in the same language or two different languages, there was an increase in the basal ganglia activity during language switching (Crinion et al., 2006). The third level of the anterior loop involving the thalamic structures is another component involved in language control. A patient with tumor growth in the thalamus showed inappropriate language mixing (Fabbro et al., 1997). Taken together, it becomes evident that the anterior loop is an important neural circuit involved in language control of mixing and switching.

As seen previously in this section, bilingualism is associated with certain advantages such as improved attentional control and enhanced flexibility to switch between tasks. When the effect of bilingualism on dementia was studied, it was found to favorably delay the disease onset (Bialystok et al., 2007). In fact, bilingualism is increasingly being recognized as a cognitive reserve factor (Gold, 2015). Therefore, in the following section, cognitive advantages of bilingualism in dementia will be discussed.

2.7 Bilingualism as a Cognitive Protective Factor

Bilingualism has been found to be a protective factor in the development of dementia. In their study, Bialystok et al. (2007) reported that bilingualism delays the onset of dementia by 4.1 years as compared to monolinguals. However, the rate of cognitive decline as assessed
by the Mini Mental State Examination (MMSE) was similar between the two groups; scores did not differ statistically over a period of four years from the time of diagnosis (Bialystok et al., 2007). This suggests that bilingualism does not affect the process of neuronal loss and neurotoxicity, but enables the brain to protect itself against these changes thus conferring a delay but not prevention. In light of this finding, bilingualism can be seen as a cognitive reserve (Gold, 2015).

Bilingualism enhances the executive control (EC) function mediated by the frontoparietal (connections between frontal lobe and parietal lobe) and frontostriatal pathways (connections between frontal lobe and basal ganglia) (Gold, 2015). The underlying potential molecular mechanisms facilitated by bilingualism within the EC pathways are described herein. Bilingualism leads to increased neuronal activity within the EC pathways and the corresponding increase in delivery of oxygen and glucose may be beneficial in a cascade fashion (Gold, 2015). Bilingualism-associated increase in neuronal activity increases myelination facilitated by glial cells (cells that surround neurons to provide support and insulation) (Bradl & Lassman, 2010; Gyllensten & Malforms, 1963), which in turn is beneficial to neurons because myelin provides lactate, an energy producing substance (Rinholm, Hamilton, Kessaris, Richardson, Bergersen, & Attwel, 2011; Lee, et al., 2012). Increased lactate metabolism in the neurons leads to the formation of new blood vessels and protection against senility-induced loss of blood supply to the frontostriatal circuits (Schmidtke & Hull, 2005). Additionally, bilingualism has been associated with increased neuron and glial cell interactions. In the bilingual brain, astrocytes (a type of glial cell) form a link between glutamate-releasing neurons and capillaries. Following activity in the glutamate-releasing neuron, glial cell is
activated to result in dilatation of the capillaries, thus increasing blood flow locally (Takano et al., 2006).

Bilingualism facilitated synaptic changes may include preservation of presynaptic terminals and receptors (Petrosini et al., 2009), neuronal branching, and an increase in the numbers of synapses within the EC pathway (Gelfo, De Bartolo, Giovine, Petrosini, & Leggio, 2009; Greenough, Volkmar, & Juraska, 1973). Furthermore, bilingualism modulates the activities of three neurotransmitters, namely dopamine, glutamate, and norepinephrine in the EC circuit (Gold, 2015). Effect on dopamine corresponds to a beneficial effect on executive functions mediated by the prefrontal cortex (Robbins & Arnsten, 2009). Beneficial effects on glutamate are associated with better language control mediated by the thalamus (Fabbro et al., 1997; Sherman, 2014). In addition, bilingualism through its beneficial effect on norepinephrine increases executive control functions such as attention and inhibitory control (Chamberlain, et al., 2007; Grefkes, Wang, Eichkhoff, & Fink, 2010). In summary, bilingualism positively affects the neurons, glial cells, and neurotransmitters in the frontostriatal and frontoparietal pathways (see Figure 2.2).

In Alzheimer’s disease patients who are bilingual, there is a tendency to regression to the L1 (McMurtray et al., 2009). Also, L2 function can deteriorate with normal aging.
(Mendez et al, 1999). The following section discusses L2 attenuation and the factors associated with it.

2.8 Language Function in Dementia and in Healthy Aging Individuals

Although bilingualism is a protective factor in advancing the onset of dementia, it has been reported that L2 loss is an early sign of dementia. In a case report, regression to L1 was reported in two elderly individuals with dementia (McMurtray et al., 2009). One of the explanations for L2 loss in dementia relies on increased cognitive load in bilinguals as compared to monolinguals (Mendez et al., 1999). Therefore, individuals with dementia find it hard to maintain proficiency in two different languages. Even in cognitively health elderly individuals, there is an observed trend for regression to L1 despite lifelong bilingualism (Goral, 2004). Furthermore, aging is associated with cross-language interference, particularly because bilingual individuals cannot separate the two languages completely, there is a possibility of the regressing language to interfere with the language that is in use (L1) (Mendez et al., 1999). Taken together, bilingual individuals are susceptible to L2 loss due to factors such as increased cognitive load and cross-language interference. Therefore, in the present study, L2 deficits in Alzheimer’s disease patients were assessed and compared to healthy elderly individuals. The following section is going to describe the various limitations associated with language decline with a particular emphasis on L2 decline.

2.9 Effects of Language Deficits

Health-related quality of life (HRQL) refers to the impact of a disease condition on the person’s level of satisfaction with their lives. The various dimensions included in the evaluation of HRQL are physical wellness, mental wellness, functioning in the family, and
social performance. In people with language deficits or aphasia, there is a reduction in HRQL corresponding to poor mental health, depression, reduced physical activity levels, and low levels of functional communication (Hilari & Byng, 2009). Apart from negative consequences on the individual, language deficits, particularly in L2, can have effects on caregiving aspects. It may become essential to find a caregiver who knows the native language (Jiji, 2013). This could have implications for immigrants; finding a caregiver who speaks the native language of the patient could become challenging. Additionally, occupational aspects could be affected in immigrants. Another interesting aspect of L2 decline is that it could affect the decision-making process. Thought processes and decision-making depend on two types of mental processes. One type of mental resource is analytic and systematic, while the other one involves emotional and intuitive aspects. Because L2 usage places an increased cognitive load on the mental processes, it is possible to expect that L2 would depend on the emotional and intuitive aspect of thinking and judgment (Keysar, Hayakawa, & An, 2005). However, individuals react less emotionally to words expressed in the L2 as compared to the L1 that convey love, stigma, or reproach (Aycicegi & Harris, 2004; Dewaele, 2004). This shows that L2 is rooted to a lesser extent in the emotional aspect of thinking as compared to L1. This observation is confirmed by the Keysar et al. (2012) study, which found that decision-making is less biased due to reduced dependence on the emotional system when individuals make choices in their L2 as compared to their L1. In this study, participants received $15 at the beginning of the study and were asked to remove $1 during each round of the bet and decide whether to participate in the bet or remain with the $1. The participant had to guess heads or tails as the experimenter flipped a coin; if the guess was right, participants earned $1.5, otherwise they lost $1. Individuals were more open to
taking bets in their foreign language as compared to when the task was presented in their native language. The authors proposed that individuals are very sensitive to loss when they make choices in native language; they call this “myopic loss aversion” (Keysar et al., 2012, p. 7). They suggest that making decisions in one’s L2 could bring investment and savings gains in the long-term (Keyser et al., 2012). The above discussion states the difficulties that may be associated with L2 loss and the following section is going to describe the various factors related to regression to L1.

