

Chapter One

Introduction

1.1 Background to the Study

The role of diet and nutritional factors in brain aging is attracting much research attention, and midlife dietary habits play a critical role in shaping cognitive health later in life. A growing body of evidence links dietary habits and pattern with cognitive impairment as a persistent and advancing condition marked by cognitive deterioration, drawing significant global health attention in recent years¹. It currently impacts approximately 55 million individuals around the world, with projections indicating a threefold rise by 2050². This neurodegenerative disorder imposes substantial economic strain on individuals, families, and healthcare infrastructures, effects that are particularly acute in low- and middle-income nations such as Nigeria^{2,3}.

In Nigeria, various investigations have highlighted the growing prevalence and health complications associated with cognitive disorders, particularly among adults in their middle age and older⁴. Notably, a systematic review assessing cognitive decline rates in Nigeria found that both incidence and reported cases have increased significantly over the last 20 years, while responses and interventions remain inadequate⁵. The Ibadan Study of Ageing (ISA) group, known for its extensive research in south-western Nigeria, has made notable contributions to understanding the relationship between cognitive health and dietary habits in that region^{6, 7, 8}. However, current epidemiological data from the other five geopolitical zones in Nigeria remain largely unavailable.

Population-based investigations into cognitive health within Nigeria have been sporadic and inconsistent, a concern that was also highlighted across low- and middle-income nations⁹. A study emphasized the need for comprehensive data collection and increased awareness to facilitate policy-making and intervention strategies for cognitive impairment¹⁰.

Evidence from observational studies suggests that a “healthy” diet is associated with better cognitive function. Some studies employed an *a priori* (hypothesis-based) approach to show that degree of adherence to a pre-defined “healthy” diet (using diet quality indices) was related to cognitive function in older individuals, although these studies used only a brief and limited measure of global cognition, the Mini-Mental State Examination (MMSE)¹¹. By contrast, one study reported a lack of association¹². However, the *a priori* method makes assumptions about which specific food items constitute a healthy diet based on current nutritional research and does not take into account the complexity of the full diet¹³. Although the south-west zone of Nigeria has produced a fair number of studies on cognitive health, there remains a noticeable lack of research on modifiable risk factors, particularly among middle-aged men living in urban centers like Lagos. Meanwhile, scientific literature has consistently demonstrated that dietary behavior is an influential lifestyle factor in the development of Alzheimer’s disease, the most common type of dementia¹⁴. Diets that are abundant in fruits, vegetables, whole grains, and healthy fats have been shown to help maintain cognitive function. In contrast, diets high in saturated fats, refined sugars, and processed foods, especially among aging men, have been linked to increased risk.

The present study is designed to explore dietary patterns and cognitive abilities of middle-aged men in Lagos, Nigeria. It aims to identify dietary behaviors that may heighten vulnerability to cognitive decline, with the goal of informing preventative and management strategies targeted at this demographic.

1.2 Statement of the Problem

Human epidemiologic studies provide convincing evidence that dietary patterns practiced during adulthood are important contributors to age-related cognitive function and the risk of impairment. Diets high in fat, especially trans and saturated fats, adversely affect cognition, while those high in fruits, vegetables, cereals, and fish are associated with better cognitive function and lower risk of decline¹⁶. Scientific studies indicate that what people eat significantly influences brain health. Diets abundant in plant-based foods and healthy fats have been associated with a lower likelihood of experiencing cognitive deterioration, whereas frequent consumption of highly processed foods, added sugars, and saturated fats appears to increase that risk¹⁷. However, despite this knowledge, few studies in Nigeria have specifically explored the role of diet in cognition risk among middle-aged male adults.

Diet plays a critical role in cognitive integrity and decline in older adults. However, little is known about the relationship between diet, dietary habits and cognitive integrity in middle age¹⁸. Education is sometimes used as a proxy measure for both prior cognitive ability and social background but no study to date has been able to control for prior cognition. Without a valid measure of early-life cognitive ability, we cannot properly examine the extent to which diet influences cognition in middle age or conclude that adhering to a particular diet contributes to successful cognitive aging¹⁹.

1.3 Justification

Recent research has highlighted that dietary choices play a pivotal role in the risk of cognitive impairment. Nutritional patterns characterized by high intakes of fruits, vegetables, whole grains, and beneficial fats have been linked to decreased dementia risk. Conversely, diets rich in sugars, processed items, and unhealthy fats may elevate the risk of developing cognitive disorders. Given the current lack of empirical data focusing specifically on the dietary behaviors of middle-aged Nigerian men and their relationship with cognitive function, this study aims to close that gap. Insights gained could form the basis for developing dietary guidelines and interventions to reduce the burden in this group.

1.4 Aims and Objectives of the Study

The aim of this study is to assess the Influence of dietary patterns and the cognitive abilities of middle-aged men in Lagos State

The specific objectives is to:

- assess the influence of Sdietary habits and patterns of middle-aged men (aged 44-60 years) in Eti – Osa LGA, Lagos State.
- i. analyze the cognitive functions of middle-aged men (aged 44-60 years) in Eti-Osa LGA, Lagos State.
- ii. examine the factors that influence the dietary habits and patterns of middle-aged men in Eti-Osa LGA, Lagos State.
- iii. examine the relationship between dietary habits, pattern and cognitive functions of middle-aged men (aged 44-60 years) in Eti – Osa LGA, Lagos State.

1.5 Research Questions

The following research questions will be used for this study;

1. What are the predominant dietary habits and patterns of middle-aged men in Eti-Osa LGA, Lagos State?
2. What are the factors that influence the dietary habits and patterns of middle-aged men (aged 44-60 years) in Eti – Osa LGA, Lagos State?
3. What are the risk factors that influence the cognitive abilities of middle-aged men (aged 44-60 years) in Eti – Osa LGA, Lagos State?
4. What are the specific dietary habits and patterns associated with the cognitive functions of middle-aged men (aged 44-60 years) in Eti – Osa LGA, Lagos State?

1.6 Significance of the Study

This study aims to bridge an existing knowledge gap by examining the impact of dietary habits and pattern on the cognitive function of middle age Nigerian men. The insights generated could serve as a foundation for developing public health strategies that encourage nutritious and healthy dietary practices to curb the rise of cognitive decline in Lagos and comparable regions. Moreover, the research will enrich the global conversation on the diet–cognitive health relationship by offering a localized narrative from a low- and middle-income country perspective, thereby adding cultural and regional relevance to international findings.

1.7 Scope of the study

The investigation focuses specifically on the relationship between dietary behavior and the cognitive abilities of middle-aged men in Lagos State, Nigeria. The target population is defined as males aged 44 to 60 years living in both urban and peri-urban communities

within the Eti-Osa Local Government Area. These individuals constitute the study's core demographic for data collection and analysis.

1.8 Limitation of the Study

This research is geographically restricted to Eti-Osa LGA in Lagos State and demographically limited to men aged 44-60 years. Additionally, engaging with some participants during the data collection phase posed communication barriers, which may have influenced the completeness and accuracy of responses. The work was limited to only middle-aged men in Eti-Osa Local Government Area of Lagos State and specifically to the male gender.

1.9 Definition of Terms

Dietary Pattern: This can be seen as the quantities, proportions, variety, or combination of different foods, drinks, and nutrients in diets, and the frequency with which they are customarily consumed.

Cognitive Abilities or Cognitive Skills: These are set of aptitudes, attitudes, processes and faculties of our mind that allow us to perceive, attend, process, select, interpret, analyze and respond to everything and everyone around us.

Middle Aged Men: These are men within the age of 44-60 years

Cognitive Decline: This is a deterioration of mental capabilities like memory, thinking, and judgment, ranging from mild forgetfulness to severe dementia.

Cognitive Health: This refers to the overall health of the brain and its ability to work effectively.

Endnotes

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Chapter Two

Literature Review

2.1 Conceptual Review

2.1.1 Cognitive Health and Functions

There has been a longstanding debate about whether males and females differ in terms of cognitive abilities. Males are often supposed to outperform females in visual spatial tasks, while females outperform males in terms of verbal and episodic memory tasks¹.

While these sex stereotypes are well accepted in our society, there is a non-negligible number of studies showing exactly the opposite, namely that men and women do not differ in most of the cognitive tasks, also referred to as the “Gender Similarity Hypothesis”^{2,3}. Based on previous research investigating specific cognitive tasks, it has been established that males and females use different cognitive processing strategies: Males seem to use a rather holistic processing style with a focus on global aspects of the task (i.e. having in mind the whole map of a city when performing a spatial navigation task). Females instead use a decomposed processing style with a focus on more local aspects of the task (i.e. remember more details of a given word list). Similar sex differences in terms of a global versus local focus have been found for other tasks such as

mental rotation tasks, number-comparison-task and Navon paradigms^{4,5}. Although sex-related differences in cognitive processing styles do not necessarily result in differences in performance in everyday life, i.e. males and females perform equally good in an everyday multitasking paradigm, they give rise to the question of whether males and females do not only differ in single cognitive abilities. Rather, the two sexes might generally differ in the overall composition of their cognitive abilities. So far, studies mostly focus on cognitive profiles that are predefined based on specific cognitive theories or derived from data-driven approaches (e.g. principal component analyses)^{6,7}. For example, single cognitive abilities are often categorized into cognitive domains, such as attention, memory and executive functions, based on correlations between performance in the tasks administered⁷.

2.1.2 Dietary Patterns; Concept and Importance in Cognitive Performance

Diet has a profound impact on brain health, particularly in middle-aged and older adults, who are at increased risk of cognitive decline and neurodegenerative diseases⁸.

Dietary patterns refer to the overall diet and the combination of foods and nutrients consumed habitually. Nigerian diets typically consist of various staples, including grains (rice, maize), tubers (yam, cassava), and legumes, often supplemented with vegetables, fruits, and proteins from both plant and animal sources. Despite the nutritional variety, disparities exist due to socioeconomic factors, affecting the availability and quality of food consumed⁹. However, various dietary patterns, including the Mediterranean diet (MedDiet), Dietary Approaches to Stop Hypertension (DASH), and Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diets, have been linked to improved cognitive function. While the relative effectiveness of these diets on brain

health is generally supported by evidence, variability in study results suggests that further research is needed to fully understand their effects across diverse populations. Studies assessing the impact of the MedDiet, DASH, MIND, and Western diets on cognitive function in middle-aged and older adults were prioritized. The research findings were synthesized to identify common and unique recommendations across these dietary patterns. The MedDiet consistently showed beneficial effects on cognitive health, including improved memory, processing speed, and long-term protection against neurodegenerative conditions. The DASH and MIND diets demonstrated potential benefits, particularly for specific cognitive domains, but the results were more mixed and inconclusive. In contrast, adherence to a Western diet was associated with negative cognitive outcomes, including cognitive decline and smaller brain volumes⁸. A growing body of evidence suggests that diets such as the Mediterranean (MedDiet), DASH (Dietary Approaches to Stop Hypertension), and MIND (MediterraneanDASH Intervention for Neurodegenerative Delay) are associated with better cognitive outcomes^{10,11}. However, these findings are not entirely consistent, with some studies observing variations in cognitive outcomes across different populations, including those with diverse baseline cognitive statuses. This highlights the complexity of diet's role in brain health^{12,13}. Research in this area spans a wide range of dietary patterns and populations. While some studies have focused on the adverse effects of Western style dietary patterns particularly in relation to cognitive decline and changes in brain structure others emphasize the neuroprotective potential of diets rich in plant based foods and healthy fats, such as the Mediterranean and DASH diets¹⁴. Despite the accumulating evidence, several gaps persist in the literature. For instance, there are few studies that

directly compare the effects of multiple dietary patterns, and Nutrients much of the research focuses on older adults, with limited data available for middle-aged populations who could benefit from early dietary interventions.

2.1.3 Influence of Dietary Pattern on Cognitive Development

Cognitive function is mainly related to knowledge and acquiring information. It is composed of various subgroups: attention, executive function, visuo-spatial skills and memory (short-term and long-term). All these segments are essential components to cognition and knowledge. Attention compels the brain to focus on the important/relevant matters and reduce the effect of any interference. Executive function is how people use all the knowledge and skills they have in order to plan, organize and perform several tasks and solve problems. Visuo-spatial skills are part of the short-term working memory and are accountable for the storage and perception of visual and spatial stimuli. Long-term memory has no expiration date. Episodic memories are part of the long-term memory; it is where life episodes or events are used. The Hippocampal part of the brain is the main part related to memories; it translates short-term memories into long-term ones¹⁵. The hippocampus is responsible for episodic and spatial memory. Spatial memory is a form of object-location memory; it is spatial recognition like geographical knowledge and remembering an item's location. In cases of cognitive decline, the hippocampal brain area is the first area to become impaired. Given the numerous subgroups of the cognitive function, various cognitive tests have been developed to measure each subgroup. These tests could include questionnaires, blood tests, brain scans, personal history and a specific cognitive test or more than one test.

The brain and body function like a data superhighway. As the center of the nervous system the brain receives and processes all the various stimuli which the body absorbs throughout the day. The traveling of this information through the synaptic relays is what allows humans to have feeling and thoughts. Together the mind and body form a complex system, and like any other system it must be properly developed and well maintained. In doing so, one component which must be considered is proper nutrition. The effect that diet has upon both mental and physical health is exceedingly important¹⁶.

A healthy diet is a key component for mental development, mood alteration, behavior, and physical health. It has been found that nutrition is important all throughout the lifespan of an individual¹⁷. It affects people at every stage of their lives. People form eating habits, whether poor or healthy, based on many different circumstances.

Preservation of cognitive abilities is central to the maintenance of independence and quality of life among older adults. A healthy diet may prevent cognitive decline during aging either alone, or in conjunction with chronic diseases and a poor lifestyle¹⁸. For example, epidemiological studies have shown that diet and certain lifestyle factors such as physical activity and social engagement may have an impact on cognitive health during aging¹⁹. Other work has indicated diet-related conditions such as abdominal girth, obesity and metabolic diseases such as hypertension, type 2 diabetes and vascular disease to be risk factors for cognitive decline and dementia²⁰. Consequently, a poor diet, sedentary lifestyle, obesity, and other vascular risk factors such as smoking could increase risk of cognitive decline operating via chronic disease.

Although certain nutrients such as dietary lipids (total, trans, saturated, mono- and polyunsaturated fats), B-vitamins (folate, B₁₂, B₆), antioxidants (vitamins C, E,

carotenoids, polyphenols, flavonoids), and minerals (zinc, selenium, manganese) may act as modulators of cognitive health, research findings have been contradictory^{21,22}. Little is known about how specific nutrients interact within the diet. At the same time, suboptimal nutrient intakes rarely occur in isolation but generally signal a poor-quality diet overall. Thus, the total diet or even a specific dietary pattern (such as the Mediterranean diet), may have a greater impact on cognitive health than individual nutrients²³. A recent review concluded that recommendations to consume a good quality diet based on consumption of healthy fats, certain vegetables and avoidance of sweetened and high glycemic-index foods could lower risk of diet-related cardiovascular and metabolic disorders, and may constitute a relatively inexpensive, easily accepted non-pharmacologic approach for preventing cognitive decline with aging.

Poor cognitive capabilities affect the ability to plan, organize, and adapt behavior to new situations, making it a condition that profoundly disrupts an individual's quality of life. It has therefore been described as a global public health crisis by numerous scholars, not only because of its rising prevalence but also due to its profound social, economic, and healthcare implication. The growing prevalence, particularly in low- and middle-income countries, has been attributed to increasing life expectancy and aging populations, a trend projected to continue over the coming decades²⁴. This trend is projected to escalate over the coming decades, creating a disproportionate burden in regions with limited resources for early diagnosis and long-term care. Globally, an estimated 47.5 million individuals currently live with cognitive disorder, and strikingly, about two-thirds of these cases are found in economically disadvantaged regions, including large parts of Africa, where access to specialized healthcare services and support systems remains minimal.

While there is currently no cure for cognitive disorders, adopting certain dietary patterns can help manage and potentially alter the progression of cognitive disorders²⁵. Emerging evidence from epidemiological research and randomized controlled trials (RCTs) suggests that assorted dietary patterns such as the Mediterranean diet (MedDiet), the Dietary Approaches to Stop Hypertension (DASH) diet, the Mediterranean-DASH diet Intervention for Neurodegenerative Delay (MIND), and the ketogenic diet may offer neuroprotective effects, potentially reducing cognitive decline²⁶⁻²⁸. These patterns are characterized by their specific food compositions and nutrient profiles, which have been studied for their potential health benefits, particularly in relation to cognitive function and neuroprotection²⁹. Dietary modification is also recognized as a potential approach to decrease the overall incidence of dementia in the population. Observational, and interventional studies indicate that certain food groups common to these diets—including fruits, vegetables, legumes, whole grains, nuts, and olive oil—could enhance cognitive functions^{30,31,32}. Additionally, specific nutrients like unsaturated fatty acids and antioxidants have been linked to a lower risk of cognitive deterioration³³. Given the potential impact of dietary patterns, it is essential to explore their effects on cognitive health.

Moreover, cognitive decline coexists with other psychiatric and medical comorbidities, such as depression, cardiovascular disease, or diabetes, further compounding its complexity and the challenges of multidisciplinary care³⁴. The consequences thus extend well beyond the affected individuals, impacting their caregivers, healthcare providers, and even entire communities, particularly in settings where formal support services are lacking. Research consistently indicates that cognitive disorder significantly affects daily

autonomy, with marked functional decline occurring in mid-to-late life stages, ultimately reducing an individual's ability to live independently³⁵. Emerging evidence has identified nutritional intake as a modifiable factor in the onset and progression of cognitive decline. Diets lacking essential micro- and macronutrients, such as vitamins B12, C, and iron, have been linked to exacerbated cognitive decline. Additionally, deficiencies in vitamin B9 (folate) and calcium have been specifically associated with neurodegenerative changes in the aging brain, suggesting that dietary inadequacy may accelerate cognitive deterioration³⁶.

2.1.4 Relationship Between Dietary Patterns and Cognitive Functions

Cognitive health is influenced not only by genetic predisposition and environmental exposures but also by the quality, diversity, and timing of nutrient intake throughout life. Evidence suggests that diets which promote cardiovascular health, reduce oxidative stress, and maintain metabolic efficiency can also help delay or mitigate neurodegenerative processes. One such example is the DASH (Dietary Approaches to Stop Hypertension) plan, initially developed to control high blood pressure. By emphasizing fruits, vegetables, whole grains, lean proteins, and low-fat dairy, the DASH diet helps maintain vascular integrity, reduce inflammation, and stabilize glucose metabolism, factors strongly implicated in cognitive decline. Since both hypertension and cardiovascular dysfunction are recognized dementia risk factors, the cardioprotective effects of DASH indirectly translate into enhanced cognitive function and lower dementia risk³⁷.

