5-9-2024

Type 2 Diabetes as a Risk Factor for the Development of Cognitive Dysfunction in U.S. Adults

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This is to certify that the doctoral dissertation by

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Walden University
2024

Abstract

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by

Bobbi R. Johnson

BS, University of Florida, 2008

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health

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May 2024

Abstract

There is no consensus as to whether having type 2 diabetes increases an individual's risk for developing cognitive dysfunction. Though cognitive dysfunction is considered a risk factor for type 2 diabetes, it is also classified as a newly formed diabetes-related complication. The purpose of the study was to evaluate if self-reported healthcare – diagnosed diabetes has any association in the risk of the development of cognitive dysfunction in individuals living with type 2 diabetes. Self-determination theory served as the theoretical framework. The research questions concerned whether (a) an association exists between diabetes and cognitive dysfunction, (b) ethnicity plays a vital role in modifying the effect of the association between diabetes and cognitive dysfunction, and (c) gender modifies the effect of the association between diabetes and cognitive dysfunction. A quantitative, cross-sectional research design was applied with a secondary data set from the data collected in National Health and Nutrition Examination Survey (NHANES). The results of logistic regression analysis indicated an association between diabetes and cognitive dysfunction, OR = 1.76, CI [1.32, 2.33], $p < .05$. Other findings indicated the existence of an association with certain gender and ethnic groups. Recommendations for future research include evaluating the impact, if any, that race has on the association between hemoglobin A1c levels and cognitive dysfunction. The study may promote positive social change by increasing health care professionals' awareness of the association between diabetes and cognitive dysfunction. By imparting this information to patients, health care professionals may be able to motivate patients to maintain better control of their diabetes.

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Dedication

This study is dedicated to my paternal grandparents, Mr. G.W. Johnson and Mrs. Ruth Johnson. I would like to also dedicate this study to my maternal grandparents, Mr. John Patterson and Mrs. Georgia Patterson.

Acknowledgments

First, I would like to thank my Lord and Savior, Jesus Christ. Second, I would like to thank my committee members, Dr. Srikanta Banerjee and Dr. Thomas O'Grady. Dr. Banerjee, thank you so much for guiding me throughout this process. You have been an exceptional chairperson. I thank you for your patience and also for your guidance throughout this journey. I have learned so much from you, and I thank you for your support. Dr. O'Grady, I would like to thank you for your guidance and positive feedback.

I want to give a tremendous amount of honor and thanks to my parents, Mr. Bobby Johnson and Mrs. Mariea Johnson. You have been riding this wave with me for a long time, and I am forever grateful for your encouraging words and insurmountable support. Thank you for your unconditional love. Last, I would like to thank my immediate and extended family members and friends for all of their support and love. I love you all.

Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction to the Study

According to the World Health Organization, diabetes affected more than 400,000,000 people worldwide (Ortiz et al., 2022; Wang et al., 2022). Individuals living with type 2 diabetes mellitus (T2DM) have an increased risk of developing cognitive dysfunction (Rizzo et al., 2022). Cognitive dysfunction, or cognitive declination, has been identified as a new form of diabetes-related complication (Umegaki, 2018). A combination of vascular and neurodegenerative damaging is perceived as the cause of cognitive dysfunction in patients living with T2DM (Rizzo et al., 2022). To further explicate, deficiencies with insulin receptor sensitivity, oxidative stress, intracellular signaling, neuroinflammation state, and mitochondrial metabolism play a pivotal role in cognitive dysfunction (Rizzo et al., 2022). The importance of maintaining good glycemic control could have an impact or decrease the likelihood of dementia in individuals living with T2DM (Rizzo et al., 2022). However, the results remain inconclusive regarding whether hemoglobin A1c levels have any association with the development of dementia.

In this study, I explored T2DM and its relationship to cognitive dysfunction. The study may promote positive social change by informing health care professionals about the potential relationship between diabetes and cognitive dysfunction; these providers could, in turn, increase patients' awareness of potential risk factors. Patients may be able to make the necessary lifestyle changes to better manage their own diabetes. In this chapter, I will focus on the background of the study. I will also provide an overview of the study, which includes the problem statement, purpose of study, research questions (RQs) and hypotheses, theoretical foundation, nature of study, assumptions, definitions,

scope and delimitations, limitations, and significance of the study. The self-determination theory underpinned the study and the development of the RQs. I used a quantitative cross-sectional research design featuring a secondary data analysis. I assessed whether self-reported diabetes has an impact on the presence of cognitive dysfunction.

Background

An association exists between T2DM and the increased risk of developing cognitive dysfunction, research shows (Rizzo et al., 2022). Diabetes mellitus has been recognized as one of the causes and amendable risk factor for the development of dementia (Sebastian et al., 2023). In comparison to individuals living without diabetes, patients diagnosed with the chronic illness are 1.5 times more than likely to experience cognitive dysfunction and the beginning stages of dementia (Lin et al., 2022). Cognitive dysfunction has been receiving more attention due to diabetes being identified as an independent risk factor for it (Barloese et al., 2022). The magnitude of cognitive dysfunction varies from subtle decrements to major neurocognitive disorders (Fang et al., 2022). The subtle decrement includes episodic memory, attention, and executive functioning, which are associated with the neurocognitive development of dementia, or Alzheimer's disease, in particular (Fang et al., 2022).

Glycemic control plays a fundamental role with cognitive dysfunction. Cognitive dysfunction is stimulated due to the presence of low-grade inflammation, weakening of insulin signaling, and pathways directly associated to chronic hyperglycemia (Biessels & Despa, 2018). Abnormal glucose levels, including fasting and postprandial hyperglycemia, not only result in neural dysfunction but also nervous system disorders,

such as cognitive decline or dysfunction (Sebastian et al., 2023). Although elevated hemoglobin A1c levels can have an impact on cognitive dysfunction, there is a lack of consensus in the literature in regard to this relationship. There is evidence that supports the elevation of hemoglobin A1c and cognitive dysfunction. However, there is also literature that does not support this logic (Umegaki, 2018).

Problem Statement

With regard to type 2 diabetes, the correlation between elevated hemoglobin A1c levels, or glucose control, and cognitive dysfunction remains inconclusive. Previous researchers have reported on the connection between insulin irregularity in individuals living with type 2 diabetes and cognitive dysfunction (Bendlin, 2022; Tumminia et al., 2018). It has been recognized that where there is consistency in elevated hemoglobin A1c levels, poor glucose control can induce the presence of Alzheimer's disease or dementia (Callisaya et al., 2018; Tumminia et al., 2018). Amyloid beta, identified as Abeta, has been identified as the defining factor in the development of Alzheimer's disease, according to Agrawal and Agrawal (2022). They noted that Abeta is destroyed by the advanced glycation end products and an enzyme, referred to as insulin-degrading enzyme, that vies with insulin. As a result, insulin initiates the secretion of Abeta and, furthermore, prompts brain inflammation. Agrawal and Agrawal also noted that hyperglycemia is responsible for the changes in synapse plasticity and also incites cognitive dysfunction. Insulin resistance has been correlated to dysexecutive syndrome (impairment of executive functions and linkage to frontal lobe damage), frontal lobe

impairment, and hyperinsulinemia, which greatly increases the risk for Alzheimer's disease (Agrawal & Agrawal, 2022).

However, the literature also indicated that there was no association between elevated hemoglobin A1c levels, or hyperglycemia, and cognitive dysfunction (Umegaki, 2018). Though researchers have found that hyperglycemia and hypoglycemia levels are associated with the risk of developing cognitive dysfunction, such as dementia, they have identified the connection as ambiguous (Biessels et al., 2018). The inconclusive nature of this relationship indicates a gap in the literature.

Purpose of the Study

The purpose of the study was to evaluate whether self-reported health care diagnosed diabetes had any association with the risk of developing cognitive decline, or dementia in individuals living with type 2 diabetes. In this study, I applied a quantitative, cross-sectional research design featuring a secondary data analysis. The intent of this study was to explore whether there is an association between diabetes and cognitive dysfunction. The independent variable was diabetes. The dependent variable was cognitive dysfunction. The covariates consisted of the following: diabetes, gender, age, ethnicity, and poverty–income ratio.

Research Questions and Hypotheses

RQ1: Is there an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity?

$H_0$1: There is no association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity.

H_a1: There is an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity.

RQ2: Does race modify the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio?

$H_0 2$: Race does not modify the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio.

$H_a 2$: Race does modify the effect of the association between cognitive diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio.

RQ3. Does sex modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio?

$H_0 3$: Sex does not modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio.

$H_a 3$: Sex does modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio.

Theoretical Framework

The theory most suitable for the study was self-determination theory. In 1985, psychologists Edward Deci and Richard Ryan (2008) developed self-determination theory. Self-determination theory has been widely used in fields, such as sports, education, and health care (Ryan et al., 2008). Self-determination is defined as the natural propensity an individual develops while modifying and sustaining health-related behavior over time (Ryan et al., 2008). Self-determination theory is focused on human motivation (Gagne et al., 2022). According to this theory, individuals are motivated in three ways:

intrinsically, extrinsically, and through the use of amotivation (Ryan & Deci, 2000). I used self-determination theory in developing the RQs, which concerned the possible association between diabetes (diagnosed using self-reported data) and cognitive dysfunction. The theory, RQs, and hypotheses will be further discussed in Chapters 2 and 3.

Nature of the Study

I used a quantitative, cross-sectional research design featuring a secondary data analysis. The data was National Health and Nutrition Examination Survey (NHANES) data from the years of 2014 (for the cognitive dysfunction responses) and the 2017-March 2020 pre-COVID-19 pandemic. The cross-sectional research design was used to establish whether there is an association between self-reported diabetes and cognitive dysfunction for individuals living with type 2 diabetes. Quantitative researchers describe events by collecting numerical data, which can be further evaluated utilizing mathematically based methods (Stockemer, 2019). In essence, quantitative researchers can test a theory by analyzing relationships between the variables being studied (Stockemer, 2019).

This study consisted of key variables. The independent variable was diabetes (type 2 diabetes). The dependent variable was cognitive dysfunction. It was dependent upon the individual (in how one manages their own diabetes), if cognitive decrements, or cognitive dysfunction would be considered a topic of discussion. The study also included covariates. The covariates comprised of the following: diabetes (type 2), gender (male or

female), age (age of participants), ethnicity (racial identity), and cognitive dysfunction (i.e., the inability to process speed and memory loss).

Definitions

The following are key terms that are used in the study:

Amyloid beta oligomers: A cluster of robust neurotoxins that mediate inflammation. The neurotoxins are connected to insulin resistance in the central nervous system (Sousa et al., 2020). They are responsible for the impediment of creating new memories (Sousa et al., 2020).

Autonomous motivation: The ability to recognize the significance of, and to identify with, an activity's value while incorporating the values into practice (Deci et al., 2008).

Cognitive decrement: A slight change an individual can recognize within themselves regarding their ability to process speed, memory, and functioning (Biessels & Despa, 2018).

Controlled motivation: External motivation through reward or punishment (Deci et al., 2008).

Hemoglobin A1c: A blood test given to determine the average blood glucose levels over a 3-month period, the result of which is used to identify prediabetes, diagnose diabetes, and assess control (Fenelon et al., 2022).

Glycation end product: Inflammatory events triggering diabetes and its complications that are emitted due to a high-fat diet or hyperglycemia (Asadipooya & Uy, 2019).

Mild cognitive impairment (MCI): A temporary and conceivably amendable stage between what is perceived as normal cognitive progression (or aging) and dementia (Li et al., 2019).

Assumptions

There is general consensus in the previous literature that elevated hemoglobin A1c levels contribute to cognitive dysfunction. Some researchers have explored what takes place in the human body when an individual living with type 2 diabetes encounters insulin resistance for a long period of time or experiences hyperglycemia on a consistent basis (Sousa et al., 2020; Tumminia et al., 2018). Insulin has been shown to play a significant role in facilitating learning and memory (Tumminia et al., 2018). However, when insulin resistance has remained constant—a situation that can lead to hyperglycemia—and then induced to elevated hemoglobin A1c levels, the development of brain disorders has been the predicted outcome (Tumminia et al., 2018). Yet, there is literature that does not support this conclusion. The assumption of ambiguity undergirded this investigation about this particular topic.

Scope and Delimitations

Cognitive dysfunction is recognized as a risk factor for type 2 diabetes. However, there is a key element that is also identified as a contributor to the development of cognitive dysfunction in individuals living with type 2 diabetes. There is conflict regarding whether elevated hemoglobin A1c levels, or a consistency with hyperglycemia, plays a significant role in cognitive dysfunction. Some of the literature supports this conclusion, whereas other literature negates this idea (Agrawal & Agrawal, 2022;

Tumminia et al., 2018; Umegaki, 2018). I focused on obtaining more understanding of what effect, if any, diabetes has on the development of cognitive dysfunction. I drew from the self-determination theory. A conceptual framework that could have been applied is the transactional model of stress and coping. This conceptual framework has been employed to assess the process of coping with stressful events (Shavaki et al., 2020). For example, some literature supports that the elevation of hemoglobin A1c levels contributes to the development of cognitive dysfunction. Stress-induced hyperglycemia occurs when blood glucose levels are significantly increased under stress (Zhang et al., 2023). Stress can lead to hyperglycemia, and the prolonged hyperglycemia glucose levels can lead to elevated hemoglobin A1c levels, which according to research, can contribute to cognitive dysfunction. If the cause of stress can be identified, individuals living with type 2 diabetes may be better able to manage their glucose levels under stressful events, which might minimize of the development of cognitive dysfunction in the future.