2.10 Possible Modifiable Factors Associated with L2 Decline in Alzheimer’s Disease

Regression to L1 has been linked to factors such as the age at which the L2 was acquired, L2 proficiency (Hope et al., 2015), and numbers of years spent using the L2 (Pearce, 2005). These factors may be seen as non-modifiable due to their retrospective significance. Therefore, there is a need to identify modifiable factors associated with L2 decline in order to improve the quality-of-life of patients with Alzheimer’s disease.

Probable factors that may be associated with language deficits in Alzheimer’s patients include depression and sleep disturbances. Hypothetical basis for this assumption is that stroke patients with aphasia experience depression (Kauhanen et al., 2000). In addition, children with Down’s syndrome show an increase in sleep disturbances leading to poor language outcomes (Edgin et al., 2015). In fact, in stroke patients with aphasia, the rates of depression are as high as 62-70% (Kauhanen et al., 2000).

Depression is associated with cognitive deficits such as psychomotor retardation and an overall decline in intellectual functioning (Christensen, Griffiths, Mackinnon, & Jacomb, 1997). Furthermore, executive functions such as planning, thinking ahead, and making
strategies mediated by the frontal lobe are susceptible to impairment by depression (Fossati, Ergis, & Allilaire, 2002). In a study that involved patients with depression, a negative correlation between verbal fluency and depression was reported (Fossati, Guillaume, Ergis, Allilaire, 2003). Depressed patients produced fewer words in the semantic task as compared to control subjects. However, the phonemic ability did not vary significantly between the two groups. Because the cluster size (number of words produced under a particular semantic category or cluster) did not vary between the two groups, but depressed patients faced more difficulty to switch between the clusters, the authors concluded that depression affects verbal fluency by impairing executive functions and not by altering semantic memory (Fossati et al., 2003). However, in a meta-analysis involving 42 studies and 2,206 individuals, Henry and Crawford (2005) reported decline in verbal fluency to be associated with the generalized cognitive slowing seen in depression, but not to executive functioning impairment. The authors stated that if verbal fluency in depression is a function of executive control impairment alone, then the extent of decline in phonemic and semantic tasks should be comparable with those having frontal lobe lesions. However, there was a discrepancy between the extent of verbal fluency decline seen in depressed patients versus those with focal frontal lobe lesions; therefore, verbal fluency deficits in depression cannot be linked to frontal lobe functions alone. They proposed that there is an overall decline in cognitive speed that leads to verbal fluency attenuation (Henry & Crawford, 2005). In addition, depression negatively interferes with attention, ability to concentrate, and ability to retrieve (Norris et al., 1995).

In children with Down’s syndrome, greater impairments in lexical and syntactic aspects of language are observed with poor sleep (Edgin et al., 2015). Because Down
syndrome is positively correlated with Alzheimer’s disease (“Down Syndrome and Alzheimer Disease,” n.d., para 1-4), it is plausible to expect some underlying behavioral similarities. Furthermore, a growing body of evidence is suggestive of sleep to correspond to better language outcomes even in healthy individuals. In adults, there is an increased recall of novel words following night-time sleep as compared to an equal period of wake time (Tamminen, Payne, Stickgold, Wamsley, & Gaskell, 2010). Additionally, sleep disturbances interfere with frontal lobe functions and impair verbal fluency (Harrison & Horne, 1997; Horne, 1988). There is an increase in retrieval difficulties when circadian rhythm (sleep-wake cycles) is disturbed (Fekete, van Ree, Niesink, & Wied, 1985). Therefore, based on this discussion, the correlation between L2 deficits, sleep disturbances, and depression was examined in the present study.

2.11 Chapter Summary

Alzheimer’s disease represents a growing health concern in the elderly population. Increased life expectancy along with reduced fertility rates in many parts of the world are associated with a substantial increase in the proportion of older individuals. Alzheimer’s disease pronounces the adverse impact of other co-existing mental and physical disorders and the cost-of-care is significantly high. Additionally, Alzheimer’s disease is associated with significant mortality and morbidity (World Alzheimer Report, 2015).

Although there are numerous symptomatic manifestations of Alzheimer’s disease, challenges in language are associated with increased clinical significance (Ferris & Farlow, 2013). Language impairments manifest as decline in verbal fluency in the early stages of the disease, and it progressively deteriorates until the severe stage (Salvatierra et al., 2007).
Bilinguals are becoming increasingly common (Ansaldo & Saidi, 2014) and therefore, there is a need to include them in language research in order to simulate reality. Bilingualism acts as a cognitive reserve mechanism (Gold, 2015) in delaying the onset of dementia (Bialystok et al., 2007). However, it has been reported that Alzheimer’s disease patients regress to using their L1 if they are bilingual (McMurtray et al., 2009). Even with normal aging, individuals may experience a decline in L2 (Mendez et al., 1999). Therefore, in the present study, L2 deficits as measured by verbal fluency tests were assessed in Alzheimer’s disease patients as compared to healthy elderly individuals. Furthermore, in the present study, an exploration of the possible neuropsychological factors that correlate with L2 deficits was attempted. Depression and sleep disturbance were the candidates under investigation in this study because of their negative impact on language performance (Edgin et al., 2015; Fossati et al., 2003).
Chapter Three

Methodology

3.0 Introduction

This chapter includes information regarding the study design, population, sampling, and data collection techniques. Furthermore, ethical consideration such as confidentiality, consent, assent, and debriefing are discussed.

3.1 Research Design

The objective of the present study was to assess the relationship between L2 deficits, depression, and sleep quality in Alzheimer’s disease patients as compared to cognitively healthy elderly individuals. Because the data were collected from the study participants at a specific point in time rather than following them over a period of time, this study was cross-sectional. The study used inferential statistics, specifically, correlations and planned comparisons.

3.2 Population

Alzheimer’s disease patients in the current study were members of the Alzheimer’s and Dementia Association of Kenya (ADOK; http://www.alzkenya.org/), all of whom were bilingual. Home-visits were performed to collect data from Alzheimer’s patients and their caregivers. Control participants were community-dwelling elderly bilingual volunteers. The control group was matched for age, gender, level of education, and level of bilingual proficiency with the Alzheimer’s group. In order to assess the level of bilingual proficiency, individuals were asked to self-report (control group) or report on behalf of patients.
(Alzheimer’s disease group). A 5-point Likert scale on how well they understood and spoke their L1 and L2 was administered. A score of 1 corresponded to: “cannot understand at all” or “cannot speak at all”, while a score of 5 corresponded to: “native speaker-like proficiency” (Salvatierra et al., 2007; see Appendix A). Participants who could speak and understand both L1 and L2 with a score ≥ 3 to 5 were included in the study.

3.3 Sampling Technique

Study participants were sampled based on convenience. Therefore, the technique was non-probability based and non-randomized. In the control group, snowballing was used as the sampling technique. With respect to the Alzheimer’s group, an invitation to participate in the study was sent to the members of the ADOK by its Chair person, and individuals who consented for their loved ones to participate were contacted.

3.4 Sample Size and Justification

The number of participants was 10 in each group; this is based on a Spanish/English study involving Alzheimer’s patients conducted by Salvatierra et al. (2007) that involved 11 participants in each group.