Similarly, the Mediterranean diet, which prioritizes the intake of plant-based foods, legumes, olive oil, whole grains, nuts, seeds, moderate fish, and limited red meat, has consistently been linked to lower rates of cognitive decline and dementia onset³⁸. This

diet's high antioxidant and anti-inflammatory nutrient profile helps protect neurons against oxidative stress and amyloid-beta toxicity, while its emphasis on omega-3-rich fish supports synaptic plasticity and neurotransmission.

Empirical studies reveal a nuanced relationship between diet and cognitive health, with regional variations influenced by cultural eating habits and food availability. For instance, research conducted among older adults in Malaysia showed that a diet rich in salty snacks, fish, and processed seafood was associated with heightened dementia risk, possibly due to high sodium and heavy metal exposure. Conversely, diets emphasizing tropical fruits and whole grains like oats were found to have protective effects against cognitive decline³⁹. These findings suggest that not all “traditional” diets are inherently protective, and nutrient balance plays a more important role than dietary labels.

A more recent dietary framework, the MIND (Mediterranean-DASH Intervention for Neurodegenerative Delay) diet, merges the core principles of the Mediterranean and DASH diets. It places a stronger emphasis on leafy green vegetables, berries, nuts, olive oil, and fish, all considered particularly beneficial for brain health. A review of the MIND diet found that adherence to this plan significantly improved memory and cognition, highlighting its preventative benefits against age-related cognitive decline⁴⁰. Importantly, even moderate adherence to the MIND guidelines has been correlated with a notable reduction in Alzheimer's disease risk, demonstrating that partial lifestyle modifications still yield measurable neuroprotective benefits⁴¹. Similarly, studies confirm that consistent adherence to Mediterranean dietary principles is linked to reduced dementia incidence among the elderly⁴². The mechanisms proposed include lower oxidative stress,

improved insulin sensitivity, reduced vascular inflammation, and preservation of hippocampal volume, all of which are essential for cognitive longevity.

Diet, however, is not an isolated factor. Lifestyle elements such as nutrition, physical activity, quality of sleep, and social interaction collectively influence cognitive health. Research indicates that these factors synergistically delay the onset of age-related cognitive impairment, reducing dementia risk, a leading cause of disability in aging populations⁴³. For example, combining a nutrient-dense diet with moderate exercise and mental stimulation yields significantly greater benefits than diet alone.

Emerging findings suggest that modifying lifestyle factors, including dietary choices and exercise, may offer a non-pharmacological approach to prevent or slow dementia progression⁴⁴. Aging, which remains the primary risk factor for dementia, is associated with declines in mitochondrial metabolism, bioenergetic efficiency, and neuronal repair mechanisms. Nutrient-rich diets can counteract these age-related changes, reducing neuronal loss. Because Type 2 diabetes is a well-established risk factor for Alzheimer's disease, adopting dietary patterns that stabilize blood glucose and improve insulin sensitivity may also indirectly support cognitive health⁴⁵. This explains why foods integral to the Mediterranean, MIND, and DASH diets, leafy greens, berries, nuts, olive oil, legumes, and fatty fish, are believed to preserve brain function, although their exact biological pathways are still being explored.

Beyond these conventional diets, alternative approaches such as ketogenic diets and intermittent fasting have gained attention for their potential cognitive benefits⁴⁶. Ketogenic dietary regimens, which prioritize high-fat and very low carbohydrate intake, shift the body's metabolism towards ketone production. Ketones are believed to provide a

more efficient energy source for neurons, enhancing mitochondrial function and reducing amyloid-beta buildup, a hallmark of Alzheimer's pathology. However, findings on ketogenic diets remain population-specific, and some results suggest that their benefits may vary based on genetic background, baseline metabolic health, or overall lifestyle⁴⁷. Similarly, intermittent fasting may improve insulin sensitivity, autophagy, and neurotrophic factor production, but long-term human studies are still limited. Despite these variations, the broader body of research strongly advocates for healthful eating practices as a flexible and accessible preventive strategy against cognitive impairment⁴⁸.

Another study emphasized the cost-effectiveness of diet-based prevention strategies for cognitive decline, noting that diets rich in legumes, fruits, seeds, nuts, cereals, moderate dairy, poultry, fish, and minimal red meat and wine intake are protective against Alzheimer's disease⁴⁹. Such diets are relatively low-cost, culturally adaptable, and sustainable, making them ideal for large-scale public health interventions. Further evidence supports the neuroprotective potential of these diets, showing improved global cognition and reduced dementia cases⁵⁰. Populations with higher consumption of plant-derived foods, antioxidants, and anti-inflammatory compounds show slower cognitive deterioration. This reinforces the view that dietary patterns rich in plant bioactives provide a feasible, natural approach to dementia prevention, especially in communities with limited access to medical therapies.

Ketogenic dietary regimens, which prioritize high fat and low carbohydrate intake, have been shown to enhance mitochondrial function and reduce amyloid-beta buildup, a hallmark of Alzheimer's disease pathology⁵¹. However, some results suggest that benefits may differ by population or be influenced by confounding factors like overall lifestyle⁵².

Despite these variations, the broader body of research strongly advocates for the adoption of healthful eating practices as a flexible and accessible preventive strategy against dementia⁵³.

While numerous studies highlight the cognitive benefits of specific nutrients and diets, meta-analyses of randomized controlled trials have shown mixed results, revealing inconsistencies in study design, sample size, adherence rates, and outcome measurements^{54, 55}. Nutrition research faces challenges such as dietary self-report bias, population heterogeneity, and confounding lifestyle factors, making it difficult to draw definitive causal conclusions.

A recent update intended for clinicians provided insights into current evidence supporting diet-based cognitive impairment prevention. It emphasized that plant-based diets, such as the Mediterranean, Nordic, and MIND diets, along with foods rich in omega-3 fatty acids, B vitamins, flavonols, and certain multi-nutrient formulations (e.g., Fortuyn Connect), help reduce dementia incidence in older adults⁵⁶. Moreover, emerging research suggests that personalized nutrition strategies, tailored to an individual's genetic, metabolic, and gut microbiome profile, could enhance dementia prevention efforts. By identifying people at higher risk and designing customized dietary plans, future interventions may yield more consistent and powerful cognitive benefits.

2.1.5 Factors That Influence Dietary Habits and Patterns of Middle Age Adult

It is empirically evident that dietary preferences vary with age and sex. For example, taste preferences vary with age and sex⁵⁷. In terms of food intake, men tend to eat more meat, whereas women prefer fish. Sex-based differences have also been observed in the

consumption of fruits ^{58,59}. Furthermore, the foods consumed also differ by age. Analysis of the quantitative index for dietary diversity in the Japanese population has shown that dietary diversity increases in men in their 40s and women in their 50s and decreases in both sexes after 60 years of age ⁶⁰. Many previous studies have used the dietary diversity score (DDS)^{61,62}. However, given the issues associated with food mixing and matching in diets, the DDS cannot adequately capture dietary patterns. The dietary patterns of Japanese people and age-specific differences in the frequency of individual foods have been examined. For example, meat intake among Japanese individuals is high among 15–19 year-olds and declines thereafter. Fish and vegetable intake increases with age, with the highest intake reported for those aged 60–69 years⁶³. Principal component analysis (PCA) has been used in many cases to analyze the dietary patterns of Japanese people. They conducted a meta-analysis of 80 articles and reported that healthy Japanese dietary patterns were rich in mushrooms, seaweeds, potatoes, fruits, legumes, and pickles ⁶⁴. Dietary patterns may change in response to life events such as disease diagnosis, social factors, or new nutrition knowledge⁶⁵.

Dietary patterns differ significantly and are influenced by one's culture and lifestyle.⁶⁶ Pathological, physiological, economic, and social factors associated with aging may impair one's capacity to pick a diet of adequate quality and diversity to meet daily nutrient demands.⁶⁷ Some factors affecting dietary habits and patterns of middle age adults are further discussed below.

i) Socioeconomic Status

Across all genders and age groups, one of the strongest determinants of eating habits is the socioeconomic status.⁶⁸ The components of socioeconomic status include: education

level, financial security, opinion of social status and income. For instance, a study found a positive relationship between socioeconomic status and countries with high incomes when compared to low- and middle-income countries like Nigeria.⁶⁹ The higher the socioeconomic status, the healthier the eating choices, therefore the higher the consumption of fruits and vegetables. Study in Mauritius revealed that middle and high socioeconomic status groups consume more dairy products than low socioeconomic status groups, with this difference attributed to the higher cost of dairy products. Low-income populations are also said to consume fast food more frequently because they can reach fast food restaurants more readily than supermarkets with fresh items. However, it should be emphasized that fast food consumption is higher among both high- and low-income individuals, thus they consume more energy. It has been reported that in Mauritius, an individual's food choice is influenced by the price of the food. Mushrooming of grocery stores and fast foods outlets influence the eating habits of low socio-economic status individuals. Furthermore, the price per weight of food products influences food choices among low-income Mauritius residents. Low-income population prefer low-fat protein foods like pulses because they are less expensive per pound⁷⁰. However, studies carried out show that middle-income participants were found to consume more beans and pulses than low-income participants.

ii) Price of Food Items

Many things, including food, are affected by rising energy and transportation costs. Therefore, people will pay more for food in shops and restaurants as fuel prices rise due to Russia and Ukraine conflict. High cost of food item affects dietary habits of individuals of all ages. For instance, as fat and sugar rich food provide energy at a low

cost. To add, excessive consumption of added sugars and fat, as well as sedentary lifestyles, have been linked to the current obesity epidemic. However, an increase in cost in soda and pizza lead to a reduced consumption of daily energy intake and a reduction in weight was also noted among individuals.⁷¹

iii) Marital Status

The structure and composition of a family are important social determinants that can influence health behaviours and outcomes. Food is frequently shared and represents a means of connecting with others: sharing meals and cooking for loved ones. Single elderly of both genders consumed more nutrients than married ones. Moreover, social isolation and loneliness are linked to decreased appetite, lower food intake, decreased physical activity, and an increased risk of malnutrition in midlife.

Married individual specially men were found to be healthier and impose lower risk of mortality than those who are unmarried. This barrier to healthy eating and adequate energy intake is because unmarried, single, separated or widowed male elderly do not know how to prepare meal and groceries for themselves. Furthermore, a strong link was observed between all unmarried states for example those who are single, never married, divorced, and widowed and an increased risk of cardiovascular disease mortality due to changes in eating habits as a result of social isolation⁷².

iv) Psychological Factors

Depression affects many older persons, while it does not affect everyone and it might be a risk factor for poor nutrition. Depression can also cause a loss of interest in activities such as shopping, cooking, and eating. This can eventually lead to a poor

diet. Prospective and epidemiological studies have now proven a link between nutrition and depression. Studies from Turkey, Iran, Isfahan and south Africa demonstrated a relatively poor diet quality, as well as a lower intake of fruits, vegetables and oily fish among depressed middle age adults. Higher consumption of sweet foods was found to be associated with emotional eating. Evidence have shown that food consumption and depressed symptoms have a bidirectional link; it can either lead to a lack of interest in eating resulting in reduced food intake and weight loss or an increase in food intake and obesity⁷³.

v) Changes in Sensory Functioning

Chemosensory (smell and taste) losses are prominent examples of sensory changes associated with aging. Around the age of 60, the sense of taste started to decrease significantly. According to several research, reduced odour has been linked to hunger suppression, weight loss, and malnutrition in middle adults. Also, the decline in chemosensory abilities can also lead to bad eating patterns, as people tend to increase their sugar and salt consumption to compensate for their inability to taste these substances in food at lower amounts.⁷⁴

Vi) Access to Food Commodities

Close vicinity and greater accessibility to any kind of food reseller were observed to be linked with lower ultra-processed foods ingestion in the majority of cases, with slightly robust relationship with exposure to restaurants and supermarkets. The lower the consumption of lower ultra-processed foods, the lower the energy intake, the lower the risk of consuming poorer overall diet quality and the lower the risk of having high BMI.

Greater food consumption is linked to food accessibility, hence resulting in a higher energy intake. To add, access to transportation is difficult in some rural areas, and food insecurity may result because of poor health and restricted access to food due to a lack of food commodities in the area.

vii) Nutrition Knowledge

Increased nutrition knowledge, including the capacity to translate nutrition knowledge into improved health practices, may be linked to education. Even though nutrition knowledge is usually linked to better eating habits, the literature on the link between eating habits and nutrition knowledge is contradictory. While some researchers have found a strong and positive link between nutrition knowledge and eating habits, others have found little association between nutrition knowledge and actual food choices time.

Poor educational status is linked to high-carbohydrate, low-fibre diets, and increased ingestion of sweets and red meats, whereas good nutritional status is associated to increased consumption of fruits, vegetables, and fish, as well as increased nutritional diversity and lower caloric intake.

viii) Medication Factors

Multiple medications can influence eating behaviour and, as a result, nutrient absorption, leading to the onset of nutritional issues as well as increased morbidity and mortality. Medicines can reduce appetite in a variety of ways; some medications can make one feel sick; some can influence taste; and some can be the source of depression. Another review reported that a higher usage of medications was linked to more frequent consumption of fruits and vegetables, skimmed milk and fish, as well as

less frequent consumption of sugary meals, soft drinks, artificial juices, and fatty meat. In order to maximize absorption, many medications include specific dose recommendations based on food intake.

2.1.6 Major Dietary Approaches in Cognitive Management.

Various dietary strategies have been highlighted across scholarly literature for their potential role in Cognitive health and functions. Nutritional interventions are increasingly recognized as non-pharmacological therapies that may improve or maintain neurocognitive function by influencing metabolic pathways, reducing oxidative damage, and modulating neuroinflammation. Among the most extensively studied dietary interventions are the ketogenic, Mediterranean, DASH, and MIND diets, each of which targets neurocognitive health through different mechanisms of action and nutrient profiles. The ketogenic diet (KD) is a highly specialized nutritional approach characterized by an extremely low intake of carbohydrates (typically less than 10% of daily energy intake) and a very high proportion of dietary fats⁷⁶. By severely limiting carbohydrate consumption, the body undergoes a metabolic shift from glucose-dependent energy metabolism to fat-derived energy. In this state, fatty acids from dietary intake and adipose tissue are oxidized in the liver, producing ketone bodies, primarily β -hydroxybutyrate (β -OHB) and acetoacetate (AcAc), which then serve as alternative fuel sources for the brain. Under normal dietary conditions, the human brain relies almost exclusively on glucose as its main energy substrate, consuming approximately 100–120 grams per day, which accounts for about 20% of the body's basal metabolic energy output⁴¹. However, when

glucose availability diminishes, fats from dietary sources and adipose tissue are broken down, leading to the synthesis of ketones in the liver^{77, 78}. These ketone bodies are subsequently released into circulation and transported to the brain, where they substitute for glucose as the primary energy source for neurons.

This metabolic adaptation induced by KD closely mimics the physiological state of fasting, during which the body naturally transitions to ketone-based energy metabolism to sustain brain function when dietary glucose is scarce⁷⁹. Within several hours of adopting a ketogenic diet, blood levels of glucose and insulin drop, while glucagon levels rise, triggering glycogenolysis and increased fatty acid oxidation^{80, 81}. By the second or third day of sustained ketosis, hepatic mitochondria become fully engaged in ketogenesis, producing sufficient ketones to meet the brain's energy demands⁸². Though normal blood ketone levels are relatively low (<0.3 mM), during prolonged fasting or sustained ketosis, levels may exceed 4 mM, making ketones a viable cerebral energy substrate^{83, 84, 85}. However, it is important to note that in pathological conditions such as uncontrolled diabetes, ketone levels can become excessively elevated (exceeding 25 mM), leading to dangerous ketoacidosis, which is a life-threatening complication distinct from the mild ketosis seen in dietary interventions⁸⁶.

The traditional formulation of the ketogenic diet delivers approximately 90% of total caloric intake from fats, with protein and carbohydrate contributing about 6% and 4% respectively⁸⁷. This nutrient profile adheres to a 4:1 fat-to-protein-plus-carb ratio⁸⁸. Most ketogenic meal plans rely on long-chain fatty acids (LCFAs) as the primary fat source. However, newer modifications of KD incorporate medium-chain triglycerides (MCTs), which are more rapidly absorbed and efficiently converted into ketones, allowing for

greater dietary flexibility even with modest carbohydrate intake^{89, 90}. A well-known variant, the Atkins diet, follows a similarly high-fat model but restricts carbohydrates to no more than 5% of total caloric intake⁹¹.

Unlike other organs, the central nervous system (CNS) cannot metabolize fatty acids directly due to the blood–brain barrier (BBB). Instead, the ketone bodies produced from β -oxidation of fatty acids are converted into acetyl-CoA, which can fuel the Krebs cycle for ATP production. Ketones can also form acetone and β -OHB through specific enzymatic reactions^{92, 93, 94}. Importantly, ketone bodies are able to cross the BBB efficiently via monocarboxylate transporters, thereby supplying neurons with an alternative, reliable energy source during glucose scarcity^{95, 96}.

Ketones are reported to be metabolized more efficiently than glucose, as they enter the cellular energy cycle without requiring glycolysis. This metabolic shortcut enhances ATP production via oxidative phosphorylation, reduces mitochondrial workload, and lowers the dependence on glycolytic pathways^{97, 98}. Consequently, KD promotes better mitochondrial performance, increases serum ketone levels, and simultaneously reduces serum glucose concentrations. These metabolic shifts are believed to protect neurons against oxidative damage, excitotoxicity, and apoptosis, which are hallmarks of neurodegenerative conditions.

In addition to metabolic effects, KD influences gene expression in neural tissues. Several studies have shown that it upregulates hippocampal genes associated with mitochondrial enzymes and energy metabolism⁹⁹. This process, known as therapeutic ketosis, has been proposed as a form of metabolic neurotherapy. Improved energy status reduced oxidative stress, lowered inflammation, and elevated neurotrophic factors (e.g., BDNF, NT-3,

GDNF) have all been documented in preclinical studies¹⁰⁰. Additionally, KD is believed to stabilize synaptic function by increasing the availability of Krebs cycle intermediates, shifting neurotransmission towards GABAergic (inhibitory) pathways, and activating ATP-sensitive potassium channels. These mechanisms collectively improve neuronal resilience, reduce abnormal excitability, and protect against progressive neurodegeneration.