In regard to the inclusion criteria, the age of the participants were at least 60 years of age or older and in non-institutionalized populations. The participants comprised of non-Hispanic Whites, non-Hispanic Blacks, Hispanics, and individuals who reported their ethnicity as Other. The exclusion criteria consisted of individuals who had not received a diabetes prognosis and living in an institutionalized population. Regarding the generalizability of the sample population, NHANES researchers applied a multistage probability sampling design. This type of sampling involves a series of steps before collecting data from the actual participants. The sampling design is valid, reliable, and

can be applied to represent the general population for those living with type 2 diabetes (Akinbami et al., 2022).

Limitations

The use of a multistage probability sampling design limited my ability to include all of the survey participants. To further expound, the study outcomes are not always exact. The goal of multistage probability sampling is to minimize variance across the groups, yet it is challenging to validate whether the demographic groups eliminated from the study is effective.

In regard to bias, attrition bias was a potential challenge during the sampling process. In order to become an active participant, there were various stages that each individual must have gone through. The use of a multistage probability sampling method may mean a loss of participants from one stage to the next. To curtail this problem, I used sampling weights. This will be discussed in detail in Chapter 3 of the study.

Significance

There is a need to better understand cognitive dysfunction as a risk factor for type 2 diabetes. However, the association of diabetes with cognitive dysfunction is not widely accepted. Previous researchers have provided evidence of the correlation; nonetheless, there is literature that does not support this conclusion (Umegaki, 2018). There is literature discussing what takes place during insulin resistance and prolonged hyperglycemia, but there is insufficient evidence supporting the elevation of the hemoglobin A1c levels and cognitive dysfunction (Biessels et al., 2018). By conducting this study, I sought to clarify whether the two variables are connected. In addition to

adding to the literature, the results from this study may motivate health care professionals to recognize the connection, if any, and educate patients on risk factors for cognitive dysfunction. These potential outcomes may promote positive social change. The more research that is conducted on this topic, the more inclined health care professionals may be in discussing the significance during conference meetings, social gatherings, and to their patients. In return, when patients are cognizant of the connection between diabetes and cognitive dysfunction, they may be more likely to maintain better control of their diabetes.

Summary

In Chapter 1, I discussed how individuals are affected by type 2 diabetes and may have an increased risk for the development of cognitive dysfunction. This chapter also included the background and problem statement of the study, which focused on the correlation between elevated hemoglobin A1c levels and glucose control. In addition, I identified the gap in the literature. The purpose of the study was briefly discussed, followed by the theoretical framework, which underpinned the RQs and hypotheses for the study, which were also presented in the chapter. The research design was acknowledged, as well as the reasoning for the design choice. In Chapter 2, I will synthesize the literature on which this research study was based. The chapter includes the literature search strategy and further discussion of the study's theoretical framework. The literature review includes key findings regarding type 2 diabetes and cognitive dysfunction.

Chapter 2: Literature Review

Introduction

Diabetes is a chronic condition, if not managed properly, has a negative impact on the human body. Cognitive dysfunction has played a significant role with diabetes (Zhu, 2020). Type 2 diabetes greatly increases cognitive dysfunction, which is divided into 3 stages. The stages are as follows: cognitive decrements, MCI, and dementia. In regard to cognitive decrement, this has been recognized as the slight change an individual can recognize within themselves on the ability to process speed, memory, and functioning (Biessels & Despa, 2018). These decrements have been most likely to surface during the pre-diabetes stage, and within time (years), exacerbated at a rate of 50% more in comparison to those who go through normal cognitive ageing (Biessels & Despa). The connection between diabetes and cognitive dysfunction, or the manifestation thereof, has been recognized in the diminishment of learning and memory, executive ability, attention, and emotions (Cui et al., 2021). Cognitive impairment, or cognitive dysfunction has been a common, yet overlooked complication of diabetes (Cui et al., 2021). A consequence of living with diabetes for an extended period of time has been the possible development of mild cognitive decrement, or decline (Dyber et al., 2018). Dementia has been the final stages of cognitive dysfunction for individuals living with type 2 diabetes (Zhu, 2020; Biessels & Despa, 2018). In fact, type 2 diabetes has accounted for 95% of all its dementia cases (Sho et al., 2021). Although cognitive decline has been considered as a risk factor for type 2 diabetes, it has also been identified as a new form of a diabetes-related complication (Umegaki, 2018).

Type 2 diabetes has been linked with an increased risk of various diseases, which are labeled as microvascular origins (Houben & Stehouwer, 2020). These origins have included the following: retinopathy, chronic kidney disease, neuropathy, stroke, depression, cognitive decline, and heart failure (Houben & Stehouwer, 2020). The precedence of low-grade inflammation, the weakening of insulin signaling, and the initiation of pathways have been directly associated to chronic hyperglycemia, which has been known to activate the onset of cognitive decline (Little et al., 2021). Glycemic control has played a significant role in cognitive decline (Biessels & Despa, 2018). Elevated hemoglobin A1c levels have been correlated with diabetes-associated cognitive decline, or decrements (Biessels & Despa, 2018; Mimenza-Alvarado et al., 2020). Yet, such contradiction remains. There has also been evidence that such an association is regarded as inadequate, or does not exist (Umegaki, 2018). Emerging literature has denoted that hyperglycemia and hypoglycemia levels are associated in the risk of developing dementia (Biessels et al., 2018). However, determining if hemoglobin A1c levels have any association with the risk of developing dementia has not been certain (Biessels et al., 2018).

Literature Search Strategy

The literature used for this study comprised peer-reviewed professional articles. To retrieve the peer-reviewed articles, I used Google Scholar. The key words included *type 1 diabetes* AND *cognitive dysfunction* and *NHANES*, *type 1 diabetes* AND *cognitive dysfunction*, *diabetes* AND *cognitive dysfunction* AND *NHANES*, *diabetes* AND *cognitive dysfunction* AND *race*, *diabetes* AND *cognitive dysfunction* AND *comorbidity*,

type 2 diabetes AND *comorbidity*, *cognitive decline* AND *diabetes-related complication*, *cognitive decline* AND *type 2 diabetes*, *diabetes* AND *Alzheimer's disease*, *oxidative stress* AND *Alzheimer's disease* AND *type 2 diabetes*, and *cognitive decrements* AND *diabetes*. Most of the literature review consisted of peer-reviewed articles conducted and published within the past 5 years.

Theoretical Framework

Psychologists, Edward Deci and Richard Ryan (2008) have developed the self-determination theory. The theory has been widely used in sports, education, and health care (Deci & Ryan, 2008). All of the preliminary work for self-determination theory stems back to the 1970's. Nonetheless, the first all-inclusive statement of self-determination theory had surfaced in the mid 1980's (Deci & Ryan, 2008). It had not been until the early 2000's when the theory became extensively used in research (Deci & Ryan, 2008). Self-determination theory has been studied previously as it pertains to chronic diseases, such as diabetes (Walker, 2012).

Self-determination theory focuses on human motivation. The theory has expressed that there are three indispensable ways to stay motivated: a) amotivation (lack of motivation), b) intrinsic (to learn, to explore, an inclination toward discovering challenges, and exercising one's abilities), and c) extrinsic (attaining motivation to carry out action through various sources) (Ryan & Deci, 2000). Ryan and Deci believed that individuals innately embody three psychological needs: autonomy, competence, and relatedness (Taylor et al., 2012). When the psychological needs are met, this is when individuals become more committed or self-motivated (Taylor et al., 2012). Individuals

have been more susceptible in accomplishing goals when they feel they have the independence or autonomy to make their own behavioral decisions, which has been correlated to improved glycemic control (Patrick & Williams, 2012). According to Mohn et al. (2015) and Williams et al. (2004) the support of health care professionals might have made a significant impact with the level of autonomy individuals can have when managing their own diabetes. With self-determination theory, autonomy support from health care professionals have assisted in enabling the internalization of autonomous motivation in individuals living with diabetes (Williams et al., 2004). And, as a result, the autonomy supported from health care providers can directly and indirectly promote improved diabetes self-management and glycemic control (Patrick & Williams, 2012; Mohn et al., 2015). Previous longitudinal study findings have reported evidence of the association between diabetes and cognitive dysfunction (Anderson et al., 2019). With the current study, the purpose was to assess if self-reported health care diagnosed diabetes has any association in the risk of developing cognitive dysfunction. To evaluate this, self-determination theory would be best suitable.

Literature Review Related to Key Variables and/or Concepts

Cognitive Dysfunction

Cognitive performance has been based upon how an individual stands apart from what is deemed as normal cognitive performance (Biessels et al., 2018). Type 2 diabetes has been associated as a risk factor for cognitive dysfunction, which has been identified as a new-found type of diabetes-related complication (Umegaki, 2018). While there has been an array of cognitive domains that have been compromised in individuals living

with type 2 diabetes, executive functioning and processing speed are most commonly reported to be weakened in the older population (Umegaki, 2018). In comparison to individuals who do not live with diabetes, and as part of a diabetes-related complication, in this observational study, individuals living with diabetes have been at a 2.5-fold increased risk of developing dementia (Liu et al., 2020). Diabetes has been a multifaceted disease, which has been correlated to other metabolic and health complications (Liu et al., 2020). Nevertheless, its core conditions, hyperglycemia and insulin resistance have been identified as the linkage between diabetes and cognitive decline (Liu et al., 2020).

Regarding cognitive dysfunction, neuroimaging studies have displayed that diabetes-related cognitive damage is exemplified by the same pathological characteristics as vascular dementia and brain atrophy (Dybjer et al., 2018). Individuals suffering from cognitive decline have been projected to double every 20 years (Albai et al., 2019). It has been estimated by 2040 that 81,000,000 individuals will suffer from cognitive decline (Albai et al., 2019). Cognitive dysfunction has been identified as revealing itself in various stages (Biessels et al., 2018). Diabetes-associated decrement has been considered as the mildest dysfunction stage, which can be presented in type 1 and type 2 diabetes (Biessels et al., 2018). Additional cognitive degeneration has been known to progress slowly over time (Biessels et al., 2018).

Though cognitive decline has been known to surface at any given age, the development of dementia typically emerges at an older age (Albai et al., 2019). There has been several diabetes mellitus-related mechanisms which have been known to further expedite cognitive functioning deterioration (Albai et al., 2019). The mechanisms have

been comprised of insulin-resistant syndrome, hyperinsulinemia, and disruption of insulin homeostasis in the brain (Albai et al., 2019). These mechanisms have also exacerbated to more advanced conditions, such as dementia (Albai et al., 2019).

Cognitive Decrements

With type 2 diabetes, there has been minor cognitive decrements known to surface. In regard to cognitive decrement, this has been recognized as a slight change an individual can recognize within themselves on the ability to process speed, memory, and functioning (Biessels & Despa, 2018). These decrements have been most likely to surface during the pre-diabetes stage, and within years further exacerbates at a rate of 50% more in comparison to those who go through normal cognitive ageing (Biessels & Despa). The decrements have been an aberration from normal cognitive functioning, but not serious enough to be categorized as an impairment (Biessels & Whitmer, 2020). The decrements are normally identified, or observable after a cognitive test performance has been conducted (Biessels & Whitmer, 2020). The subtle decrements have been recognized as possibly having an effect on memory, the ability to process speed, and executive functioning (Biessels & Whitmer, 2020).

Cognitive Dysfunction-Dementia

Dementia has become a major public health concern (Callisaya et al., 2018). Poor metabolic health (blood glucose control) has been associated with a high risk for dementia (Callisaya et al., 2018). Poor metabolic control, or metabolic disturbances involving high hemoglobin A1c levels, hyperglycemia and high cholesterol has been noted for inducing the presence of dementia, which is known as Alzheimer's disease

(Tumminia et al., 2018). There are longitudinal studies with findings which have indicated that type 2 diabetes is linked to an increased decline in executive function, processing speed, verbal fluency, and memory (Callisaya et al., 2018). Dementia is a disease that has been characterized by the weakening of the memory and the ability to maintain self-care (Hsiao, 2019). Type 2 diabetes has been known to increase the risk of dementia (Andersen et al., 2019). As a matter of fact, type 2 diabetes has doubled in the risk of the development of dementia (Callisaya et al., 2018; Dyber et al., 2018). There has been an estimated 4,600,000 newly developed dementia cases worldwide each year (Albai et al., 2019). Dementia has been distinguished by underdeveloped cognitive performance, such as, language, memory, visuospatial, and executive functions (Biessels et al., 2018; Shao et al., 2021). Type 2 diabetes has been accounted for 95% of all dementia cases (Shao et al., 2021). Most individuals living with diabetes, who become diagnosed with dementia, have been typically over 65 years of age due to the onset of MCI and dementia not occurring (or rarely occurring) under 60 to 65 years of age (Biessels et al., 2018)

Individuals living with type 2 diabetes who have poor glycemic control, severe hypoglycemia, longer duration of diabetes, and pre-existing micro- or macrovascular complications have been at a heightened risk of developing dementia (Yu et al., 2020). Uncontrolled diabetes has been correlated with dementia in individuals living with type 2 diabetes (Yu et al., 2020). High blood glucose can perhaps weaken cerebral functioning in individuals who have been living with type 2 diabetes as well (Hsiao, 2019). Insulin treatment and the longevity of diabetes played a contributing factor in dementia (Yu et

al., 2020). Yet, research remained inconclusive as to if high glucose leads to dementia for type 2 diabetics (Yu et al., 2020). Yu explains that poor glucose control has been linked to dementia in type 2, but there has also been support, or evidence of high glucose not being associated to dementia (as cited in Xu, 2009, p. P127). On the contrary, poor glucose control has been in connection to cognitive decline between the ages of 70 to 79 years (Yu et al., 2020). However, there has been less of a decline in individuals over 80 years of age living with type 2 diabetes (Yu et al., 2020).

