Differences in Diet Quality and Concurrent Chronic Diseases by Level of Glycemic Control in US Adults

Thesis

Presented in Partial Fulfillment of the Requirements for the Degree Master of Science in the Graduate School of The Ohio State University

By

Stephanie Marissa Fanelli, BS

Graduate Program in Allied Medicine

The Ohio State University

2019

Thesis Committee

Christopher A. Taylor, PhD, RDN, LD, FAND, Adviser

Jessica L. Krok-Schoen, PhD, MA

Randell K. Wexler, MD, MPH, FAAFP

Copyrighted by

Stephanie Marissa Fanelli

2019

Abstract

Background: Along with rising rates of diabetes come increased prevalence of common comorbidities: obesity, hyperlipidemia, and hypertension. Diet is a key element in the prevention and treatment of such diseases. Therefore, the aim of this study was to identify differences in diet quality by degree of glycemic control and chronic diseases in a nationally representative sample of adults.

Methods: Dietary, anthropometric, and laboratory data from 23,708 adults, aged 31 years and older, were gathered from the 2005-2016 NHANES. Glycated hemoglobin (%A1c) classified participants by level of glycemic control: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); and poorly controlled diabetes (≥7%). Dietary data gathered from 24-hour recalls were used to calculate diet quality (HEI-2015) by glycemic level. Chronic disease prevalence was evaluated for overweight or obesity (BMI ≥25), hyperlipidemia (total cholesterol ≥200 mg/dL), and hypertension (BP ≥120/80 mm Hg).

Results: Rates of hypertension and obesity were highest in adults with diabetes, where more than 50% presented with hypertension and over 90% with overweight or obesity. Prevalence of hyperlipidemia was greatest in the prediabetes group. Adults with diabetes had significantly poorer diet quality than those with normal glycemia, and overall diet quality was lower in the presence of hypertension and overweight or obesity.

Conclusions: Adults with diabetes had higher rates of chronic diseases and poorer diet quality than adults with normal glycemia. Furthermore, diet quality was poorer when another chronic disease was present. These findings support the need for nutrition therapy to target overall diet quality in the population with diabetes.

Acknowledgments

I would like to thank my advisor, Dr. Christopher Taylor, for his mentorship throughout the beginning of my research career, his integral role in answering these research questions, and his continual patience while training me in secondary data analysis. His efforts extend far beyond the walls of this thesis and are deeply appreciated. Additionally, I would like to recognize committee members Dr. Jessica Krok-Schoen and Dr. Randy Wexler for their vital contributions, perspectives, and constructive feedback in this thesis. The committee is credited with the success of this work. Of course, I sincerely thank my family, namely my parents, for their unconditional support and encouragement throughout my educational career and far beyond. Lastly, I acknowledge Dr. Owen Kelly, Dr. Steven Hertzler, and Dr. Satya Jonnalagadda at Abbott Nutrition for providing funding for this research.

Vita

January 18, 1994 .... Born; Pittsfield, MA

2012 ....Pittsfield High School

2016 .... B.S. in Dietetics, Nutrition, and Food Sciences; University of Vermont

2016 to 2017 ....Researcher, Food and Brand Lab; Cornell University

2018 to Present.................................. Delegate, Council of Graduate Students; The Ohio State University

2019 .... M.S. in Allied Medicine; The Ohio State University

2019 .... Graduate Research Associate; Division of Medical Dietetics; The Ohio State University

Publications

Fanelli S, Kelly O, Luo M, Krok-Schoen J, Taylor C. Prevalence of Physical Function Limitations by Protein Intake Levels in US Older Adults. *Journal of the Academy of Nutrition and Dietetics.* 2018;118(10):A132.

Fanelli S, Kelly O, Luo M, Krok-Schoen J, Taylor C**.** Differences in Micronutrient Intakes by Levels of Glycemic Control in US Adults. *Journal of the Academy of Nutrition and Dietetics.* 2018;118(10):A133.

Fanelli RD, Fanelli SM. Intraoperative endoscopy: An important skill for general surgeons. *Techniques in Gastrointestinal Endoscopy*. 2018;20:166-171. doi:10.1016/j.tgie.2018.09.003.

Fields of Study

Major Field: Allied Medicine

Specialization: Medical Dietetics

Table of Contents

List of Tables

List of Figures

List of Definitions

Cardiovascular disease	A group of disorders of the blood vessels and heart
Diabetes mellitus	Chronic diseases characterized by hyperglycemia
Diet quality	Comprehensive adequacy of one's diet as a whole
Dietary patterns	Eating routines of foods and food components
Hyperlipidemia	Abnormally elevated levels of blood lipids
Hyperglycemia	High blood glucose
Hypertension	High blood pressure
Impaired fasting glucose	Higher than normal fasting glucose levels yet not as high as in diabetes; typically seen in prediabetes
Impaired glucose tolerance	Partial inability to regulate blood glucose yet not as advanced as in diabetes; typically seen in prediabetes
Insulin resistance	Cells fail to respond to the presence of insulin, leading to chronic hyperglycemia
Insulin sensitivity	Cells successfully respond to the presence of insulin
Metabolic syndrome	A cluster of conditions that increase risk for chronic diseases such as diabetes and cardiovascular disease
NHANES	A continuous series of cross-sectional studies examining health trends across America

List of Abbreviations

ACC/AHA	American College of Cardiology/American Heart Association
ADA	American Diabetes Association
AMPM	Automated Multiple Pass Method
BMI	Body mass index
CDC	Centers for Disease Control
CVD	Cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DGA	Dietary Guidelines for Americans
FNDDS	Food and Nutrition Database for Dietary Studies
FPED	Food Patterns Equivalents Database
A1c	Glycated hemoglobin
HDL-C	High-density lipoprotein cholesterol
HEI	Healthy Eating Index
HLD	Hyperlipidemia
HTN	Hypertension
IFG	Impaired fasting glucose
IGT	Impaired glucose tolerance
LDL-C	Low-density lipoprotein cholesterol
MEC	Mobile examination center
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
USDA	United States Department of Agriculture

Chapter 1. Introduction

Diabetes Mellitus Background

Diabetes affects more than 30 million adults in the US alone and prevalence is expected to rise as a result of increasing rates of obesity, physical inactivity, urbanization, aging, and population growth[1–3]. Along with rising rates of diabetes, long-term complications and comorbidities, such as microvascular disease and macrovascular conditions, will likely increase as well[4]. Contributing to the development of and risk for diabetes are both nonmodifiable and lifestyle factors. Among the modifiable risk factors are dietary patterns, which have been a focus of diabetes prevention research[4,5].

Assessing dietary patterns among those with already diagnosed diabetes is an additional area of research and it has been discovered that individuals with diabetes tend not to meet recommendations for fruit or vegetable[6], fiber[7–9], added sugar[10], saturated fat[6–9,11,12], and sodium intakes[7,8,13]. Typically, failure to meet the recommendations for one dietary component is accompanied by that of additional dietary components, yielding inadequate quality of overall diet[10,14]. However, insufficient literature exists evaluating overall diet quality in Americans with diabetes nor diet quality stratified by levels of glycemic control. Inadequate diet quality may contribute to the risk for additional chronic diseases, such as hypertension and hyperlipidemia, which serve as risk factors for the development of comorbidities, including cardiovascular disease[15]. Not only would the development of comorbidities contribute to increased mortality rates among those with diabetes, national and personal healthcare costs would rise, as well[16,17].

In an effort to fill the gaps in current literature, the aim of this study was to assess the differences in diet quality and markers of chronic diseases by level of glycemic control in the US adult population. This information would aid in the development and improvement of public health initiatives aimed at improving diet quality and reducing the development of subsequent chronic conditions in adults with diabetes.

Research Questions

1. How does diet quality in adults differ by glycemic level (normal glycemia: A1c <5.7%; prediabetes: A1c 5.7-6.4%; controlled diabetes: A1c 6.5-6.9%; and poorly controlled diabetes: A1c ≥7%)?
2. Do adults with diabetes have more markers of chronic diseases (overweight or obesity, hyperlipidemia, and hypertension) than those without diabetes?
3. Is diet quality poorer in adults with diabetes and concurrent chronic diseases?

Chapter 2. Review of Literature

Background of Diabetes

Diabetes mellitus refers to a group of chronic, progressive diseases that are characterized by hyperglycemia and altered metabolism due to decreased insulin secretion and sensitivity[3,4,16]. The three primary subtypes of diabetes mellitus include type 1 diabetes, type 2 diabetes, and gestational diabetes[3,18]. Type 2 diabetes is the most common form of all diabetes cases and will be the focus of this thesis[1]. The remainder of this thesis will refer to type 2 diabetes as diabetes.

Past trends show a rising rate of diabetes in both developed and developing countries[3]. In 2000, more than 17 million adults (8% of the population), ages 20 through 79, in the United States had diabetes[19]. In 2015, an estimated 30.2 million US adults ages 18 and older (12.2% of the population) had either diagnosed or undiagnosed diabetes and 84.1 million US adults (33.9% of the population) had prediabetes[1,2]. By 2030, it is projected that 30.3 million people in the US will have diabetes and by 2045 approximately 35.6 million will have the chronic disease[2,16]. Considering type 2 diabetes accounts for 90% to 95% of all diabetes cases, it can be assumed that case estimations closely represent type 2 diabetes rates specifically[1].