Ketogenic Diet is being investigated as a potential metabolic therapy for conditions like Alzheimer's disease and mild cognitive impairment. By bypassing impaired glucose metabolism in the aging brain, ketones may act as an alternative energy source, sustaining neuronal activity in regions affected by glucose hypometabolism, a common feature in Alzheimer's pathology. Preliminary studies suggest KD can improve cognitive performance, attention, and memory in some patients with early-stage dementia, although long-term outcomes remain under investigation. However, KD may not be suitable for all populations due to its restrictive nature, potential side effects (such as gastrointestinal discomfort or dyslipidemia), and variable adherence rates in older adults. Therefore, more controlled clinical trials are required to determine its safety, sustainability, and long-term cognitive benefits.

Neuroinflammation and oxidative stress are key contributors to Alzheimer's disease and related dementias¹⁰¹. Inflammatory processes in the brain are often initiated by microglial activation, leading to the release of proinflammatory cytokines such as IL-1 β , IL-6, and TNF- α . These cytokines amplify oxidative stress, impair synaptic function, and disrupt neuronal signaling. In addition, chronic inflammation suppresses the release of neuroprotective factors, creating a self-perpetuating cycle of damage that particularly

affects memory-related regions like the hippocampus¹⁰². The ketogenic diet (KD) has been shown to exert significant anti-inflammatory effects by targeting multiple signaling pathways that drive neuroinflammation. One of the key mechanisms involves the inhibition of the NF- κ B signaling pathway, which is a major transcription factor responsible for upregulating inflammatory genes¹⁰³. Downregulation of NF- κ B leads to the suppression of proinflammatory enzymes such as cyclooxygenase-2 (COX2) and inducible nitric oxide synthase (iNOS). As a result, the levels of neurotoxic cytokines including IL-1 β , IL-6, TNF- α , and monocyte chemoattractant protein-1 (MCP-1) are markedly reduced.

Another important pathway modulated by KD involves the activation of peroxisome proliferator-activated receptor gamma (PPAR γ), a nuclear receptor known to have anti-inflammatory and neuroprotective roles¹⁰⁴. Activation of PPAR γ by ketone bodies may also mitigate NMDA receptor-induced excitotoxicity, which is one of the critical processes that contribute to neuronal death in Alzheimer's disease. Ketone bodies also activate microglial cells in an anti-inflammatory manner and promote neurotrophic factors and chaperone proteins that protect against cellular stress and protein misfolding¹⁰⁵. In this altered microglial state, ketones also stimulate the production of neurotrophic factors such as BDNF (brain-derived neurotrophic factor) and heat shock proteins or molecular chaperones. These proteins help stabilize misfolded proteins, protect neurons from cellular stress, and maintain proteostasis, all crucial in limiting the accumulation of amyloid-beta plaques and tau tangles.

Thus, KD appears to attenuate neuroinflammation through multiple overlapping mechanisms, including inhibition of proinflammatory signaling cascades, modulation of

glial cell activity, and promotion of neuroprotective molecular mediators. This anti-inflammatory profile may be a major reason why ketogenic states are being investigated as adjunctive therapies for neurodegenerative diseases.

Neuroimaging studies have shown that cerebral glucose hypometabolism can occur decades before clinical symptoms appear. This early dysfunction correlates with reduced expression of GLUT1 and GLUT3 transporters, which are responsible for transporting glucose across the blood-brain barrier and neuronal membranes^{106, 107}. In parallel, a diet high in refined sugars and simple carbohydrates has been strongly associated with insulin resistance, which exacerbates impaired glucose utilization in the brain and increases the risk of Alzheimer's disease¹⁰⁸. This has led to the concept of Alzheimer's as a "type 3 diabetes," where neurons progressively lose their ability to respond to insulin signaling.

The ketogenic diet provides an alternative energy pathway that circumvents these metabolic blockages by supplying ketone bodies as direct fuel for neurons. Clinical trials have demonstrated that KD and medium-chain triglyceride (MCT) supplementation can improve memory, attention, and executive function, particularly in older adults with mild cognitive impairment (MCI), a precursor stage to dementia¹⁰⁹. When the brain suffers from chronic glucose deprivation, there is a cascade of metabolic failure, including reduced cerebral perfusion and impaired neuronal signaling, which accelerates neurodegeneration^{110, 111}.

Furthermore, the accumulation of advanced glycation end products (AGEs), which result from chronic hyperglycemia, increases oxidative stress and protein cross-linking in neurons, exacerbating neurodegenerative processes^{112, 113}. By maintaining stable ketone levels, KD not only provides alternative energy substrates but also reduces the formation

of AGEs and improves neuronal resilience. These metabolic benefits may explain why KD has been associated with short-term cognitive improvements in dementia patients and holds potential as a disease-modifying nutritional strategy.

These abnormalities result in a chronic energy shortfall, especially in areas responsible for memory and cognition, such as the hippocampus¹¹⁴. One hallmark of Alzheimer's pathology is a shift from normal glucose metabolism to a hypometabolic state, resulting in reduced cerebral glucose availability and utilization¹¹⁵. Interestingly, while glucose utilization declines with aging, the brain's ability to metabolize ketones often remains intact or may even increase, offering a compensatory energy pathway through ketone body metabolism¹¹⁶. This observation forms a key rationale for using KD in cognitive management; by supplying ketones, it bypasses faulty glucose pathways, maintaining neuronal energy balance.

At the cellular level, mitochondrial dysfunction and oxidative stress represent key mechanisms driving neuronal damage in Alzheimer's disease and related dementias. These processes generate high levels of reactive oxygen species (ROS), which are known to damage essential macromolecules such as nucleic acids, lipids, and structural proteins, ultimately contributing to cell death¹¹⁷. The ketogenic diet offers a means to bypass impaired glucose pathways by supplying the brain with ketone bodies, alternative energy substrates that can reduce ROS production, improve mitochondrial function, and provide metabolic flexibility¹¹⁸.

One significant molecular adaptation to ketogenic therapy involves modulation of the NAD⁺/NADH redox ratio, a balance critical for cellular energy metabolism. Elevating this ratio helps to maintain redox stability, enhance mitochondrial biogenesis, and support

consistent neuronal signaling¹¹⁹. In animal studies, rodents maintained on ketogenic regimens for as little as two days exhibited notable increases in NAD⁺/NADH levels in the brain's cortex and hippocampus, indicating rapid systemic metabolic reprogramming¹⁰¹. This metabolic shift triggers the activation of sirtuin 1 (SIRT1), a key regulator of gene expression involved in DNA repair, mitochondrial biogenesis, and anti-aging pathways^{120, 121}. SIRT1 activation enhances neuronal survival and synaptic plasticity, both critical for preserving memory.

Furthermore, the ketogenic diet enhances the function of the electron transport chain (ETC) by increasing expression of uncoupling proteins (UCPs) in the hippocampus. These proteins serve to stabilize mitochondrial membrane potential and prevent excessive production of ROS and reactive nitrogen species (RNS), both of which contribute to neuroinflammation and neuronal degeneration^{122, 123, 124}. Ketogenic states also block voltage-gated sodium and calcium channels in neurons, contributing to a neuroprotective membrane environment and maintaining proper ion homeostasis¹²⁵.

Another mitochondrial benefit is the regulation of mitochondrial permeability transition (mPT). Under pathological conditions, abnormal opening of mPT pores leads to loss of mitochondrial integrity and triggers apoptosis. Ketone bodies help prevent this pore opening, thereby preserving mitochondrial stability and neuronal viability¹²⁶. Additionally, polyunsaturated fatty acids (PUFAs) such as DHA, EPA, and arachidonic acid, which are often present in high-fat diets, contribute synergistically by reducing ROS production, suppressing neuroinflammatory mediators, and enhancing neuronal excitability through channel regulation¹²⁷.

Beyond direct energy production, ketogenic interventions have been shown to increase glutathione levels and stimulate glutathione peroxidase (GSH-Px) activity, key components in neutralizing free radicals in the brain, especially within the hippocampus¹²⁸. These antioxidants bolster the brain's endogenous defense systems, further reinforcing its resistance to oxidative stress.

The mTOR (mammalian target of rapamycin) pathway, which controls cellular growth and metabolic activity, is also influenced by the ketogenic diet. Studies have shown that ketogenic states suppress insulin signaling and reduce phosphorylation of Akt and S6 kinase, thereby inhibiting mTOR activity¹²⁹. This inhibition is associated with improved autophagy, mitochondrial turnover, and reduced protein aggregation, all of which are highly relevant in Alzheimer's disease. Additionally, ketosis has been associated with elevated ATP and phosphocreatine concentrations in the brain, suggesting enhanced energy reserves and improved metabolic efficiency¹³⁰.

Together, these biochemical and molecular modifications demonstrate how the ketogenic diet may mitigate neurodegenerative progression by restoring energetic balance, protecting mitochondria, and modulating oxidative and inflammatory stressors. While the ketogenic diet (KD) has demonstrated therapeutic benefits, particularly for neurological and metabolic conditions, it is not without its limitations and potential health risks. These risks become more pronounced during long-term adherence, or in individuals with preexisting medical conditions. Understanding these adverse effects is critical, especially when applying KD in vulnerable populations such as the elderly or those with neurodegenerative diseases.

In otherwise healthy adults, the short-term adverse effects of KD are relatively predictable. They often emerge during the initial adaptation period, sometimes referred to as the “keto flu.” During this transitional phase, individuals may experience hypoglycemia, electrolyte imbalances, and dehydration, resulting from the shift in metabolic fuel from glucose to ketones¹³¹. Such symptoms typically resolve as the body adapts to sustained ketosis, but they may discourage some patients from maintaining the diet.

Historically, ketone bodies were perceived as harmful, primarily due to their association with diabetic ketoacidosis (DKA), a condition marked by excessive ketone production, acidosis, and dangerously high blood glucose levels. In DKA, ketone concentrations may exceed 20 mM, a level considered toxic but reversible with insulin administration¹³². Importantly, diet-induced ketosis differs fundamentally from DKA, as it is accompanied by normal or low blood glucose and regulated insulin levels. However, in individuals with insulin deficiency, ketone production may spiral into hyperketonemia, causing severe acidosis and potentially fatal complications¹³³.

Among the most frequently reported side effects in patients using the ketogenic diet for epilepsy management are digestive disturbances, weight loss, and temporary dyslipidemia¹³⁴. Digestive issues such as nausea, constipation, vomiting, and reduced appetite are particularly common during the early phases of the diet. These disturbances may interfere with nutrient absorption and overall adherence to the regimen¹³⁵. Weight loss, while beneficial for overweight or obese individuals, can become problematic if uncontrolled reduction in lean body mass occurs. In some cases, muscle wasting and

energy deficiencies may develop, especially if the diet is poorly planned or lacks adequate protein and micronutrient support.

Another notable metabolic effect of KD is its impact on lipid profiles. Clinical observations show that an initial rise in total cholesterol, LDL cholesterol, and triglycerides often occurs during the first stages of ketogenic therapy. Fortunately, these lipid elevations typically return to baseline levels within 6–12 months of sustained adherence¹³⁶. Other clinical reports list complications such as Hypomagnesemia, hyponatremia, and hyperuricemia due to shifts in fluid and electrolyte balance, pancreatitis and elevated liver enzymes, particularly in individuals with preexisting liver or pancreatic conditions, and Renal stress, including the formation of kidney stones, which may result from increased urinary calcium and altered acid-base balance¹³⁷. These risks underline the importance of medical supervision and periodic biochemical monitoring for anyone following a strict KD.

Extended adherence to ketogenic diets raises additional long-term concerns. Prolonged ketosis has been associated with potential risks such as atherosclerosis, cardiac hypertrophy, kidney stones, and hepatic dysfunction. Moreover, because KD significantly restricts carbohydrate intake, there is a heightened risk of nutrient deficiencies, especially fat-soluble vitamins (A, D, E, K) and key minerals like calcium, iron, and selenium¹³⁸. There are also rare but documented cases of optic neuropathy, anemia, neuropathy, and reduced bone density, which may result from inadequate micronutrient intake or excessive protein restriction over time. Such complications are particularly concerning in older adults, where preexisting malnutrition or sarcopenia can exacerbate these effects.

In elderly patients or those with neurodegenerative conditions, additional barriers may affect the sustainability of ketogenic interventions. Symptoms like apraxia, dysphagia, sensory deficits, and behavioral disturbances can hinder regular food consumption, increasing the risk of malnutrition¹³⁹. Loss of taste or olfactory sensitivity may also reduce food appeal, further complicating dietary compliance¹⁴⁰. Caregivers may also find it difficult to maintain strict ketogenic meal plans for individuals with advanced cognitive impairment.

Despite these concerns, some studies suggest the ketogenic diet may be safe in certain populations when properly managed. One investigation involving 83 obese adults found no significant adverse outcomes over a 24-week ketogenic intervention period¹⁴¹. Ultimately, individualized nutritional assessment, close medical supervision, and appropriate supplementation are essential to minimize risks and ensure safety during prolonged KD use. KD should be tailored based on patient age, metabolic status, and comorbid conditions, rather than applied universally.

The Mediterranean diet (MD) is widely recognized for its health-promoting properties, particularly in relation to cognitive preservation and neurodegenerative disease prevention¹⁴². Unlike the ketogenic diet, which emphasizes low carbohydrate and high fat intake, the MD is balanced and diverse, drawing inspiration from the traditional eating patterns of countries bordering the Mediterranean Sea.

This diet is centered around frequent consumption of plant-based foods, including vegetables, fruits, legumes, nuts, and whole grains, along with moderate to high intake of fish and seafood. Olive oil serves as the primary fat source, replacing animal fats and saturated oils¹⁴³. These dietary elements collectively provide a high density of

antioxidants, unsaturated fats, vitamins, and polyphenols, which are believed to support neuronal health, reduce oxidative stress, and maintain vascular integrity.

Epidemiological studies have reported strong associations between adherence to the Mediterranean diet and a decreased incidence of cognitive decline and Alzheimer's disease¹⁴⁴. Evidence suggests that individuals who closely follow MD guidelines exhibit a substantially lower risk of cognitive impairment, while poor adherence correlates with an increased likelihood of developing dementia-related disorders. However, while observational studies are consistent in their findings, they fall short of establishing a definitive cause-and-effect relationship¹⁴⁵. Controlled trials are more challenging to conduct due to dietary variability, cultural influences, and long-term adherence issues.

According to existing literature, the mechanisms through which the Mediterranean diet may confer cognitive benefits are multifaceted. These include improved cardiovascular health, better regulation of glucose and lipid metabolism, and suppression of chronic inflammation, factors closely linked to the pathophysiology of dementia¹⁵⁰. Furthermore, specific components of the diet, such as omega-3 fatty acids from fish, polyphenols from fruits and vegetables, and monounsaturated fats from olive oil, are individually linked to neuroprotective functions¹⁵¹.

Despite promising outcomes, some researchers caution that current evidence remains inconclusive in confirming the Mediterranean diet as a definitive preventive therapy for cognitive disorder. Nonetheless, a growing number of studies report cognitive benefits and decreased dementia incidence among individuals who incorporate this dietary model consistently into their lifestyle. A notable meta-analysis exploring the relationship between the Mediterranean diet and age-related cognitive disorders, including general

cognitive impairment, demonstrated that high adherence to the diet is associated with a statistically significant reduction in disease risk¹⁵². The review emphasized the potential of MD as a key element in global strategies aimed at delaying cognitive aging. A subsequent study further validated the protective association between MD adherence and reduced dementia occurrence, particularly among older adults¹⁵³.

While there is broad support for the Mediterranean diet's neuroprotective benefits, practical implementation remains a challenge. The dietary framework may differ markedly from that of typical Western diets, which are often high in saturated fats, sugars, and processed foods¹⁵⁴. Consequently, long-term commitment to the MD may be difficult to sustain in populations unfamiliar with its components or cultural background¹⁵⁶. This highlights the importance of culturally tailored dietary education and public health policies that encourage more sustainable adoption of Mediterranean-style eating patterns. Programs that adapt Mediterranean principles to local foods and traditions may help overcome barriers and promote wider adherence.

The DASH diet, short for Dietary Approaches to Stop Hypertension, was originally developed as a non-pharmacological strategy to manage and lower high blood pressure, but over time, its benefits have been recognized to extend beyond cardiovascular health¹⁵⁷. Increasingly, research has linked DASH adherence to improved cognitive outcomes in both middle-aged and elderly populations. This diet emphasizes foods rich in potassium, magnesium, and calcium, nutrients that play a vital role in maintaining neuronal membrane potential, synaptic transmission, and vascular integrity. In practical terms, the DASH diet promotes a high intake of fruits, vegetables, low-fat dairy, whole grains,

legumes, and nuts, while limiting sodium, red meats, processed foods, and added sugars¹⁵⁸.

The MIND diet (Mediterranean-DASH Intervention for Neurodegenerative Delay) is a more recent innovation that combines key elements of the DASH and Mediterranean diets but is specifically designed to promote brain health and prevent neurodegenerative conditions¹⁵⁹. Unlike its predecessors, the MIND diet includes explicit recommendations for food portions and frequency to meet the nutritional needs of the aging brain¹⁶⁰. It also strongly limits foods high in saturated fats, such as butter, cheese, fried foods, and pastries, which are known to promote oxidative stress and inflammatory processes detrimental to neural tissue¹⁶¹.

Although both DASH and MIND diets contribute to reducing the risk of chronic diseases, only the MIND diet was developed with dementia prevention as its primary objective¹⁶². Key food groups emphasized in the MIND diet include leafy green vegetables, berries, olive oil, whole grains, nuts, and lean protein sources such as poultry and fish¹⁶³. These food items are selected based on strong evidence for their positive influence on memory and cognitive function.

Empirical studies support a strong inverse relationship between MIND diet adherence and the risk of Alzheimer's disease. Participants with higher MIND diet scores were found to have slower cognitive decline and lower incidence of Alzheimer's-related symptoms^{164, 165}. In fact, some longitudinal studies showed that even moderate adherence to MIND principles could lead to significant neuroprotective benefits, suggesting that partial dietary improvements still positively influence brain health. However, scholars

continue to emphasize the need for more robust randomized controlled trials to determine whether this correlation is causal.

However, despite these promising associations, scholars emphasize the need for more rigorously designed randomized controlled trials to determine whether the observed benefits are causal or simply correlated with other healthy lifestyle factors. Nonetheless, the body of observational research remains compelling, showing that dietary patterns such as MIND, DASH, and the Mediterranean diet (MD) significantly reduce cognitive disorders across diverse populations^{166, 167, 168}.