Individuals who have been diagnosed with dementia while living with type 2 diabetes could also be accompanied with other chronic complications, reduced physical and mental functions, and increased mortality (Albai et al., 2019). Nonetheless, cognitive impairment with its connection to the development of dementia can also be induced from lipid disorder, poor glucose control, poor diet control, and hypoglycemic medication (Albai et al., 2019). As a result, early detection of MCI progression has been vital and necessary (Albai et al., 2019).

Mild Cognitive Impairment

MCI has been a temporary and conceivably amendable stage between what is perceived as normal cognitive progression (or aging) and dementia (Li et al., 2019). Prediabetes has correlation to mild cognitive decrements (Dybjer et al., 2018). In comparison to healthy individuals, those living with MCI have been more than likely at a higher risk of developing dementia (Li et al., 2019). Type 2 diabetes has not only been a contributing factor for the development of MCI, but a driving force in the progression of MCI into the dementia stage (Li et al., 2019). As a result of this, it has been crucial to

induce treatment in individuals living with type 2 diabetes and MCI (Li et al., 2019). It has been vital due to the improvement of diagnosis (Li et al., 2019). For individuals living with type 2 diabetes, depression has also become a contributing factor for MCI (Li et al., 2019). Although type 2 diabetes has been associated with MCI, there has also been a possibility that the use of a glucose-lowering drug treatment could help improve cognitive functioning (Li et al., 2019).

Glucose Control/Metformin

For individuals living with type 2 diabetes, metformin has been deemed as the first line of defense for the treatment of lowering blood glucose levels (Hsiao, 2019). Metformin has been presented as having the ability to decrease the risk of atherosclerotic diseases and cancers in type 2 diabetes (Hsiao, 2019). However, the impact metformin has on dementia remains unknown (Hsiao, 2019). Research has indicated that not only the use of metformin by itself, but also the use of metformin and sulfonylureas (drug treatment for blood glucose) shows a lower risk of dementia for type 2 diabetes (Hsu et al., 2011, as cited in Hsiao, 2019, p. 38). In comparison to metformin and sulfonylureas, research addressed how participants taking thiazolidinediones, a drug treatment for blood glucose, could have an increased risk of dementia (Cheng et al., 2014, as cited in Hsiao, 2019, p. 38). Contrarily, there has been an increased risk of dementia linked to the use of metformin (Kuan et al., 2017; Imfeld et al., 2012, as cited in Hsiao, 2019, p. 38).

Cognitive Dysfunction-Alzheimer's Disease

Alzheimer's disease is the most prevalent form of dementia (Caberlotto et al., 2019). It has been regarded as an advanced neurodegenerative disease found in the

central nervous system, and depicted by its histopathological, molecular, and biochemical abnormalities (Caberlotto et al., 2019; Yuan et al., 2020). Associations have been found between type 2 diabetes and Alzheimer's disease (Chornenkyy et al., 2019). In fact, the likelihood of being at risk of Alzheimer's disease has increased 2–3 times more for individuals living with diabetes mellitus (Zhang et al., 2021). Stanciu et al. (2020) stated that individuals living with diabetes have a 65% increased risk of acquiring Alzheimer's disease. In addition to this, there has also been a decrease in cognitive skills, which consisted of the following: perceptual speed, memory, and learning (Stanciu et al., 2020).

The underlining connection between type 2 diabetes and Alzheimer's disease has been within the metabolic disturbances (diabetic phenotype), which include: hyperglycemia, hyperinsulinemia, and hypercholoesterolemia (Tumminia et al., 2018). These metabolic disturbances have been affiliated with brain atrophy and not to mention, the pathological features of Alzheimer's disease (Tumminia et al., 2018).

Problems Associated With Alzheimer's Disease

The problems associated with type 2 diabetes have included the following: high blood pressure, hyperglycemia, dyslipidemia, insulin resistance, and abdominal obesity. These problems have been known to enhance the likelihood of heart disease and any other additional coronary issues (Matos et al., 2017). Intriguingly, all the associated problems listed have been identified as key indicators of individuals that exhibit, or at risk of the advancement of MCI to Alzheimer's disease (Matos et al., 2017). Increased cognitive declination have been most likely found in individuals with Alzheimer's disease who displayed the aforementioned problems listed (Matos et al., 2017). The

problems have not only correlated to insulin resistance (in reference to type 2 diabetes), but have also been linked to vascular endothelial dysfunction (Matos et al., 2017).

Another contributing factor that played a vital role in Alzheimer's disease is oxidative stress and neuroinflammation (Matos et al., 2017). The diabetes-related underlying cause for these factors has been extensive (Matos et al., 2017). For example, with type 2 diabetes, insulin resistance can prompt hyperglycemia, which can therefore, induce tissue damage and oxidative stress (Matos et al., 2017). Oxidative stress, chronic inflammation, and hypertension have been known to contribute to the pathophysiological modifications that are in connection to hyperglycemia, insulin resistance, and dyslipidemia (Matos et al., 2017). All these factors have an impact on the endothelial dysfunction (Matos et al., 2017). This, in turn, has been identified in the possible development of vascular cognitive impairment by the decreasing of blood flow into the brain (Matos et al., 2017). The oxidative enzymes in lipid metabolism have been noted to add to the inflammation and endothelial damage, which are both most likely to be discovered in individuals living with diabetes and Alzheimer's disease (Matos et al., 2017).

Insulin Resistance

Brain insulin resistance has been a vital, however, disregarded feature of Alzheimer's disease (Chatterjee et al., 2018). When insulin has been unimpeded from the pancreas, it is then transferred to the brain through the blood brain barrier using a receptor-mediated mechanism (Chatterjee et al., 2018). Initially, the brain was deemed as a non-insulin target organ (Chatterjee et al., 2018). However, numerous studies have

denoted a widespread distribution of insulin receptors (in the brain) indicating a complex neuroregulatory role for insulin (Chatterjee et al., 2018).

Tumminia et al. (2018) suggested insulin play an integral role in neuronal survival and brain functioning. Not only has insulin been essential for facilitating learning and memory, but also for neuronal survival (Tumminia et al., 2018). Insulin has contributed to the activation of dendritic spine and synapse formation, neuronal stem cell activation, neurite growth and repair, and neuroprotection (Tumminia et al., 2018). As a result, modifications to insulin signaling and metabolism in the central nervous system have greatly impacted the development of various brain disorders (Tumminia et al., 2018). Insulin-signaling dysregulation has been identified as a possible essential component for the early detection of Alzheimer's disease pathogenesis (Tumminia et al., 2018).

Insulin Inflammation

Amyloid beta oligomers are a cluster of robust neurotoxins that have mediated inflammation, which has been in connection to insulin resistance in the central nervous system (Sousa et al., 2020). This has been believed to be the central reasoning for the impediment of creating new memories (Sousa et al., 2020). Insulin levels are depleted from individuals living with type 2 diabetes who have been diagnosed with Alzheimer's disease (Sousa et al., 2020). The pancreas is responsible for secreting insulin, which has been recognized as a hormone produced by beta cells (Sousa et al, 2020). The insulin receptors are located in the synaptic membranes of the brain (Sousa et al., 2020). The insulin substrates, referred to as IRS, have contributed to the role of not only learning ability, but also memory (Sousa et al., 2020). The existence of a certain formation of the

amyloid beta oligomers has been in connection to the malfunctioning of the insulin receptor substrates (Sousa et al., 2020). In essence, for individuals living with type 2 diabetes, if there has been a consistency with insulin resistance, the functioning of insulin receptors will diminish, which can enhance the onset of Alzheimer's disease (Sousa et al., 2020).

Summary and Conclusions

In Chapter 2, I discussed how cognitive decline is not only a risk factor but is also recognized as a diabetes-related complication in type 2 diabetes. The likelihood of experiencing cognitive dysfunction can occur as early as the prediabetes stage. Cognitive dysfunction is presented in the form of three stages: cognitive decrement, MCI, and dementia. In comparison to individuals who do not live with type 2 diabetes, the decrements that are associated with cognitive dysfunction tend to grow at an alarming rate for type 2 individuals, which is reasonable enough to deem this as a diabetes-relatable complication. Chapter 2 also identifies the literature search strategy, theoretical framework, and discloses an in-depth look into the three stages of cognitive dysfunction among individuals living with type 2 diabetes. Chapter 3 will discuss the research design method, population, recruitment process, data collection, and ethical issues.

Chapter 3: Research Method

Introduction

The development of cognitive declination has been a risk factor for individuals living with type 2 diabetes. The process of the decline has been identified in the form of three stages: (a) cognitive decrement, (b) MCI, and (c) dementia (Biessels & Despa, 2018; Zhu, 2020). It has been a newly formed diabetes-related complication (Umegaki, 2018). The purpose of the study was to evaluate if self-reported diabetes has any association with the risk of developing cognitive declination, or dementia in individuals living with type 2 diabetes. In accordance with the literature review, glycemic control has played an integral part in cognitive dysfunction. Although evidence has indicated that poor glycemic control can bring on the presence of cognitive dysfunction, there has also been inconclusiveness of any correlation between glycemic control and cognitive dysfunction. For this study, the independent variable is glycemic control, while the dependent variable is identified as cognitive dysfunction. This chapter will further expand on the research design and approach and will address the population, sampling size, threats to validity, and ethical concerns.

Research Design and Rationale

A quantitative, cross-sectional research design, with a secondary data analysis was applied to the study. The approach was utilized to assess if there was any association between diabetes and cognitive dysfunction. The sole purpose of utilizing a secondary data analysis was the ability to provide scientific knowledge through an alternative viewpoint (Johnston, 2017). The quantitative approach was the most applicable for

gaining a better understanding, if any, of the association between diabetes and cognitive dysfunction. The research variables were selected based upon topic of interest, literature review findings, and accessibility of data through NHANES. Each of the variables were measured using a Computer-Assisted Personal Interview (CAPI) system. The study variables were as follows:

- Diabetes: NHANES developed a questionnaire asking participants if a doctor, or health professional has ever told them they have diabetes (with the exception of during pregnancy). The responses and codes were (a) "yes" (coded as 1) and (b) "no" (coded as 0).
- Gender: Participants from NHANES were asked if their gender was either male or female. A code, or value was labeled for the male or female response. The male participants received a code of 1 and the female participants, a code of 2. Participants whose gender were missing were eliminated from the study.
- Age: The participants of NHANES were asked of their age at the time of screening. The participants' ages ranged from 0 to 150 years. Participants aged 0–79 were identified or received a code pertaining to their own respective age. In contrast, participants 80 years of age and older received a code of 80. Participants whose age was missing were eliminated from the study.
- Ethnicity: Each participant self-reported their racial identity. The participants identified themselves as (a) non-Hispanic White (coded 1), (b) non-Hispanic

Black (coded 2), (c) Hispanic (coded 3), and (d) Other (coded 4). Participants whose ethnicity was missing were eliminated from the study.

- Cognitive dysfunction: Each participant reported to have some form of cognitive dysfunction. The participants were identified as displaying (a) high cognitive form (coded 0) or (b) low cognitive form (coded 1). Participants with missing data were eliminated from the study.
- Poverty-income ratio: Each participant reported their total annual household income. The ratio varied from 0-4.98. Participants who had a ratio between 0-4.98 received a code pertaining to their own respective value.

Table 1 provides an additional description of each variable along with its variable code.

Table 1

Study Variables

Variable name	Variable code	Description
Diabetes2	DIQ10	This describes diabetes diagnostics (does not include type 1versus type 2).
Gender	Riagendr	Gender
Age	Ridageyr	Age (continuous variable)
Ethnicity	Ethnicity	Ethnicity has four main categories for participants to select from.
Cognitive dysfunction	Cognitive	This explains the level, or form of dysfunction, displayed.
Poverty–income ratio	Indfmpir	This is compared the 2000 standard census and is a reflection of the federal poverty guidelines.

Research Questions and Hypotheses

RQ1: Is there an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity?

$H_0$1: There is no association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity.

H_a1: There is an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity.

Figure 1 illustrates the hypothesized relationship between diabetes and cognitive dysfunction.

Figure 1

Hypothesized Relationship Between Diabetes and Cognitive Dysfunction

RQ2: Does race modify the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, and poverty–income ratio?