Diabetes was the sixth leading cause of disability and the seventh leading cause of death in the US in 2015[1,20]. In 2017, approximately 4 million people died from diabetes around the world[16]. As the prevalence of obesity rises, the prevalence of diabetes is expected to rise coincidingly[3]. The increasing incidence of this chronic disease threatens to further increase rates of common complications. Diabetes oftentimes leads to macrovascular complications such as hypertension (HTN), hyperlipidemia (HLD), coronary artery disease, stroke, myocardial infarctions, cerebral vascular disease, and peripheral vascular disease, as well as microvascular conditions such as retinopathy, nephropathy, and neuropathy[4]. Macrovascular disease is the primary cause of morbidity and mortality in those with diabetes[3]. Additionally, compared to those without diabetes, patients with diabetes have a 15% higher risk of all-cause mortality[20].

Managing diabetes requires considerable medical supplies, medications, and laboratory testing, resulting in an economic burden for both the individual and the nation. In the US, for those of ages 20 through 79, the total healthcare expenditure on diabetes was $348 billion in 2017, with $11,638 as the mean healthcare expenditure per person with diabetes[16]. Costs are expected to surpass $500 billion by 2025 in the US alone[17]. These projections assume the mean expenditure per person and diabetes prevalence rate remain stable with only demographic changes, yielding modest cost predictions that will likely be exceeded[16].

Etiology of Diabetes

Diabetes is a progressive disease[16]. Before disease onset, patients develop impaired glucose tolerance (IGT) in prediabetes, during which pancreatic islet beta-cells fail due to the presence of insulin resistance, likely caused by obesity or excess adipocytes[21,22]. Prior to diabetes diagnosis, up to 50% of pancreatic beta-cells may have already failed[20]. This pancreatic beta-cell decline contributes to the decrease in metabolic control and the increase in chronic hyperglycemia during disease progression[16,21]. Mild to moderate symptoms may manifest during prediabetes, yet they typically present themselves after disease onset. Typical symptoms of diabetes include increased thirst, frequent urination, tiredness, blurred vision, slow-healing wounds, recurrent infections, and tingling sensations or numbness in hands and feet[16].

In metabolically healthy individuals, high blood glucose levels lead to the release of insulin from pancreatic beta-cells, triggering uptake of amino acids, fatty acids, and glucose from the blood into tissues[15,23]. In addition, insulin travels to the liver to promote the conversion of glucose into glycogen, the storage form of glucose, for later energy use[24,25]. These mechanisms help in lowering blood glucose levels to return to homeostasis[24]. Alternatively, when blood glucose concentrations are low, cells in the pancreas release glucagon, which signals for the breakdown of glycogen into glucose within the liver[22,24]. This glucose is released into the bloodstream, raising blood glucose levels[24]. Communication between insulin-sensitive peripheral tissues and pancreatic beta-cells signal for the release of these pancreatic hormones, insulin or glucagon, when blood glucose levels disrupt homeostasis[23].

Diabetes is characterized by insulin resistance in target organs, hyperinsulinemia, and insulin deficiency due to pancreatic beta-cell failure[20]. Insulin resistance, present prior to hyperglycemia, refers to when the cells in peripheral tissues and target organs do not respond to the presence of insulin and subsequently prevent the uptake of glucose into cells from the bloodsteam[15,16]. In the presence of insulin resistance, signals for further insulin secretion are sent to pancreatic beta-cells in an effort to maintain plasma glucose homeostasis, leading to hyperinsulinemia[23]. Over time, this repeated signaling can lead to inadequate insulin production from pancreatic beta-cells and, in combination with insulin resistance, contributes to chronic hyperglycemia[16,23]. The degree of beta-cell failure helps to determine the degree of hyperglycemia[23]. However, hyperglycemia can further worsen pancreatic beta-cell function, leading to continued hyperglycemia and fueling a vicious cycle of worsened metabolic outcomes and disease progression[15].

Chronic hyperglycemia may play a role in damaging target cells by impairing their ability to limit glucose uptake from the blood[26]. This leads to intracellular hyperglycemia and promotes mitochondrial synthesis of reactive oxygen species, which activate multiple metabolic mechanisms identified to play a role in the pathogenesis of microvascular and macrovascular disease[26,27].

The American Diabetes Association (ADA) is responsible for setting diagnostic criteria for normal glycemia, prediabetes, controlled diabetes, and poorly controlled diabetes[18]. To diagnose diabetes, at least one of the criteria in Table 1 must be met[18]. The following glycated hemoglobin (A1c) increments help to identify diabetes status: less than 5.7% is normal glycemia, 5.7 to 6.4% is prediabetes, 6.5 to 6.9% is controlled diabetes, and 7% or greater is poorly controlled diabetes[18]. This analysis considers an A1c of 7% or greater to be classified as poorly controlled diabetes because levels less than 7% are typically recommended for proper blood glucose management[18]. Therefore, A1c levels greater than this recommendation indicate improper blood glucose management.

Must meet at least 1 of the following criteria:
Fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L)
2-hour plasma glucose ≥ 200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test using 75 grams of anhydrous glucose dissolved in water
Glycated hemoglobin (A1c) ≥ 6.5% (48 mmol/mol)
A random plasma glucose ≥ 200 mg/dL (11.1 mmol/L) in patients with symptoms of hyperglycemia

Table 1. American Diabetes Association Criteria for Diabetes Diagnosis[18]

Risk Factors for Diabetes

Common risk factors for diabetes include both nonmodifiable and modifiable factors that may contribute to disease development and progression[5]. Some factors highlighted below include genetics, demographics, weight status, physical inactivity, metabolic characteristics, smoking, sleep deprivation, and dietary patterns[4].

Individuals with a family history in a first degree relative have a 2- to 3-fold increased risk of developing diabetes, and a 5- to 6-fold risk in those with both a maternal and paternal history of diabetes[28,29]. This increased risk is thought to be due to environmental as well as genetic factors, such as diabetes-associated loci[4,30]. More than 75 loci have been identified in genome-wide association studies with the majority of the loci associated with impaired beta-cell function as opposed to impaired insulin sensitivity[4,30]. Further research is needed to identify additional variants that account for the majority of genetic susceptibility[30].

Additional nonmodifiable risk factors for diabetes include age and sex[3]. The majority of people with diabetes in the US are greater than 45 years of age[1]. Biological changes manifest with age due to a decline in beta-cell function and insulin secretion[17]. Additionally, diabetes distribution varies across sexes, and some estimates assume higher incidence in men and others in women[1,31–33]. Women with polycystic ovarian syndrome have an increased risk for diabetes[34].

Data show that the risk for developing diabetes is higher for Asians, Hispanics, and African Americans than non-Hispanic whites[35,36]. Differences in risk for diabetes among ethnicities are most likely due to both biological and behavioral factors, including genetic predispositions, age, psychosocial factors, socioeconomic status, and cultural lifestyle habits[37].

The term diabesity refers to the commonly simultaneous presence of diabetes and obesity[3]. The risk for diabetes, as well as IGT, increases with higher body weight. Increase in body mass index (BMI) has been found to be the most important factor examined, compared to age and race or ethnicity, to predict diabetes, accounting for 50% of the increase in diabetes prevalence in men and 100% in women[38]. Additionally, central or abdominal obesity is linked to the highest incidence of diabetes and the greatest degree of insulin resistance[39,40].

A sedentary lifestyle reduces physical activity and therefore energy expenditure, and likely will contribute to weight gain and the risk for diabetes[39]. However, it has been shown that physical inactivity, even in the absence of weight gain, still increases risk for diabetes[41]. On the other hand, a study showed that regular physical activity, including both aerobic and strength-training, improves blood glucose control, lipid profiles, and blood pressure[21]. A previous intervention study showed that risk for diabetes may be lessened by as much as 60% with combined weight loss and physical activity[42].

Insulin resistance is one of the best metabolic predictors of diabetes[15]. Insulin resistance itself is characterized by HTN and HLD[21]. Additional metabolic characteristics that increase risk for diabetes include gestational diabetes and any criteria for prediabetes, such as IGT, impaired fasting glucose (IFG), and metabolic syndrome[3,43]. These metabolic factors seen in prediabetes are associated with a 5-fold increase in developing diabetes in the future and about 25% of patients with either IFG or IGT will develop diabetes within 3 to 5 years after onset[21,43].

Modifiable lifestyle and behavioral risk factors for diabetes include smoking, sleep deprivation, and dietary patterns. It is hypothesized that smoking impairs insulin sensitivity and is linked to greater abdominal fat and waist-to-hip ratio[39]. Data have shown that risk for diabetes increases along with the number of daily cigarettes smoked per day and that diabetes risk may remain heightened for ten years following smoking cessation[40]. Additionally, sleep deprivation is suspected to augment insulin resistance, HTN, hyperglycemia, and HLD[21]. These effects may contribute to the development and progression of diabetes. A U-shaped relationship has been identified between the hours of sleep and risk for diabetes[44]. The lowest risk for diabetes was seen at 7 to 8 hours of sleep per day[44].

Lastly, assessing dietary patterns is preferred over single food groups due to the comprehensive nature of eating a variety of foods in any given day[45,46]. There is a paucity of literature aimed at identifying associations between dietary patterns and diabetes prevention[47]. Diets high in refined grains, red meat, processed meats, and sugar sweetened beverages are associated with a higher risk for diabetes[48,49]. On the contrary, dietary patterns that are richer in fruits, vegetables, nuts, legumes, and whole grains are associated with diabetes prevention when compared to the former dietary pattern[48–50]. While dietary patterns examine habitual food intakes, diet quality evaluates overall nutritional adequacy.

Diet Quality in Individuals with Diabetes

Diet quality is measured by indices which compare how well an individual's eating patterns align with the recommendations[46,51]. For instance, the Healthy Eating Index (HEI)-2015 assesses the compliance of dietary patterns with the evidence-based 2015-2020 Dietary Guidelines for Americans (DGA)[46,52,53]. Higher HEI-2015 scores signify greater adherence to the 2015-2020 DGA and the maximum HEI score of 100 represents perfect adherence to its associated DGA. In general, HEI scores greater than 80 indicate good dietary habits, whereas scores of less than 51 are considered to be poor[54,55]. Scores between those cutoffs are thought to need improvement[55].