Taken together, these diets represent accessible, non-invasive strategies to mitigate neurodegenerative risk. They also complement other lifestyle interventions, such as physical activity, social engagement, and mental stimulation, which synergistically enhance cognitive reserve and resilience.

2.1.7 Factors That Affect Cognitive Functions In Middle Age Adult

Maintaining cognitive function is an important component of healthy aging¹⁶⁹. The aging process is associated with changes in cognition and, while these gradual changes may vary markedly between persons, they do not impair everyday activities. Cognitive changes that interfere in day-to-day activities, such as difficulty following instructions or failing to attend scheduled appointments, may be indicative of more serious health conditions, such as mild cognitive impairment or dementia¹⁷⁰.

While evidence suggests some decreases in executive and memory functions with age there is increasing recognition of the extraneous factors which may expedite the typical cognitive aging process, including medical, psychological, and sensory concerns¹⁷¹.

A wide array of modifiable and non-modifiable factors has been shown to impact the cognitive abilities of middle-aged populations, particularly men. Studies have examined these determinants from biological, behavioral, and social perspectives. A study based on primary healthcare data in older adults revealed that physical inactivity, depressive symptoms, and hypertension were among the most prevalent and influential risk factors for dementia¹⁷². Other contributing elements included monolingualism, poor visual health, and inconsistent consumption of fruits and vegetables. Interestingly, the analysis found no significant sex-based differences in risk factor prevalence. However, regression models revealed that both education level and age group were statistically significant predictors, with lower educational attainment and older age being associated with higher risk of cognitive disorder.

Another large-scale investigation focusing on midlife health determinants found that obesity, diabetes, active smoking, hypercholesterolemia, and elevated systolic blood pressure were linked to a 41% to 78% increase in dementia risk¹⁷³. However, sensitivity analysis revealed that the associations with high cholesterol and blood pressure were less robust and require confirmation in further high-quality studies. Moreover, the systematic review identified three additional factors, elevated homocysteine levels, chronic psychological stress, and excessive alcohol intake, as significantly increasing dementia susceptibility. Conversely, engagement in physical activity, maintaining a nutrient-rich diet, and hormone therapy in midlife were associated with reduced cognitive decline risk.

A separate two-year longitudinal study comparing middle-aged and older adults identified several baseline predictors of future cognitive deterioration¹⁷⁴. Across both assessments, low educational background, history of traumatic brain injury, and smoking

remained constant risk factors. Additionally, the prevalence of hypertension and sedentary behavior increased over time, while social isolation decreased, suggesting shifts in behavioral patterns. Notably, participants with baseline depressive symptoms had a statistically higher probability of experiencing cognitive decline during follow-up.

Sleep patterns have also emerged as a significant, though less discussed, contributor to cognitive aging. A study examining the relationship between sleep disorders and cognitive function found that sleep deficiencies, particularly insomnia, were linked to heightened risk for MCI, diabetes, and obesity¹⁷⁵. While insomnia prevalence was higher in females, sex-specific patterns for other sleep disorders remain unclear and require further study

In a population-based cross-sectional study conducted in the Tibetan region, researchers found that factors such as older age, single marital status, low education, obesity, hypertension, diabetes, and history of cardiovascular disease were positively associated with dementia prevalence¹⁷⁶. Interestingly, the study found no association between religious activity frequency and dementia incidence in this population.

The risk factor that influences cognitive functions of middle-aged men is discussed as follows:

Physical inactivity has been identified as a substantial contributor to the rising prevalence of dementia across diverse age groups, particularly among older adults¹⁷⁷. It disrupts physiological processes such as vascular integrity, neuronal regeneration, and cognitive reserve, all of which are essential in protecting brain health and mitigating neurodegenerative¹⁷⁸. Studies suggest that individuals engaging in regular physical

activity may experience up to a 20% reduced risk of developing dementia compared to those leading sedentary lifestyles¹⁷⁹. The most substantial impact is observed in individuals who transition from a completely inactive state to engaging in even minimal levels of activity. Evidence further underscores that maintaining consistent physical exercise during midlife provides a significant protective effect against cognitive deterioration.

Elderly individuals are not exempt from these benefits. One study tracking daily movement patterns among older adults found that those in the lowest activity percentile (bottom 10%) were over twice as likely to develop Alzheimer's disease compared to their highly active counterparts (top 10%)¹⁸⁰. While the role of exercise in reducing risk of MCI is well-established, ongoing research is required to identify which types, durations, and intensities of physical activity offer the most effective neuroprotective benefits¹⁸¹.

Diabetes mellitus has been widely recognized as a significant metabolic risk factor associated with cognitive decline, especially among aging populations¹⁸². The condition arises from impaired insulin production or reduced sensitivity of the body's cells to insulin, resulting in persistent hyperglycemia¹⁸³. Insulin, a hormone synthesized in the pancreas, plays a central role in regulating blood glucose levels and energy metabolism, both of which have direct implications for brain function. Diabetes manifests in two primary forms: Type 1 diabetes, an autoimmune condition typically diagnosed early in life, and Type 2 diabetes, a metabolic disorder that develops gradually due to insulin resistance or insufficient insulin production¹⁸⁴.

Research has linked Type 2 diabetes to an elevated risk of dementia, with the likelihood increasing in tandem with the duration and severity of the condition¹⁸⁵. While not every

diabetic individual will develop dementia, the presence of diabetes substantially amplifies vulnerability, particularly when glucose levels are poorly controlled. Moreover, individuals with Type 1 diabetes are also at increased risk when experiencing frequent episodes of hyperglycemia or hypoglycemia, both of which can compromise neuronal integrity¹⁸⁶.

Obesity, defined as excessive fat accumulation adversely affecting health, is another recognized risk factor implicated in cognitive health. Its risk contribution is multifactorial and is often mediated by comorbid conditions such as Type 2 diabetes, hypertension, systemic inflammation, and metabolic syndrome, all of which are independently linked to cognitive decline. According to the National Health Service (NHS), a Body Mass Index (BMI) of 30 kg/m² or higher qualifies an adult as obese¹⁸⁷. Epidemiological evidence indicates that midlife obesity exerts a stronger influence on dementia development than weight gain later in life, emphasizing the importance of early intervention and long-term weight management strategies.

A meta-analysis of 19 long-term studies concluded that individuals who were obese between the ages of 35 and 65 were approximately 30% more likely to develop dementia later in life. Interestingly, individuals categorized as merely overweight, but not obese, did not exhibit the same elevated risk¹⁸⁸. Moreover, obesity has a compounding effect on other dementia-related conditions. Those with obesity are two to three times more likely to develop diabetes and hypertension, both of which have well-documented links to cognitive impairment¹⁸⁹. Furthermore, obesity is also associated with depression, reduced physical activity, and increased social withdrawal, compounding the overall risk for dementia.

From a biological standpoint, obesity contributes to systemic inflammation through the release of proinflammatory adipokines such as TNF- α , IL-6, and leptin, which may cross the blood-brain barrier and promote neuroinflammation. Chronic low-grade inflammation disrupts neuronal signaling, increases oxidative stress, and impairs synaptic plasticity, all of which accelerate the pathogenesis of dementia. Furthermore, obesity can reduce cerebral perfusion by worsening vascular health, thereby increasing vulnerability to vascular cognitive impairment.

Tobacco use is a notable modifiable risk factor for dementia, including both Alzheimer's disease and vascular dementia¹⁹⁰. Smoking contributes to the progression of cognitive decline through several mechanisms, most notably its destructive impact on vascular health. Cigarette smoking induces atherosclerosis and damages the endothelium, impairing the brain's microcirculation and oxygen supply. Reduced cerebral blood flow increases susceptibility to ischemic events such as transient ischemic attacks (TIAs) and strokes, both of which are strongly linked to vascular cognitive impairment and dementia progression.

The toxic chemicals in tobacco smoke also promote oxidative stress and persistent neuroinflammation, both of which are central to the neuronal degeneration¹⁹¹. Some evidence suggests that smoking hampers the brain's ability to eliminate beta-amyloid plaques, which are hallmark features of Alzheimer's disease pathology¹⁹². While nicotine may offer short-term cognitive stimulation and improved alertness due to enhanced cholinergic activity, chronic nicotine exposure disrupts neurotransmitter balance, contributes to dopaminergic dysfunction, and ultimately worsens neural degeneration over time¹⁹³.

Smoking further increases the probability of developing other chronic conditions, such as diabetes, hypertension, and cardiovascular disease, all of which compound the risk for dementia through independent and synergistic pathways¹⁹⁴. The cumulative impact of smoking, vascular injury, oxidative damage, inflammatory cascades, and interactions with other systemic diseases, amplifies cognitive vulnerability, making smoking cessation a critical intervention in dementia prevention strategies.

Educational attainment, or lack thereof, plays a pivotal role in the prevalence of dementia, with multiple studies highlighting low education levels as a key social determinant of cognitive decline¹⁹⁵. The Cognitive Reserve Theory provides an explanatory framework for this relationship. It postulates that individuals with higher education levels develop more robust and flexible cognitive networks, enabling them to compensate for neurodegenerative damage more effectively and delay the onset of clinical symptoms¹⁹⁶. By contrast, those with minimal formal education are presumed to have lower cognitive reserves, making them more vulnerable to dementia¹⁹⁷.

Educational disadvantage is also strongly associated with reduced access to preventive healthcare services, lower health literacy, and fewer healthy behaviors, such as maintaining an optimal diet, engaging in regular exercise, and managing chronic diseases¹⁹⁸. These factors elevate exposure to multiple dementia risks, including hypertension, diabetes, and obesity. Furthermore, individuals with limited education are more likely to occupy lower socioeconomic strata, which may restrict access to mentally stimulating environments, quality medical care, and nutritious food¹⁹⁹. Additionally, certain types of employment associated with lower education levels may involve repetitive manual labor with little cognitive engagement or greater exposure to

environmental toxins, both of which have been correlated with poorer long-term brain health²⁰⁰.

Research has also shown that limited cognitive stimulation in early life may impede optimal brain development, laying a foundation for diminished cognitive function in later years²⁰¹. Enhancing early educational opportunities not only increases cognitive reserve but also improves health literacy, socioeconomic status, and lifestyle behaviors, thereby reducing cumulative dementia risk over the lifespan.

Hypertension, often referred to as elevated blood pressure, is widely acknowledged as a leading modifiable risk factor for dementia in numerous studies. When blood pressure remains persistently high over time, it can severely damage the vascular system, particularly the small blood vessels in the brain²⁰². Additionally, chronic hypertension has been associated with the emergence of white matter hyperintensities and small vessel diseases, both of which are established indicators of cognitive decline.

Scientific investigations have documented that prolonged uncontrolled hypertension may lead to microvascular strokes, often undetected, which gradually degrade cognitive functions prior to the manifestation of overt dementia symptoms²⁰³. Epidemiological evidence suggests that elevated blood pressure during midlife is significantly correlated with increased risk of cognitive impairment later in life²⁰⁴. Importantly, lifestyle interventions such as dietary regulation, physical activity, and antihypertensive medications have demonstrated efficacy in minimizing these risks. This underscores the importance of early hypertension screening and management as a strategic public health intervention to mitigate dementia burden globally²⁰⁵.

Social isolation, characterized by a lack of meaningful interpersonal connections or engagement in social settings, has emerged as a substantial psychosocial risk factor contributing to cognitive decline and dementia incidence²⁰⁶. Unlike loneliness, which refers to subjective emotional distress, social isolation is more objective, it concerns the frequency of social interaction, living arrangements, and participation in communal activities.

Evidence reveals that individuals who are socially isolated often engage in fewer cognitively stimulating activities, which leads to a diminished cognitive reserve, a known risk factor for early-onset dementia²⁰⁷. Moreover, social isolation tends to promote mental health challenges such as chronic stress and depression, both of which can adversely affect the hippocampus, a region critically involved in learning and memory formation²⁰⁸.

Biologically, socially disconnected individuals often exhibit increased levels of cortisol and inflammatory markers, which may lead to accelerated neural degradation²⁰⁹. Behaviorally, isolation is frequently accompanied by negative health practices, including sedentary behavior, poor dietary choices, tobacco use, and inconsistent adherence to medical therapies, all of which are contributors to dementia pathogenesis²¹⁰. Neuroimaging research supports these findings, showing that individuals with robust social networks tend to maintain larger brain volumes and greater neural connectivity, while socially isolated individuals display atrophic brain changes linked to cognitive dysfunction^{211, 212}.

Depression stands as one of the most prevalent and underdiagnosed neuropsychiatric disorders among middle-aged men, and it plays a significant role in the etiology of cognitive disorder. This affective condition exerts its deleterious effects on the brain via

prolonged psychological stress, which in turn alters neuroendocrine responses and neural plasticity.

A primary mechanism through which depression increases dementia risk is elevated cortisol secretion, a hallmark of chronic stress, which has been shown to shrink the hippocampus, the brain region responsible for encoding new memories^{213, 214}. Damage to the hippocampus has been consistently linked to both Alzheimer's disease and vascular cognitive impairment. For midlife males, undiagnosed depression may lead to long-term structural and functional brain alterations, making them particularly vulnerable to developing dementia in later life.

Furthermore, depression is often comorbid with vascular risk factors such as hypertension and diabetes, further amplifying the risk of cerebrovascular-related cognitive decline²¹⁵. The stigma surrounding mental health in many societies often prevents men from seeking treatment, exacerbating the long-term neurological impact. Integrating mental health care into primary and geriatric services is thus vital for early detection and intervention.

Cerebrovascular disease represents a significant etiological contributor to the progression of dementia, particularly among aging males. A variety of vascular dysfunctions associated with CVD are known to exacerbate cognitive decline, highlighting a complex interplay between neurovascular pathology and cognitive deterioration in later life stages²¹⁶. As men advance in age, the burden of cerebrovascular insults becomes more pronounced, forming a critical component in dementia risk modeling.

Hypertension is a principal driver within the CVD spectrum. Persistent elevation of blood pressure exerts damaging effects on the integrity of arterial walls, particularly the small

vessels supplying the brain. These changes lead to endothelial injury, vascular stiffness, and arterial narrowing, culminating in compromised cerebral perfusion²¹⁷. Among middle-aged and elderly males, such hypertension is frequently tied to lifestyle determinants including stress exposure, excessive sodium intake, alcohol consumption, and sedentary behavior. Over time, this constellation of factors elevates the likelihood of ischemic damage, which is strongly associated with vascular dementia.

Another well-established contributor is atherosclerosis, a condition marked by the deposition of lipid-rich plaques in arterial walls. These plaques impede or obstruct cerebral blood flow, setting the stage for transient ischemic attacks (TIAs) and strokes, both of which are common precursors to cognitive impairment²¹⁸. Men with poor dietary habits, limited physical activity, or smoking behaviors fall into high-risk groups for developing atherosclerotic complications, which may trigger or accelerate dementia-related changes²¹⁹.

In tandem with vascular damage, Type 2 diabetes mellitus significantly heightens the risk of cerebrovascular compromise. Chronic hyperglycemia leads to both macrovascular and microvascular injury, promoting inflammation and capillary dysfunction in the brain²²⁰. Among men in their midlife, unregulated insulin resistance contributes to gradual, often asymptomatic cerebrovascular degeneration. Structural changes such as white matter hyperintensities, microinfarctions, and cortical atrophy are commonly observed in diabetic individuals, linking metabolic dysfunction to the pathophysiology of the brain vessels.

Collectively, these interconnected vascular and metabolic mechanisms illustrate how cerebrovascular pathology serves as a foundation for cognitive impairment. Preventive

strategies aimed at reducing modifiable risks, hypertension control, diabetes management, lifestyle interventions, are critical for mitigating long-term cognitive decline among vulnerable male populations.

Beyond biomedical and lifestyle determinants, psychosocial elements such as marital status have emerged as influential factors in the onset of cognitive decline. Accumulating research suggests that individuals, particularly middle-aged men, who remain unmarried, divorced, or widowed face a disproportionately higher risk of developing dementia compared to their married counterparts²²¹. The absence of a life partner often corresponds with reduced emotional support, irregular health habits, social disconnection, and delayed engagement with health services.

One major pathway through which unmarried status contributes to cognitive decline is social isolation. Marital partnerships frequently offer built-in structures for daily interaction, emotional regulation, and health monitoring, elements that are essential for maintaining cognitive resilience²⁰⁴. Without these, individuals may experience loneliness and decreased cognitive stimulation, both of which are independently associated with neurodegeneration²²². The richness of frequent, meaningful social contact has been shown to protect cognitive functions, while its absence allows decline to proceed unchecked.

Empirical studies affirm that social engagement acts as a cognitive buffer, supporting memory and executive function. In contrast, unmarried men often lack access to such consistent stimulation, which diminishes their cognitive reserve and accelerates vulnerability to dementia. Moreover, spouses often act as facilitators for timely healthcare, scheduling check-ups, encouraging adherence to medication, and recognizing

early signs of mental decline. Unmarried individuals, particularly men, may delay seeking medical attention or disregard subtle symptoms, allowing pathological changes to progress undetected^{223,224}.

From a psychological perspective, unmarried men are statistically more likely to experience depression, anxiety, and chronic stress, all of which have adverse effects on brain health. These emotional states can dysregulate neuroendocrine pathways, notably through elevated cortisol levels, which can impair memory systems and damage the hippocampus over time^{225, 226}. Life transitions such as retirement, loss of loved ones, or health setbacks can further exacerbate emotional instability in the absence of a supportive partner.

Unmarried status also correlates with the adoption of unhealthy lifestyle behaviors. Studies have shown that single men are more prone to smoking, heavy alcohol intake, physical inactivity, and poor dietary practices, all of which elevate dementia risk either directly or through cardiovascular and metabolic intermediaries²²⁷. In contrast, married men typically benefit from shared health responsibilities, mutual encouragement, and a structured lifestyle that fosters better long-term health.

2.2 Theoretical Framework

2.2.1 Health Belief Model (HBM)

The Health Belief Model (HBM) is a psychological framework developed in the 1950s by social psychologists Hochbaum, Rosenstock, and Kegels at the U.S. Public Health Service. It was initially designed to explain and predict health-related behaviors,

particularly the uptake of preventive health services such as screenings and immunizations. Since its inception, the model has become one of the most widely used theories in health education, health promotion, and behavioral medicine. Its enduring relevance lies in its simplicity, adaptability, and capacity to explain a broad range of health behaviors across different populations and health conditions.