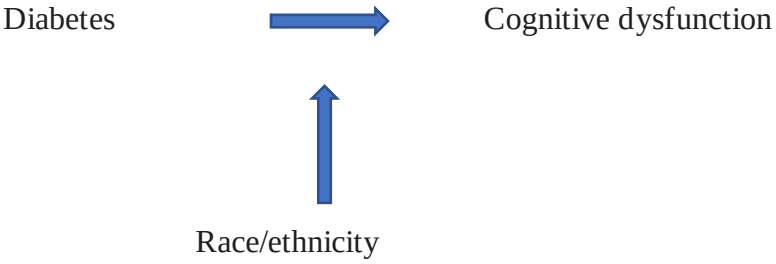
$H_0$2: Race does not modify the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio.

H_a2: Race does modify the effect of the association between cognitive diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio.

Figure 2 illustrates the hypothesized relationship between diabetes, cognitive dysfunction, and race/ethnicity.

Figure 2

Hypothesized Relationship Between Diabetes, Cognitive Dysfunction, and Race/Ethnicity



RQ3: Does sex modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio?

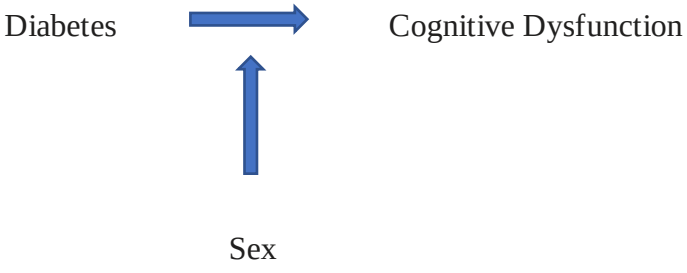
$H_0$3: Sex does not modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio.

H_a3: Sex does modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio.

Figure 3 illustrates the hypothesized relationship between diabetes, cognitive dysfunction, and sex.

Figure 3

Hypothesized Relationship Between Diabetes, Cognitive Dysfunction, and Sex



Methodology

Population

Type 2 diabetes has been considered a worldwide epidemic (Alaofè et al., 2022). The number of adults diagnosed with type 2 diabetes has been expected to increase between 75% and 80% in the lower and middle-income countries by 2045 (Alaofè et al., 2022). The initial increase has been expected at 48% (Alaofè et al., 2022). NHANES is a program designated to evaluate the stance of health and nutrition with children and adults residing in the United States. NHANES relevancy stems from the National Center for Health Statistics (NCHS). The NCHS is part of the Centers for Disease Control and Prevention. The sole responsibility of NCHS is the production of the vital and health statistics for the world. The target population had consisted of participants living with type 2 diabetes who were 60 years of age and older, non-Hispanic White, non-Hispanic Black, Hispanic, and Other (Akinbami et al., 2022). According to NHANES, data distributed for public use has been released in 2-year cycles. NHANES's latest data released were for 2017 through March 2020, a period before the COVID-19 pandemic. Prior to this data, NHANES released data for the 2017-2018 year. The next 2-year cycle would have been the 2019–2020 year, which would have been published in 2021, however, due to the pandemic, the data collected during the 2019–2020 cycle year had been delayed (Akinbami et al., 2022). Data for the 2019–2020 cycle year had only been collected until March 2020 (Akinbami et al., 2022). As a result, the data collected prior to March 2020 was merged into the 2017-2018 cycle year to reflect the representation of the general population (Akinbami et al., 2022).

Sampling and Sampling Procedures

The sampling and sampling procedures will be obtained through NHANES. A multistage, probability sampling design will be utilized for the study. The inclusion criteria consisted of participants who were 60 years of age and older and the exclusion criteria comprised of participants who have not received a diabetes prognosis and part of an institutionalized population. The multistage probability sampling design had been employed to select participants who collectively represented the civilian, non-institutionalized U.S. population (NCHS, n.d.). The multi-stage sampling has also been referred to as the multi-stage cluster sampling (Rahman et al., 2022). Multi-stage probability has been regarded as one of the most intricate forms of sampling due to its serial selection process (Rahman et al., 2022). In essence, various steps have been included with the multi-stage probability sampling. From NHANES, the multi-stage sampling consisted of four stages. The stages comprised the following: (a) primary sampling units (PSUs; including counties, groups of tract within counties, or combination of adjacent counties), (b) segments (census blocks or combinations of blocks), (c) dwelling units (DUs; particularly within segments), and (d) people (within households) (Akinbami et al., 2022).

First Stage Sampling: Primary Sampling Units

According to NHANES, the PSU was chosen in with probabilities in proportion to the measure of size (MOS) (Akinbami et al., 2022). The MOS for each PSU was supported by preexisting, well-known criteria for acquiring survey estimates for subgroups established by sex, race, income, age group, and Hispanic origin (Akinbami et

al., 2022). MOS has been defined as the weighted average of population counts, in which the weights are estimated, or calculated, in order to have a greater probability of selection to the PSUs with a relatively superior proportion of people within the subgroups that are selected for oversampling (Akinbami et al., 2022). The weights (sampling fraction) that were used to ascribe the appropriate contribution relative to each race in its computation of MOS had been assessed (see Appendix). The ultimate reasoning for NHANES stratification of PSUs was to make certain the chosen sampling units were evenly distributed (Akinbami et al., 2022).

The NHANES 2015–2018 and 2019–2022 sampling data consisted of oversampling within some of the population subgroups (Akinbami et al., 2022). It was the oversampling of subgroups that governed the sampling domains. The population subgroups selected for oversampling were used to establish the sampling domains that are utilized to choose the sample at all stages. The subgroups selected for oversampling included the following: Hispanics, non-Hispanic Blacks, non-Hispanic, non-Black Asians, non-Hispanic Whites (and other races and ethnicities that are equal to, or below 185% of the federal poverty level). However, the subgroup names used for the sampling design and weighting process for oversampling were categorized distinctively from the data files released to the general public (using RIDRETH3 and RIDRETH1). In order to determine eligibility, the subgroups had the option of identifying themselves as Hispanic, non-Hispanic Black, non-Hispanic, non-Black Asian, and non-Hispanic White (other races and ethnicities equal to, or below 185% of federal poverty level). However, the subgroups racial identification data (coded as RIDRETH and RIDRETH1) released to the

public was categorized as (a) non-Hispanic White, (b) non-Hispanic Black, (c) non-Hispanic Asian, and (d) other races.

Second Stage Sampling: Segments

The second stage of the sampling process was the segment. With regard to this stage, each PSU was split into segments, which comprised of one or more contiguous census blocks (Akinbami et al., 2022). The segments were sampled based upon the MOS, which is the sum of the MOS for every census block within the segment (Akinbami et al., 2022). To ensure an adequate sample, every segment had to meet the minimum size for the MOS (Akinbami et al., 2022). The segment MOS was subtly bedded by the density of minority populations. This ensured that race and the Hispanic-origin distribution of the sample mirrored the overall distribution of the PSU (Akinbami et al., 2022).

Third-Stage Sampling: Dwelling Units

DUs refer to the actual location where an individual lives (Akinbami et al., 2022). This is comprised of apartment complexes, single-family homes, dormitories, or even shelters (Akinbami et al., 2022). Every DU from the sampled segments was listed. Each DU was sampled at rates created to deliver a national and equal probability sample (Akinbami et al., 2022).

Fourth-Stage Samling: People

In order to establish final eligibility, all of the chosen DU addresses were screened to decide if any of the participants would be great for the inclusion criteria (Akinbami et al., 2022). The participants selected in this phase had the following information collected from them: race, Hispanic origin, age category, sex, and income (Akinbami et al., 2022).

Sample Weights

Sample weights had been identified as the measurement of the number of individuals within the given target population symbolized by each participant (Akinbami et al., 2022). Sample weighting comes in the form of three steps. The first step encompassed the calculation of base weights in compensating for disproportionate probabilities of selection within the sample domains (Akinbami et al., 2022). Second, there was a modification for nonresponses to decrease potential bias (internal validity) (Akinbami et al., 2022). Third, there was a calibration of the sampled weights to its selected population (Akinbami et al., 2022).

Base Weights. With regard to the first step in the sampling weight process, base weights, NHANES calculation was defined as the product of the initial base weight and three adjustment factors: $W_{i\,(base,\,screener)} = 1/r_k\ (f_{i\,(release)}\ f_{i\,(inc)}\ f_{i\,(stratum)})$ (Akinbami et al., 2022). The preliminary base weight for the individual participants within a sampling domain (k) was the exact same for all individuals in that domain and also equaled the inverse of the sampling rate (r_k) in the sampling domain (Akinbami et al., 2022). However, when it came to estimated response rates and alternating population distributions, the base weight for each domain was distinct (Akinbami et al., 2022). The initial base weights were modified to include the following: (a) $f_{i\,(release)}$, the proportion of DUs released for screening in a PSU; (b) $f_{i\,(inc)}$, the increase in the DU sample size required in some PSUs; and (c) $f_{i\,(stratum)}$, the factor to adjust for the limited sample (Akinbami et al., 2022).

Nonresponsive Adjustment. According to NHANES, the second step to sampling weights was the nonresponsive adjustment. With the nonresponsive adjustment, what was taken into consideration was that not every household was going to be screened (Akinbami et al., 2022). Some of the individuals from the sampling population (SP) screened declined from being interviewed, and some of the interviewed participants had chosen not to proceed any further with the process (Akinbami et al., 2022). Therefore, to minimize bias, the base weights were adjusted for the nonresponse at the screening, interview, and examination stages (Akinbami et al., 2022). The information, or the amount of, that was utilized for the adjustment, typically increased at each stage (Akinbami et al., 2022). Meanwhile, person-specific information during the interview was available to adjust mobile examination centers (MEC) weights (Akinbami et al., 2022). The non-response adjustment formula encompassed computing the following:

$$F_{i\,(NR)} = \frac{\text{Sum of stage base weights in the adjustment cell}}{\text{Sum of stage base weights of the participants in the adjustment cell}}$$

and, applying these to the survey weights as:

$$w_{i\,(NR,\,stage)} = w_{i\,(base,\,stage)}\, f_{i\,(NR,\,stage)}$$

independently with the nonresponse cells (Akinbami et al., 2022). These nonresponse cells were identified by categorical characteristics by participants and nonrespondents (Akinbami et al., 2022). There was very little known for those who did not complete the screening (regarding the household). Therefore, for these particular nonresponsive individuals, there was an adjustment cell set in place. Also, there was a nonresponsive adjustment for those participants during the interview and examination stages (Akinbami et al., 2022). In order for the adjustment cells to formulate, different variables were

utilized to help recognize the cells within certain age groups: 0-5, 6-19-20-39, 40-59, and 60 years of age and older (Akinbami et al., 2022).

Weight trimming was necessary to decrease the influence of any extreme weight on estimation (Akinbami et al., 2022). Though, if not vigilant, the trimming of sampled weights could have initiated estimation bias (internal validity) (Akinbami et al., 2022). Therefore, weight trimming was not applied for all sampled weights (Akinbami et al., 2022). So, in deciding to conduct weight trimming for samples or subsets of samples, NHANES had assessed the distribution of weights within each sampling domain that was inspected (Akinbami et al., 2022). NHANES threshold for the sampling interview weights was demarcated as being 4.75 times the sampling domain mean (Akinbami et al., 2022). There was a total of four extra weights which superseded this threshold (Akinbami et al., 2022). The values of these radical weights were deduced to the threshold weights (Akinbami et al. 2022). Also, a weight adjustment was made with the weights of all cases within the same sampling domain, so the total sum of the weights within every sampling domain equated to the corresponding weighted sum just before weight trimming (Akinbami et al., 2022). The sampling examination weights threshold was demarcated as being five times the sampling domain mean (Akinbami et al., 2022). There was a total of 13 weights that superseded this threshold, therefore, weight trimming took place (Akinbami et al., 2022). NHANES formula for trimming sampled weights was defined as the following:

$$T_i = \{W_{i\,(NR)}, \text{ if } w_{i\,(NR)} <\text{- threshold}$$
$$\text{threshold, otherwise}$$

Then the actual trimming factor $f_{i\,(TR)}$ was defined as:

$$F_{i\,(TR)} = t_i/w_{i\,(NR)} \cdot \sum_i^{nk}=1\ w_{i\,(NR)}/\sum_i^{nk}=1\ t^i$$

whereas the n_k is identified as the sample size of the *k*th race-Hispanic-origin-income-sex-age sampling domain, and:

$$w_{i\,(TR,\,stage)} = w_{i\,(NR,\,stage)}\ f_{i\,(TR,\,stage)}$$ (Akinbami et al., 2022)

Calibration. Last was the calibration of sampled weights to its selected population total. NHANES utilized raking as a form of calibrating. It was conducted during the screening, interviewing, and examination stages (Akinbami et al., 2022). To further explicate on the process of calibration (raking), it started with the number of individuals who shared the same characteristics within a target population that was a representation, or signified participant's sampled weight (Akinbami et al., 2022). For example, within a demographic subgroup, the sum of all of the participants' weights could have been deemed as the total number of individuals being represented (for this particular subgroup) (Akinbami et al., 2022). With calibration, the participants' sampled weight (from the demographic subgroup) was modified, so the total sum of the sample weights within the group equated to the population from an independent data source (Akinbami et al., 2022).