The average Total HEI-2010 score in US adults is 58 for those of ages 18 to 64 years, and the average score is 65.5 for those older than 64 years of age[56]. These scores signify that the average US adult's diet quality needs improvement. Although the overall diet quality of the US population has been examined previously[55,57–60], there is a lack of literature evaluating the impact of diet quality in the adult population with diabetes in the US[6]. Literature assessing diet quality by degree of glycemic control is lacking, as well. Among the limited existing literature are inconsistencies, as one study identified higher HEI scores in those with diabetes versus in those without diabetes in their sample, yet the mean HEI-2010 score for the diabetes group (57.7) was lower than that of the general US adult population in another study[56,61]. Although more research is needed to assess diet quality in individuals with diabetes, there is some literature available that assessed specific dietary components of eating patterns in this target population[6–8,11,12,62–64].

The adult population with diabetes has previously been found to consume fewer than 5 servings of fruits and vegetables combined per day, indicating that the recommended intake (1.5 to 2 cups of fruits and 2 to 3 cups of vegetables per day[65]) was not met[6]. Fruits and vegetables contribute not only micronutrients yet also dietary fiber, a type of complex carbohydrate. Although data suggest an increase in total carbohydrate intake among American adults with diabetes from 1988 to 2004, there was no increase in fiber intake. This indicates a higher intake of simple carbohydrates, as found in refined grains and added sugars, as opposed to complex carbohydrates, as found in fruits and vegetables[8]. Additionally, a significant reduction of fiber intake in Americans with diabetes from 1988 to 2012 was identified, and average intakes did not meet the fiber recommendations (14 grams per 1000 calories)[7,9].

In 2013 to 2014, nearly 56% of the US adult population, not limited to those with diabetes, had an added sugar intake that surpassed the recommendation set by the 2015-2020 DGA (less than 10% of daily calories from added sugars[66])[14,63]. In fact, added sugar intakes exceeding recommendations were identified in more than 71% of people evaluated in a 2005 to 2010 NHANES study[10]. One study has indicated agreeably high added sugar intake in the population with diabetes[12], whereas others have hypothesized lower intakes due to diabetes-related nutrition education on sugar's effect on blood glucose control[61,67].

Literature also suggests that adults with diabetes surpass the daily recommended limit of saturated fat in the 2015-2020 DGA (less than 10% of daily calories from saturated fat[66])[6–9,11,12]. This increased saturated fat intake may be due to the combination of high red meat and processed meat intake as well as added fats[11]. Saturated fat intakes between 1988 and 2012 remained relatively stable, yet consistently exceeded recommendations[7,8].

On average, Americans consume 3,436 mg per day of sodium whereas Americans with diabetes have been found to consume an average of 3,214 mg or greater of sodium per day[8,13]. These intake levels surpass the 2015-2020 DGA recommendation (less than 2,300 mg per day of sodium[66]) as well as the American Heart Association's limit of 1,500 mg per day of sodium[13]. A trends study identified increases in sodium intake between 1988 and 2012 in the US population with diabetes as well as consistent insufficient potassium intakes[7]. These high sodium and low potassium intakes are of concern to the population with diabetes, in particular, due to increased risk for HTN and cardiovascular disease (CVD)[13]. Overall, noncompliance to one subcomponent of the DGA is typically linked to noncompliance of additional subcomponents, leading to overall poor dietary patterns that may impact the development of chronic diseases[10,14].

Long-term Complications of Diabetes and Chronic Diseases

These trends in dietary patterns identified in the adult population with diabetes may contribute to the development of additional chronic diseases as a result of impaired glucose and lipid metabolism in the presence of diabetes[68]. Diabetes is oftentimes accompanied by metabolic outcomes that serve as risk factors for the development of macrovascular disease, such as HTN, HLD, and CVD[15]. The increased prevalence of diabetes has amplified the amount of CVD attributable to diabetes over the last 60 years[21,69]. It is estimated that 60% of people with diabetes have HTN, and the combined presence of HTN and diabetes increases risk for CVD by 4-fold[13,70]. Managing blood pressure and lipid profiles is essential to the prevention of macrovascular disease, just as controlling blood glucose is vital to prevent microvascular complications[21].

Dietary contributors to the development of HTN include excess sodium intake and insufficient potassium intake[13]. As evidenced, adults with diabetes exceed sodium recommendations. Therefore, it may be hypothesized that poorer diet quality in those with diabetes is associated with a higher incidence of HTN and a greater subsequent risk for CVD.

HLD consists of high triglycerides, low high-density lipoprotein cholesterol (HDL-C), and increased low-density lipoprotein cholesterol (LDL-C) levels[68,71]. Epidemiology data suggest that high saturated fat intakes negatively impact metabolic profiles, such as increasing LDL-C, triglycerides, A1c, and inflammatory biomarkers in those with diabetes[72,73]. On the contrary, adherence to the saturated fat intake recommendations has been associated with significantly lower LDL-C and BMI in adults with diabetes[12].

Failure to adhere to added sugar recommendations is associated with poorer lipid profiles and increased inflammatory markers than lower added sugar intake in individuals with diabetes[72]. Adherence to the added sugars intake recommendation is significantly associated with lower triglycerides and higher HDL-C in adults with diabetes, independent of BMI[12]. Epidemiologic data demonstrate that people whose added sugar consumption exceeds the recommendations are at an increased risk for obesity, HLD, HTN, and CVD[10]. This CVD risk remains heightened even after adjusting for blood pressure and total serum cholesterol[10]. Additionally, fiber intake of 15 grams or greater per 1,000 calories has been associated with improved plasma lipid profiles, decreased A1c, and lower BMI compared to those of lower fiber intake in the adult population with diabetes[12,72]. However, as evidenced, fiber recommendations in individuals with diabetes are not being met[7–9]

Diet quality metrics have been linked to chronic conditions and mortality. High HEI scores are found to be inversely associated with risk of all-cause mortality, CVD, and cancer for men and women without diabetes[45,63,74,75]. Assessing specific components of dietary patterns allows for more targeted associations between diet and outcomes yet evaluating overall diet quality would produce more comprehensive findings for the adult population with diabetes.

Conclusions

The nationwide prevalence and progressive nature of diabetes make it a focal point in research. Considering diabetes is heavily influenced by lifestyle factors, future research should aim to fill the gaps in current diet-related diabetes literature. Preexisting literature demonstrates that Americans with diabetes do not meet the recommendations for specific components of dietary patterns, alike that of the American population without diabetes, yet the impact of diet quality in the various stages of disease progression has seldom been explored. Assessing the diet quality of Americans by diabetes status would assist in the understanding of how diet impacts disease progression as well as allow for the improvement of nutrition interventions. In turn, this may help to reduce the progression of the disease and the development of additional chronic conditions.

Chapter 3. Methods

Overview of Study

The data in this cross-sectional analysis were taken from the 2005 to 2016 NHANES and included adults aged 31 years and older at the time of data collection. Data were used to assess differences in diet quality (HEI-2015) as well as markers (BMI, blood pressure, and total cholesterol) indicative of chronic conditions (obesity, HTN, and HLD) stratified by level of glycemic control. Glycemic control was organized into groups by glycated hemoglobin (A1c) values: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); and poorly controlled diabetes (≥7%). Laboratory values were measured from blood samples taken during the in-person exam and dietary intakes were collected during the dietary interview of the NHANES process. Estimations of nutrient intakes were made using the Food and Nutrition Database for Dietary Studies. Obtaining Institutional Review Board approval was not necessary for this study, as all data were taken from publicly available sources containing de-identified information.

Research Questions

1. How does diet quality in adults differ by glycemic level (normal glycemia: A1c <5.7%; prediabetes: A1c 5.7-6.4%; controlled diabetes: A1c 6.5-6.9%; and poorly controlled diabetes: A1c ≥7%)?
2. Do adults with diabetes have more markers of chronic diseases (overweight or obesity, hyperlipidemia, and hypertension) than those without diabetes?
3. Is diet quality poorer in adults with diabetes and concurrent chronic diseases?

Overview of NHANES

The National Health and Nutrition Examination Survey (NHANES) is a program facilitated by the Centers for Disease Control (CDC) and Prevention's National Center for Health Statistics (NCHS)[76]. It first began in 1971 as three individual installments yet transitioned to a continuous program in 1999 that examines a broader range of health topics annually[77]. Types of data collected include demographic, socioeconomic, dietary, and other health-related information gathered through interviews, as well as medical, dental, physiological assessments and laboratory tests[76]. The NCHS Research Ethics Review Board is responsible for assessing and granting permission for the NHANES to be conducted[78]. The aim of NHANES is to provide information on trends in the health status of individuals in the US that may be used for health research and initiatives[76,77].

Data Collection

Participants

In preparation of conducting the NHANES, 15 distinct sections of the US were identified and 1 county from each section was randomly selected for participation[79]. About 20 to 24 smaller groups within each county were then chosen for continuation in the random sampling process. From there, about 30 households from within the selected smaller groups were visited by NHANES interviewers and demographic information was collected. Lastly, an algorithm was used to determine which members of the household were eligible for inclusion in the survey[79]. This allowed for a nationally representative sample of the noninstitutionalized resident population. Exclusion criteria included individuals in institutionalized settings, and active-duty military personnel[76]. Additionally, the proximity of participants' homes to the mobile examination center (MEC) was taken into consideration during selection, and typically results in high response rates[76].

Certain populations were oversampled during specific years in an effort to allow meaningful data collection of minority groups and to increase generalizability to the US population. The 1999 to 2006 NHANES oversampled Mexican-Americans whereas the 2007 to 2010 NHANES included oversampling of the Hispanic population[76]. Additionally, the 2011 to 2014 NHANES oversampled the Asian population[76].