3.5 Data Collection Strategies and Methods

Patients with an established diagnosis for Alzheimer’s disease from a neurologist were included in the Alzheimer’s group. Additionally, their Mini-Mental Status Examination (MMSE) score at baseline was evaluated. Patients in the control group were assessed with the help of MMSE to rule out cognitive impairment. The sensitivity (ability to correctly exclude those without the condition) of MMSE is 100%, while specificity (ability to correctly
exclude those without the condition) is 55% (Sabe et al., 1993). The MMSE assesses aspects such as orientation to space and time, attention, concentration, memory, calculation, language, and praxis (see Appendix B). Scores on MMSE can range from 0 to 30; a score > 24 is considered to be normal (Folstein, Folstein, & McHugh, 1975). An interview with participants (for the control group) or caregivers (for the Alzheimer’s group) was conducted to gather information such as age, gender, bilingual status, languages used, educational level, and level of bilingual proficiency.

Assessment of verbal fluency was done under the phonemic and semantic categories. The test was administered in English to all participants. In the phonemic task, individuals were asked to produce words that began with F, A, and S during the first, second, and the third minute, respectively. Participants were asked not to include proper names. Each correct response earned 1 point. Words beginning with the same sound but a different letter were marked as incorrect (e.g., phone under letter F). Numbers was considered as a correct response. Final score in the phonemic task was calculated as an average of words produced under each letter. Assessment of semantic ability included a 1-minute trial involving the participants to name as many animals (semantic category) as they could. Identification of a superordinate category (e.g., birds) was considered as a correct response. Subsequently, if for example, “crow” was identified, both birds and crow were scored as correct answers. Additionally, gender discriminants (e.g., cow, bull) and age discriminants (e.g., calf, cow) were allowed. The total score was computed under the semantic category for each participant (Salvatierra et al., 2007). The audio responses were recorded for later scoring. Verbal fluency tests are found to be associated with an inter-rater variability of $r = .98$ (Norris et al., 1995). Test-retest reliability with retesting occurring at 6 months is associated with a reliability co-
efficient of \( r = .74 \) (Ruff et al., 1996). The semantic verbal fluency task has a sensitivity of 68% and specificity of 83% in Alzheimer’s disease (Coen et al., 1999).

Assessment of depression was using the Geriatric Depression Scale-15 (see Appendix C); self-report version was used in the control group, while an informant version was used in the Alzheimer’s group. A score of 0-4 corresponds to no depression, while 5-10 and > 11 connotes mild depression and severe depression, respectively (Sheikh & Yesavage, 1986). Sensitivity and specificity of the GDS-15 in detecting depression in the elderly are 97% and 95%, respectively. Test-retest reliability with retesting occurring within two weeks is associated with Cronbach’s alpha of 0.80 and the inter-rater reliability was found to be \( r = .94 \). Based on these statistics, Nyunt et al. (2009) report that the GDS-15 is a reliable and valid screening tool for depression. Furthermore, in their study, Nyunt et al. (2009) found that GDS-15 can be used irrespective of age, gender, ethnicity, and presence or absence of chronic diseases.

Sleep quality was measured by the Pittsburgh Sleep Quality Index (PSQI; see Appendix D); the questionnaire was self-reported in the control group, while it was reported by informants in the Alzheimer’s group. A score of 5 or greater on the PSQI corresponds to poor quality sleep (Buysse et al., 1988). Test-retest reliability of PSQI score with retesting occurring after 2 days to several weeks was found to be \( r = .87 \). There is a high correlation between PSQI score and sleep log data obtained from sleep diary. PSQI score > 5 is associated with sensitivity of 98.7% and specificity of 84.4% in individuals with sleep disturbances (Backhaus et al., 2002).
3.6 Ethical Issues

3.6.0 Confidentiality

To maintain confidentiality, participants’ identification details were detached from the questionnaires immediately after receiving the responses. A unique identifier was given to the responses in order to ensure anonymity during data analysis.

3.6.1 Consent and Assent

Study participants had the right to express consent or dissent to participate in the study. A consent form (see Appendix E) was given to the participants before collecting any data from them. Signed forms were securely stored in a location only accessed by the principal investigator. Because participants in the Alzheimer’s group were not in a position to provide informed consent, consent from a legally authorized person was obtained (American Psychological Association, 2012).

3.6.2 Debriefing

The study involved no potential risk to participants. However, a debrief form (see Appendix F) was given to study participants, in the event that they experienced any distressing reactions, they were asked to seek psychological support. A list of recommended psychotherapy centers was given to them, if help was needed.
3.7 Chapter Summary

The study involved 10 individuals with Alzheimer’s disease and 10 healthy elderly individuals. The MMSE was administered at baseline in order to assess the level of cognitive functioning. Verbal fluency was assessed by the semantic and the phonemic fluency tasks. Participants were evaluated for depression by the GDS-15, while sleep quality was evaluated with the PSQI. The study adhered to ethical principles pertinent to confidentiality, informed consent (for participants in the control group), obtaining consent from a legal guardian (for participants in the Alzheimer’s group), and debriefing of participants.
Chapter Four

Results

4.0 Introduction

This chapter discusses the participant characteristics such as age, level of education, and the bilingual proficiency of the Alzheimer’s group and the control group. In addition, the baseline MMSE scores of the two groups are presented. Furthermore, comparisons of the depression and the sleep quality scores between the Alzheimer’s group and the control group are discussed. A comparative analysis of the semantic and the phonemic tasks in Alzheimer’s group as well as the control group has been included. Finally, a correlative analysis of depression, sleep quality, and verbal fluency deficits in the two study groups has been discussed.

4.1 Descriptive Statistics

The study involved 10 bilingual individuals diagnosed with Alzheimer’s disease affiliated to the ADOK. The control group comprised of 10 bilingual healthy community-dwelling individuals. The two groups were matched for age, education, and the level of bilingual proficiency. The mean age (with SD) of the Alzheimer’s group and the control group was 69.1 years (10.62) and 62.2 years (11.58), respectively. The mean number of years of education (with SD) in the Alzheimer’s group and the control group were 10.7 years (5.47) and 13.4 years (5.89), respectively. A t-test was performed to compare age and education levels between the two groups. There were no significant differences in age, $t(9) = 1.481, p = .17$, and level of education, $t(9) = 1.003, p = .342$, between the two groups. The degree of
bilingualism was matched by selecting dyads (one individual from Alzheimer’s group and one individual from control group) that had the same score on the proficiency assessment of their first language and second language. All individuals had a score ≥ 3 for their ability to understand and speak first and second languages.

MMSE was performed to assess the level of cognitive functioning. The mean scores (with SD) for the control group and the Alzheimer’s group were 25.9 (2.99) and 4.7 (1.64), respectively. Alzheimer’s patients scored significantly lower on the MMSE as compared to healthy individuals, $t(9) = 9.614, p < .001$.

### 4.2 Sleep Quality in Alzheimer’s Patients and Control Participants

Assessment of sleep habits with PSQI revealed no significant differences between the Alzheimer’s group ($M = 7, SD = 4.13$) and the control group ($M = 5, SD = 2.16$), $t(9) = -1.399, p = .195$. However, component 7 on the PSQI that corresponds to daytime dysfunction varied significantly between the two groups. Alzheimer’s patients ($M = 1.3, SD = 1.15$) showed significantly higher scores for component 7 as compared to the control group ($M = .2, SD = .42$), $t(9) = 2.538, p = .032$. This shows that Alzheimer’s patients experience higher levels of disturbances during the day that are related to sleep quality as compared to healthy older individuals. Between the two questions that comprise component 7, the one that is pertinent to reduced enthusiasm during the day showed higher values as compared to the one that measures daytime sleepiness, $t(9) = -2.905, p = .017$. 
4.3 Depression in the Alzheimer’s Group and the Control Group

Assessment of depression with the GDS-15 revealed that Alzheimer’s patients had higher rates of depression as compared to healthy elderly individuals, \( t(9) = -4.680, p = .001 \), see Figure 4.1. One participant (10%) in the control group showed a value > 4 on GDS-15, while 8 out of 10 participants (80%) in the Alzheimer’s group showed a value > 4. Any value > 4 corresponds to symptomatic depression.