Calibration entailed of using a ratio adjustment to the survey weights (Akinbami et al., 2022). The formula was as follows:

$$F_{i(C)} = \frac{N_C}{\text{Sum of nonresponse adjusted and trimmed weights of the demographic subgroup}}$$

And, the calibrated weights were calculated as:

$$W_{i\,(C,stage)} = w_{i\,(TR,\,stage)}\ f_{i\,(C,\,stage)}$$ (Akinbami et al. 2022).

To reiterate, calibration used a ratio adjustment to the survey weights. Regarding this particular step, the numerator (N_C) was the population control total (for the demographic subgroup) (Akinbami et al., 2022). The denominator was the sum of the nonresponse adjusted and trimmed weight of the demographic subgroup (Akinbami et al., 2022). With regard to the interview and MEC examination weights, they were calibrated by utilizing a four-dimensional raking (Akinbami et al., 2022). The raking included: race-Hispanic-origin-age-sex demographic subgroups, race-Hispanic-origin-sex-education-level subgroups (20 years of age and older), area-level household income, and urbanicity (Akinbami et al., 2022).

Regarding the sample size, the initial sample size for NHANES started with roughly 27,066 participants during the screening stage (NCHS, 2023). However, during the interview stage, the total number of participants was 15,560. There was a total of 7,721 male participants to 7,839 female participants. As for the examination stage, the total came to 14,300 total participants, with 7,085 men and 7,215 women engaging in the study.

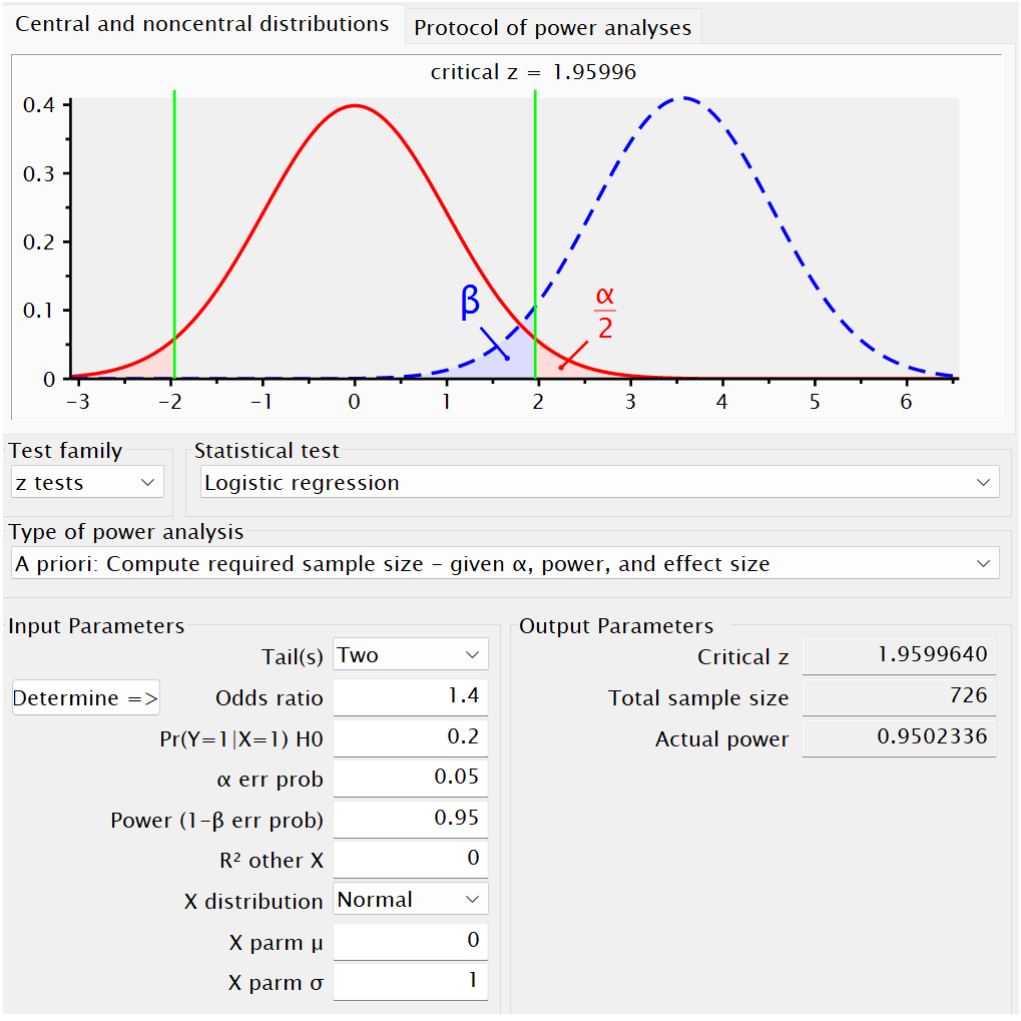
Power Analysis

In order to determine the minimum sample size required to observe an effect, a power analysis was conducted using G*Power (see Figure 4). The main analysis for this study was a logistic regression. For a logistic regression analysis, sample sizes of 726 will yield a power of 95% with $\alpha = 0.05$ and a minimum detectable odds ratio of 1.4 with diabetes as the predictor variable and cognitive dysfunction as the outcome variables when testing the different hypotheses. The minimum detectable odds ratio of 1.4 was

used because based on the odds ratio from other research. Additionally, according to certain literature, each predictor variable conservatively must have ten outcome events per predictive variable in order to prevent overfitting, under special circumstances less than ten is permissible. In light of these calculations in support of the power analyses, there was confidence that the sample size used in this study was adequate.

Figure 4

Power Analysis



Sampling Strengths and Weaknesses

The central focus of sampling was to collect a smaller group of participants who were a representation of a significantly larger group, or population (Rahman et al., 2022). From the smaller group, and its findings, the researcher could construct applicable, or reasonable generalizations that could represent the larger population (Rahman et al., 2022). Although multi-stage probability sampling was complex, the multiple steps it took to divide larger populations into smaller groups ensured primary data collection to be more effective and economical (Rahman et al., 2022). Typically, researchers have utilized this type of sampling to evade the challenges that come from randomly sampling a larger group of individuals (Rahman et al., 2022). On the contrary, multi-stage probability sampling did not cover all survey participants (Rahman et al., 2022). In essence, a researcher's study outcomes have not always been 100% accurate (Rahman et al., 2022). Multi-stage probability sampling has been seeking to decrease variance within and across groups, however, it was difficult to verify if the demographics removed from a study was effective (Rahman et al., 2022).

Data Analysis Plan

In this study, logistic regression was used to determine the relationship in the main model between diabetes and cognitive dysfunction. There are multiple ways to determine effect modification. As described by Van Ness et al. (2006), the relationship was further assessed separately in a stratified model in order to run effect modification. With regard to effect modification,determined by stratified analysis, evidence provided the statistical significance was tested separately for the non-Hispanic White population,

non-Hispanic Black population, Hispanic population, and Others. The effect modification was assessed by gender where separate model was created through sub-group stratified analysis. The effect modification was assesed to see how the exposure and outcome differed according to the levels of a third variable, whis would be the effect modifier. In order to assess for this relationship SPSS v. 28.0.1.0 was used.

Threats to Validity

For this study, attrition bias can contribute to the threat of internal validity. At the screening, interview, and examination stages, there can be variability with the number of individuals either having the desire to partake and cannot, or who are active participators in the study. In general, with any study, attrition bias can be a problem. Incorporating a complex sampling method, such as multi-stage sampling, can increase a decline in participants from one step to another. Nevertheless, in order to minimize this, sampling weights were adjusted. With external validity, the sampling bias can present itself to be a problem as well. However, with the multi-stage sampling method, it utilizes a 4-step process in selecting participants. By utilizing this method, it minimizes the use of selection bias.

Ethical Procedures

Unlike any study conducted, ethical concerns should be addressed. For NHANES, their main priority is to protect the privacy of any and every individual. With regard to confidentiality, NHANES assured participants all of the information that explained identifiable characteristics of participants, establishment, or even practice would only be utilized for statistical analyses (NHANES, 2022). The participants were made aware that

staff, contractors, and agents involved would not divulge or release any responses in any identifiable form without the actual consent of the participant (NHANES, 2022). This was in accordance with the Public Health Service Act (42 U.S.C. 242m(d)), section 308(d) and the Confidential Information Protection and Statistical Efficiency Act of 2002 (NHANES, 2022). The participants were also privy to knowing that each staff member, contractor, and agent associated with the National Center for Health Statistics (NCHS), or NHANES, had taken a pledge to abide by the rules of not divulging any type of information (without the participant's consent) (NHANES, 2022). If this pledge was broken, the individuals would be subjected to losing their job, which included serving up to 5 years in prison, being fined up to $250,000, or both (NHANES, 2022). All staff and other individuals who were involved with NHANES, had to sign the pledge (as an act of compliance) before taking an active role in having any involvement with future studies, or anything else pertaining to NHANES (NHANES, 2022). In maintaining privacy, participants were also informed about how individuals affiliated with NHCS, or NHANES had to adhere to certain guidelines when handling private information (NHANES, 2022). The individuals would have to ensure that any evidence which could reveal who the participants were, was removed immediately (NHANES, 2022). This could contain any information, but not limited to, the participant (themselves), the participant's family names, personal addresses, phone numbers, and workplace (NHANES, 2022). Second, all individuals affiliated with NCHS or NHANES had access to computers that are password protected. All data is encrypted for transmission by utilizing a secure data network, which was on a secure server that is only accessible to

authorized personnel (NHANES, 2022). For this study, Walden University's Institutional Review Board had to approve the secondary data collection. The approval number is 10-04-23-0264837.

Confidentiality

With NHANES, participants' privacy was imperative. The names of all participants was not linked to any of the studies administered. Each participant was de-identified, or in essence, given a distinctive ID, so the participant's personal information was not exploited. In addition to being de-identified, with regard to confidentiality purposes, each participant would be given a consent form to complete. With NHANES, which is an extension of the Centers for Disease Control and Prevention, data was released to the public every 2 years. Therefore, because the data were a combination of NHANES's 2017–2020 collection, there were actually two separate consent forms. However, the difference between the two was the form (dated in 2017) had the assurance of confidentiality statement toward the bottom of the form, whereas, the confidentiality statement was omitted on the 2019 form.

Summary

A quantitative, cross-sectional research design, utilizing a secondary data analysis was applied to the study. This approach was used to depict the features of a population and having the ability to understand the determinants of health (Wang & Cheng, 2020). Chapter 3 introduced the significance of cognitive dysfunction and the pivotal role it plays in type 2 diabetes. The variables, codes for each variable, and description of the variables were discussed. The research design and RQs have been identified in this

chapter, and the reasoning for adapting a particular approach was reviewed in the study as well. This chapter expanded on the target population and size, sampling and sampling procedures, and nonetheless, sample weights. The strengths and weaknesses of sampling have been identified, along with the threats to validity (internal and external), and ethical issues for conducting the study. Next, will be the transition to data collection.

Chapter 4: Results

Introduction

The purpose of this study was to evaluate if self-reported health care diagnosed diabetes had any association in the risk of developing cognitive decline, or dementia in individuals living with type 2 diabetes. However, from the NHANES's website, there was no specific data set which pertained exclusively to the hemoglobin A1c levels. However, there was data made accessible to the public that focused on the association between cognitive dysfunction and self-reported diabetes. The study encompassed three RQs. The first question inquired if there is an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity. The null hypothesis stated there was no association between cognitive dysfunction and diabetes after control. The alternative hypothesis mentioned there is an association between cognitive dysfunction and diabetes after control.

The second RQ asked if race modified the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, and poverty–income ratio. The null hypothesis stated that race does not modify it, however, the alternative hypothesis mentioned that race does modify it. Last, the third RQ asked if sex modified the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio. The null hypothesis stated that sex does not modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio. However, the alternative hypothesis mentioned that sex

does modify the effect of the association between each variable. This chapter will focus on data collection and results from the study.

Data Collection

Time Frame

NHANES runs on a 2-year data cycle. However, for the 2017-2020 pre-COVID-19 pandemic data files, there was a minor change in how the data were collected. NHANES combined the 2017-2018 data cycle with the 2019–March 2020 cycle. These data were unique due to the time frame of the collection, which was on a 3-year cycle instead of the usual 2-year time frame (Akinbami et al., 2022). The data were taken from the PSUs chosen using two different sample designs. The 2017–2018 data were taken from the 2015–2018 sample design, and the 2019–March2020 data were taken from the 2019–2022 sampling design (Akinbami et al. 2022). Although the 2017–2018 cycle was already published, data collection from the 2019–2020 cycle came to a halt as of March 2020 for safety concerns due to the coronavirus outbreak (Akinbami et al., 2022). As a result of this, the data collected was not sufficient enough (or nationally represented). Therefore, data from the 2017-2018 cycle had to be incorporated, thus creating the 2017-March 2020 pre-pandemic data files (Akinbami et al., 2022).