Approximately 5,000 persons have been examined across the 15 study locations per year for NHANES, and data were collected for those of all ages[76]. However, this particular study used participants 31 years and older at the time of the survey, as prevalence of the chronic diseases in question is greater in this population[80,81]. Participation in the survey was voluntary and confidential[77].

Physical and Serum Laboratory Assessments

Once selected, participants who consented completed an interview and a physical examination in the MEC. During this time, they had blood drawn for laboratory analysis. Among the assessments were height, weight, blood pressure, A1c, and a lipid panel.

During the physical examination, standing height and weight were measured for each participant. Standing height was assessed by positioning participants on the stadiometer platform while following proper protocol, such as instructing participants to stand upright with heels together and toes apart as well as ensuring the back of the head, shoulders, buttocks, and heels were touching the backboard. Meanwhile, weight was measured in kilograms using a digital scale and participants were directed to stand in the center of the scale with their hands by their sides. These two values were used to determine BMI per participant. The following BMI criteria were used to categorize weight status: underweight (BMI <18.5); normal weight (BMI of 18.5-24.9); overweight (BMI of 25.0-29.9); and obese (BMI ≥ 30.0)[82]. BMI values indicating both overweight and obese were considered to be positive markers of one of the chronic diseases examined in this analysis.

Diagnostic criteria outlined in the 2017 American College of Cardiology and American Heart Association (ACC/AHA) Guidelines for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults were used in this analysis as opposed to those from the 2003 Seventh Report or the 2014 Eighth Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure[83–85] (Table 2). The 2003 and 2014 guidelines both designated HTN at ≥140/≥90 mm Hg for the general population. In contrast, the 2017 ACC/AHA guidelines recommend diagnosing HTN at ≥130/≥80 mm Hg. This lowered diagnostic threshold translates to a greater proportion of the sample having HTN, yet aligns with the most recent guidelines of clinical practice and therefore was selected for use in the current study[86].

Blood pressure was measured during the physical examination in the MEC, and both systolic and diastolic measurements were used to assess blood pressure and hypertensive stages in adults. Blood pressure was collection followed standardized protocol and participants were instructed to rest for at least 5 minutes in a seated position prior to the blood pressure measurements to reduce inaccurate readings. Additionally, blood pressure was measured three times for each participant, and the averaging rules for determining mean blood pressure from the NHANES Physician Examination Procedures Manual were followed[82]. In this analysis, participants were excluded if they did not have more than two complete blood pressure readings recorded. The first blood pressure reading was discarded and the remaining readings were averaged per participant. This average was then used for further analyses in this study.

A1c values were represented as a percentage to demonstrate the percent of hemoglobin, a protein found in blood, that was bound by glucose. This value indicates the degree of blood glucose control for the prior 90 days. Table 3 shows the A1c values and their designated categories of diabetes status. Measuring A1c is a preferred method for assessing and monitoring diabetes status compared to fasting plasma glucose and oral glucose tolerance test[18,87]. Some advantages to utilizing A1c are that patients are not required to be fasting, the preanalytical stability is greater, the value is less affected by acute exacerbations of stress on the body, and it provides longer term blood glucose information as opposed to a single snapshot in time[18,87]. However, a disadvantage to measuring A1c is lower test sensitivity[18]. Additionally, those with conditions that involve increased red blood cell turnover, such as sickle cell disease, pregnancy, hemodialysis, recent blood transfusions or loss, or erythropoietin therapy should use plasma glucose criteria to diagnose diabetes rather than A1c[18].

Serum lipid profiles are laboratory analyses performed that require a blood sample. Lipid panels measured the concentration of total cholesterol, HDL-C, and triglycerides[88]. An equation was then used to calculate LDL-C. Fasting, generally at least 9 hours without eating or drinking anything aside from water, is not necessary to measure total cholesterol and HDL-C yet is required for an accurate triglyceride measurement[88]; therefore, participants' fasting status was recorded before the blood draw. Table 4 demonstrates healthy blood cholesterol levels for US men and women over the age of 20. This analysis considered total cholesterol levels of ≥200 mg/dL to be considered as elevated.

Dietary Data Collection

What We Eat in America is the dietary interview portion of the NHANES which was conducted for each participant. Each dietary interview included the Dietary Recall section, the Supplement and antacid use section, and the Post-Recall section[89]. The Automated Multiple Pass Method (AMPM), a computer-assisted dietary interview system, was developed by Westat under the United States Department of Agriculture's (USDA) Agricultural Research Service and was used for the interview. The AMPM was comprised of 5 steps (Figure 1) to improve accuracy of the 24-hour dietary recall by maximizing participants' chances to remember and report consumed foods. The AMPM was shown to yield accurate assessments of energy and sodium intakes and is therefore a reliable tool for gathering dietary intake data[90,91]. The 24-hour dietary recall measured food and beverage intake from midnight to midnight. The interview took place in person in the MEC[89]. During this interview, information regarding time and place of consumption and title of meal or snack was gathered. At the end of this interview, participants were asked about their typical intakes, salt use for the day prior to the interview (both at the table and during cooking), and any special diets followed. In an effort to improve accurate estimations of portion sizes, 3-dimensional measuring guides (such as glasses, bowls, mugs, mounds, circles, thickness tools, spoons, water bottles, and food models) were provided to participants.

Data Preparation

Food and Nutrients Database for Dietary Studies

Information gathered from the 24-hour dietary recalls was coded and linked to the Food and Nutrients Database for Dietary Studies (FNDDS) and the Food Patterns Equivalents Database (FPED). The FNDDS converts information gathered from the What We Eat in America dietary interviews into individual food files and total nutrient intake files. These files contain the designated USDA food codes, quantities of foods consumed (in grams), and the nutrients in each food. An updated FNDDS is released each time What We Eat in America data are released and currently includes 65 nutrients.

FPED is a tool used to assess dietary patterns and works by converting foods and beverages in the FNDDS files into 1 of 37 mutually exclusive food components per 100 grams of each ingredient (Table 5)[92]. FPED uses cup equivalents of Fruits, Vegetables, and Dairy; ounce equivalents of Grains and Protein Foods; teaspoon equivalents of Added Sugars; gram equivalents of Solid Fats and Oils; and number of alcoholic beverages.

Healthy Eating Index-2015

The HEI-2015 is a scale used to measure diet quality and to compare how dietary intakes align with the DGA. The scale was first developed in 1995, was then revised in 2005 and again in 2010, with the latest revision taking place in 2015 using the 2015-2020 DGA as the reference standard[56,93]. The HEI-2015 demonstrates construct validity, criterion validity, and reliability, and therefore is deemed as an appropriate method to assess diet quality in relation to the 2015-2020 DGA[52]. As new DGA are released every 5 years, the HEI will too be updated to reflect the new precedents[53].

The HEI-2015 includes 13 components, 9 of which are adequacy components and are recommended in a healthy diet, whereas 4 are moderation components that should be limited in a healthy diet (Table 6)[52,63]. The maximum score of 100 indicates complete adherence to the 2015-2020 DGA. Some changes to note in the HEI-2015 include that legumes now fit into 4 components: Total Vegetables, Greens and Beans, Total Protein Foods, and Seafood and Plant Proteins[63]. Additionally, Empty Calories from the HEI-2010 was removed and Saturated Fats, originally from the HEI-2005, was reinstalled in the HEI-2015 along with Added Sugars. The HEI-2015 also considers all energy from alcohol within the total energy[53,63]. This scale is density-based and measures amounts per 1,000 calories[53].

Data Analysis

To conduct the analyses of the included data while accounting for the oversampled populations, SPSS Complex Samples (Version 24) was used. This provided a nationally representative sample. Participants were stratified into groups of diabetes status based on A1c values: normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); and poorly controlled diabetes (≥7%). Differences in diet quality and markers of chronic disease by diabetes status were assessed by a Chi square and one-way analysis of variance. Analysis of covariance tested for differences between BMI, blood pressure, and total cholesterol by diabetes status, controlled for age, sex, race, ethnicity, and percent of the federal poverty level. Chi square tested for differences in categories of diet quality by diabetes status among participants included in the sample. Descriptive statistics were used to summarize the data and produce population-based means and sample-based standard errors for all variables. Significance was set *a priori* at $\alpha \leq 0.05$.

Blood Pressure Category	Systolic Blood Pressure		Diastolic Blood Pressure
Normal	<120 mm Hg	and	<80 mm Hg
Elevated	120-129 mm Hg	and	<80 mm Hg
Hypertension			
Stage 1	130-139 mm Hg	or	80-89 mm Hg
Stage 2	≥140 mm Hg	or	≥90 mm Hg

Individuals with SBP and DBP in 2 categories should be designated to the higher BP category. BP indicates blood pressure (based on an average of ≥ 2 careful readings obtained on ≥ 2 occasions, as detailed in Section 4); DBP, diastolic blood pressure; and SBP, systolic blood pressure[83].