![Figure 4.1](Image)

*Figure 4.1. Mean GDS-15 scores observed in the two study groups. GDS-15 refers to the Geriatric Depression Scale-15 that assesses severity of depression in older individuals.*

4.4 Assessment of Semantic and Phonemic Task Scores in Alzheimer’s Patients and Healthy Elderly Individuals

Assessment of semantic ability using the animal category showed that control participants had higher semantic ability as compared to those in the Alzheimer’s group, \( t(9) = \)
11.513, \( p < .001 \) (see Table 4.1). In addition, scores on the phonemic task varied significantly between the control and the Alzheimer’s groups, \( t(9) = 7.151, p < .001 \) (see Table 4.1).

Furthermore, the number of words produced in the semantic category was higher than the words produced in the phonemic task in the control group, \( t(9) = 3.939, p = .003 \). However, there was no such difference in the Alzheimer’s group, likely because their performance was so weak in both tasks to warrant any difference between tasks.

Table 4.1

*Mean Performance Scores on the Semantic and the Phonemic Tasks of the Alzheimer’s Group and the Control Group*

<table>
<thead>
<tr>
<th>Category</th>
<th>Control group</th>
<th>Alzheimer group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Semantic category</td>
<td>13.5 (1.21)</td>
<td>0.3 (0.21)</td>
</tr>
<tr>
<td>Phonemic category</td>
<td>8.56 (3.26)</td>
<td>0.23 (0.73)</td>
</tr>
</tbody>
</table>

*Note: SD = standard deviation.*

PSQI score did not show a correlation between the semantic and phonemic abilities in the Alzheimer’s group. Similarly, the global PSQI score did not correlate with the semantic and phonemic tasks in the control group. However, in the control group, component 4 of the PSQI that corresponds to habitual sleep efficiency had a significant effect on the semantic task. A two-way analysis of variance (ANOVA) conducted with gender (male vs. female) and sleep efficiency (poor efficiency < 85% vs. good efficiency > 85%) as independent variables and the number of words produced under the semantic category as the dependent variable, showed the main effect of sleep efficiency to be significant \( (F(1,6) = 6.069, p = \)
Higher sleep efficiency corresponded to higher semantic task scores. There was no significant effect for gender on semantic ability and there was no significant interaction between gender and sleep efficiency. There was no correlation between depression, as assessed by GDS-15 and the semantic and phonemic abilities in Alzheimer’s group as well as the control group.

4.5 Chapter Summary

There were no significant differences in the global PSQI scores between the Alzheimer’s group and the control group. However, component 7 of the PSQI that corresponds to daytime dysfunction, showed significantly higher values in the Alzheimer’s group. Depression as assessed by the GDS-15 was significantly higher in the Alzheimer’s group. Both the semantic and the phonemic task scores were significantly higher in the control group as compared to the Alzheimer’s group. In the Alzheimer’s group, there was no difference between the semantic and the phonemic abilities; however, individuals in the control group scored significantly higher on the semantic task as compared to the phonemic task. There was no correlation between sleep quality, depression, and verbal fluency attenuation in the Alzheimer’s group as well as the control group. Nevertheless, sleep efficiency as recorded by component 4 of the PSQI showed a significant main effect on semantic ability in the control group.
Chapter Five

Discussion, Conclusions, and Recommendations

5.0 Introduction

Alzheimer’s disease significantly impairs language abilities and evidence shows verbal fluency impairments to be one of the early signs of language attenuation (Ferris & Farlow, 2013). However, language difficulties manifest in healthy aging individuals as well (Goral, 2004). Therefore, the present study evaluated the extent of verbal fluency deficits in Alzheimer’s disease patients as compared to healthy elderly individuals. Furthermore, language impairments are linked to depression as well as sleep disturbances. Studies have shown sleep as opposed to wakefulness to increase the ability to recall novel words (Tamminen, 2010). Additionally, sleep disturbances impair the ability to retrieve appropriate words (Fekete et al., 1985) and negatively impact verbal fluency (Harrison & Horne, 1997; Horne, 1998). Therefore, the current study evaluated the association between sleep disturbances and verbal fluency in Alzheimer’s disease patients. As observed with sleep, depression interferes negatively with language outcomes as well. Depression diminishes verbal fluency, ability to retrieve words, attention, and concentration (Henry & Crawford, 2005; Norris et al., 1995). Lower rate of cognitive processing seen in depression is one of the factors implicated in reduced language functions (Henry & Crawford, 2005). Therefore, the study evaluated the correlation between depression and verbal fluency deficits seen in Alzheimer’s patients.
5.1 Summary of Findings

The current study did not find a correlation between depression and verbal fluency in Alzheimer’s disease patients as well as control participants. Additionally, there was no correlation between sleep disturbances and verbal fluency in the Alzheimer’s group and the control group. However, the rate of depression was significantly higher in the Alzheimer’s group as compared to the control group. The global PSQI scores did not vary between the control and Alzheimer’s group; however, the PSQI sub-component corresponding to daytime dysfunction showed higher values in Alzheimer’s patients. Further analysis of this sub-component revealed daytime dysfunction to be significantly contributed by reduced daytime enthusiasm as compared to daytime sleepiness. Higher rate of depression and reduced daytime enthusiasm reveal an underlying affect dysregulation in Alzheimer’s patients. Additionally, an analysis of the semantic and phonemic task scores in the Alzheimer’s group showed both the abilities to be affected to comparable extent. On the other hand, individuals in the control group scored significantly higher on the semantic task as compared to the phonemic task. Although there was no correlation between global PSQI score and verbal fluency measures in the control group, sleep efficiency as evaluated by component 7 of the PSQI had a significant main effect on the semantic ability.

5.2 No Correlation between Depression, Sleep Disturbances, and Verbal Fluency Tasks in Alzheimer’s Group

There was no correlation between depression scores and PSQI scores and verbal fluency tasks in the Alzheimer’s group in the present study. A possible explanation for this observation could be that the output in semantic and phonemic tasks was very low in the Alzheimer’s group. Patients with higher levels of verbal fluency may be evaluated in order to
determine if a correlation exists between depression, sleep, and verbal fluency. In addition, the small sample size of the study would have been a factor for not observing a correlation between the aforementioned variables.

**5.3 Increased Daytime Dysfunction and Depression in the Alzheimer’s Group**

Although the global PSQI scores did not vary significantly between the Alzheimer’s group and the control group, there was a significantly higher level of daytime dysfunction in Alzheimer’s patients. Within the daytime dysfunction domain of the PSQI, there was a significant difference between daytime enthusiasm and daytime sleepiness. Individuals scored significantly higher on reduced enthusiasm levels as compared to daytime sleepiness. This shows that dysfunction during the day is likely to be contributed by mood dysregulation rather than sleepiness. In fact, this finding corroborates well with the high rate of depression observed in the Alzheimer’s group. The mean score for depression as evaluated by the GDS-15 was significantly higher in the Alzheimer’s group as compared to the control group.