Recruitment

Since 1999, continuous NHANES has been consistent in utilizing a multiyear, stratified, clustered four-stage sampling method in recruiting participants (Akinbami et al., 2022). This 2017–March 2020 pre-COVID-19 pandemic study was no exception to it. In recruiting participants, NHANES applied the multistage, probabilistic sampling

design. It is one of the most complex forms of sampling due to the serial selection process, in which the selected participants (or sample) collectively represents the non-institutionalized civilian population living in the 50 states and the District of Columbia and is considered nationally representative data (NCHS, n.d.).

For this study, NHANES's 2013–2014 participants were selected from different 4-year sample designs (Akinbami et al., 2022). The participants were recruited in four specific stages (from the two different sampling cycles). In the first stage, all of the states were split into four health groups—(a) A (healthiest indicators), (b) B, (c) C, and (d) D (least healthiest indicators) —according to the health index values (Akinbami et., 2022). Next, the PSUs in each state were split into three or four major strata within each health group. This generated 14 major strata in total, which is based upon the urban-rural population distribution and other characteristics of the neighborhood. Third, one of the PSUs was chosen from each stratum, for every year, with a probability related to the MOS. This led to an evenly balanced number of sampled PSUs per major stratum for the survey design. In addition to this, another PSU was selected (encompassing a large metropolitan area and MOS), for which its addition in the survey is warranted. The sampling designs from the 2 cycles included 15 PSUs (sampled from each year), for a combined total of 30 PSUs for every 2-year cycle. Nevertheless, due to other characteristics and population size that influence the major strata membership change over time.

Data Collection Discrepancies

Although NHANES's data collection runs on a 2-year data cycle, data for the 2013 and 2014 years were complete. . Due to the COVID-19 pandemic, NHANES data collection was suspended for the remainder of the year. As a result, the data collected from 2013 to March 2014 did not give a nationally representative view of the U.S. population for this time frame. With regard to this issue, impartial estimates could not be formed based upon data being partially collected (Akinbami et al., 2022). The NHANES researchers did not consider such impartial representation to be reliable.

The first stage of the sampling process consisted of utilizing both sampling designs and classifying states into 4 health groups (according to the health index values). Subsequent to this, the PSUs were separated into three or four major strata (within each health group). This formed 14 major strata (in total), which was created on the urban-rural population distribution and other characteristics of the locality, or area. From each stratum and each year, one PSU was chosen with a probability in relation to their MOS, thus resulting in an equivalent number of sampled PSUs per major stratum for each NHANES survey design. In addition to this, another PSU was selected. This additional PSU (selected for each year) consisted of a large metropolitan area and MOS. In coalescing the two sampling designs, the end result came to a total of 15 PSUs for each sampled year, 30 PSUs for every 2-year cycle, and 60 PSUs for the 4-year period covered by the sampling design. Due to the characteristics and population size (which control the major strata over time), the strata encompassed diverse PSUs in the 2013-2014 NHANES dataset. In addition, sampling weights will be applied. The process of sampling weights

will incorporate modifications due to non-responses and the calibrations of the sampling weights to the underrepresentation of the selected population.

Results

Secondary data from NHANES (through the Centers for Disease Control and Prevention) was utilized for conducting my research study. Due to the data being made available to the general public, no additional permission was required to obtain the data set. The data set attained from NHANES was from the 2013-2014 year (for the cognitive dysfunction responses).. In order to evaluate the data provided from NHANES, I used SPSS (28th version) software. Three RQs were designed from the research study and a logistic regression analysis was conducted to determine the findings from the RQs formulated. The findings from NHANES's data set will be addressed from the RQs composed.

Descriptive/Baseline Characteristics

Table 2 displayed the baseline descriptive and demographic characteristics of participants. The total population size amassed to 53,421,769 participants. With regard to the baseline characteristics, the participants identified themselves as either male (46.3%) or female (53.7%) living with diabetes (19.5%) or not living with the chronic illness (80.5%). As for the ethnicity, participants were identified as non-Hispanic White (80%), non-Hispanic Black (8.5%), Hispanic (7.1%), or other (4.5%). Nonetheless, participants had some form of cognitive dysfunction, whether it was highly recognizable (52.6%) or

on the lower end (47.4%). Table 3 included information on the sample design. As shown in the table, the number of unweighted cases totaled 1,468.

Table 2

Categorical Variable Information

		Weighted Count	Weighted Percent
Cognitive[a]	0	28093128.755	52.6%
	1[b]	25328641.146	47.4%
Diabetes	.00	42989520.113	80.5%
	1.00	10432249.788	19.5%
Ethnicity	1	42712985.009	80.0%
	2	4537767.372	8.5%
	3	3783924.414	7.1%
	4	2387093.107	4.5%
Gender	1	24710677.921	46.3%
	2	28711091.980	53.7%
Population Size		53421769.901	100.0%

a. Dependent variable
b. Reference category

Table 3

Sample Design Information

		N
Unweighted Cases	Valid	1468
	Invalid	8707
	Total	10175
Populaton Size		53421769.901
Stage 1	Strata	15
	Units	30
Sampling Design Degrees of Freedom		15

Research Questions and Hypotheses

RQ1 asked if there was an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity. The null hypothesis suggested there was no association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity. However, the alternative hypothesis proposed there was an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity. The mean age of participants was 69. The average ratio of family income to poverty was 3.0743.

With regard to RQ1, Table 4 presented the pseudo R^2 values, or the model summary. These values encompassed the percentage of the variation explained by the relationship between two variables. For Nagelkerke, the R^2 was .329. In essence, 32% of the variation was explained between the indpendent and dependent variables. Table 5, the model of effects, displayed the input in each of the independent variables to the model and its statistical significance.

Table 4

Pseudo R^2 *Values for Research Question 1*

Cox and Snell	.247
Nagelkerke	.329
McFadden	.205

Dependent Variable: Cognitive (reference category=1)
Model: (Intercept), Diabetes2, Ethnicity, Gender, Poverty-income ratio, Age

Table 5

Tests of Model Effects for Research Question 1

Source	df1	df2	Wald F	Sig.
(Corrected Model)	7.000	9.000	38.116	<.001
(Intercept)	1.000	15.000	149.284	<.001
Diabetes2	1.000	15.000	17.819	<.001
Ethnicity	3.000	13.000	30.134	<.001
Gender	1.000	15.000	11.708	<.004
Poverty-income ratio	1.000	15.000	90.856	<.001
Age	1.000	15.000	213.158	<.001

Dependent Variable: Cognitive (reference category =1)
Model: (Intercept), Diabetes2, Ethnicity, Gender, Poverty-income ratio, Age

Table 6 presented the odds ratio for RQ1. So, in order to answer the question, the tests of model effects and odds ratio had to be assessed. Looking at the independent variable, diabetes2 (from Table 5), the *p*-value was less than 0.05. This highlighted the statistical significance of the diabetes2 variable in conjunction to the dependent variable, cognitive dysfunction. However, the odds ratio determined the validity of the significance from the tests of model effect. The RQ inquired if there was an association between cognitive dyfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity. From the findings in SPSS, there was a relatively high association between diabetes and cognitive dysfunction when controlling for the other variables. The odds ratio was determined through the results of the logistic regression analysis with lower and higher confidence interval along with its estimate value. According to the output, the true odds ratio was between 1.321 and 2.333 with an estimate (or middle value) at 1.756. Since the estimate was at 1.756, which definitely was between 1.321 and 2.333, there was

95% confidence that there was an association between diabetes and cognitive dysfunction.

Table 6

Odds Ratio for Research Question 1

			95% Confidence Interval	
	Cognitive	Odds Ratio	Lower	Upper
Diabetes .00 vs 1.00	0	1.756	1.321	2.333

Dependent Variable: Cognitive (reference category=1)
Model: (Intercept), Diabetes2, Ethnicity, Gender, Poverty-income ratio, Age[a]

Factors and covariate used in the computation are fixed at the following values: Diabetes2=1.00; Ethnicity=4; Gender=2; Ratio of family income to poverty=3.0743; Age in years at screening=69.32

RQ2 asked whether race modified the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, and poverty–income ratio. The null hypothesis predicted that race did not modify the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio. The alternative hypothesis suggested that race did modify the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio. RQ2 will be answered in four parts (in reference to race).

Table 7 showed the categorical variable information for non-Hispanic White Participants. It contained the percentage of non-Hispanic White participants (ethnicity = 1). A total of 57.8% non-Hispanic White participants displayed high cognitive dysfunction and 42.2%, low cognitive dysfunction. With regard to the variable, diabetes2, 82.6% of non-Hispanic White participants did not have diabetes, however, 17.4% participants exhibited having diabetes. The subpopulation size was at 42,712,985. The

average age of the non-Hispanic White participants was 69 years of age, and the ratio of family income to poverty for those participants was 3.2595.

Table 7

Categorical Variable Information for non-Hispanic White Participants

		Weighted Count	Weighted Percent
Cognitive[a]	0	24681569.634	57.8%
	1[b]	18031415.375	42.2%
Diabetes2	.00	35272763.282	82.6%
	1.00	7440221.727	17.4%
Subpopulation Size		42712985.009	100.0%

Subpopulation: Ethnicity=1
a. Dependent Variable
b. Reference Category

Table 8 displayed the psuedo R^2 values for RQ2. The variation ranged from 16% to 27%. For Nagelkerke, the R^2 was .275. In essence, 27% of the variation was explained between the independent and dependent variables.

Table 8

Pseudo R^2 *Values for non-Hispanic White Participants*

Cox and Snell	.204
Nagelkerke	.275
McFadden	.168

Subpopulation: Ethnicity=1
Dependent Variable: Cognitive (reference category=1)
Model: (Intercept), Diabetes2, Poverty-income ratio, Age

Table 9 consisted of the categorical variable information pertaining to non-Hispanic Black participants (ethnicity = 2). There was a total of 24.9% non-Hispanic Black participants who displayed high cognitive dysfunction and 75.1% had been accounted for low cognitive dysfunction. Meanwhile, under the variable, diabetes2,

27.4% of non-Hispanic Black participants exhibited having diabetes. On the other hand, 72.6% did not exhibit such chronic illness. The subpopulation size of non-Hispanic Black participants was at 4,537,767. The mean age of non-Hispanic Black participants was 67 years of age, and the ratio of family income to poverty was 2.3014.

Table 9

Categorical Variable Information for non-Hispanic Black Participants

		Weighted Count	Weighted Percent
Cognitive[a]	0	1127653.953	24.9%
	1[b]	3410113.420	75.1%
Diabetes2	.00	3293955.450	72.6%
	1.00	1243811.922	27.4%
Subpopulation Size		4537767.372	100.0%

Subpopulation: Ethnicity = 2

a. Dependent Variable
b. Reference Category

Table 10 showed the pseudo R^2 values for non-Hispanic Black participants. As a result, the variation ranged from 15% to 23%. For Nagelkerke, the R^2 was .230. In essence, 23% of the variation is explained between the independent and dependent variables.

Table 10

Pseudo R^2 *Values for non-Hispanic Black Participants*

Cox and Snell	.155
Nagelkerke	.230
McFadden	.150

Subpopulation: Ethnicity = 2

Dependent Variable: Cognitive (reference category=1)
Model: (Intercept), Diabetes2, Poverty-income ratio, Age

Table 11 consisted of the categorical variable information in relation to Hispanic participants (ethnicity = 3). There was a total of 28.5% participants who displayed high cognitive dysfunction and 71.5% who exhibited low cognitive dysfunction. Meanwhile, variable, diabetes2, showed that 68.8% of Hispanic participants displayed no form of diabetes. However, 31.2% of Hispanic participants exhibited having diabetes.The subpopulation size of Hispanic participants was at 3,783,924. The average age of the participants was 67 years of age, and ratio of family income to poverty was 2.0324.

Table 11

Categorical Variable Information for Hispanic Participants

		Weighted Count	Weighted Percent
Cognitive[a]	0	1078190.745	28.5%
	1[b]	2705733.668	71.5%
Diabetes2	.00	2603196.999	68.8%
	1.00	1180727.415	31.2%
Subpopulation Size		3783924.414	100.0%

Subpopulation: Ethnicity = 3

a. Dependent Variable
b. Reference Category

Table 12 showed the pseudo R^2 values for Hispanic participants. The variation ranged from 19% to 29%. For Nagelkerke, the R^2 was .293. In essence, 29% of the variation was explained between the independent and dependent variables.

Table 12

Pseudo R^2 *Values for Hispanic Participants*

Cox and Snell	.204
Nagelkerke	.293
McFadden	.191

Subpopulation: Ethnicity =3
Dependent Variable: Cognitive (reference category=1)
Model: (Intercept), Diabetes2, Poverty-income ratio, Age

Table 13 consisted of the categorical variable information in relation to participants identified as "other" (ethnicity = 4). There was a total of 50% participants who displayed high cognitive dysfunction and 49.5% who exhibited low cognitive dysfunction. The variable, diabetes2, showed that 76.2% displayed no form of diabetes. Contrarily, 23.8% exhibited having diabetes. The subpopulation size of the "other" participants was at 2,387,093. The mean age of the participants was 68 years of age, and ratio of family income to poverty of the participants was 2.8818.