Table 2. American College of Cardiology and American Heart Association Guidelines for Blood Pressure Thresholds in Adults[83]

Glycated Hemoglobin (A1c)	Diabetes Status
< 5.7%	Normal glycemia
5.7 to 6.4%	Prediabetes
6.5 to 6.9%	Controlled diabetes
≥ 7%	Poorly controlled diabetes

Table 3. Glycated Hemoglobin (A1c) Values to Categorize Diabetes Status[18]

Demographic	Total Cholesterol	Non-HDL	LDL	HDL
Men ≥20 years old	125-200 mg/dL	<130 mg/dL	<100 mg/dL	≥40 mg/dL
Women ≥20 years old	125-200 mg/dL	<130 mg/dL	<100 mg/dL	≥50 mg/dL

Table 4. Healthy Total Cholesterol Levels for US Adults[94]

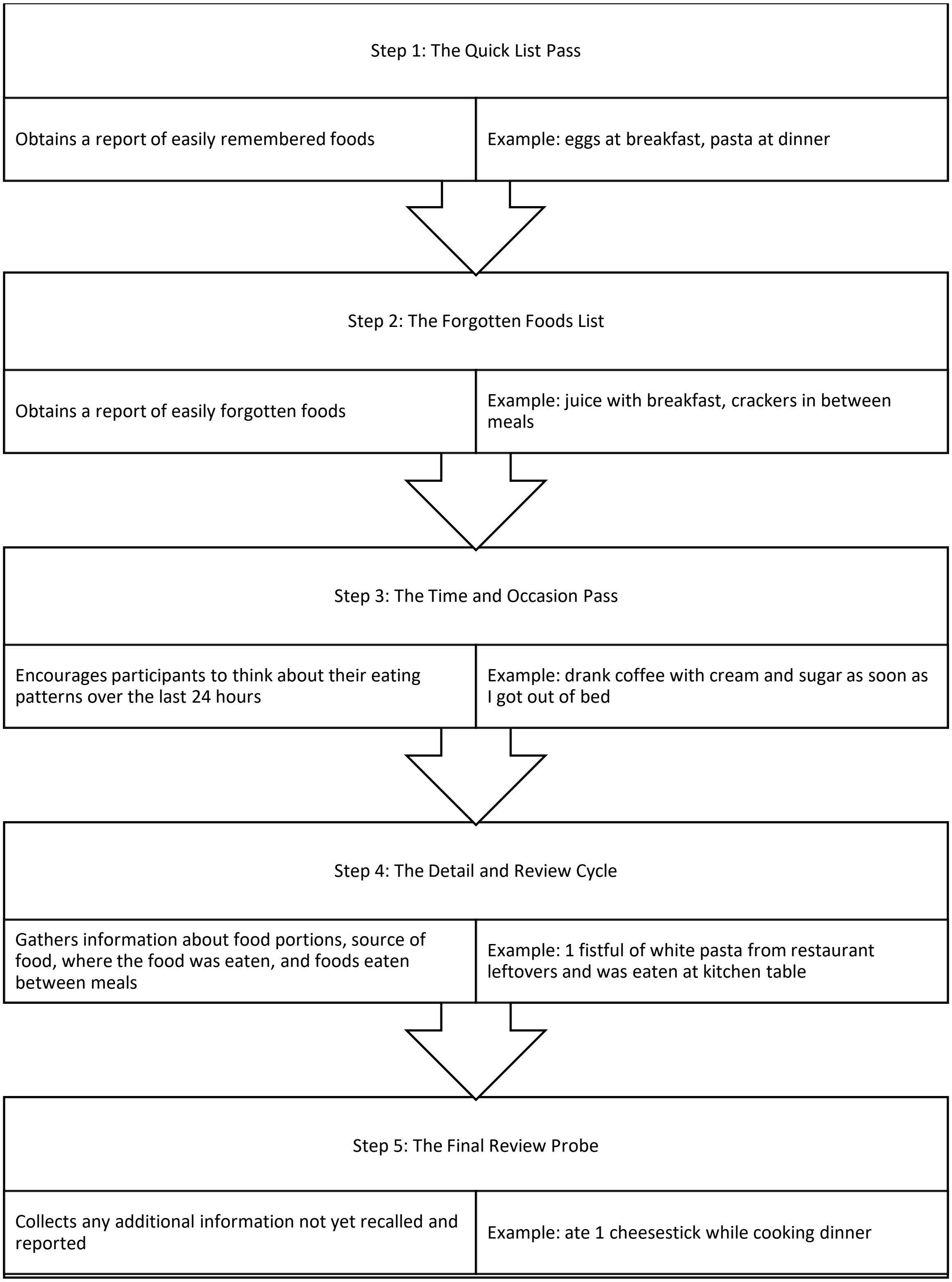

Figure 1. The 5 Step Automated Multiple Pass Method[89]

Main Components	FPED Food Components for 2015-2016
Fruit	1 Total fruit
	2 Citrus, melons, and berries
	3 Other fruits
	4 Fruit juice
Vegetables	5 Total vegetables
	6 Dark green vegetables
	7 Total red and orange vegetables
	8 Tomatoes
	9 Other red and orange vegetables (excludes, tomatoes)
	10 Total starchy vegetables
	11 Potatoes (white potatoes)
	12 Other starchy vegetables (excludes white potatoes)
	13 Other vegetables
	14 Beans and peas computed as vegetables
Grains	15 Total grains
	16 Whole grains
	17 Refined grains
Protein Foods	18 Total protein foods
	19 Total meat, poultry, and seafood
	20 Meat (beef, veal, pork, lamb, game)
	21 Cured meat (frankfurters, sausage, corned beef, cured ham and luncheon meat made from beef, pork, poultry)
	22 Organ meat (from beef, veal, pork, lamb, game, poultry)
	23 Poultry (chicken, turkey, other fowl)
	24 Seafood high in n-3 fatty acids
	25 Seafood low in n-3 fatty acids
	26 Eggs
	27 Soybean products (excludes calcium fortified soy milk and mature soybeans)
	28 Nuts and seeds
	29 Beans and peas computed as protein foods
Dairy	30 Total dairy (milk, yogurt, cheese, whey)
	31 Milk (includes calcium fortified soy milk)
	32 Yogurt
	33 Cheese
Oils	34 Oils
Solid Fats	35 Solid fats
Added Sugars	36 Added sugars
Alcoholic Drinks	37 Alcoholic drinks

Table 5. Food Patterns Equivalents Database Food Components for 2015-2016

Component:	Maximum Points:	Standard for Maximum Score:	Standard for Minimum Score of Zero:
		Adequacy:	
		Intakes per 1,000 kcals	
Total Fruits[2]	5	≥0.8 cup equiv.	No fruit
Whole Fruits[3]	5	≥0.4 cup equiv.	No whole fruit
Total Vegetables[4]	5	≥1.1 cup equiv.	No vegetables
Greens and Beans[4]	5	≥0.2 cup equiv.	No Dark Green Vegetables or Legumes
Whole Grains	10	≥1.5 oz. equiv.	No Whole Grains
Dairy[5]	10	≥1.3 cup equiv.	No Dairy
Total Protein Foods[6]	5	≥2.5 oz. equiv.	No Protein Foods
Seafood and Plant Proteins[6,7]	5	≥0.8 oz. equiv.	No Seafood or Plant Proteins
Fatty Acids[8]	10	(PUFAS+MUFAS)/SFAs≥2.5	(PUFAS+MUFAS)/SFAs≤1.2
		Moderation:	
Refined Grains	10	≤1.8 oz. equiv.	≥4.3 oz. equiv.
Sodium	10	≤1.1 gram	≥2.0 grams
Added Sugars	10	≤6.5% of energy	≥26% of total energy
Saturated Fats	10	≤8% of energy	≥16% of total energy

1. Intakes between minimum and maximum are scored proportionately.
2. Include 100% fruit juice.
3. Includes all forms except juice.
4. Includes legumes (beans and peas).
5. Includes all milk products, such as fluid milk, yogurt, cheese, and fortified soy beverages.
6. Includes legumes (beans and peas).
7. Includes seafood, nuts, seeds, soy products (other than beverages), and legumes (beans and peas).
8. Ratio of polyunsaturated fatty acids (PUFAs) and monounsaturated fatty acids (MUFAs) to saturated fatty acids (SFAs).

Table 6. Healthy Eating Index 2015 Scoring Criteria[53]

Chapter 4. Results and Discussion

Results

Demographics

The final sample consisted of 23,708 adults, aged 31 years and older, from the 2005 to 2016 NHANES (Table 7). Diabetes was more prevalent in males (52.1-53.7%) than in females (46.3-47.9%). Diabetes was more prevalent in adults who were obese (>65.7%), married (>64%), had stage 2 HTN (>30.8%), and had some college education or an associate's degree (>29.8%).

Diet Quality by Level of Glycemic Control

Mean diet quality scores demonstrate that adults with prediabetes and diabetes have significantly poorer diet quality compared to the normal glycemia group as evidenced by lower Total HEI-2015 scores (P<0.001, Table 8). Regardless of glycemic category, diet quality was poor for Whole Grains, Greens and Beans, Sodium, Total Fruit, Whole Fruit, and Seafood and Plant Proteins (HEI-2015 <50% of maximum score).

Of the thirteen food components measured, there were significant differences among groups for Total Fruit, Dairy, Seafood and Plant Proteins, Refined Grains, Sodium, Added Sugars, and Saturated Fat. Adults with prediabetes and diabetes had lower diet quality for all food components except for Dairy (controlled diabetes had higher score), Total Protein Foods (poorly controlled diabetes had highest score), and Added Sugars (poorly controlled diabetes was highest, while diabetes groups had significantly higher scores than the normal glycemia and prediabetes groups). Scores for Total Protein Foods were similar across all groups, yet adults with diabetes consumed significantly more saturated fat than adults with normal glycemia, suggesting greater intake of fatty meats in the diabetes population.

Markers of Chronic Diseases by Level of Glycemic Control

To identify whether adults with diabetes have more markers of chronic conditions than those without diabetes, A1c, blood pressure, total cholesterol, and BMI were analyzed (Table 9). Data show highest rates of normal weight in adults with normal glycemia (30.6%) and lowest rates in those with controlled diabetes (8.3%). Likewise, the normal glycemia group had greatest rates of overweight (36.7%), which were lower in prediabetes (32.8%) and diabetes groups (25.2% and 23.5%). Obesity rates were highest in the controlled (66.5%) and poorly controlled (65.7%) diabetes adults, and lower in the normal glycemia (31.2%) and prediabetes (48.4%) groups. Although lower when compared to the diabetes groups, obesity was still largely prevalent in adults with prediabetes.