The rate of depression observed in the present study was very high (80%). Prevalence rates of major depression in Alzheimer’s patients as reported elsewhere vary between 4.8% and 50% depending upon the assessment tool and the diagnostic criteria (Modrego, 2010). Higher rate of occurrence of depression in the present study could be due to the fact that the mean MMSE score was 4.7, which corresponds to severe cognitive impairment. A study evaluating the rate of occurrence of depression as a function of the severity of cognitive impairment would confirm the above proposition. With severe cognitive impairment, day-to-day activities would be significantly adversely affected and this can be challenging to Alzheimer’s patients (Ferris & Farlow, 2013). Furthermore, their ability to communicate
reduces drastically, which can be observed from the mean semantic task and phonemic task scores in the present study. Therefore, Alzheimer’s patients may find it challenging to express their feelings verbally. Increased dependence on others for everyday activities and significantly reduced verbal fluency may be seen as the contributing factors for the high rate of depression.

Depression can be debilitating to the Alzheimer’s patients and their caregivers. It reduces the quality-of-life of the affected individuals and can negatively interfere with the psychological well-being of their caregivers. In addition, depression affects mental functioning, day-to-day activities, and induces behavioral changes such as wandering and aggression that are particularly problematic in Alzheimer’s patients (Modrego, 2010). Furthermore, comorbid depression in Alzheimer’s disease pronounces the debilitating effect of Alzheimer’s disease when compared to those patients who do not have depression (Ronver et al., 1989). Neurobiological basis for depression in Alzheimer’s disease is linked to reduced levels of acetyl choline and monoamines such as serotonin and norepinephrine (Modrego, 2010). Although reduced levels of acetyl choline in the brain are typically related to reduced cognitive performance, there is evidence to support that decreased acetyl choline contributes to psychiatric symptoms such as aggression, apathy, anxiety, depression, and sleep disturbances (Cummings & Back, 1998). Another study found reduced number of serotonergic neurons in certain brain regions in Alzheimer’s patients with depression as compared to healthy elderly individuals. An additional neurobiological explanation for depression in Alzheimer’s disease is based on reduced levels of norepinephrine; in fact, a study reported that there is a 10% to 20% reduction in norepinephrine levels in cerebral cortices of dementia patients with comorbid depression as compared to healthy control
participants (Zubenko, Moossy, & Kopp, 1990). Increased presence of neurofibrillary tangles accounts for another factor related to depression in Alzheimer’s patients (Wilson et al., 2003).

Treatment of depression in Alzheimer’s patients includes drug therapies and non-drug approaches. Drugs include selective serotonin reuptake inhibitors (SSRIs) such as sertraline, citalopram, and fluoxetine. Psychotherapy represents another option for the treatment of depression (Modrego, 2010). Behavioral therapy involving interventions for Alzheimer’s patients and their caregivers has been found to be useful. Creating pleasant events for patients and increasing problem solving abilities in caregivers was beneficial (Teri, Logsdon, Uomoto, & McCurry, 1997). In another study, exercise for patients in combination with support for caregivers was associated with improved depression symptoms as compared to control treatment that involved no intervention (Teri et al., 2003). Usefulness of cognitive therapy in the treatment of depression in Alzheimer’s patients has been evaluated by a meta-analysis. This report suggested cognitive therapy to be associated with improvement in both depressive symptoms and everyday activities (Sitzer, Twanley, & Jeste, 2006).

Given the very high rate of occurrence of depression in Alzheimer’s patients with severe cognitive impairment, there is an important need to both screen Alzheimer’s patients for depression and formulate effective interventions. Challenges that may be encountered while formulating treatment interventions in this subset of patients would be their significantly reduced ability to communicate feelings and reduced ability to carry out activities such as physical exercise. Therefore, interventions need to be focused more on caregivers and to create a pleasant atmosphere around the patient for improved outcomes to accrue (Teri et al., 1997).
5.4 Equivalent Deterioration of Semantic and Phonemic Abilities in Alzheimer’s Patients

Individuals in the Alzheimer’s group produced significantly fewer words under the semantic and phonemic categories as compared to the control group. This replicates other findings that show reduced verbal fluency in Alzheimer’s patients (Salmon et al., 1999; Salvatierra et al., 2007). However, in the present study, both semantic and phonemic scores did not vary significantly in Alzheimer’s patients. This is contrary to the findings that Alzheimer’s patients experience greater difficulty with semantic task as compared to the phonemic task (Salvatierra et al., 2007). This difference in the semantic and the phonemic task abilities may be related to the differences in the extent of cognitive functioning observed in the two studies. The mean MMSE score was 21.2 in the Salvatierra et al. (2007) study and in the present study, the mean MMSE score was 4.7. Salmon et al. (1999) in their 3-year longitudinal study found the rate of decline in semantic ability to be higher than the rate of deterioration of phonemic ability and the difference between the two abilities reduced gradually. When the findings from the present study are combined with those of Salmon et al. (1999) and Salvatierra et al. (2007), it can be speculated that as Alzheimer’s disease progresses, the phonemic representations in the brain are lost as well, leading to the rate of decline in the two abilities to reach a plateau. Studies that examine the cut-off MMSE score at which both the phonemic and semantic abilities level off may confirm this explanation.

Another possible explanation for reduction in the phonemic and semantic abilities could rely on deficits associated with word retrieval rather than the loss of phonemic and semantic representations. However, this explanation is not favored by existing data on gradual deterioration of semantic representations in the brain. In a study conducted by Martin
and Fedio (1983), individuals in the Alzheimer’s group produced fewer words under each semantic category (e.g., vegetables) when compared to healthy control participants. Hodges, Salmon, and Butters (1992) found that irrespective of the word retrieval condition, there was no change in the semantic task output. Further support in favor of gradual deterioration of the semantic representation as opposed to a retrieval deficit is the finding from Salmon et al. (1999) study that shows a consistent pattern in the failure to produce words under specific categories in Alzheimer’s disease patients. In this study, once individuals failed to produce a word that was previously preserved under a specific category, they did not produce it in the subsequent evaluations (the study involved one baseline evaluation and three annual evaluations). Based on this, it has been proposed that deterioration in the semantic task in Alzheimer’s disease is independent of the word retrieval methods (Salmon et al., 1999).

The findings from the Salvatierra et al. (2007) study when combined with findings from the Melrose et al. (2009) study give a directional idea of the brain regions affected during the course of Alzheimer’s disease. Temporal lobe regions and inferior frontal gyrus are associated with semantic functions, whereas left inferior frontal gyrus alone is associated with phonemic functions (Melrose et al. 2009). Based on this, the direction of verbal fluency loss (semantic loss → phonemic loss) may be related to temporal region atrophy → atrophy of the left frontal gyrus.