The HBM is grounded in the assumption that individuals are rational actors who will engage in health-related behaviors if they believe that doing so will reduce their risk of illness or improve their health outcomes. It posits that health behavior is influenced by personal beliefs or perceptions about a disease and the strategies available to decrease its occurrence. The model comprises several key constructs: perceived susceptibility, perceived severity, perceived benefits, perceived barriers, cues to action, and self-efficacy²²⁸. Each of these elements contributes to the decision-making process that precedes health behavior change.

Perceived susceptibility refers to an individual's belief about the likelihood of contracting a health condition. When individuals perceive themselves to be at higher risk, they are more likely to engage in behaviors that mitigate that risk. Conversely, if they believe they are unlikely to be affected, they may disregard health messages or delay taking preventive action. Perceived vulnerability is one of the most consistent predictors of health behavior, particularly in preventive contexts such as vaccination and screening²²⁸.

Perceived severity involves the belief about the seriousness of a health condition and its potential consequences. This includes both medical implications, such as pain or disability, and social consequences, such as stigma or financial burden. A person who believes that a disease will have severe outcomes is more motivated to take preventive

action. Perceived severity is often a weaker predictor than susceptibility but gains influence when paired with strong beliefs in personal risk²²⁸.

Perceived benefits refer to an individual's belief in the efficacy of the advised action to reduce the risk or severity of impact. If the perceived benefits of taking action outweigh the perceived costs or barriers, the individual is more likely to adopt the behavior. Belief in the effectiveness of a behavior is a central motivator, especially in preventive health measures like quitting smoking or adopting healthier diets²²⁸.

Perceived barriers are the individual's assessment of the obstacles to performing a behavior. These can be practical, such as cost or time, or psychological, such as fear or stigma. Among all HBM constructs, perceived barriers are considered the most powerful single predictor of behavior²²⁸. Interventions that aim to reduce these barriers, through financial support, education, or access to services, are often more successful in achieving behavioral change.

Cues to action are external or internal stimuli that trigger the decision-making process. These cues can take many forms, such as media campaigns, reminders from healthcare providers, or the onset of symptoms. They serve as activating factors, prompting individuals to make a health-related decision. While cues to action are less studied empirically compared to other constructs, they are nonetheless critical in initiating behavior, particularly in populations that are otherwise unaware or disengaged²²⁸.

Self-efficacy, which was later added to the model by Bandura in the 1980s, refers to an individual's confidence in their ability to perform a specific behavior²²⁸. This construct addresses the question of whether an individual believes they can execute the action required to achieve the desired health outcome. High self-efficacy is associated with

greater persistence, resilience, and success in behavior change. Incorporating self-efficacy into the HBM improved its predictive power, particularly in behaviors requiring long-term maintenance, such as exercise or dietary adherence²²⁸.

Despite its strengths, the HBM has limitations. Critics argue that it does not adequately account for emotional, habitual, or social influences on behavior. For example, peer pressure, cultural norms, or structural barriers are not explicitly integrated into the model. Additionally, the model assumes that behavior is the result of logical decision-making, which may not fully capture the complexities of human behavior. Nonetheless, it remains a valuable tool in designing, implementing, and evaluating health promotion programs²²⁸. It provides a clear structure for understanding the cognitive factors that influence health decisions and is frequently used in conjunction with other models to address its limitations.

A central construct of the HBM, perceived barriers, is directly relevant to this study's focus. Perceived barriers refer to an individual's evaluation of the obstacles that may prevent them from engaging in a recommended behavior, such as adhering to a therapeutic diet. These barriers can be tangible, such as financial costs or lack of access to healthy food, or intangible, such as low motivation, taste preferences, or lack of family support. Perceived barriers are the strongest and most consistent predictor of health-related behavior within the HBM framework¹¹⁵. In the context of dietary adherence, these barriers may influence whether a patient follows a low-sodium diet for hypertension, a gluten-free diet for celiac disease, or a carbohydrate-controlled plan for diabetes.

Other constructs of the HBM also contribute to understanding adherence behaviors. Perceived susceptibility and perceived severity describe a patient's belief about their risk

of experiencing negative health outcomes if they do not adhere to dietary recommendations. For example, a diabetic patient who believes they are at high risk for complications like neuropathy or vision loss due to poor dietary habits may be more motivated to comply with nutritional guidelines⁹. Conversely, a patient who does not perceive their condition as serious or who feels asymptomatic may not prioritize dietary changes. Both perceived severity and susceptibility influence intention but may require reinforcement through education and communication for sustained behavior change.

Perceived benefits are equally important in this analysis. They refer to the individual's belief in the positive outcomes of performing the recommended behavior, in this case, the perceived advantages of maintaining a therapeutic diet. Patients who believe that adherence to dietary therapy will result in better disease control, reduced medication dependency, or improved energy levels are more likely to engage consistently with their diet plans. Belief in efficacy must be strong enough to outweigh the perceived barriers for behavior to occur. In diet therapy, this means that benefits must be understood not only as long-term health improvements but also as meaningful short-term gains such as improved digestion, better sleep, or increased physical activity.

Cues to action and self-efficacy further contextualize adherence behavior in this thesis. Cues to action are the triggers that stimulate the decision to engage in a health behavior. These can include advice from healthcare providers, family encouragement, physical symptoms, or exposure to health information. In diet therapy, cues may come from follow-up appointments, nutrition counseling, or observing health decline due to poor dietary habits. These cues often determine the timing and urgency of the patient's decision to act²²⁸. Self-efficacy, or the belief in one's ability to execute the required

dietary behavior, is another key determinant. Self-efficacy strongly influences the maintenance of health behaviors²²⁸. In the case of diet therapy, self-efficacy reflects a patient's confidence in their ability to shop for healthy ingredients, prepare meals, resist unhealthy foods, and maintain dietary control in social settings.

2.2.2 Cognitive Reserve Theory

The Cognitive Reserve Theory (CRT) has emerged as one of the most influential frameworks for understanding variability in dementia onset and progression. First proposed by Stern the theory posits that individuals differ in their capacity to withstand neuropathological damage without manifesting clinical symptoms of cognitive decline. This “reserve” is thought to be shaped by a combination of genetic, lifestyle, and environmental factors that either enhance or diminish resilience against dementia. CRT emphasizes two main constructs: brain reserve and cognitive reserve²²⁹. Brain reserve refers to the structural aspects of the brain, such as volume, synaptic density, and neuronal count, while cognitive reserve refers to the functional efficiency, flexibility, and adaptability of neural networks. Diet can influence both constructs. For example, nutrient-rich diets—such as the Mediterranean or MIND diets—are high in antioxidants, polyphenols, and omega-3 fatty acids, which promote neuronal health, synaptic plasticity, and neurogenesis. These biological effects contribute to greater brain reserve by maintaining structural integrity. At the same time, consistent consumption of balanced diets supports efficient neural functioning, thereby strengthening cognitive reserve.

In contrast, poor dietary patterns characterized by excessive intake of refined carbohydrates, saturated fats, and processed foods have been associated with chronic inflammation, oxidative stress, and metabolic disorders such as obesity and diabetes.

These conditions accelerate neurodegenerative processes and reduce both structural and functional resilience of the brain. Among middle-aged men, such dietary practices can diminish their cognitive reserve, leaving them more vulnerable to the clinical manifestation of dementia in later life.

The relevance of CRT to this study also lies in its lifespan perspective. The theory underscores that cognitive reserve is not fixed but accumulates or declines throughout life depending on lifestyle choices. Midlife, typically considered between ages 44 and 60, is a crucial period for intervention because it represents a window in which protective lifestyle modifications—particularly dietary adjustments—can exert long-term benefits. Building cognitive reserve during this stage may delay the onset or reduce the prevalence of dementia in later years.

Furthermore, CRT integrates well with other risk factors identified in dementia research, such as education, physical activity, and social engagement¹³³. For middle-aged men, adopting dietary patterns that support cardiovascular and metabolic health also complements these other factors by reducing systemic risks that indirectly affect brain health. For instance, diets that lower hypertension and improve glycemic control help maintain cerebrovascular integrity, thereby supporting neural efficiency and adaptability. In practical terms, CRT suggests that dietary interventions are not merely nutritional but also cognitive health strategies. By promoting dietary patterns that enhance brain plasticity and resilience, public health policies and individual practices can effectively increase the cognitive reserve of middle-aged men. This, in turn, may mitigate the prevalence of dementia in this population.

2.3 Review of Empirical Studies

Cognitive decline, a progressive and irreversible condition affecting cognitive capacity, is classified into multiple subtypes according to their underlying biological causes²³⁰. While it is most prevalent among older adults, emerging data suggests that middle-aged populations are increasingly affected, particularly in low- and middle-income countries such as Nigeria²³¹. One global review analyzing dementia prevalence by region, age, and gender revealed substantial increases in prevalence with age. Individuals aged 100 years and above had rates up to 6,592 per 10,000, compared to just 27 per 10,000 in the 50–59 age group, demonstrating a more than 240-fold increase with advanced age²³². These data illustrate the exponential nature of dementia risk, highlighting age as the most potent and consistent risk factor. Importantly, while age-related neurodegeneration is a universal biological phenomenon, its clinical onset and severity are shaped by lifestyle, socioeconomic status, education level, and access to healthcare.

Gender disparities were also clearly observed in global prevalence data. Females generally exhibited higher overall dementia prevalence compared to males (788 vs. 561 per 10,000). Within the 60–69 age range, Alzheimer's disease prevalence was nearly double in women compared to men, likely due to a combination of hormonal changes post-menopause, longer life expectancy, and differences in cognitive reserve. In contrast, vascular dementia showed a slight male predominance (56 vs. 32 per 10,000), reflecting higher midlife cardiovascular risk factors in men²³³. Geographic disparities were also reported, with higher rates in Europe and North America compared to regions in Asia,

Africa, and South America. However, these differences were attributed more to study design and diagnostic access than actual biological or demographic variation.

A systematic review focusing on cognitive abilities in Nigeria underscored the scarcity of nationwide, high-quality epidemiological data. Nevertheless, available studies estimated a 4.9% (95% CI: 3.0–6.9%)²³⁴. Most of these studies were concentrated in southwestern Nigeria, meaning regional disparities remain underexplored. Importantly, higher cognitive decline among women was consistently observed, echoing global trends. Risk factors such as age, sex, and body mass index (BMI) emerged as consistent predictors of dementia occurrence. The review also concluded that cognitive decline has been steadily increasing in Nigeria over the past two decades, largely due to demographic shifts (aging population), urbanization, changing diets, and limited early intervention or diagnostic infrastructure.

Research from Singapore revealed similarly high cognitive decline rates in people aged 60 and above, with older age (especially 75+), lower educational attainment, and retired or homemaker status being associated with increased risk²³⁵.

Decline in cognitive capabilities continues to pose an increasing global health burden, with projections indicating a marked rise in prevalence over the coming decades²³⁶. As populations age, the economic, social, and emotional toll of dementia is expected to increase disproportionately in low- and middle-income countries, where healthcare systems are less equipped to manage chronic neurodegenerative disorders. While no definitive cure currently exists, accumulating evidence suggests that modifiable lifestyle factors, particularly nutrition, play a vital role in prevention, especially when interventions are implemented during the early stages of cognitive decline. Nutritional

epidemiology has increasingly revealed that specific dietary habits and overall diet quality can influence the onset, severity, and trajectory of dementia, particularly in older adults. However, the scientific consensus remains complex and sometimes inconsistent, largely due to differences in study design, dietary assessment tools, cultural eating patterns, and genetic predispositions.

Not all longitudinal investigations have found a clear-cut relationship between diet and dementia. For instance, one 20-year longitudinal study exploring the relationship between midlife dietary habits and cognitive decline reported no statistically significant association between long-term dietary patterns and dementia incidence²³⁸. This finding suggests that some dietary changes may not exert measurable effects on cognitive decline over very long periods, methodological limitations, such as recall bias in dietary reporting or insufficient sample sizes, may mask true associations, and diet alone might not override the influence of other non-modifiable risk factors, such as age or genetic predisposition.

Conversely, evidence from culturally specific populations has shown more positive associations between traditional diets and reduced risk of cognitive decline. A Japanese prospective cohort study examining the influence of dietary changes over time found a protective association between greater adherence to traditional Japanese eating habits (high in fish, vegetables, soy products, and green tea) and lower dementia risk²³⁹. Participants who maintained or improved their adherence to these traditional dietary patterns experienced reduced incidence rates, while those who deviated toward more Westernized diets, high in refined sugars and saturated fats, demonstrated increased impairment vulnerability. This suggests that dietary consistency over time, especially

diets rich in nutrient-dense, anti-inflammatory foods, may serve as a protective factor for cognitive health.

Another investigation sought to explore whether genetic predispositions to Alzheimer's disease modify the effects of diet on cognitive decline. Interestingly, while the study confirmed significant associations between dietary habits and overall dementia incidence, it found no detectable interaction between genetic markers (such as APOE-ε4 allele) and dietary patterns in the development of Alzheimer's disease specifically²⁴⁰. The findings suggest that healthy diets may reduce general dementia risk, but for individuals with strong genetic risk factors, the protective effects of diet alone may be limited. Importantly, participants who adhered to healthy, plant-based diets showed lower dementia risk, whereas those following Western-style dietary patterns, rich in saturated fats, processed meats, and refined sugars, demonstrated significantly elevated risk levels.

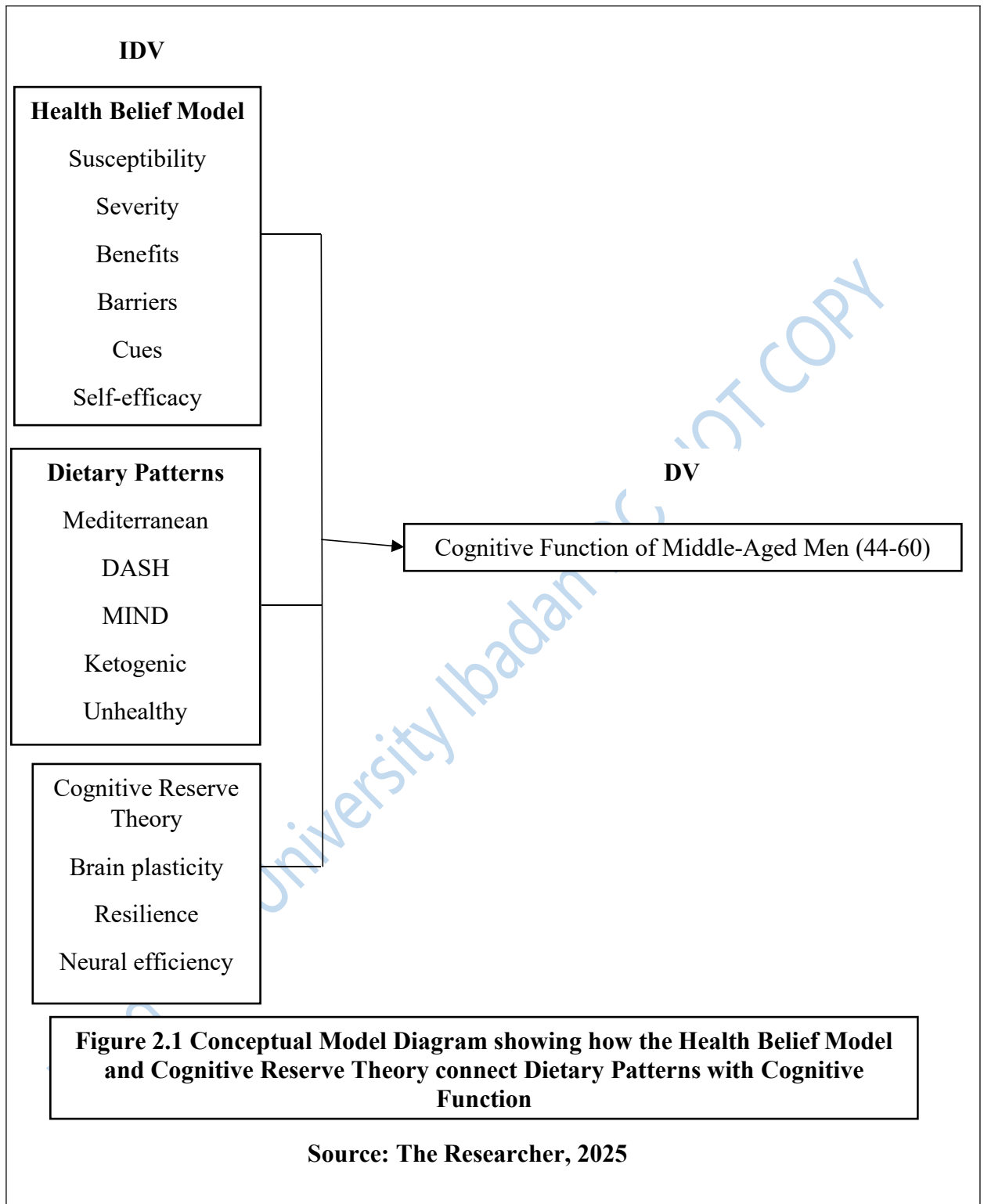
An extensive systematic review of dietary habits and their correlation with Alzheimer's disease and age-related cognitive dysfunction further emphasized the neuroprotective potential of certain eating patterns. It concluded that Mediterranean-style diets, as well as emerging models like DASH and MIND diets, are consistently associated with slower cognitive decline and reduced Alzheimer's disease rates. The study further examined controversial food items. For instance, while eggs and red meat have often been regarded as detrimental, their roles may be context-dependent. High intakes of saturated fats were consistently linked to cognitive decline, whereas diets rich in fish, monounsaturated fats, and omega-3 polyunsaturated fatty acids (PUFAs) were associated with enhanced brain health²⁴¹.

Further evidence from a large-scale observational study reinforced the importance of overall diet quality. Participants with high adherence to nutrient-dense and balanced dietary patterns, sometimes referred to as “high diet quality”, had a significantly lower overall risk of dementia and Alzheimer’s disease, compared to those consuming low-quality, nutrient-poor diets¹⁵. The study also revealed that dementia risk was influenced by age, gender, and duration of dietary adherence, indicating that the cognitive benefits of diet accumulate over time. Therefore, adopting healthy dietary practices earlier in life may yield more substantial protective effects than dietary changes implemented late in life.