Adults with normal glycemia had highest rates of normal blood pressure (47.1%), with lower rates in across prediabetes (32.0%) and diabetes groups (25.4% and 28.0%). Prevalence of elevated blood pressure and stage 1 and stage 2 HTN were greater in the diabetes and prediabetes groups compared to diabetes-free adults. Adults with controlled (30.8%) and poorly controlled diabetes (31.4%) had greatest rates of stage 2 HTN.

Contrary to the previous findings, adults with diabetes (controlled diabetes: 67.2%; poorly controlled diabetes: 60.4%) had greater rates of desirable cholesterol levels than those without diabetes (normal glycemia: 50.6%; prediabetes: 52.8%). Rates of moderate cholesterol levels were lowest in the diabetes groups (controlled diabetes: 22.8%; poorly controlled diabetes: 23.8%). High risk levels fluctuated between groups, with prediabetes adults claiming the greatest percentage (17.3%).

Diet Quality in Adults by Level of Glycemic Control and Markers of Chronic Diseases

Adults with normal glycemia had better diet quality than their prediabetes and diabetes counterparts with the exceptions of highest Total HEI-2015 scores in adults with controlled diabetes for the elevated cholesterol, normal blood pressure, and elevated BMI groups (Table 10). All of the lowest diet quality scores belonged to the diabetes or prediabetes groups. Overall, diet quality was poorer in adults with both poorly controlled diabetes and obesity (50.9 ± 0.4) as opposed to diabetes alone (50.7 ± 0.39).

Discussion

Medical nutrition therapy for adults with diabetes emphasizes overall diet quality, yet the lay public focuses on individual dietary components[95–98]. This study contributes to the body of evidence supporting the need for an emphasis on overall diet quality in adults with diabetes and in those with comorbidities. Diet quality and prevalence of markers of chronic diseases were examined in US adults with normal glycemia, prediabetes, controlled diabetes, and poorly controlled diabetes. This study found that adults with diabetes and prediabetes have significantly poorer diet quality than those without diabetes. Prevalence of obesity and stage 2 HTN is higher in adults with diabetes, yet lower for elevated cholesterol levels compared to those with normal glycemia and prediabetes. Lastly, diet quality is poorer in the presence of elevated markers for chronic diseases.

Assessing overall diet quality provides insight into the risk for disease development and progression[46,51]. The HEI indicates compliance to the DGA, which are aimed at the general population[46,52,53]. Core tenants of the DGA include emphasis on fruit and vegetable consumption, whole grains as opposed to refined grains, and lean proteins as opposed to fatty meats[66]. These same core principles are shared across multiple recommended dietary patterns, such as the Dietary Approaches to Stop Hypertension (DASH) eating plan and the Mediterranean diet, as well as in the prevention and treatment of diabetes and other chronic diseases[66,99–101]. Therefore, assessing the overall diet quality through these indices provides insight into the risk of chronic disease development in the general population as well as in adults with diabetes.

US adults have an overall poor diet quality. A previous study found a mean diet quality score (Total HEI-2015) of 58 out of 100 for adults in the general population[56]. Although slightly lower, our findings are in alignment, with a total score of 53 out of 100 for US adults greater than 30 years of age without diabetes. Our findings confirm poor overall diet quality in the general population of US adults.

Furthermore, we place particular interest in the population with diabetes. The presence of diabetes itself serves as a risk factor for morbidity and mortality[13,21,69,70]. In addition, previous studies have identified inverse associations between diet quality and CVD, cancer, and all-cause mortality[45,63,74,75,102]. The simultaneous presence of diabetes and poor diet quality is a cause for concern and warrants the evaluation of diet quality in this population. A prior study identified a Total HEI-2010 score of 57.7 for adults with diabetes, and report higher diet quality in those with the disease than those without[61]. However, the current study reports lower diet quality results of 50.8 in adults with diabetes, supporting our original hypothesis that adults with diabetes have poorer diet quality than their diabetes-free counterparts. In the current analysis, the range of total diet quality scores in all glycemic groups fits into the lowest quintile of diet quality from a previous study, which is associated with a 12-28% increased risk for CVD, cancer, and all-cause mortality[74].

A benefit of the current analysis is the ability to examine which dietary elements are responsible for the overall poor diet quality of the diabetes population in addition to identifying which areas are the most successful. One area that the diabetes population reigns in is added sugar consumption. Previous studies have identified conflicting results regarding added sugar intake[10,12,63,67,103]. Our findings align with those reporting significantly lower intakes and moderate adherence to the DGA recommendation in adults with diabetes[12,63,67,103]. These lower intakes are likely due to greater exposure to nutrition education and awareness of dietary sugar's affect on blood glucose[67]. A cross-sectional analysis identified an inverse dose response between duration of diabetes as well as frequency of blood glucose self-monitoring and sugar-sweetened beverage consumpsion, supporting the prior hypotheses[103].

Although added sugar intake is reduced in adults with diabetes, our data inform us of the many remaining areas of concern in this target population that pose as threats to the exacerbation of diabetes, comorbidities, and mortality. Adults with diabetes consume significantly higher amounts of saturated fat than those without diabetes. Considering total protein intakes were fairly consistent across glycemic groups, we infer that adults with diabetes consume more fatty meats, including red meats and processed meats, and more added fats than their diabetes-free counterparts[11]. These results align with those of previous analyses, in which intakes of adults with diabetes surpass the recommended limit of saturated fat[6–9,11,12].

Across all levels of glycemia in our sample, participants have especially poor intakes of whole grains, green vegetables, beans, fruit, seafood, and plant proteins, and excessive sodium intakes. Previously, it has been reported that adults with diabetes do not meet recommended fruit, vegetable, whole grain, and fiber intakes[6–9]. Additionally, their sodium consumption has increased all the while maintaining inadequate potassium intakes[7]. Our findings support the notion that US adults, with and without diabetes, do not meet recommendations for such dietary components. These inadequacies are problematic, as these food categories serve as the main pillars of the DGA, the DASH diet, and the Mediterranean diet, which have been linked to reduced risk for morbitiy and mortality[45,63,74,75]. Conversely, dietary patterns that do not comply with the recommendations may contribute to the development or progression of diabetes and common comorbidities, such as hypertension, hyperlipidemia, and obesity[13,15,68].

The current study found that more than 50% of adults with diabetes have HTN, 30% of which are classified as stage 2. Our rates are slightly lower than previous estimates that state 60-80% of adults with diabetes have concurrent hypertension[1,13,70,83]. Previous research also found that it is twice as common in the diabetes population than in the normal glycemia population[83]. Our findings support that adults with diabetes have double the rate of stage 2 HTN than normal glycemia adults. By following the 2017 ACC/AHA's lowered diagnostic thresholds for elevated blood pressure and both stages of HTN, our data nearly double the proportion of adults with markers of HTN. The benefit to utilizing the 2017 thresholds includes outcomes that reflect most recent protocol and clinical practice guidelines. Along with prevalence, we found that diet quality worsened in the presence of HTN for all glycemic groups. Considering this study's data suggest noncompliance to key principles of the DASH diet, including emphasis on whole grains, fruits, vegetables, and lean proteins as well as limited sodium and sufficient potassium intake, it can be presumed that diet quality plays a role in the high rates of HTN in this target population[13,104,105].

Epidemiology data associate high intakes of saturated fat and added sugars with worsened total cholesterol values in adults with diabetes[10,12,72,73]. HLD has been reported in more than 70% of adults with diabetes[106]. However, results from the current study differ from previous prevalence estimations and show that only 33-40% of adults with diabetes have HLD, whereas nearly 50% of adults with prediabetes and normal glycemia have elevated cholesterol levels. These findings may be due to use of statin medications in the diabetes population. A national analysis identified that 58-67% of adults with diabetes were on lipid-lowering statin therapy[1]. Medication data were not included in this current analysis due to their unavailability from NHANES at the time of analysis. This absence of medication data inhibits our ability to determine whether cholesterol values are metabolic or pharmacologic. This notion may explain the absence of significant differences in diet quality seen among adults with normal versus elevated total cholesterol levels. However, even in the presence of pharmacologic blood lipid control, diet quality remains to be relevant in the prevention and management of comorbidities and mortality[10,12,72].

Obesity is highly prevalent among the diabetes population, and an increase in BMI is considered to be one of the strongest predictors of diabetes in both sexes[3,38]. In this study, more than 90% of adults with diabetes are either overweight or obese, mirroring a previous prevalence estimate[1]. Although more prominent in the diabetes groups, nearly 50% of adults with prediabetes and more than 30% of those without diabetes are obese. These data reflect previous findings regarding weight trends in US adults[107]. Although multifaceted, one potential contributor to the higher overweight and obesity rates in the diabetes groups is use of insulin to manage blood glucose. Weight gain is a common side effect of insulin therapy, and therefore, may contribute to these outcomes[108]. Across all glycemic groups, diet quality was poorer when overweight or obesity was present. The high rates of overweight or obesity and reduced diet quality among all groups, including in normal glycemia and prediabetes adults, are alarming and may forebode disease development and exacerbation[3].

Strengths and Limitations

The current study has numerous strengths. Sampling weights produced nationally representative estimates for noninstitutionalized US citizens, increasing the external validity through broad generalizability. The study design of NHANES includes a large sample size determined by a multistage sampling process as well as oversampling of subpopulations, also contributing to greater generalizability. Additionally, data from six NHANES cohorts were pooled to generate stronger results that more accurately reflect the target population.