5.5 Increased Output in the Semantic Category in Healthy Individuals

Individuals in the control group scored significantly higher on the semantic task as compared to the phonemic task. A possible account for such an observation would be that phonemic task places a greater demand on the retrieval strategies (Shao, Janse, Visser, &
Meyer, 2014). In their paper, Shao et al. (2014) propose that although semantic and phonemic fluency tasks are similar, they are associated with differences in retrieval process. Semantic fluency depends on knowledge that is related to our day-to-day activities such as buying fruits or vegetables from a supermarket. Individuals can access semantic information with the help of cluster labels, cluster content, and related clusters (e.g., animals and birds). However, accessing information from a phonemic cluster does not occur on a day-to-day basis and individuals should exercise control to suppress associated words (e.g. phone or phony under the F category) and use novel strategies to access the phonemic information (Shao et al., 2014). In addition, available language resources or vocabulary size have an effect on the phonemic task, but not on the semantic task (Luo, Luk, & Bialystok, 2010). In patients with amyotrophic lateral sclerosis, there is increased reduction in phonemic ability as compared to semantic ability as well as a reduction in the prefrontal cortex activity (Quinn et al., 2012). Based on this finding, phonemic ability can be seen as an executive function. Although Shao et al. (2014) study and Salmon et al. study (1999) recorded a numerical difference between the semantic and phonemic task scores in healthy older individuals (with semantic score being higher), the difference did not reach statistical significance. While Clark et al. (2010) reported reduced phonemic ability relative to semantic ability in healthy older individuals, Tomer and Levin (1999) found older healthy individuals to be associated with significantly worse semantic task score relative to phonemic task score. This shows that there is conflicting evidence regarding the differential semantic and phonemic task abilities in healthy individuals. The present study results are in support of the aforementioned line of thought pertinent to increased cognitive demand associated with phonemic task.
5.6 Increased Sleep Efficiency and Improved Semantic Task Scores

The positive effect of habitual sleep efficiency on semantic task ability in healthy older individuals as observed in the present study could be explained by the various beneficial effects of sleep on memory. There is a large body of evidence to support that sleep affects memory processing. Memory consolidation (conversion of short-term memory to long-term memory) has been shown to be positively affected by slow-wave sleep when brain activity is significantly reduced (Plihal & Born, 1999). Also, increased slow-wave sleep corresponds to increased memory performance during the wakeful period (Durant et al., 2013; Wilhelm et al., 2011). Another important component of sleep, the sleep spindles (small positive and negative oscillations on the EEG that occur during non-REM sleep), have a beneficial effect on declarative (Clemens et al., 2005) and procedural tasks (Fogel & Smith, 2006) because of increased overnight memory retention. Direct causal association between slow-wave sleep and sleep spindles and memory performance have been reported as well. A study that externally induced slow-waves with the help of electrical stimulation found increased slow-waves to lead to increased retention of word pairs learned before sleep (Marshall et al., 2006). In a study that involved drug-induced increase in spindle activity, there was an increase in verbal memory scores during the wakeful period (Mednick et al., 2013).

Apart from the beneficial effect of sleep on memory performance, there is evidence that sleep promotes the formation, reorganization, and strengthening of semantic memory (these processes are together called as semanticization) (Hennies, 2014). Semanticization involves grouping of objects or concepts based on the regularities they have with the objects
or concepts already stored under a category. This assimilation of new information into already existing semantic information (semantic clusters) emerges from episodic memory. For example, when an individual sees a car for the first time in his/her life, the information (color, size, shape, etc.) is stored as an episodic memory. Subsequently, when he/she sees another car of a different color, the second episode gets clustered with the first episode because of statistical regularities of shape and size. With time, the cluster size increases and the cluster gets reorganized, and strengthened (Hennies, 2014).

Sleep facilitates the extraction of regularities from concepts presented and frame a rule that would help in accomplishing a task. In a study that involved a number reduction task, a rule to solve the problem could be gained if one is able to infer regularities presented in the task. Sleep, but not wakefulness, increased the participants’ ability to find the hidden rule; this suggests that semantic memory undergoes processing during sleep (Wagner et al., 2004). In addition, sleep facilitates the unification of distantly related concepts into an integrated memory representation. In a study that involved the following connected sequence (1>2>3>4>5), but presented the participants with two elements at a time (1>2, 2>3, 3>4, 4>5), noted sleep to affect the process of linking concepts. Sleep facilitated the extraction of the overall order from pairwise data presented in this study (Ellenbogen et al., 2007). When new words are learned, sleep facilitates its integration into the existing concepts and the brain activity is more (increased slow waves and spindles) if the word is clustered in a category that is less dense as compared to clusters that are very dense (Tamminen et al., 2013). Based on the above discussion, it can be seen that sleep plays a beneficial role in semanticization. In addition, poor sleep efficiency is linked to increased cortisol levels and psychological stress.
(Massar, Liu, Mohammad, & Chee, 2017). Therefore, another account for improved semantic score could rely on the triangle involving cortisol, stress, and sleep efficiency.

5.7 Conclusions

The current study found no correlation between depression, sleep disturbances, and verbal fluency tasks in Alzheimer’s patients. This could have been because of the significantly low levels of phonemic and semantic outputs in the Alzheimer’s group. However, the rate of depression in this group was very high, which suggests that severe cognitive impairment could pronounce the symptoms of depression. Depression can be debilitating to both the affected individuals as well as their caregivers (Modrego, 2010). Screening and effective treatment of depression in Alzheimer’s patients with severe cognitive impairment are needed.

Alzheimer’s patients showed significantly reduced scores on both the semantic and phonemic tasks as compared to those in the control group. This finding is consistent with other reports that suggest a decline in verbal fluency during the course of Alzheimer’s disease. However, in patients with Alzheimer’s disease, the phonemic ability is higher than the semantic ability (Salvatierra et al., 2007). The current study did not report a statistical difference between the two verbal fluency measures. This observation could be attributed to the extent of cognitive impairment seen in the two studies. With severe cognitive impairment as seen in the present study, both the semantic and the phonemic abilities deteriorate to such an extent that further impairment is not possible. Furthermore, in healthy older individuals, there was a significant difference between the semantic and phonemic task scores. Although there is conflicting data regarding the differential abilities on semantic and phonemic tasks in
healthy individuals, the results of the present study support that the phonemic task is
cognitively complex than the semantic task. Furthermore, higher sleep efficiency increased
the semantic task scores in control participants. Future studies that evaluate the protective
role of sleep on semanticization and the neuroendocrine mechanism involving cortisol, stress,
and sleep efficiency and its effect on the semantic ability may increase our understanding of
the relationship between sleep and semantic fluency.

5.8 Recommendations

The present study shows that the rate of depression is higher in Alzheimer’s disease
patients. Therefore, there is an important to detect depression in this patient group and
provide effective interventions for improved quality-of-life effect to accrue. Higher semantic
scores in healthy individuals in their L2 as compared to the phonemic task show the
cognitive complexity associated with the phonemic task. It would be interesting to examine if
phonemic and semantic abilities vary when individuals respond in their L1 and L2 and the
factors that may underlie such differences. Furthermore, an exploration of the
neurobiological factors that may contribute to increased semantic ability involving increased
sleep efficiency may enhance our understanding of the protective role of sleep in the process
of semanticization.