In another five-year longitudinal study conducted among older Malaysian adults living independently in the community, researchers assessed how different dietary patterns affected the incidence of both mild cognitive impairment (MCI). A sample of 280 adults aged 60 and above was followed, with data collected on medical history, blood biomarkers, anthropometric measures, psychosocial status, and food intake. After adjusting for confounding factors, the findings indicated that a diet high in local snacks, seafood, and salty foods was associated with higher MCI risk, while a fruit-and-oats-based diet correlated with a lower likelihood of developing AD. These results underscore the dual role diet can play, either as a protective shield or a risk factor, depending on composition.

2.4 Conceptual Model

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2.5 Summary of Gaps in Literature Reviewed

Despite the growing body of research examining the relationship between dietary patterns and cognitive abilities, several significant gaps remain, particularly about middle-aged men. Most existing studies on risk of cognitive decline have focused predominantly on older adults aged 65 and above, since this group presents the highest prevalence rates of the condition. While this focus is understandable, it leaves a major knowledge gap concerning the critical midlife period, during which dietary and lifestyle choices may significantly influence cognitive health later in life. Evidence suggests that dementia is a disease with a long preclinical phase, and the cumulative impact of diet may begin decades before symptoms appear. Yet, there is limited empirical investigation into how dietary habits and patterns of men aged 44–60 shape outcome of cognitive abilities, creating a need for targeted studies in this age group.

Much of the literature has explored diet and cognitive decline in mixed populations or has focused on women due to their higher prevalence of Alzheimer's disease. While this has provided important insights, men in midlife present distinct dietary behaviors, risk profiles, and health outcomes that are often overlooked. For instance, men are more likely to engage in high-calorie, meat-heavy, and alcohol-rich diets, which may interact differently with dementia risk factors compared to women. Current evidence is insufficient in disaggregating findings by gender to establish whether the dietary patterns protective against dementia in women apply equally to men. There are also inconsistencies in the findings regarding specific dietary patterns. While the Mediterranean and MIND diets have been widely associated with reduced cognitive decline, results regarding ketogenic, DASH, or high-protein diets remain inconclusive.

Some studies show protective effects, while others highlight potential adverse consequences, particularly concerning cardiovascular health. These contradictions limit the development of unified dietary recommendations for dementia prevention. Furthermore, many studies examine single nutrients (such as omega-3 fatty acids or antioxidants) rather than broader dietary patterns, which makes it difficult to capture the complex interactions of foods in everyday diets.

Another important gap involves the role of comorbid risk factors such as obesity, diabetes, hypertension, smoking, and physical inactivity. While these factors are recognized contributors to dementia, few studies explicitly examine how they interact with dietary patterns in shaping prevalence among middle-aged men. This lack of integrative research makes it difficult to isolate whether diet exerts independent protective effects or whether its impact is mediated through cardiovascular and metabolic pathways.

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Chapter Three

Methodology

3.1 Research Design

Descriptive cross-sectional design was adopted for this study. This is to enable the researcher to give detailed description on the Influence of dietary pattern and cognitive abilities of middle-aged men (44-60) in Eti-Osa LGA, Lagos State, Nigeria. The reason for using this design is because it does not call for manipulation of variables. But it allows an in-depth study of the subject matter, and it is suitable to described attitudes, views or opinions and behavior pattern of the people.

3.2 Study Area

The study was carried out in Eti-Osa, Local Government Area of Lagos State Nigeria. It has two normal seasons which are dry and rainy seasons. The temperature of the area is 25°C with wind blowing at an average of 13 km/h. The Local Government Area has a population of 283,791, which represents 3.11% of the state's population. 158,858 of the total population are male while the remaining 124,933 are female ¹. The local government is bounded with Ikoyi, Lekki and Victoria Island². The people of Eti-Osa are

predominantly from the Awori Yoruba and Ijebu Yoruba. However, like most parts of Lagos State, it is currently home to a diverse mix of ethnicities from different states of the country ³. The population including people from Hausa, Igbo, Yoruba and other tribes.

There are fewer industries within Eti-Osa Local Government Area. Being the former location of the nation's capital, Eti-Osa is home to many large domestic and international businesses ³. Eti-Osa is a commercial area that has several public and private institutions with Banks, hotels, clubs and modern markets where the exchange of good and services take place ³. In 2009, Eti-Osa was placed under study for commercial horticultural practices in Nigeria, where it was discovered that it had 75 gardens that were concentrated in Ikoyi and Victoria Island ⁴.

3.3 Population of the Study

The study population encompassed only middle aged men (44-60) in Eti-Osa Local Government Area of Lagos State. The study considered only middle aged male from diverse socio-economic backgrounds and family structures. It was focus on assessment of dietary pattern and the prevalence of dementia among middle aged men (44-60) in Eti-Osa LGA, Lagos State, Nigeria.

3.4 Sampling Size Determination and Sample Techniques

Cochran formula was used to determine the sample size for this study as shown below:

$$n = \frac{Z^2 pq}{e^2}$$

Where Z depends on the degree of confidence level and they are checked from the contribution table, therefore, 95% of the confidence level from the t-distribution table gives $z = \pm 1.96$.

The degree of variability is denoted as “p” and expressed as decimal; if the degree of variability is not known, then, 50% is to be used, which is equal to 0.5; “e” is the marginal error or confidence interval, or precision expressed as decimal; a precision rate +5 which is 0.05. Since the population of study is unknown, we therefore use the formula below to derive the sample size for this study:

$$n = \frac{Z^2 pq}{e^2}$$

$z = 1.96$; Confidence level = 95%

$p = 50\%$

$e = \pm 5\%$

$q = 1 - p$

Therefore, $z^2 = 1.96^2$; $p = 0.5$ and $e = 0.05$, $q = 1 - 0.5$

$$\frac{1.96^2 \times 0.5 \times (1 - 0.5)}{0.05^2}$$

$n = 384.16$

10% attrition

10% of 385 = 38.5 approximately 39

Total Sample = 385 + 39 = 424

The sampling population is approximately 424 respondents, which is statistically suitable given the population size and the desired precision level. A sample of 424 ensures that data can be collected efficiently while maintaining the validity and reliability of the study findings. This sample size is adequate for drawing meaningful conclusions and generalizations about the target population.

For this study, a multistage sampling technique was employed to select respondents for the study in Eti-Osa Local Government Area of Lagos State. The multistage sampling approach ensures a systematic and representative selection process, covering different demographic groups within the middle aged population as showing below:

1. **Stage One: Stratified Sampling:** The study employed a stratified sampling technique to ensure a fair representation of middle aged men across selected areas in Eti-Ose LGA, Lagos State. Areas within the Local Government Area were categorized based on specific strata such as public and private areas to capture diverse socio-economic backgrounds. This stratification helped in selecting participants from different areas to ensure a balanced study population.
2. **Stage Two: Simple Random Sampling:** Random selection of respondents from public and private areas was done.

3.5 Instrument for Data Collection

The instrument for data collection was a well-structured questionnaire. The questionnaire was developed from the literature review and research questions, and it was titled:

Influence of dietary pattern and the prevalence of dementia among middle aged men (44-60) in Eti-Osa LGA, Lagos State. The instrument (questionnaire) consisted of five

sections, A, B, C and D. Section A the factors that influence the dietary habits and patterns of middle-aged men in Eti-Osa LGA, Lagos State; section B assess dietary habits and patterns by using the Food Frequency Questions; C, analyze the cognitive functions using the 10 Word Delay Recall Test and D, examines the relationship between dietary habits, pattern and cognitive functions.

The questionnaire consists of 46 items based on the research questions 1 to 4 of the study. It was structured using daily, 2-6 times, weekly and occasionally answering method except for section A which focussed on the social demography and socioeconomic characteristics of the respondents.

The 10-Word Delay Recall Test (10-WDRT) was used for cognitive assessment. The 1-WDRT, adapted from the Consortium to Establish a Registry of Alzheimer's Disease 10-word learning list, is test of memory. It has been to be valid tool, largely unaffected by educational level⁵.

3.6 Validation of Instruments

The questionnaire was validated by 10 respondents who could read and understand. They were examined and all the ambiguous and difficult items indicated on the Influence of dietary habits and patterns on the cognitive abilities of middle-aged men (44-60) in Eti-Osa LGA, Lagos State, Nigeria were removed. Thereafter, the validity of the instrument was established through content and face validation by the researcher's supervisor.

3.7 Method of Data Collection

The structured questionnaires were distributed to the selected number of respondents across the selected area in Eti-Osa Local Government Area using simple random and

purposive sampling techniques. The respondents' responses were also recorded for clarity. The researcher and two (2) trained field assistants distributed and retrieved the questionnaire within the study period to be able to guide, monitor, and retrieve at least 95% of the distributed questionnaires. This was done to ensure a good representation across the selected area in Eti-Osa Local Government Area.

3.8 Data Analysis

After the data collection process was completed, each questionnaire was checked for completeness and accuracy. The quantitative data were analyzed using Statistical Package for Social Science (SPSS). And the result was expressed using descriptive statistics (means and percentages).

Data were summarized using descriptive statistics such as frequency and percentage, mean and standard deviation. Independent t-test and ANOVA were used to test for mean differences between 10-Word Delay Recall Test (10-WDRT) and Associated Factors.

All data were analyzed using the IBM SPSS Statistics version 25.

Level of statistical significance was set at 5%.

Endnotes

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Chapter Four

Results and Discussion of Findings

4.1 Socio-demographic and economic characteristics of respondents

The sociodemographic characteristics of the respondents is shown in Table 4.1. All the participants of the study were males. Meanwhile, more than half of the respondents were aged between fifty and fifty-nine years (57.9%), some were less than fifty years old (32.5%) while others (9.6%) were sixty years and above. The median age was fifty years. Over one-third of the respondents (40.7%) were Yoruba, some were Igbo (32.8%), Hausa (24.6%) while very few of them belonged to another ethnicity (1.9%). About half of them (49.0%) practiced Islam, some practiced Christianity (44.0%) while others (6.9%) practiced African Traditional Religion. Majority of them (68.7%) were married, other were divorced (12.2%), widowed (10.3%), single (6.9%) or separated (1.9%). The respondents had a household size ranging from four and seven (46.7%), more than seven

(21.8%), and less than four (18.4%), although few of them (13.2%) did not disclose their household size. The median household size was six.

While the socio-economic characteristics of the respondents is shown in Table 4.2. About two-thirds of the respondents (61.2%) attained tertiary education, other attained secondary education (16.7%), and primary education (1.9%), while some others did not have a formal education (20.1%). Their occupation includes self-employed (39.0%), government employee (33.0%), private sector employee (14.6%), and farming (6.9%), although very few of them were unemployed (4.5%). The average monthly income of the respondents ranged from less than fifty thousand naira (4.3%), between fifty thousand but less than one-hundred thousand naira (9.6%), between one-hundred thousand and two-hundred thousand naira (21.5%), and above two two-hundred thousand naira (4.1%), although, about two-thirds of the respondents (60.5%) did not disclose their average monthly income. The respondents mostly resided in the urban centres (86.6%).

Table 4.1a Socio-demographic and Economic Characteristics of Respondents

Characteristics	Frequency (%)
Age	
40 – 49 years	136 (32.5)
50 – 59 years	242 (57.9)
> 60 years	40 (9.6)
Median (IQR)	50 (49 – 55)
Ethnicity	
Yoruba	170 (40.7)
Hausa	103 (24.6)
Igbo	137 (32.8)
Others	8 (1.9)
Religion	
Christianity	184 (44.0)
Islam	205 (49.0)
Traditional	29 (6.9)
Marital Status	
Single	29 (6.9)
Married	287 (68.7)
Divorced	51 (12.2)
Widowed	43 (10.3)
Separated	8 (1.9)
Household Size	
< 4	77 (18.4)
4 – 7	195 (46.7)
> 7	91 (21.8)
Undisclosed	55 (13.2)
Median (IQR)	6 (4 – 8)

Table 4.1a Socio-demographic and Economic Characteristics of Respondents

Level of Education	
No formal	84 (20.1)
Primary	8 (1.9)
Secondary	70 (16.7)
Tertiary	256 (61.2)
Occupation	
Government employee	138 (33.0)
Self employed	163 (39.0)
Private sector employee	61 (14.6)
Farm/Hunting/Fishing	37 (6.9)
Unemployed	19 (4.5)
Average Monthly Income	
<₦50,000	18 (4.3)
₦50,000 - <₦100,000	40 (9.6)
₦100,000 – ₦200,000	90 (21.5)
>₦200,000	17 (4.1)
Undisclosed	253 (60.5)
Median (IQR)	₦100,000 (₦75,000 – ₦120,000)
Residence	
Rural	23 (5.5)
Peri-urban	33 (7.9)
Urban	362 (86.6)

4.2 Dietary Habit of Respondents

Less than half of the respondents (44.3%) had two meals per day, others had three, one and more than one meal per day (35.2%, 16.7% and 3.8%, respectively). Meal skipping was very high among the respondents (78.2%). The frequency of meal skipping ranged from every day (48.6%), weekends only (26.0%), weekdays only (23.9%) and rarely (1.5%).

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Table 4.2 Dietary Habits

Habit	Frequency (%)
Number of Meals Per Day	
One	70 (16.7)
Two	185 (44.3)
Three	147 (35.2)
More than three	16 (3.8)
Meal Skipping	
Yes	327 (78.2)
No	91 (21.8)
If yes, How Often	
Everyday	159 (48.6)
Weekdays only	78 (23.9)
Weekends only	85 (26.0)
Rarely	5 (1.5)

4.2.1 Frequently Skipped Meals

The frequently skipped meals are shown in Figure 4.1 below. The frequently skipped meals were lunch (54.7%), breakfast (36.4%) and dinner (8.9%).

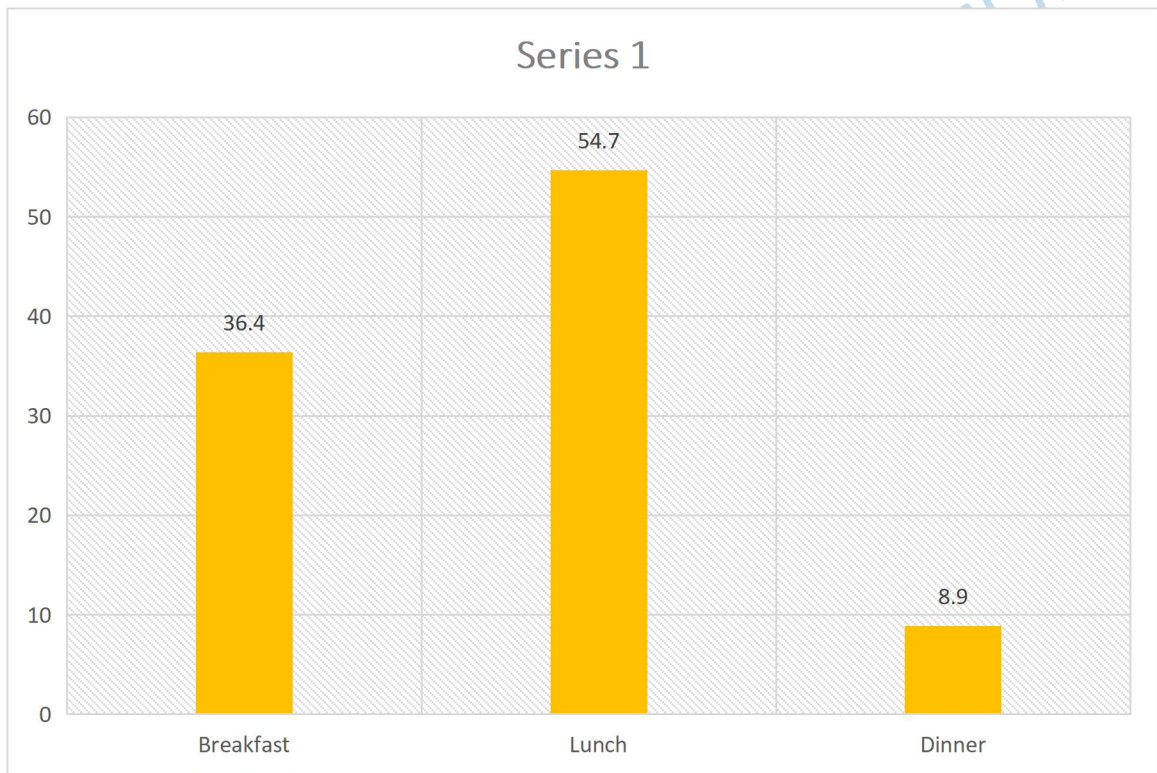


Figure 4.1 Frequently Skipped Meals

Source: Research, 2025

4.2.2 Reasons for Skipping Meals

The reasons respondents skipped meals include usual habit (44.6%), time constraints (29.8%), financial constraints (18.2%), loss of appetite (6.5%), and other reasons (0.9%).

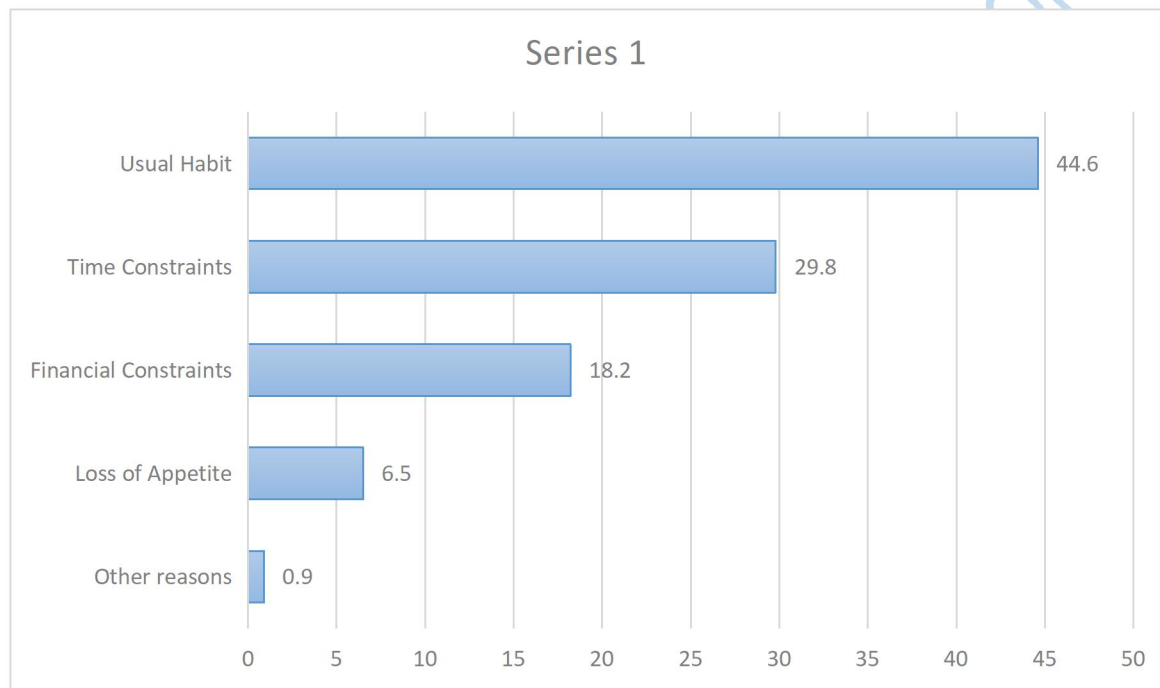


Figure 4.2 Reasons for Skipping Meals

Source: Research, 2025

4.2.3 Frequency of Food Consumption by Respondents

There was a daily consumption of grains (82.8%) and root and tuber (45.2%); a regular consumption of pulses (33.5%), beverages (40.2%) and fried food (39.5%); an occasional consumption of organic meat (42.3%), poultry (52.6%), fish and sea foods (38.8%), and dark-green leafy vegetables (68.9%); a rare consumption of nuts and seeds (47.1%), milk and dairy products (36.4%), processed meat (44.7%), vitamin-a-rich vegetables (48.1%), vitamin-a-rich fruits (55.7%), other vegetables (58.1%), and sugary food (34.2%).