Additional strengths of the current analysis include following standardized protocols to gather dietary data and perform laboratory and physical assessments. Dietary data were gathered from 24-hour dietary recalls utilizing the AMPM. The AMPM increases the accuracy of the recall, producing more thorough intake estimates[90,91]. One limitation is the use of data from only one day of intake per participant, which prevents us from identifying dietary trends in each individual. However, the large sample size of the current analysis allows for cumulative trends and nationally representative results.

Blood pressure was measured following standardized procedures, including using an appropriately sized cuff, allowing each participant to rest seated for five minutes prior to the assessment, and measuring blood pressure three times per visit. However, the 2017 ACC/AHA guidelines recommend the averaging of blood pressure readings from two or more separate visits, whereas all three readings were taken in one single visit to the MEC in NHANES.

Lastly, the absence of prescription medication data serves as a limitation to the current analysis, as it prevents us from determining which markers of chronic conditions have been influenced by pharmacological intervention. These data would be particularly useful in interpreting the HLD data, as adults with diabetes had highest prevalence of desirable cholesterol levels. Our data may underrepresent the proportion of adults with HLD that are masked by pharmacologic control.

Implications for Research and Clinical Practice

Due to inconsistent results in the body of evidence, additional research examining overall diet quality in the diabetes population is needed to either support or dispute our findings that diet quality is poorer in adults with diabetes and prediabetes compared to those with normal glycemia. Furthermore, future studies should aim to assess the impact that the aging population has on diet quality in those with diabetes. This would allow for a unique viewpoint, as better diet quality has been reported in older adults yet poorer diet quality has been reported in adults with diabetes[58].

Although no causal relationship can be confirmed by cross-sectional analyses, our findings support the need for emphasis on overall diet quality in medical nutrition therapy for both the population with and without diabetes. Our results demonstrate the successful campaign of discouraging added sugar intake among adults with diabetes in an effort to better manage blood glucose; yet in doing so, attention has been shifted away from other necessary aspects of dietary patterns, resulting in decreased overall diet quality.

Lastly, our analyses provide unique insights into the progression of diet quality along with the stages of diabetes. Our results demonstrate that diet quality is poorer in adults with less glycemic control, with prediabetes scores significantly poorer than those of adults with normal glycemia, and poorly controlled diabetes scores lower than those of controlled diabetes. Considering that the criteria for prediabetes, such as IGT, increase the risk for diabetes development by 5-fold and that poor diet quality is associated with increased risk for diabetes, the population with prediabetes is at an especially critical point[21,43]. Medical nutrition therapy efforts should be encouraged in this population in an effort to delay or prevent the disease progression to diabetes and its comorbidities[97,109].

Conclusions

Poorer diet quality is associated with greater risk for CVD, cancer, and all-cause mortality in the general population[45,63,74,75]. Presence of diabetes and the comorbidity triad of HTN, HLD, and obesity further increase these risks[3,4,20,106]. Our findings support the notion that adults with diabetes and additional chronic diseases have poorer diet quality than the general population. These simultaneous risk factors for morbidity and mortality create a sense of urgency for lifestyle interventions to target diet quality in adults with diabetes and comorbidities. Acknowledging the aspects of diet quality in which adults with diabetes are doing well and are doing poor allows for better targeted nutrition therapy, improving the overall diet quality, and in turn, reducing morbidity and mortality.

		Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)
			n (%)		
Gender	Male	7165 (47.1%)	2881 (46.5%)	484 (52.1%)	1071 (53.7%)
	Female	7676 (52.9%)	3042 (53.5%)	439 (47.9%)	950 (46.3%)
Weight Status	UW (<18.5)	234 (1.4%)	58 (0.9%)	0 (0%)	3 (0.2%)
	NW (18.5-24.9)	4447 (30.6%)	1018 (17.8%)	98 (8.3%)	248 (10.6%)
	OW (25-29.9)	5365 (36.7%)	1983 (32.8%)	253 (25.2%)	518 (23.5%)
	OB (≥30)	4655 (31.2%)	2796 (48.4%)	556 (66.5%)	1205 (65.7%)
Marital Status	Single/Widowed/Divorced	5095 (30.7%)	2353 (35.7%)	359 (36%)	790 (35.5%)
	Married/Living as Married	9741 (69.3%)	3569 (64.3%)	564 (64%)	1228 (64.5%)
Education Level	<9th grade	1425 (4.5%)	880 (8.6%)	170 (11.4%)	424 (13.3%)
	9-11th grade	1976 (9.5%)	927 (13.3%)	172 (16.9%)	348 (14.1%)
	High School/GED	3233 (21.4%)	1467 (26.4%)	208 (22.9%)	461 (26%)
	Some college or AA degree	4156 (30.3%)	1541 (28.7%)	226 (29.8%)	528 (30.1%)
	College graduate	4042 (34.3%)	1101 (23.1%)	147 (19.1%)	255 (16.5%)
Total Cholesterol	Desirable (<200)	7849 (50.6%)	3171 (52.8%)	612 (67.2%)	1192 (60.4%)
	Moderate risk (200-239)	4705 (33.5%)	1778 (29.9%)	202 (22.8%)	477 (23.8%)
	High risk (≥240)	2150 (16%)	903 (17.3%)	88 (10%)	316 (15.8%)
Blood Pressure	Normal (<120/<80)	6414 (47.1%)	1709 (32%)	240 (25.4%)	494 (28%)
	Elevated (120-129/<80)	2336 (16.1%)	1027 (18.4%)	162 (19.2%)	350 (19.3%)
	Stage 1 (130-139 or 80-89)	2910 (21.3%)	1382 (24.8%)	196 (24.7%)	430 (21.3%)
	Stage 2 (≥ 140 or ≥ 90)	2479 (15.4%)	1485 (24.9%)	271 (30.8%)	626 (31.4%)
			Mean (SEM)		
Age (years)		50.5 (0.2)	59.6 (0.3)	61.4 (0.6)	58.6 (0.4)

Unweighted counts and population percentages.
Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).

Table 7. Demographic Characteristics of Sample Population by Level of Glycemic Control

HEI-2015 Score (Score range)	Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)	P
		Adjusted means (SEM)			
Total Fruit (0-5)	2.3 (0.03)[a]	2.2 (0.05)[b]	2.0 (0.10) [b]	2.1 (0.07) [b]	0.011
Whole Fruit (0-5)	2.3 (0.03)	2.2 (0.05)	2.1 (0.10)	2.1 (0.08)	0.330
Total Vegetables (0-5)	3.2 (0.02)	3.2 (0.04)	3.2 (0.08)	3.2 (0.06)	1.000
Greens and Beans (0-5)	1.7 (0.04)	1.6 (0.04)	1.5 (0.10)	1.5 (0.07)	0.061
Whole Grains (0-10)	2.6 (0.04)	2.5 (0.08)	2.8 (0.16)	2.6 (0.12)	0.764
Dairy (0-10)	4.9 (0.05)[a]	4.8 (0.06)[a]	5.4 (0.13)[b]	4.9 (0.10)[a]	0.007
Total Protein Foods (0-5)	4.3 (0.01)[a]	4.3 (0.03)[ab]	4.3 (0.07)[ab]	4.4 (0.04)[b]	0.046
Seafood and Plant Proteins (0-5)	2.5 (0.03)[a]	2.4 (0.05)[b]	2.4 (0.12)[ab]	2.3 (0.08)[b]	0.018
Fatty Acids (0-10)	5.3 (0.06)	5.1 (0.07)	5.1 (0.15)	5.2 (0.13)	1.000
Refined Grains[1] (0-10)	6.4 (0.05)[a]	6.0 (0.08)[b]	5.5 (0.17)[b]	5.7 (0.12)[b]	<0.001
Sodium[1] (0-10)	4.6 (0.04)[a]	4.0 (0.08)[b]	3.4 (0.18)[c]	3.5 (0.12)[c]	<0.001
Added Sugars[1] (0-10)	6.8 (0.05)[a]	6.7 (0.08)[a]	7.5 (0.14)[b]	7.7 (0.12)[b]	<0.001
Saturated Fat[1] (0-10)	6.3 (0.05)[a]	5.9 (0.08)[b]	5.6 (0.14)[c]	5.6 (0.13)[c]	<0.001
Total HEI Score (0-100)	**53 (0.2)[a]**	**50.8 (0.3)[b]**	**50.8 (0.6)[b]**	**50.7 (0.4)[b]**	**<0.001**

Adjusted for age, race, ethnicity, sex, marital status, and percent of federal poverty rate.
Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).
[1]Moderation components; higher scores indicate lower consumption.

Table 8. Differences in Healthy Eating Index 2015 Scores by Level of Glycemic Control in US Adults

		Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)	
		n (%)				P
Cholesterol	Desirable (<200)	7849 (50.6%)	3171 (52.8%)	612 (67.2%)	1192 (60.4%)	<0.001
	Moderate (200-239)	4705 (33.5%)	1778 (29.9%)	202 (22.8%)	477 (23.8%)	
	High (≥240)	2150 (16.0%)	903 (17.3%)	88 (10.0%)	316 (15.8%)	
Blood pressure	Normal (<120/<80)	6414 (47.1%)	1709 (32.0%)	240 (25.4%)	494 (28.0%)	<0.001
	Elevated (120-129/<80)	2336 (16.1%)	1027 (18.4%)	162 (19.2%)	350 (19.3%)	
	Stage 1 (130-139 or 80-89)	2910 (21.3%)	1382 (24.8%)	196 (24.7%)	430 (21.3%)	
	Stage 2 (≥140 or ≥90)	2479 (15.4%)	1485 (24.9%)	271 (30.8%)	626 (31.4%)	
Obesity	UW (<18.5)	234 (1.4%)	58 (0.9%)	0 (0.0%)	3 (0.2%)	<0.001
	NW (18.5-24.9)	4447 (30.6%)	1018 (17.8%)	98 (8.3%)	248 (10.6%)	
	OW (25-29.9)	5365 (36.7%)	1983 (32.8%)	253 (25.2%)	518 (23.5%)	
	OB (≥30)	4655 (31.2%)	2796 (48.4%)	556 (66.5%)	1205 (65.7%)	

Adjusted for age, race, ethnicity, sex, marital status, and percent of federal poverty rate.
Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).