5.9 Chapter Summary

In the current study, there was no correlation between verbal fluency and both
depression and sleep disturbances in the Alzheimer’s group. However, Alzheimer’s patients
showed higher rates of depression as compared to control group participants. Although there
was no correlation between verbal fluency, depression, and sleep disturbances in control
group participants, habitual sleep efficiency (one of the seven components of sleep quality assessed by the PSQI) had a significant main effect on the semantic task. Additionally, semantic ability was significantly higher than the phonemic ability in control group participants. In the Alzheimer’s group, the extent of deterioration of the phonemic and the semantic abilities were comparable.
References


Appendix A: Bilingual Proficiency Data and Biographical Information

Age: ______
Gender: ______
Number of years of formal education (from class I): __________
Highest education: _____________________
Languages used: _____________________
Degree of fluency in (Circle those that apply):

First language

Ability to understand

1. Virtually nothing (Cannot understand at all)
2. Limited (Can understand sentences if spoken slowly and repeated if needed; vocabulary limited to a few basic words (e.g., greetings, food, enquiring for directions))
3. Relatively well (Can understand nearly everything thing spoken by a native speaker)
4. Quite well (Can understand native speakers even when they speak quickly using colloquial language)
5. Excellent (Language is exactly like a native speaker)

Ability to speak

1. Virtually nothing (Cannot speak at all)
2. Limited (Can speak basic words and sentences)
3. Relatively well (Can talk to native speakers about everyday activities. Grammar is fairly good)
4. Quite well (Can find words for everything one intends to say; grammar is good)
5. Excellent (Can speak like a native speaker)

**Second language**

Ability to understand

1. Virtually nothing (Cannot understand at all)
2. Limited (Can understand sentences if spoken slowly and repeated if needed; vocabulary limited to a few basic words (e.g., greetings, food, enquiring for directions))
3. Relatively well (Can understand nearly everything thing spoken by a native speaker)
4. Quite well (Can understand native speakers even when they speak quickly using colloquial language)
5. Excellent (Language is exactly like a native speaker)

Ability to speak

1. Virtually nothing (Cannot speak at all)
2. Limited (Can speak basic words and sentences)
3. Relatively well (Can talk to native speakers about everyday activities. Grammar is fairly good)
4. Quite well (Can find words for everything one intends to say; grammar is good)
5. Excellent (Can speak like a native speaker)
### Appendix B: Mini Mental State Examination

<table>
<thead>
<tr>
<th>Maximum Score</th>
<th>Patient’s Score</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td></td>
<td>“What is the year? Season? Date? Day? Month?”</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td>“Where are we now? State? County? Town/city? Hospital? Floor?”</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>The examiner names three unrelated objects clearly and slowly, then the instructor asks the patient to name all three of them. The patient’s response is used for scoring. The examiner repeats them until patient learns all of them, if possible.</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td>“I would like you to count backward from 100 by sevens.” (93, 86, 79, 72, 65, …) Alternative: “Spell WORLD backwards.” (D-L-R-O-W)</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>“Earlier I told you the names of three things. Can you tell me what those were?”</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>Show the patient two simple objects, such as a wristwatch and a pencil, and ask the patient to name them.</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td>“Repeat the phrase: ‘No ifs, ands, or buts.’”</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>“Take the paper in your right hand, fold it in half, and put it on the floor.” (The examiner gives the patient a piece of blank paper.)</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td>“Please read this and do what it says.” (Written instruction is “Close your eyes.”)</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td>“Make up and write a sentence about anything.” (This sentence must contain a noun and a verb.)</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td>“Please copy this picture.” (The examiner gives the patient a blank piece of paper and asks him/her to draw the symbol below. All 10 angles must be present and two must intersect.)</td>
</tr>
</tbody>
</table>

| 30 | TOTAL |

79
A Guide to Interpreting MMSE Scores

<table>
<thead>
<tr>
<th>Method</th>
<th>Score</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single Cutoff</td>
<td>&lt;24</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Range</td>
<td>&lt;21</td>
<td>Increased odds of dementia</td>
</tr>
<tr>
<td></td>
<td>&gt;25</td>
<td>Decreased odds of dementia</td>
</tr>
<tr>
<td>Education</td>
<td>21</td>
<td>Abnormal for 8th grade education</td>
</tr>
<tr>
<td></td>
<td>&lt;23</td>
<td>Abnormal for high school education</td>
</tr>
<tr>
<td></td>
<td>&lt;24</td>
<td>Abnormal for college education</td>
</tr>
<tr>
<td>Severity</td>
<td>24-30</td>
<td>No cognitive impairment</td>
</tr>
<tr>
<td></td>
<td>18-23</td>
<td>Mild cognitive impairment</td>
</tr>
<tr>
<td></td>
<td>0-17</td>
<td>Severe cognitive impairment</td>
</tr>
</tbody>
</table>
### Appendix C: Geriatric Depression Scale-15

<table>
<thead>
<tr>
<th>No.</th>
<th>Question</th>
<th>Answer</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Are you basically satisfied with your life?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Have you dropped many of your activities and interests?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Do you feel that your life is empty?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Do you often get bored?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Are you in good spirits most of the time?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Are you afraid that something bad is going to happen to you?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Do you feel happy most of the time?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Do you often feel helpless?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Do you prefer to stay at home, rather than going out and doing new things?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>Do you feel you have more problems with memory than most people?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>Do you think it is wonderful to be alive?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>Do you feel pretty worthless the way you are now?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>Do you feel full of energy?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>Do you feel that your situation is hopeless?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>Do you think that most people are better off than you are?</td>
<td><strong>YES / NO</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>TOTAL</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*(Sheikh & Yesavage, 1986)*
Appendix D: Pittsburgh Sleep Quality Index

Instructions:
The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, when have you usually gone to bed at night?
   USUAL BED TIME ________________

2. During the past month, how long (in minutes) has it usually take you to fall asleep each night?
   NUMBER OF MINUTES ________________

3. During the past month, when have you usually gotten up in the morning?
   USUAL GETTING UP TIME ________________

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)
   HOURS OF SLEEP PER NIGHT ________________

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you...
   (a) Cannot get to sleep within 30 minutes
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (b) Wake up in the middle of the night or early morning
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (c) Have to get up to use the bathroom
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (d) Cannot breathe comfortably
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (e) Cough or snore loudly
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (f) Feel too cold
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (g) Feel too hot
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (h) Had bad dreams
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
   (i) Have pain
      Not during the past month ______ once a week ______ twice a week ______ times a week ______
(i) Other reason(s), please describe __________________________________________

How often during the past month have you had trouble sleeping because of this?
Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________

6. During the past month, how would you rate your sleep quality overall?
Very good ________
Fairly good ________
Fairly bad ________
Very bad ________

7. During the past month, how often have you taken medicine [prescribed or "over the counter"] to help you sleep?
Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?
Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?
No problem at all ________
Only a very slight problem ________
Somewhat of a problem ________
A very big problem ________

10. Do you have a bed partner or roommate?
No bed partner or roommate ________
Partner/roommate in other room ________
Partner in same room, but not same bed ________
Partner in same bed ________

If you have a roommate or bed partner, ask him/her how often in the past month you have had...
(a) Loud snoring
Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________

(b) Long pauses between breaths while asleep
Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________

(c) Legs twitching or jerking while you sleep
Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________

(d) Episodes of disorientation or confusion during sleep
Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________

(e) Other restlessness while you sleep; please describe __________________________________________

Not during the __________ past month ________ Less than ________ Once or ________ Three or more ________
year ________ once a week ________ twice a week ________ times a week ________
Appendix E: Sample Consent Form

I am Ambika Shivashanmugam, a graduate student at United States International University-Africa, where I am pursuing a Master of Arts in Clinical Psychology. As part of my degree requirements, I am completing a research study and I would like to include you in the study. Dr. Dana Basnight-Brown, my research supervisor at USIU-A, may be contacted by email at dbrown@usiu.ac.ke, if you have any questions at any time. I can be contacted by email at ashivashanmugam@usiu.ac.ke or phone 0704892588.

Your written consent is required so that I can confirm that you have been informed of the study and that you agree to participate. You are free to decline or discontinue your participation at any time during the study, if you wish to do so. All information obtained in this study will be kept confidential; a number will be assigned to any research questionnaire to ensure that your privacy is protected. Your name or identity will not be given in any report or publication.

The purpose of the study is to understand factors associated with language difficulties in Alzheimer’s disease patients. You will be asked to complete questionnaires that assess your cognitive abilities, verbal fluency, symptoms of depression (if any), and sleep disturbances (if any). This is not an exam or a test and there are no right or wrong answers, simply answer the questions as honestly as you can. The questionnaires may take about 30 minutes to complete in one sitting.