Table 13

Categorical Variable Information for Participants Identified as "Other"

		Weighted Count	Weighted Percent
Cognitive[a]	0	1205714.424	50.5%
	1[b]	1181378.683	49.5%
Diabetes2	.00	1819604.382	76.2%
	1.00	567488.382	23.8%
Subpopulation Size		2387093.107	100.0%

Subpopulation: Ethnicity = 4

a. Dependent Variable
b. Reference Category

Table 14 showed the pseudo R^2 values for "other" participants. The variation ranged from 11% to 19%. For Nagelkerke, the R^2 was .198. In essence, 19% of the variation is explained between the independent and dependent variables.

Table 14

Pseudo R^2 *Values for Participants Identified as "Other"*

Cox and Snell	.149
Nagelkerke	.198
McFadden	.116

Subpopulation: Ethnicity = 4

Dependent Variable: Cognitive (reference category=1)
Model: (Intercept), Diabetes2, INDFMPIR, RIDAGEYR

In response to RQ2, Table 15 included data to answer the question whether race modified the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, and poverty–income ratio. With regard to effect modification, determined by stratified analysis, the effect modification for race and sex were studied separately. The evidence provided the statistical significance for the non-Hispanic White population, non-Hispanic Black population, Hispanic population, and Others. The effect modification was assesed to see how the exposure and outcome can differ according to the levels of a third variable, whis would be the effect modifier (Ness et al., 2006). In this case, the effect modifier has been identfied by race. For the non-Hispanic White population, there was 95% confidence that the lower confidence interval was at 1.283 and the upper confidence level was at 2.796. The true odds ratio was at 1.894.

Table 15

The Influence of Race on the Association Between Diabetes and Cognitive Dysfunction After Controlling for Age, Sex, and Poverty–Income-Ratio

Race	*OR*	95% CI	*p*-value
Non-Hispanic White	1.894	1.283-2.796	.003

Non-Hispanic Black	1.316	0.585-2.963	.482
Hispanic	1.273	0.496-3.268	.593
Other	2.644	1.028-6.798	.044

Because the true odds ratio was in between the lower and upper confidence levels (1.283 and 2.796), the *p*-value only confirmed its statistical significance, which was .003. In essence, the *p*-value was p<.01. Therefore, the results showed that this particular group played a vital role in modifying the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, and poverty–income ratio. Second, with the non-Hispanic Black population, there was 95% confidence that the lower confidence interval was at .585 and the upper confidence levels was at 2.963. The true odds ratio was at 1.316. Yes, the true odds ratio was in between the lower and upper confidence levels (.585 and 2.963), however, there was a wide variation between the two levels. The lower confidence level was below 1, while the upper confidence level was above 1. As a result, the *p*-value only confirmed of its statistical insignificance, which was .482. In essence, the *p*-value greater than .05. Therefore, the results demonstrated that this particular group did not play a vital role in modifying the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio.

Third, with the Hispanic population, there was 95% confidence that the lower confidence interval was at .496 and the upper confidence level was at 3.268. The true odds ratio was at 1.273. Although, the true odds ratio was in between the lower and upper confidence levels (.496 and 3.268), there was a wide variation between the two levels.

The lower confidence level was below 1, while the upper confidence level was above 1. As a result, the *p*-value only confirmed of its statistical insignificance, which was .593. In essence, the *p*-value was greater than .05. Therefore, the results show that this particular group did not play a vital role in modifying the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, poverty–income ratio. Last, with the "other"population, there was 95% confidence that the lower confidence interval was at 1.028 and the upper confidence level was at 6.798. The true odds ratio was at 2.644. Since the true odds ratio was in between the lower and upper confidence levels (1.283 and 2.796), the *p*-value only confirmed of its statistical significance, which was .044. In essence, the *p*-value was less than .05. Therefore, the results illustrate that this particular group played a vital role in modifying the effect of the association between diabetes and cognitive dysfunction after controlling for age, sex, and poverty–income ratio.

RQ3 asked does sex modify the effect of the association between diabetes and cognitive dsyfunction after controlling for age and poverty–income ratio. The null hypothesis stated that sex does not modify the effect of the association between diabetes and cognitive dsyfunction after controlling for age and poverty–income ratio. Meanwhile, the alternative hypothesis stated that sex does modify the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio.

Table 16 showed the categorical variable informaton pertaining to all of the male participants in the study (gender = 1). There was a total of 49.1% of men who displayed high cognitive dysfunction and 50.9% exhibiting low cognitive dysfunction. The

variable, diabetes2, indicated that 79.5% revealed no presence of diabetes. In contrast, 20.5% exhibited having diabetes. The subpopulation size was at 24,710,677. The mean age of the participants was 69 years of age, and ratio of family income to poverty of the participants was 3.2812.

Table 16

Categorical Variable Information for Male Participants

		Weighted Count	Weighted Percent
Cognitive[a]	0	12131909.244	49.1%
	1[b]	12578768.677	50.9%
Diabetes2	.00	19644218.770	79.5%
	1.00	5066459.151	20.5%
Subpopulation Size		24710677.921	100.0%

Subpopulation Gender = 1

a. Dependent Variable
b. Reference Category

Table 17 showed the pseudo R^2 values for male participants. The variation ranged from 18% to 30%. For Nagelkerke, the R^2 was .306. In essence, 30% of the variation is explained between the independent and dependent variables.

Table 17

Pseudo R^2 *Values for Male Participants*

Cox and Snell	.230
Nagelkerke	.306
McFadden	.188

Subpopulation Gender = 1

Dependent Variable: Cognitive (reference category = 1)
Model: (Intercept), Diabetes2, Poverty-income ratio, Age

Table 18 showed the categorical variable information for all of the participants identified as women (gender = 2). There was a total of 55.6% (15,961,219) of women who displayed high cognitive dysfunction and 44.4% (12,749,872) exhibiting low cognitive dyfunction. The variable, diabetes2, showed that 81.3% (23,345,301) revealed no presence of diabetes. However, 18.7% (5,365,790) displayed having diabetes. The subpopulation size was at 28,711,091. The mean age of the participants was 69 years of age, and ratio of family income to poverty of the participants was 2.8963.

Table 18

Categorical Variable Information for Female Participants

		Weighted Count	Weighted Percent
Cognitive[a]	0	15961219.511	55.6%
	1[b]	12749872.469	44.4%
Diabetes2	.00	23345301.343	81.3%
	1.00	5365790.638	18.7%
Subpopulation Size		28711091.980	100.0%

Subpopulation: Gender = 2

a. Dependent Variable
b. Reference Category

Table 19 displayed the pseudo R^2 values for female participants. The variation ranged from 14% to 24%. For Nagelkerke, the R^2 was .246. In essence, 24% of the variation was explained between the independent and dependent variables.

Table 19

Pseudo R^2 *Values for Female Participants*

Cox and Snell	.184
Nagelkerke	.246
McFadden	.148

Subpopulation Gender = 2

Dependent Variable: Cognitive (reference category=1)
Model: (Intercept), Diabetes2, Poverty-income ratio, Age

In response to RQ3, Table 20 provided data to answer the question of whether sex modified the effect of the associaton between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio. With regard to effect modification, evidence provided the statitical significance pertaining to the men and women who were involved with the study. For the male population, there was 95% confidence that the lower confidence interval was at 1.251 and the upper confidence interval was at 3.491. The true odds ratio was at 2.090. Because the true odds ratio was in between the lower and upper confidence intervals (1.251 and 3.491), the *p*-value only confirmed its statistical significance, which was .008.

Table 20

The Influence of Sex on the Association Between Diabetes and Cognitive Dysfunction After Controlling for Age and Poverty–Income Ratio

Gender	*OR*	95% CI	*p*-value
Men	2.090	1.251-3.491	.008
Women	1.682	0.984-2.875	.057

Therefore, the results indicated that the male population played a vital role in modifying the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio. In essence, the *p*-value was $p<.05$. With the female population, there was 95% confidence that the lower confidence interval was at .984 and the upper confidence level was at 2.875. The true odds ratio was at 1.682.

Although the odds ratio was in between the lower and upper confidence intervals (.984 and 2.875), there was a wide variation between the two intervals. The lower confidence level was below 1, while the upper confidence level was above 1. As a result, the *p*-value only confirmed of its statistical insignificance, which was .057. In essence, the *p*-value was p>.05. Therefore, the results demonstrate that the female population did not play a vital role in modifying the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio.

Summary

Chapter 4 focused on discussing the purpose of the study, data collection, and the results. The data collection delved into the time frame for collecting data, recruitment process, discreptancies with data, and the baseline and demographic characteristics of the population assessed. However, a susbstantial amount of the content was centered on the RQs asked in the study. I sought to answer three RQs: (a) Is there an association between cognitive dysfunction and diabetes after controlling for age, sex, poverty–income ratio, and ethnicity? (RQ1), (b) Does race modify the effect of the association between diabetes and cognitive dysfunction controlling for age, sex, poverty–income ratio? (RQ2), and (c) Does sex modify the effect of the association between diabetes and cognitive dysfunction and controlling for age and poverty–income ratio? (RQ3). In performing a logistic regression analysis (from secondary data obtained from NHANES), it was discovered that there was an association between cognitive dysfunction and diabetes when controlling for age, sex, ethnicity, and poverty–income ratio. In regard to RQ2, it was recognized that race modified the effect of the association between diabetes and cognitive

dysfunction after controlling for age, sex, and poverty–income ratio for the non-Hispanic Whites and ethnicity groups identified as “other”. On the contrary, this was not the case for the non-Hispanic Blacks or Hispanic population. With the third RQ, data concluded that sex does modify the effect of the association between diabetes and cognitive dsyfunction and diabetes after controlling for age and poverty–income ratio in men. However, this was not the case for women.

In Chapter 5, I will reintroduce the purpose of the study and its potential significance. I will further elaborate on the study findings in relation to previous literature, as reviewed in Chapter 2). The limitations to the study will be discussed. Recommmendations for future research will be noted.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

The purpose of the study was to evaluate if any association existed between self-reported diabetes and the risk of developing cognitive dysfunction in those living with type 2 diabetes. I used a quantitative research approach by applying the cross-sectional research design. The cross-sectional research design was used to assess the association between type 2 diabetes (independent variable) and cognitive dysfunction (dependent variable) without the use of manipulation. The covariates comprised of diabetes (type 2), gender (male or female), age (age of participants), ethnicity (racial identity), and cognitive dysfunction (i.e., inability to process speed, memory loss). A secondary data analysis was utilized. The data file was obtained from data collection by the National Center for Health Statistics. The data file from NHANES used for analysis was from the 2017–2020 pre-COVID-19 pandemic years. Logistic regression analysis was performed to determine the findings from the RQs developed. The study was conducted due to the gap in the literature previous research studies regarding the association between hemoglobin A1c levels and cognitive dysfunction.

Conducting a logistic regression from the NHANES data set prompted important results. Upon assessing the relationship between type 2 diabetes and cognitive dysfunction, it was discovered that there is an association between the independent and dependent variable. Yet, when incorporating other covariates and modifiers, the association was altered. The RQs did not only pose if there was an association between the independent and dependent variables, but also inquired if ethnicity and gender could

modify the association between the two key variables. After completing the logistic regression, results determined the significance, or impact the covariates had on the key variables.

Interpretation of the Findings

Peer-Reviewed Literature

The study findings provided evidence that an association existed between diabetes and cognitive dysfunction. Results have indicated that non-Hispanic White participants and participants acknowledging themselves as "others" modified the effect of the association between diabetes and cognitive dysfunction. However, there were no modifications denoted for the non-Hispanic Black and Hispanic populations. With regard to gender, study findings have also provided evidence of the male population modifying the effect of the association between diabetes and cognitive dysfunction. Yet, this was not demonstrated for the women.

Type 2 diabetes accounts for 95% of its dementia cases (Sho et al., 2021). Cognitive dysfunction, or cognitive decline is also recently being introduced as a diabetes-related complication (Umegaki, 2018). Previous literature indicated the impact glycemic control had on cognitive decline, or cognitive dysfunction (Biessles & Despa, 2018). The elevation of the hemoglobin A1c levels have been linked to diabetes-associated cognitive decline (Biessles & Despa, 2018; Mimenza-Alvarado et al., 2020). On the contrary, previous literature also denoted deficiency in the correlation between diabetes and cognitive dysfunction (Umegaki, 2018). There is ambiguity in determining if hemoglobin A1c levels has any connection in the development of cognitive dysfunction

(Biessels et al. 2018). However, literature review findings revealed that hyperglycemia and insulin resistance is the linkage between diabetes and cognitive dysfunction (Liu et al., 2020).

In comparison to the findings identified from previous peer-reviewed literature, the results from the logistic regression analysis indicated evidence of the connection between type 2 diabetes and cognitive dysfunction. The secondary data set obtained from NHANES confirmed previous literature findings. An association exists between diabetes and cognitive dysfunction, especially when incorporating confounders such as age, sex, ethnicity, and poverty–income ratio. The results also further expands in the knowledge of previous literature findings. These findings provide evidence on the impact diabetes and cognitive dysfunction has in relation to gender and various ethnic backgrounds. With regard to the expansion of the literature, it was evident that some races played a vital role in modifying the effect of the association between diabetes and cognitive dsyfunction after controlling for age, sex, and poverty–income ratio. The non-Hispanic White population and individuals identifying themselves as “other” played a role in modifying the effect of the association between diabetes and cognitive dysfunction. On the contrary, for the non-Hispanic Black and Hispanic population, this was not evident. Additionally, when it came to gender, the results provided evidence that the male population was responsible for altering the effect of the association between diabetes and cognitive dysfunction after controlling for age and poverty–income ratio. However, the association was not the same for women.