Table 4.3 Frequency of Food Consumption by Respondents

Food Group	Daily	4-6x/week	2-3x/week	Rarely	Never
	n (%)	n (%)	n (%)	n (%)	n (%)
Grains	346 (82.8)	7 (1.7)	61 (14.6)	4 (1.0)	0 (0.0)
Roots and Tuber	189 (45.2)	18 (4.3)	128 (30.6)	82 (19.6)	1 (0.2)
Pulses	115 (27.5)	140 (33.5)	103 (24.6)	59 (14.1)	1 (0.2)
Nuts and Seeds	32 (7.7)	42 (10.0)	146 (34.9)	197 (47.1)	1 (0.2)
Milk and Dairy Products	37 (8.9)	126 (30.1)	102 (24.4)	152 (36.4)	1 (0.2)
Processed Meat	89 (21.3)	41 (9.8)	99 (23.7)	187 (44.7)	2 (0.5)
Organic Meat	115 (27.5)	54 (12.9)	177 (42.3)	71 (17.0)	1 (0.2)
Poultry	39 (9.3)	67 (16.0)	220 (52.6)	91 (21.8)	1 (0.2)
Fish and Sea Foods	37 (8.9)	115 (27.5)	162 (38.8)	104 (24.9)	0 (0.0)
Dark-green leafy vegetables	23 (5.5)	90 (21.5)	288 (68.9)	17 (4.1)	0 (0.0)
Vitamin-A-rich Vegetables	35 (8.4)	108 (25.8)	74 (17.7)	201 (48.1)	0 (0.0)
Vitamin-A-rich Fruits	50 (12.0)	58 (13.9)	76 (18.2)	233 (55.7)	1 (0.2)
Other Vegetables	44 (10.5)	53 (12.7)	78 (18.7)	243 (58.1)	0 (0.0)
Sugary Foods	33 (7.9)	116 (27.8)	122 (29.2)	143 (34.2)	4 (1.0)
Beverages	47 (11.2)	168 (40.2)	144 (34.4)	59 (14.1)	0 (0.0)
Fried Food	26 (6.2)	165 (39.5)	83 (19.9)	143 (34.2)	1 (0.2)

4.3 Relationship between Dietary Habits and Cognitive Abilities

Some of the respondents (53.1%) consume alcohol more than three times per week, participate in mentally stimulating activities (41.4%), engage in regular physical exercise (46.1%), and do not normally engage in social activities or interact with friends and family regularly (38.8%). Meanwhile, some of the respondents did not smoke cigarettes or use tobacco products regularly (51.4%), have not been diagnosed with depression or anxiety before (67.0%), did not have a family history of dementia or Alzheimer's disease (67.5%), or did not experience high levels of stress or work-related pressure (44.0%).

Table 4.4 Relationship between Dietary Habits and Cognitive Abilities

ITEMS	Strongly Agree n (%)	Agree n (%)	Disagree n (%)	Strongly Disagree n (%)
I consume alcohol more than three times a week	57 (13.6)	222 (53.1)	96 (23.0)	43 (10.3)
I smoke cigarettes or use tobacco products regularly	30 (7.2)	59 (14.1)	215 (51.4)	114 (27.3)
I engage in regular physical exercise (e.g., walking, jogging, gym workouts) at least three times a week	186 (44.5)	195 (46.7)	20 (4.8)	17 (4.1)
I normally feel isolated or lonely	70 (16.7)	64 (15.3)	140 (33.5)	144 (34.4)
I have been diagnosed with depression or anxiety before	43 (10.3)	55 (13.2)	280 (67.0)	40 (9.6)
I have a family history of dementia or Alzheimer's disease	32 (7.7)	51 (12.2)	282 (67.5)	53 (12.7)
I frequently experience high levels of stress or work-related pressure	52 (12.4)	104 (24.9)	184 (44.0)	78 (18.7)
I participate in mentally stimulating activities (e.g., reading, puzzles, learning new skills)?	173 (41.4)	142 (34.0)	28 (6.7)	75 (17.9)
I don't normally engage in social activities or interact with friends and family regularly	162 (38.8)	83 (19.9)	136 (32.5)	37 (8.9)

4.3.1 Family History of Cognitive Decline

Most of the respondents (89.0%) did not have a family history of dementia only a few had (11.0%).

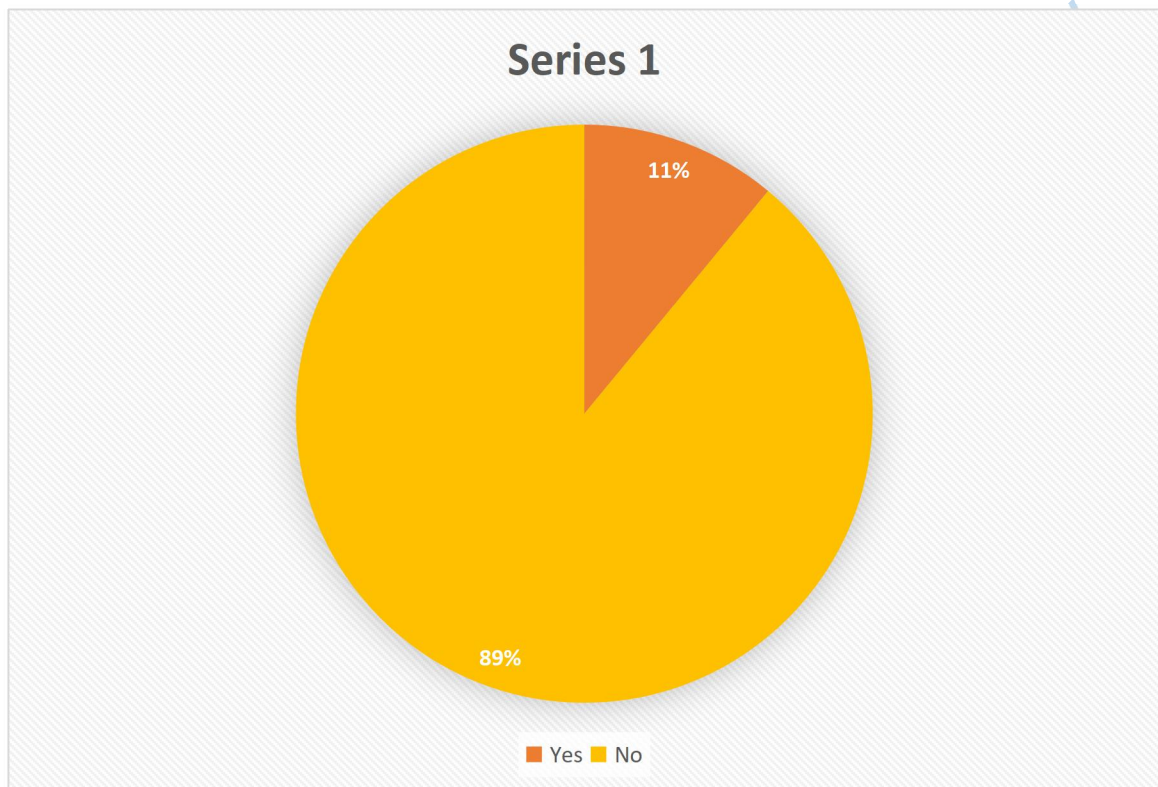


Figure 4.3 Family History of Cognitive Decline

Source: Research, 2025

4.4 10-Word Delay Recall Test (10-WDRT)

The respondents had varying degree of recall capacity; 9.3% of the respondents recalled only one of the selected words, 20.1% recalled two words, 11.5% recalled three words, 21.1% recalled four words, 10.8% recalled five words, 12.2% recalled six words, however, only 4.8%, 3.6%, 2.2% and 1.0% could recall seven, eight, nine and ten words, respectively. Meanwhile, it is worthy to note that only 3.6% of the respondents could not recall any of the words.

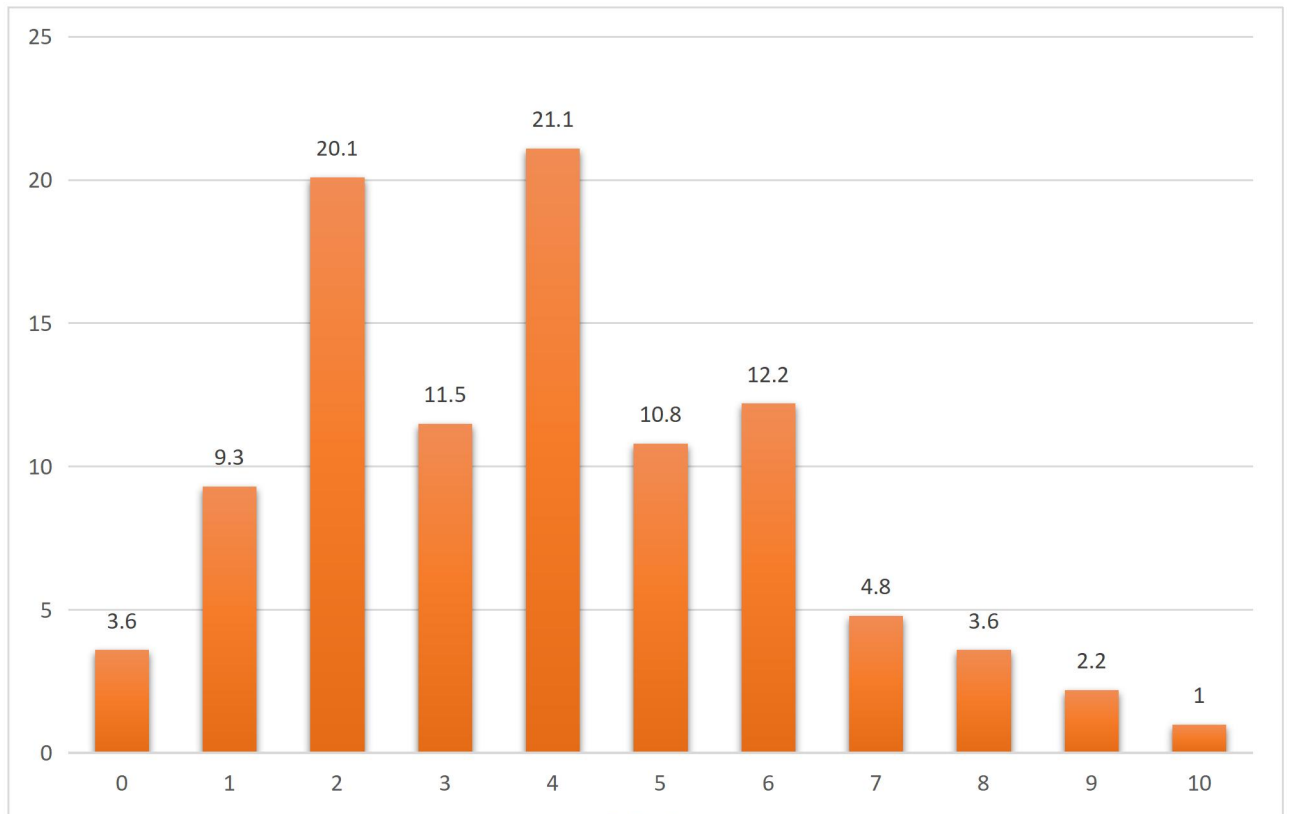


Figure 4.4 10-Word Delay Recall Test (10-WDRT)

Source: Research, 2025

4.4.1 10-Word Delay Recall Test (10-WDRT) and Associated Factors

The mean word recall of younger respondents (4.36 ± 2.23) was significantly higher than older respondents' (3.77 ± 2.06 and 2.73 ± 2.16 , respectively) ($p=0.000$). The mean word recall of respondents with higher level of education (4.43 ± 2.05) was significantly higher than the rest of the group (3.63 ± 1.92 and 2.29 ± 1.52 , respectively) ($p=0.000$). Additionally, respondents with a family history of dementia had a significantly lower word recall rate (3.68 ± 2.06) than those without a family history (5.37 ± 2.64) ($p=0.000$).

Table 4.5 10-Word Delay Recall Test (10-WDRT) and Associated Factors

Variable	Delayed Word-recall	p-value
	Score	
	Mean±S.D	
Age		0.000*
40 – 49 years	4.36±2.23	
50 – 59 years	3.77±2.06	
> 60 years	2.73±2.16	
Level of Education		0.000*
No formal	3.49±2.41	
Primary	3.63±1.92	
Secondary	2.29±1.52	
Tertiary	4.43±2.05	
Family History of Dementia		0.000*
Yes	3.68±2.06	
No	5.37±2.64	

* Significant at the p<0.05 level

4.5 Discussion of Findings

Understanding the role of diet in cognitive health is becoming increasingly critical, especially in rapidly urbanizing African settings where lifestyle patterns are shifting dramatically. Urbanization brings with it a double burden of malnutrition, the coexistence of undernutrition and emerging dietary excesses from processed, calorie-dense foods. These changes significantly influence brain health. Among middle-aged men, nutritional behavior intersects with socioeconomic pressures and limited awareness of cognitive health, forming a concerning pattern of premature cognitive decline.

This study examined the dietary patterns and cognitive abilities of men aged 44 to 60 years, identifying the associated risk factors that may contribute to cognitive impairment. The findings offer valuable insight into the unique health challenges facing urban Nigerian men in midlife, where dietary inadequacies, mental inactivity, and psychosocial stress may converge to create a hidden epidemic of cognitive vulnerability. These observations emphasize the urgent need for preventive interventions that begin much earlier than late adulthood, when dementia symptoms are more pronounced and harder to reverse.

The dietary profile of participants revealed a heavy reliance on carbohydrate-rich staples, with grains and tubers forming the core of most daily meals. While these foods provide necessary calories, they are limited in essential micronutrients. Only a small proportion of participants consumed vegetables, fruits, dairy products, or fish with any regularity. Such nutrient-dense foods are rich in omega-3 fatty acids, vitamin B12, antioxidants, flavonoids, and polyphenols, which are known to protect neuronal membranes, reduce oxidative stress, and support synaptic plasticity. Studies have shown that nutrients such as

polyphenols, flavonoids, and vitamins have the potential to be therapeutically beneficial in maintaining neuronal function by targeting oxidative stress and inflammatory pathways¹⁻². The limited intake of such nutrient-dense food groups in this study suggests a nutritional pattern that may predispose participants to cognitive vulnerability over time. In particular, the low intake of vitamin-A-rich vegetables, nuts, and dark leafy greens indicates potential deficiencies in neuroprotective nutrients that are vital in preventing age-related neurodegeneration². Vitamin A, C, E, and folate act as powerful antioxidants that neutralize reactive oxygen species linked to neuronal damage. Longitudinal studies have shown that populations with higher fruit and vegetable intake demonstrate better memory retention and slower cognitive decline compared to those with monotonous starch-heavy diets. This nutritional gap, therefore, could be a modifiable risk factor for dementia among urban African men².

Meal-skipping was another concerning trend. A majority of the participants reported skipping meals regularly, especially breakfast and lunch. The reasons cited included habitual tendencies, lack of time, and financial constraints. This pattern has been similarly observed in urban African populations, where work demands and commuting times interfere with regular meal times³. The cognitive implications of habitual meal-skipping are significant. Breakfast, often called the “most important meal of the day,” provides stable glucose supply to the brain. Skipping breakfast results in energy fluctuations that impair concentration, memory, and problem-solving abilities⁴. Over time, chronic meal-skipping can contribute to subtle but cumulative nutrient deficiencies, reducing the brain’s ability to maintain neurotransmitter balance and repair damaged neurons. In this study, many participants reported fatigue, forgetfulness, and difficulty

maintaining focus, which aligns with research linking irregular eating patterns to reduced cognitive performance in midlife.

Self-reported cognitive symptoms were alarmingly widespread, even within this relatively young cohort. Participants described forgetfulness, difficulty recognizing familiar individuals, disorientation, and challenges in completing daily tasks. These symptoms are consistent with mild cognitive impairment (MCI), a recognized transitional state between normal aging and dementia⁵. The 10-Word Delayed Recall Test (10-WDRT) further validated these complaints. Only a few participants recalled seven or more words, while a substantial subset recalled fewer than three, indicating significant early memory deficits. Globally, MCI prevalence increases sharply after age 60, but this study highlights that cognitive decline may be emerging a decade earlier in this urban population. This raises important public health concerns about the earlier onset of dementia in high-stress, low-nutrient environments such as Lagos.

Age and education were strongly associated with performance on the memory test. Men in their 40s generally performed better than those nearing 60, which aligns with global evidence showing increased risk of cognitive impairment with advancing age⁶. However, even within the younger subgroup, there were signs of memory deficits. Educational level showed a significant protective effect. Participants with tertiary education demonstrated higher recall abilities compared to those with no formal or limited education. This supports the cognitive reserve theory, which proposes that individuals with more educational attainment develop stronger neural connections that help delay the clinical expression of dementia symptoms⁷. Indeed, studies show that individuals with 12 or more years of education have up to a 40% lower dementia risk compared to those with minimal

education. This suggests that education policy can indirectly function as a public health intervention by building lifelong cognitive resilience.