The outcome of the information obtained during this research will be summarized and utilized in my thesis study. Participant names will not be utilized; a number will be assigned
to ensure the participant’s identity. I ensure that personal information will be kept confidential during and after this study is completed.

Consent to Participate:

By signing below, I consent to participate in this study.

______________________________                       _________
(Signature of Participant)                        (Date)

_____________________________                      ___________
(Principal Researcher)                                                       (Date)

Participant Number to be used on all other documents: ______________
Appendix F: Sample Participant Debrief Form

Thank you for participating in this research study. The purpose of this study is to examine if language deficits experienced by Alzheimer’s disease patients can be positively modified by altering factors such as depression and sleep disturbances. Your participation will help researchers gain more insight into language difficulties experienced by Alzheimer’s disease patients.

In the event that you experience any emotional distress due to the questions presented to you in this study, you may want to seek further support. A list of referrals has been included below for your information.

Thank you once again for your participation.

Sincerely,

Ambika Shivashanmugam

0704892588/ashivashanmugam@usiu.ac.ke
# Referral Contacts:

<table>
<thead>
<tr>
<th>Amani Counselling Center</th>
<th>Oasis Africa Counseling Center and Training Institute</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Head Office</strong></td>
<td></td>
</tr>
<tr>
<td>Mbagathi Way, Off Langata Road</td>
<td>Regent Court, Suite B7</td>
</tr>
<tr>
<td>P.O. Box 41738 - 00100, Nairobi, Kenya.</td>
<td>Argwings Kodhek Rd,</td>
</tr>
<tr>
<td><strong>Tel:</strong> +254 02 6002672/3</td>
<td>Opp. Nairobi Women’s Hospital</td>
</tr>
<tr>
<td><strong>Fax:</strong> +254 02 6002674</td>
<td>Hurlingham, Nairobi, Kenya</td>
</tr>
<tr>
<td><strong>Cell Phone:</strong> Safaricom: 0722 626 590; Airtel: 0733 263 870</td>
<td><strong>Phone:</strong> 254-725 366614/254-733 366614</td>
</tr>
<tr>
<td><strong>E-mail:</strong> <a href="mailto:info@amanicentre.org">info@amanicentre.org</a></td>
<td><strong>Website:</strong> <a href="http://www.oasisafrica.co.ke">www.oasisafrica.co.ke</a></td>
</tr>
<tr>
<td><strong>Town Office</strong></td>
<td></td>
</tr>
<tr>
<td>Nairobi CBD Office, KCS House 7th Floor</td>
<td></td>
</tr>
<tr>
<td>Mama Ngina Street</td>
<td></td>
</tr>
<tr>
<td><strong>Cell Phone:</strong> Safaricom: 0718 225 627; Airtel: 0733 388 200</td>
<td></td>
</tr>
<tr>
<td><strong>SMS Line:</strong> 0722 797 068</td>
<td></td>
</tr>
</tbody>
</table>
Appendix G: Sample Legal Guardian Consent Form

I am Ambika Shivashanmugam, a graduate student at United States International University-Africa, where I am pursuing a Master of Arts in Clinical Psychology. As part of my degree requirements, I am completing a research study and I would like to include you in the study. Dr. Dana Basnight-Brown, my research supervisor at USIU-A, may be contacted by email at dbrown@usiu.ac.ke, if you have any questions at any time. I can be contacted by email at ashivashanmugam@usiu.ac.ke or phone 0704892588.

Your written consent is required so that your loved one can participate in the study and I can confirm that you have been informed of the study and that you agree for your loved one to participate. You are free to decline or discontinue your loved one’s participation at any time during the study if you wish to do so. All information obtained in this study will be kept confidential; a number will be assigned to any research questionnaire to ensure that the privacy of your loved one is protected. Your loved one’s name or identity will not be given in any report or publication.

The purpose of the study is to understand factors associated with language difficulties in Alzheimer’s disease patients. You will be asked to complete questionnaires that assess your loved one’s cognitive abilities, verbal fluency, symptoms of depression (if any), and sleep disturbances (if any). This is not an exam or a test and there are no right or wrong answers, simply answer the questions as honestly as you can. The questionnaires may take about 30 minutes to complete in one sitting.

The outcome of the information obtained during this research will be summarized and utilized in my thesis study. Participant names will not be utilized; a number will be assigned
to ensure the participant’s identity. I ensure that personal information will be kept confidential during and after this study is completed.

Consent to Participate:

By signing below, I consent for my loved one to participate in this study.

_____________________________  ___________
(Signature of the legal guardian)  (Date)

_____________________________
(Name of the patient)

_____________________________  ___________
(Principal Researcher)  (Date)

Participant Number to be used on all other documents: ______________
Appendix H: Invitation Letter to the ADOK

To,

The Chairman,

Alzheimer’s and Dementia Association of Kenya

Nairobi, Kenya

Dear Elizabeth,

I am Ambika Shivashanmugam, a graduate student at United States International University-Africa, where I am pursuing a Master of Arts in Clinical Psychology. As part of my degree requirements I am completing a research study and I would like to include Alzheimer’s patients in the study. Dr. Dana Basnight-Brown, my research supervisor at USIU-A, may be contacted by email at dbrown@usiu.ac.ke, if you have any questions at any time. I can be contacted by email at ashivashanmugam@usiu.ac.ke or phone 0704892588.

I request your permission to work with your members on a study that is going to explore language difficulties in Alzheimer’s disease. Participants (patients and caregivers/legal guardians) will be asked to complete questionnaires that assess the patients’ cognitive abilities, verbal fluency, symptoms of depression (if any), and sleep disturbances (if any). The questionnaires may take about 30 minutes to complete in one sitting.

Participants will be free to decline or discontinue their participation at any time during the study, if they wish to do so. All information obtained in this study will be kept confidential; a number will be assigned to any research questionnaire to ensure that the
privacy of the participant is protected. Participant name or identity will not be given in any report or publication.

The outcome of the information obtained during this research will be summarized and utilized in my thesis study. Participant names will not be utilized; a number will be assigned to ensure the participant’s identity. I ensure that personal information will be kept confidential during and after this study is completed.

I sincerely look forward to receiving your permission to conduct this study.

Sincerely,

Ambika Shivashanmugam

0704892588/ ashivashanmugam@usi.ac.ke
Appendix I: IRB Approval Letter

14th March 2017,

USIU-A/IRB/17/S05
Ambika Shivashanmugam,
Master of Arts in Clinical Psychology,
Student ID NO: 644227
Email: ambika.shivashanmugam84@gmail.com

IRB-RESEARCH APPROVAL.

The USIU-A IRB has reviewed and granted ethical approval for the research proposal titled ‘The Association between Verbal Fluency Deficits in Second Language and Depression and quality of sleep among Alzheimer’s Disease Patients.’ The approval is for six months from the date of IRB. Please submit a completed copy of the study to the IRB office, soft copy is acceptable.

You are advised to follow the approved methodology and report to the IRB any serious, unexpected and related adverse events and potential unanticipated problems involving risks to subjects or others.

Should you or study participants have any queries regarding IRB’s consideration of this project, please contact irb@usiuc.ac.ke.

For Chair,

Prof. Dumary Sikalich,
Chair | IRB | USIU-Africa,
d.sikalich@usiuc.ac.ke
Office 20 3605 112.

CC: Research Office