Theoretical Framework

The purpose of this study was to evaluate if self-reported health care diagnosed diabetes had any association in the risk of developing cognitive decline, or dementia in individuals living with type 2 diabetes. The results from previous literature indicated the connection between the two key variables were inconclusive. There was evidence of an association between the two variables, but there was also evidence indicating no such connection existed. This was the gap in the literature, which prompted the use of self-determination theory as its theoretical framework. The theory proposed three ways an individual is motivated. An individual is: a) intrinsically motivated (through autonomy), b) extrinsically motivated (from outside influences), or c) non-motivated (having a lack of motivation). The significance of the theory contributed to the development of the RQs posed. From the results, there was evidence that an association existed between cognitive dysfunction and diabetes. Results also showed that non-Hispanic White participants and participants identified as "others" altered the effect of the association between diabetes and cognitive dysfunction, while the non-Hispanic Black population and Hispanic population did not. Last, the results showed, with gender, that the male population modified the effect of the association between diabetes and cognitive dysfunction, however, the women did not. With regard to self-determination theory, it can be concluded that participants are motivated in some form with the management of their own diabetes, whether intrinsically, extrinsically, or a lack of thereof.

From previous research findings, it is evident there is an increased risk of cognitive dysfunction in individuals living with type 2 diabetes (Andersen et al., 2019).

Longitudinal studies have supported evidence of type 2 diabetes having direct correlation to an increased decline in executive function, processing speed, verbal fluency, and memory (Callisaya et al., 2018). Type 2 diabetes doubles the risk of dementia (Callisaya et al., 2018; Dyber et al., 2018). To be exact, results indicated a 2.5-fold increased risk in the chances of developing dementia (Liu et al., 2020). Previous literature stated that those living with type 2 diabetes for an extended time period, who have uncontrolled, or poor glycemic control (severe hypoglycemia and hyperglycemia), have pre-existing micro- or macrovascular complications, and high cholesterol are at an increased risk of developing dementia and the weakening of the cerebral function (Tumminia et al., 2018; Hsiao, 2019; Yu et al., 2020). Findings have reported a linkage between cognitive impairment and the development of dementia, which can also be developed from lipid disorders and poor diet control (Albai et al., 2019). Literature findings have indicated there are diabetes-related mechanisms which can further exacerbate cognitive functioning deterioration, such as insulin-resistant syndrome and disruption of insulin homeostasis of the brain (Albai et al. 2019).

With regard to previous research findings, insulin resistance and insulin inflammation play a vital role in the risk, or development of cognitive dysfunction. Insulin is significant for neuronal survival and brain function (Tumminia et al., 2018). Insulin is not only responsible for facilitating learning and memory, but neuronal survival as well (Tumminia et al., 2018). Insulin is significant in the activation of the dendritic spine and synapse formation, neuronal stem cell activation, neurite growth and repair, and neuroprotection (Tumminia et al., 2018). The pancreas is responsible for secreting

insulin, as insulin is a hormone that is produced by beta cells (Sousa et al., 2020). When insulin is released from the pancreas, it is transferred to the brain through a blood brain barrier using a receptor-mediated mechanism (Chatterjee et al., 2018; Sousa et al., 2020).

Amyloid beta oligomers are a cluster of strong neurotoxins which mediate inflammation (Sousa et al., 2020). Amyloid beta oligomers are linked to insulin resistance in the central nervous system, which has been identified as the reasoning for the obstruction of creating new memories (Sousa et al., 2020). The presence of a particular formation of the amyloid beta oligomers are linked to the malfunctioning of the insulin receptor substrates (Sousa et al., 2020). Insulin receptor substrates are responsible for memory and learning ability (Sousa et al., 2020). Therefore, if insulin resistance is continuously present in individuals living with type 2 diabetes, it will further induce the deterioration of insulin receptors, which can enhance the onset of a particular cognitive dysfunction, Alzheimer's disease (Sousa et al., 2020). Any adjustments to insulin signaling and metabolism in the central nervous system can immensely have an effect on the development of various brain disorders (Tumminia et al., 2018).

From previous research findings, it is apparent that hyperglycemia and insulin resistance (which can cause hyperglycemia) can have an adverse impact for those living with type 2 diabetes. This is where self-determination theory comes into effect. In managing diabetes, it all comes to the motivation. From the results of conducting a logistic regression analysis on a secondary data set, it is not only evident of the association between diabetes and cognitive dysfunction, but in relation to various ethnic backgrounds and gender, there maintains a level of motivation. In some form,

participants are either motivated to try to maintain normal hemoglobin A1c levels or diabetes diagnosis (to prevent the risk of cognitive dysfunction), or have a lack of, or no motivation, therefore permitting the likelihood of developing a form of cognitive dysfunction.

Limitations of the Study

NHANES conducted a comprehensive evaluation of the 2017–March 2020 pre-COVID-19 pandemic data file to test the reliability of the national estimates generated (NHANES, 2021). However, in conducting a multi-stage probability sampling design, there were some limitations. NHANES was constructed to create trustworthy, or reliable health statistics for the subdomains of the general population due to the health characteristics altering by age, race, sex, income, and geographical location (Akinbami et al., 2022). To attain the adequate sample size within the subdomains, oversampling had to be conducted, but at a relatively higher rate. When subdomains were combined for analysis, an expansive range of weights occurred. This prompted an increase in variance with the results, in which the end-goal of the sampling design is designated to decrease variance across all groups. (Akinbami et al., 2022).

Another limitation to the multi-stage probability sampling design was the ability to cover all of the participants. Nevertheless, this can be challenging. The process of conducting a multi-stage probability sampling design caused an omission of potential participants (relative to target population groups) from research study. For this case, there was ambiguity knowing if the potential participants omitted could have been a great

addition to the study. Also, with the multi-stage probability sampling design, the surveys were only administered amongst non-institutionalized individuals.

Last, a third limitation with the cross-sectional study was recall bias. Due to the exposure and outcome being measured at once, or at the same time, previous knowledge of a condition could greatly impact the ascertainment of an exposure or the outcome (X. Wang, & Cheng, 2020). Participants can recall information regarding their outcomes differently depending upon the exposure (X. Wang, & Cheng, 2020). For instance, when it comes to identifying some form of cognitive dysfunction, the study can make it permissible for participants to not be exact on the development of their own cognitive decline. When the participants are being asked a series of questions at one time, this does not allow much validation of the answers provided. Therefore, permitting the participants to formulate their own timeline of when they have experienced some form of cognitive dysfunction, when the timeline could be inaccurate.

Recommendations

The study results was an extension of the same sentiments from previous literature findings regarding the association between diabetes and cognitive dysfunction. It is recognized there is an association between the two key variables. Also, there is evidence of race and gender having an impact on modifying the effect of the association between diabetes and cognitive dysfunction. With regard to this, and for future research purposes, it can be beneficial to assess the influence race has on the association between diabetes and cognitive dysfunction. There can be studies that identify if there are certain thresholds (within the hemoglobin A1c levels) that varies by race, which will make them

more susceptible to the risk of developing cognitive impairment. Previous literature discussed how various mechanisms can exacerbate cognitive development. The mechanisms include (a) insulin-resistant syndrome, (b) hyperglycemia, and (c) interruption of insulin homeostasis in the brain (Albai et al., 2019). Future studies could also delve into how these mechanisms factor into various ethnic groups. The findings could provide evidence of a specific mechanism being more prevalent amongst a particular race in comparison to others. With regard to gender, again, further research can be conducted to evaluate if there are certain thresholds (within the hemoglobin A1c levels) that will increase the onset of cognitive dysfunction.

For future studies, the use of multi-stage probability sampling is substantially effective for recruiting participants. This type of sampling is significant because it allows researchers to create, or make rational generalizations for a smaller group of participants representing a larger population. The use of this particular type of sampling is intricate, yet the process it takes to partition larger populations into significantly smaller groups certifies for data collections to be more effective and even economical. On the contrary, in utilizing multi-stage probability sampling, the outcomes are not always 100% precise because it does not cover all of the surveyed participants. Due to this, there is ambiguity if the participants, or demographics eliminated from the study, is effective. In order to remedy this situation, the use of sampling weights can be of assistance.

Implications

The impact for social change can be highly effective at the individual, family, organizational, and societal level. The study findings extend on the knowledge from

previous literature regarding the association between cognitive dysfunction and diabetes. The results can be presented and/or discussed at various professional conferences. Subsequently, this can lead to open discussions at numerous medical facilities, which can therefore prompt medical professionals to convey the findings to their patients. From this standpoint, patients are either going to make a conscious effort in minimizing their chances of becoming at risk for the development of cognitive dysfunction, or a lack thereof (with regard to maintaining normal hemoglobin A1c levels). Family members can be made cognizant of the findings as well (during routine doctor visits), especially if they are the main caretaker. Not only can the study results promote urgency in medical professionals to inform their patients of continuous findings, but also incite medical professionals and researchers to discover if there are certain mechanisms that can trigger the onset of cognitive dysfunction. If there are certain mechanisms that can be found more prevalent with one race, in comparison to another, then this can be made known to patients as well. Again, this is impactful not only for the individual, but family members, medical professionals' organizations, and the society as a whole.

The results from the study will not only be an addition to previous literature, but can inspire other health care professionals to become more cognizant of the association between diabetes and cognitive dysfunction, and to educate patients on such findings. This can result promote positive social change. The more research is being conducted on this topic, the more inclined health care professionals will be in discussing the association between the two key variables during conference meetings, social gatherings, and to their patients. In return, when patients are aware of the connection between the two variables,

it will make some, if not most, more likely to want to try to maintain better control of their diabetes.

The implications from a methodological and theoretical perspective are important for various reasons. The study evaluated if self-reported diabetes had any association with the risk of developing cognitive dysfunction. Self-determination theory was incorporated to help formulate the RQs answering if there is a relationship between the two variables. A multi-stage probability sampling design was applied to select participants. In order to randomly select a smaller group of participants from a larger population, this was the best sampling design to use. For future research purposes, studies can utilize self-determination theory to identify how participants are motivated in managing their own diabetes. Perhaps, a longitudinal study could be developed where the participants are followed for an extended period of time to monitor the degree of the motivation, the hemoglobin A1c levels, and the developmental stages of cognitive dysfunction, if any.

In regard to practice recommendations, it would be vital for patients to continue with their scheduled doctor appointments in staying well-informed about their health. As a form of good practice, it would also be recommended for patients to ensure they monitor their diet, blood glucose levels, and to take the necessary medications prescribed by their doctor. Contrarily, for the health care professionals, it would be advised for them to actively stay abreast with research findings, continue communicating those findings to patients, and to provide the resources that would be beneficial in assisting patients living with the chronic illness.

Conclusion

In conclusion, the overall purpose of the study was to gain further insight into what seemed to be pertinent, yet inconclusive to previous research studies. Ambiguity surrounded claims of the association between diabetes and cognitive dysfunction. Previous studies have alluded to an existence between the two variables; however, evidence also indicated no existence of causation. From the study findings, it is proven an association is established between diabetes and cognitive dysfunction. The diagnosis of diabetes played an integral role in the risk of developing cognitive dysfunction. Acquiring this knowledge is extremely insightful because not only can medical professionals educate their patients regarding this matter, but it can also help significantly decrease the likelihood of being at risk of developing cognitive dysfunction. This is why it is imperative to maintain a normal glucose level. If the blood glucose levels are consistently high, it is reflected when the hemoglobin A1c level is revealed. Individuals living with diabetes are already at a disadvantage with an increased risk of developing cognitive dysfunctions, but to consistently not maintain a normal glucose level can further exacerbate the likelihood of developing some type of cognitive development, or dysfunction.

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Appendix: Race and Hispanic-Origin and Income Group Sampling Fractions Used to Calculate Sample Sizes in Primary Data

Race and Hispanic origin and income group	Sampling fraction values	
	2015–2018	2019–2022
Hispanic	0.000206	0.000266
Non-Hispanic Black	0.000287	0.000382
Non-Hispanic, non-Black Asian	0.000453	0.000572
Non-Hispanic, low-income, White and other races and ethnicities	0.000184	0.000182
Non-Hispanic, non-low-income, White and other races and ethnicities	0.000086	0.000147

Note. Data are from the National Health and Nutrition Examination Surveys, 2013–2014. The data sets excluded non-Hispanic Black and non-Hispanic Asian people. Due to this, sampling weights had to be applied. Republished from *National Health and Nutrition Examination Survey, 2013-2014 File: Sample Design, Estimation, and Analytic Guidelines* (Vital and Health Statistics Series 2, No. 190), by L. J. Akinbami, T.-C. Chen, O. Davy, C. L. Ogden, S. Fink, J. Clark, M. K. Riddles, and L. K. Mohadjer, 2022, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics (https://doi.org/10.15620/cdc:115434). In the public domain.