Lifestyle-related risk factors also played a significant role. Although a majority of participants reported engaging in physical activity, only a few did so consistently, highlighting a disconnect between awareness and practice. Physical activity has been shown to reduce the risk of dementia by improving cerebral circulation, reducing inflammation, and stimulating neurogenesis⁹. A study confirmed that even moderate physical activity was associated with increased hippocampal volume and better memory performance in older adults¹⁰. The inconsistency in physical exercise among participants could therefore undermine cognitive health and contribute to the observed memory problems.

Mental inactivity and social isolation were also common among participants. Many reported that they did not regularly engage in mentally stimulating activities such as reading, puzzles, or skill learning. Others stated they had limited social interaction or felt isolated. Both factors are known to contribute to cognitive decline. The brain, like any organ, benefits from stimulation, and reduced cognitive engagement has been shown to accelerate functional deterioration¹¹. Similarly, limited social interaction has been linked to emotional distress, depression, and reduced neural plasticity, all of which heighten dementia risk¹².

The presence of stress and emotional strain also emerged as a potential risk factor. Several participants reported experiencing high levels of work-related or personal stress. Chronic stress is known to increase cortisol production, which can negatively affect brain structures such as the hippocampus and prefrontal cortex, both of which are critical for

memory and decision-making¹³. The physiological effects of prolonged stress have been observed to impair synaptic plasticity and contribute to mild cognitive impairment. Moreover, stress often co-occurs with poor sleep, unhealthy eating, and reduced physical activity, further compounding cognitive risk.

Compounding this, stress often coexists with poor sleep, unhealthy eating, and reduced physical activity, creating a vicious cycle that accelerates cognitive decline. While few participants reported a history of depression or anxiety, these conditions are likely underreported due to stigma. Midlife depression is a known predictor of cognitive decline, with evidence showing it can double Alzheimer's risk in longitudinal studies¹⁴. Depression can impair motivation, reduce neurogenesis, and affect neurotransmitter systems that are crucial for memory and learning. A study demonstrated that midlife depression was an independent predictor of Alzheimer's disease in a ten-year longitudinal follow-up¹⁴.

Family history was another area explored. While most participants denied any known family history of cognitive decline, among those who did report such a history, memory test performance was notably lower. This is consistent with previous findings that suggest a familial or genetic predisposition to Alzheimer's disease and other forms of cognitive disorder. A review highlighted that individuals with a first-degree relative affected by dementia have a two- to three-fold higher risk of developing the condition themselves⁵. However, genetic predisposition interacts with lifestyle factors, and many genetically at-risk individuals may never develop symptoms if protective behaviors are adopted early.

The cumulative evidence from this study indicates that the cognitive abilities of middle-aged men in Eti-Osa may be shaped by multiple, interacting factors. These include poor

dietary practices, irregular meal patterns, insufficient consumption of protective nutrients, low levels of physical and mental activity, psychosocial stress, and limited health literacy. All of these are modifiable factors, and their prevalence suggests that prevention or efforts to reduce the risk of cognitive decline should begin in midlife, rather than waiting until old age.

Compared to studies in other sub-Saharan African countries, the findings in this study point to a higher prevalence of Mild Cognitive Impairment symptoms. For example, a study reported that 8.3% of older adults showed signs of mild cognitive impairment, while this study identified a broader spectrum of memory complaints and functional difficulties even among younger individuals¹⁵. The unique pressures of urban living, fast-paced routines, economic uncertainty, environmental pollution, and reduced communal support, may amplify cognitive risk. Indeed, urbanization has been linked to higher dementia rates globally, as it fosters sedentary lifestyles, dietary transitions toward processed foods, and social fragmentation¹⁶.

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Chapter Five

Conclusion

5.1 Summary of Findings

The study on dietary habits and patterns on the cognitive abilities of middle-aged men in Eti – Osa Local Government Area of Lagos State which broadly aimed to examine the dietary patterns on the cognitive abilities of middle-aged men in Eti – Osa Local Government Area of Lagos State and seeks specifically to; assess the dietary patterns of middle-aged men; analyze the cognitive functions in this population; examine the factors that influence the dietary habits and patterns, examine the relationship between dietary habits, patterns and cognitive functions of middle-aged men in Eti-Osa Local Government of Lagos State.

The study adopted survey design which enable the researcher to give detail description on assessment of dietary patterns and cognitive abilities of middle-aged middle in Eti-Osa LGA, Lagos State, Nigeria. This design does not call for manipulation of variables but, it allows an in-depth study of the subject matter, and it is suitable to described attitudes, views or opinions and behavior pattern of the people. The study population encompassed only middle aged men (44-60) in Eti-Osa Local Government Area of Lagos State and include aged men of diverse socio-economic backgrounds and family structures.

A multistage sampling technique was employed to select respondents for the study in Eti-Osa Local Government Area of Lagos State. The multistage sampling approach ensures a systematic and representative selection process, covering different demographic groups within the middle aged population which include stratified sampling technique to ensure a fair representation of middle aged men across selected areas in Eti-Ose LGA, Lagos State. The Areas within the Local Government Area were categorized based on specific

strata such as public and private areas to capture diverse socio-economic backgrounds. This stratification helped in selecting participants from different areas to ensure a balanced study population. Simple Random Sampling which is stage two, was used to select from public and private areas within the study area.

The sample size was determined using Cochran (1953) formula and was put at 424 respondents. The data collected were processed and each questionnaire was checked for completeness and accuracy. The quantitative data was analyzed using Statistical Package for Social Science. And the result was expressed using descriptive statistics (means and percentage).

The dietary assessment revealed that most middle-aged men consumed starchy foods like grains and tubers daily, while the intake of nutrient-rich food groups such as vegetables, fruits, legumes, nuts, and dairy products was comparatively low. A significant number of participants engaged in irregular eating patterns, with many habitually skipping meals, particularly breakfast and lunch. The main reasons cited for meal skipping included time constraints, habitual tendencies, and financial limitations. Only a minority reported regular consumption of dark-green leafy vegetables, fish, and vitamin-A-rich produce, indicating a potential deficiency in neuroprotective nutrients essential for cognitive health.

Self-reported data showed widespread symptoms consistent with early-stage cognitive decline. A large number of participants admitted to frequent forgetfulness, repeated questioning, difficulty recognizing familiar individuals, and increased reliance on others for reminders. The 10-Word Delayed Recall Test confirmed cognitive challenges, with most respondents recalling fewer than five of the ten words. Only a few demonstrated high recall ability, and some were unable to recall any. These findings suggest a

considerable prevalence of probable dementia or mild cognitive impairment within the study population.

Several modifiable risk factors were identified. These included poor dietary quality, frequent meal skipping, low educational attainment, irregular physical activity, limited social and cognitive engagement, and high levels of self-reported stress. A small number of participants reported a family history of dementia, which was associated with lower recall scores. Depression, although underreported, and social isolation also emerged as potential contributors to cognitive decline. The combination of these behavioural, psychosocial, and lifestyle factors underscores the multifactorial nature of dementia risk in this demographic.

An inverse relationship was observed between healthy dietary practices and cognitive performance. Participants who consumed vegetables, legumes, dairy products, and fish more frequently generally demonstrated better recall scores. Conversely, those who consumed fewer protective nutrients and engaged in unhealthy eating behaviors, such as meal skipping and high intake of processed or fried foods, showed more symptoms of memory impairment. This pattern supports existing literature linking inadequate nutrition to cognitive decline and highlights the potential of diet-based interventions for dementia prevention.

5.2 Conclusion

This study has provided critical insights into the intersection of dietary behavior and cognitive health among middle-aged men in Eti-Osa Local Government Area, Lagos State. The findings reveal a concerning pattern of inadequate dietary practices, characterized by high reliance on starchy staples, infrequent consumption of nutrient-

dense foods, and widespread meal skipping. These dietary behaviors, compounded by socio-economic pressures and urban lifestyle constraints, appear to contribute significantly to the observed cognitive challenges in this population.

A notable prevalence of probable dementia symptoms was identified, even within a relatively young cohort. Many participants demonstrated clear signs of memory loss, disorientation, and impaired daily functioning, supported by poor performance on the 10-Word Delayed Recall Test. These cognitive deficits were not uniformly distributed but were significantly associated with modifiable risk factors such as low educational attainment, inconsistent physical activity, social isolation, and high stress levels. Although genetic predisposition was present in a minority, the findings emphasize the dominant role of lifestyle and environmental influences in shaping cognitive outcomes.

A relationship was observed between healthier dietary patterns and better cognitive performance. Participants with more frequent intake of vegetables, legumes, dairy products, and fish exhibited higher recall abilities, whereas those with poor dietary diversity and unhealthy eating habits reported more symptoms of cognitive decline. These associations highlight the potential of diet as a modifiable determinant in the prevention or delay of cognitive decline.

5.3 Recommendations

Based on the findings of this study, the following recommendations are proposed:

- i. It is recommended that targeted nutrition education programs be implemented at the community level to raise awareness on the importance of consuming neuroprotective nutrients.

- ii. To address the financial and accessibility constraints associated with healthy food consumption, local government authorities should collaborate with community-based organizations, agricultural stakeholders, and market associations to make nutrient-dense foods more affordable and readily available.
- iii. Given the substantial prevalence of early cognitive decline identified in this study, routine cognitive assessment and dementia screening should be integrated into existing primary healthcare services.
- iv. Cognitive and social engagement activities, such as adult literacy programs, reading clubs, cultural events, and skill-building workshops, should be encouraged to reduce social isolation and enhance mental stimulation. Mental health support services should also be made available to address stress, depression, and loneliness, which were identified as contributory factors to cognitive decline in the study population.

5.4 Contribution to Knowledge

This study makes significant contributions to the existing body of knowledge on the relationship between dietary patterns and cognitive health among middle-aged men in sub-Saharan Africa, particularly in an urban Nigerian context. While most existing literature on dementia has focused on elderly populations in high-income countries, this research provides context-specific evidence from a middle-aged demographic in Lagos State, thereby filling a critical gap in understanding the early manifestations of cognitive decline within a socio-culturally diverse African setting.

The study highlights the nutritional and lifestyle determinants of cognitive decline, demonstrating a clear association between inadequate dietary quality, characterized by

low intake of neuroprotective nutrients such as vegetables, legumes, dairy products, and fish, and an increased prevalence of early cognitive impairment. It provides empirical evidence that habitual meal skipping, irregular eating patterns, and reliance on starchy staple foods may accelerate memory decline even before old age, underscoring the importance of early dietary interventions for dementia prevention. This insight expands current knowledge on modifiable risk factors, emphasizing that preventive strategies must begin in midlife rather than waiting until later stages of aging.

Furthermore, the research illuminates the multifactorial nature of dementia risk, identifying the interplay between poor nutrition, low educational attainment, irregular physical activity, psychosocial stress, and social isolation as key contributors to cognitive decline in the study population. By incorporating socio-economic stratification within the sampling design, the study enriches the discourse on how structural inequalities and financial constraints shape health behaviors and, consequently, cognitive outcomes.

5.5 Suggested Area for Further Study

This study suggested further research in the following areas:

- i. Longitudinal studies are needed to establish causal relationships between dietary habits and dementia risk over time. While this study identified strong associations between poor dietary quality, irregular meal patterns, and early cognitive impairment, its cross-sectional design limits the ability to infer causality. Prospective cohort studies tracking middle-aged individuals over several years would help clarify how dietary changes influence the progression from mild cognitive impairment to dementia.

- ii. Future research should explore the biochemical and nutritional biomarkers associated with cognitive decline in this population. Laboratory-based assessments of micronutrient deficiencies, oxidative stress markers, and neuroinflammatory indicators could complement self-reported dietary data and provide more objective evidence linking nutritional inadequacies to cognitive outcomes.
- iii. There is a need to investigate the synergistic effects of lifestyle factors, such as physical activity, sleep quality, mental health status, and social engagement, in relation to diet and dementia risk. Since this study identified psychosocial stress, low cognitive stimulation, and social isolation as contributory factors, an integrated approach examining how these variables interact may yield more holistic preventive strategies.

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Appendix 1

Questionnaire

Influence of Dietary Habits and Pattern on Cognitive Abilities Of Middle Aged Men (44-60) In Eti-Osa L.G.A, Lagos State, Nigeria.

Letter of Introduction

Dear Respondents,

I am a final year student at the Department of Human Nutrition and Dietetics, Lead City University. I am currently conducting research on the above title. This research is for academic purposes only and all information collected shall be treated with utmost confidentiality. Participating in this study does not pose any risk to you in any identifiable way. You are free to ask any question relating to this study and I implore you to give honest responses as much as possible.

Thank you for your cooperation.

Do you agree to participate: Yes [] NO [].

Respondent Signature: _____

Questionnaire Number: _____

**INFLUENCE OF DIETARY HABITS AND PATTERN ON COGNITIVE
ABILITIES OF MIDDLE AGED MEN (44-60) IN ETI-OSA L.G.A, LAGOS
STATE, NIGERIA.**

Please kindly follow the instruction carefully and tick [√] where appropriate.

SECTION A: Demographic Information

1. Age (years) _____
2. Education: Primary [] Secondary [] Tertiary [] No Formal Education []
3. Occupation: Government Worker [] Self Employed [] Private Worker []
Unemployed [] Farming/Hunting/Fishing [] Others (specify) _____
4. Marital status? Single [] Married [] Divorced [] Widowed [] Separated []
5. Religion: Christianity [] Islam [] Traditional []
6. Ethnicity: Yoruba [] Hausa [] Igbo [] Others (specify) _____
7. Household size _____
8. Residence: Rural [] Peri-urban [] Urban []
9. Average monthly income (₦) _____

SECTION B: Dietary Habits and Patterns:

- i) No of meals per day: One [] Two [] Three [] More than three []
- ii) Do you skip meals? Yes [] No []
If yes, how often? Everyday [] Weekdays only [] Weekends only [] Rarely []
- iii) Frequently skipped meals: Breakfast [] Lunch [] Dinner []

- iv) Reason for skipping meals: Financial constraints [] Time constraints []
 Usual habit [] Loss of appetite [] Others (specify) _____

(How often do you eat the following in a typical week?)

S/N	Food Group	Daily	4- 6x/week	2- 3x/week	Rarely	Never
1.	Grains e.g rice, corn, bread, noodles, spaghetti					
2	Roots and Tubers e.g cassava, sweet potatoes, yam					
3	Pulses e.g beans, peas, green pea, kidney beans,					
4	Nut and Seeds e.g groundnut, cashew nut					
5	Milk and Dairy Products e.g milk, cheese, yoghurt					
6	Processed Meat e.g sausage, canned meat					
7	Organic Meat e.g liver, kidney, gizzard, pancreas, blood					

8	Poultry Meat e.g Chicken, turkey					
9	Fish and Sea Foods e.g shellfish, crayfish, catfish, salmon					
10	Dark Green Leafy Vegetables e.g pumpkin leaves (Ugwu), bitter leaf, okra, spinach					
11	Other Vitamin A rich vegetables e.g carrots, red pepper					
12	Other Vitamin A rich fruits e.g ripe mangoes, ripe paw-paw, sweet melon					
13	Other Vegetables e.g cucumber, eggplant, mushroom, tomato, garden eggs, cabbage					
14	Sugary Foods e.g chocolates, honey, candies, cakes, biscuits, ice-cream					
15	Beverages e.g carbonated drinks, sweetened fruit juices					

16	Fried Foods e.g crisps and chips potatoes, plantain					
----	--	--	--	--	--	--

SECTION C: 10 Word-List Memory Test

Instructions: “Now, I want to see how well you can learn a list of words. The words are printed in this notebook. As I call the words, repeat it out loud and try to remember it. After you have read all the words, I will ask you to repeat as many words from the list as you can. Any questions?”

Here are the words - Butter, Arm, Letter, Queen, Grass, Engine, Ocean, Shelter, Tree, and Nail.

Write out the words remembered after a 5-minute delay _____

Have you or a close family member been diagnosed with dementia by a healthcare professional? Yes [☐] No [☐]

Section D: Relationship Between Dietary Habits and Cognitive Function

S/No	ITEMS	Strongly Agree	Agree	Disagree	Strongly Disagree
1	I consume alcohol more than three times a week				
2	I smoke cigarettes or use tobacco products regularly				

3	I engage in regular physical exercise (e.g., walking, jogging, gym workouts) at least three times a week				
4	I normally feel isolated or lonely				
5	I have been diagnosed with depression or anxiety before				
6	I have a family history of dementia or Alzheimer's disease				
7	I frequently experience high levels of stress or work-related pressure				
8	I participate in mentally stimulating activities (e.g., reading, puzzles, learning new skills)?				
9	I don't normally engage in social activities or interact with friends and family regularly				

Anthropometric Measurement

Height (m): _____

Weight (kg)

Appendix 2
Pictures Taken during Field Work











Biodata

Personal Data

Name: Ekaette IDE
Matric No: LCU/PG/005753
Address: Gbagada Lagos
Phone no: 07035491897
Mail: ekaetteide@yahoo.com
Date of Birth: 16th Mar 1986
Marital status: Single

Core Area of Expertise: Clinical Nutrition

Education

2023/2024 – Till Date MSc Human Nutrition and Dietetics, Lead City University, Ibadan
2005- 2011 BSc Food Science and Technology University of Uyo, Uyo
1997 – 2002 West Africa Senior Secondary Examination Holy Child Girls Secondary School, Ikot Ekpene

Professional Experience

2022 – Till Date SYNLAB Nigeria Ltd

- Develop strategic sales plan to execute national sales objectives by driving the end-to-end sales process with the team within the assigned region.
- Conduct in-depth market research and competitor analysis to refine strategies and enhance SYNLAB's competitive positioning.
- Collaborate with healthcare professionals to drive brand awareness while building and maintaining professional network.
- Market Expansion: Identify and capitalize on growth opportunities by tailoring diagnostic solutions to meet diverse market demands and expanding SYNLAB's regional footprint.
- Team Development: Lead, mentor and inspire a high performing team sales team, fostering accountability, professional development and alignment with company goals.
- CSR Initiatives: Champion Corporate Social Responsibility including health programs and diagnostic outreach, to boost brand visibility and impact in underserved communities.
- Attend clinical meetings at tertiary hospitals with the respective clinic consultants

The University Compliance Certificate

This is to certify that this thesis, written by Ekaette IDE, Matric No LCU/PG/005753 in the Department of Human Nutrition and Dietetics, Faculty of Basic Medical & Medical Sciences, Lead City University, Ibadan, is in full compliance with the approved University format and style.

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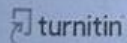
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