Table 9. Prevalence of Markers of Chronic Diseases by Level of Glycemic Control in US Adults

Chronic Disease (Measure)		Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)
		Adjusted means (SEM)			
Hyperlipidemia (Total cholesterol)	Normal	51.9 (0.3)	51.4 (0.3)	50.4 (0.7)	50.8 (0.5)
	Elevated[a]	51.9 (0.3)	51.1 (0.4)	53.2 (1.2)	51.5 (0.6)
Hypertension (Blood pressure)	Normal	52.8 (0.3)*	52.0 (0.5)	53.4 (1.1)	51.3 (0.7)
	Elevated[b]	51.2 (0.3)	51.0 (0.3)	50.9 (0.8)	50.9 (0.5)
Overweight or Obesity (BMI)	Normal	53.9 (0.3)*	53.0 (0.7)*	52.7 (1.9)	51.5 (1.3)
	Elevated[c]	51.0 (0.2)	50.8 (0.3)	51.3 (0.6)	50.9 (0.4)

Adjusted for age, race, ethnicity, sex, marital status, and percent of federal poverty rate.
Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).
[a]Elevated total cholesterol: ≥200 mg/dL.
[b]Elevated blood pressure: ≥120/<80 mm Hg, systolic blood pressure ≥130 mm Hg, or diastolic blood pressure >80 mm Hg.
[c]Elevated BMI: overweight/obesity categorized by BMI values of ≥25 kg/m^2.
*P<0.05

Table 10. Differences in Total Healthy Eating Index 2015 Scores across Glycemic Level by Markers of Chronic Diseases in US Adults

Chapter 5. Manuscript

Introduction

Type 2 diabetes affects more than 30 million adults in the US and prevalence is expected to rise as a result of increasing rates of obesity, physical inactivity, urbanization, aging, and population growth[1–3]. Additionally, nearly 20% of adults with diabetes have a triad of concurrent chronic diseases, namely hypertension, hyperlipidemia, and obesity[106]. The presence of diabetes and these chronic diseases serve as risk factors for morbidity and mortality as a result of microvascular and macrovascular complications[13,21,69,70].

Diet plays an integral role in the prevention and treatment of diabetes and comorbidities. Diet quality is measured by indices which compare how well an individual's eating patterns align with the recommendations[46,51]. The Healthy Eating Index (HEI)-2015 assesses the compliance of dietary patterns with the evidence-based 2015-2020 Dietary Guidelines for Americans (DGA)[46,52,53]. Higher HEI-2015 scores signify greater adherence to the 2015-2020 DGA and the maximum HEI score of 100 represents perfect adherence to its associated DGA. Previous studies have identified inverse associations between diet quality and cardiovascular disease, cancer, and all-cause mortality[45,63,74,75,102].

Assessing overall diet quality provides insight into the status of a population's dietary health, yet consumption of individual food components has been the primary focus of diet-related diabetes prevention and treatment research[4,5]. Adults with diabetes tend not to meet recommendations for fruit or vegetable[6], fiber[7–9], added sugar[10], saturated fat[6–9,11,12], and sodium intakes[7,8,13]. Typically, failure to meet the recommendations for one dietary component is accompanied by that of additional dietary components, yielding inadequate quality of overall diet[10,14]. However, insufficient literature exists evaluating overall diet quality in Americans stratified by level of glycemic control.

Therefore, the aim of this study was to assess the differences in diet quality and markers of chronic diseases by varying degrees of glycemic control in a nationally representative sample. This information aids in the development or improvement of public health initiatives aimed at improving diet quality and reducing the development of subsequent chronic conditions in those with diabetes.

Methods

Sample Population

Data from 23,708 adults aged 31 years and older were gathered from the 2005 to 2016 National Health and Nutrition Examination Survey (NHANES). NHANES is a series of continuous surveys conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention aimed at identifying health-related trends in the US population. Participants were selected utilizing stratified, multistage probability sampling of noninstitutionalized US citizens. Participants gave informed consent and data were deidentified before released to the public.

Dietary, anthropometric, and laboratory data were gathered to assess diet quality and severity of chronic disease markers per participant in the current analysis. Demographic characteristics, including sex, age category, marital status, and highest level of education, were assessed during in-home interviews. Anthropometric and laboratory data were assessed during in-person visits to the mobile examination center. All methods were approved by the National Center for Health Statistics Research Ethics Review Board.

Chronic Disease Measures

Anthropometric and laboratory data were gathered during the visit to the mobile examination center[89]. Percents of glycated hemoglobin (A1c) and total cholesterol levels were determined by blood samples collected during the visit and sent to the University of Minnesota for analysis[82]. Blood pressure, height, and weight were measured during the physical examination portion of the visit. This analysis eliminated participants from the sample who lacked data for these selected chronic diseases.

A1c data were used to categorize participants into the following levels of glycemic control: normal glycemia (A1c <5.7%); prediabetes (A1c 5.7-6.4%); controlled diabetes (6.5-6.9%); and poorly controlled diabetes (≥7%). Additionally, total cholesterol concentration was measured via enzymatic assay as part of the serum lipid panel. Values of 200 mg/dL or greater were considered to be elevated, with values of 240 mg/dL or greater were further categorized as high risk for hyperlipidemia in the current analysis.

Three readings of both systolic and diastolic blood pressure were measured per participant during the visit to the mobile examination center and followed standard protocols[82]. The current analysis used the 2017 ACC/AHA guidelines to categorize blood pressure levels into normal blood pressure (<120/<80 mm Hg), elevated blood pressure (120-129/<80 mm Hg), stage 1 hypertension (130-139/80-89 mm Hg), and stage 2 hypertension (≥140/≥90 mm Hg)[83]. For analysis, the first blood pressure reading was eliminated and the remaining readings were averaged. Participants with less than three readings were excluded from the sample.

Weight status was grouped by BMI (kg/m^2), which is a comparison of height to weight. Standing height and weight were among the assessments conducted during the physical examination in the mobile examination center. These two measurements were used to calculate BMI. BMI values were categorized into underweight (BMI <18.5), normal weight (BMI of 18.5-24.9), overweight (BMI of 35.0-39.9), and obese (BMI ≥30).

Dietary Intakes and Quality

Dietary data were gathered through 24-hour dietary recalls conducted by trained interviewers[89]. Dietary recalls utilized the Automated Multiple Pass Method, which is a multistep method shown to deliver improved accuracy of dietary intake data[90,91]. Recalls captured intakes from midnight to midnight the prior day. Dietary intake data were then coded and linked to the Food and Nutrient Database for Dietary Studies and the Food Patterns Equivalents Database to generate nutrient intake estimates. Diet quality was assessed with the HEI-2015. Amounts per 1,000 kcal were calculated. The HEI-2015 is comprised of 13 individually graded components, either worth 5 or 10 points for a maximum score per component. The maximum total score of 100 indicates perfect compliance with the DGA.

Statistical Analyses

Data from 2005 to 2016 NHANES were gathered from publically available sources and imported into SPSS Complex Samples (version 24, IBM SPSS Inc., Chicago, IL). A nationally representative sample of weighted population-based estimates was assembled using sampling weights provided by the Centers for Disease Control and Prevention's Center for Health Statistics. Analyses controlled for age, sex, race, ethnicity, marital status, and percent of federal poverty rate. Analysis of variance assessed differences in diet quality by glycemic category and Chi square tested for differences in diet quality by level of BMI, total cholesterol, and blood pressure by glycemic level. Descriptive statistics generated means with standard errors or unweighted counts with weighted population percentages. Significance was set *a priori* at $\alpha \leq 0.05$.

Results

Demographics

Data from 23,708 adults aged 31 years and older from the 2005 to 2016 NHANES were stratified into normal glycemia (n=14,841), prediabetes (n=5,923), controlled diabetes (n=923), and poorly controlled diabetes (n=2,021) to conduct the analysis (Table 1). Diabetes was more prevalent in males (52.1-53.7%) than in females (46.3-47.9%). Diabetes was more prevalent in adults who were obese

(>65.7%), married (>64%), stage 2 hypertensive (>30.8%), and had some college education or associate's degree (>29.8%).

Diet Quality by Level of Glycemic Control

Adults with diabetes and prediabetes had significantly poorer diet quality, as evidenced by total HEI-2015 scores, compared to the normal glycemia group (P<0.001). Regardless of glycemic category, diet quality was poor for Whole Grains, Greens and Beans, Sodium, Total Fruit, Whole Fruit, and Seafood and Plant Proteins (HEI-2015 <50% of maximum score).

Out of the thirteen food components measured, there were significant differences among groups were seen in Total Fruit, Dairy, Seafood and Plant Proteins, Refined Grains, Sodium, Added Sugars, and Saturated Fat. Adults with prediabetes and diabetes had lower diet quality for all food components except for Dairy (controlled diabetes had significantly higher score), Total Protein Foods (poorly controlled diabetes had highest score), and Added Sugars (poorly controlled diabetes was highest, while diabetes groups had significantly higher scores than the normoglycemic and prediabetes groups). Scores for Total Protein Foods were similar across all groups, yet adults with diabetes consumed significantly more saturated fat than normal glycemia adults, suggesting greater intake of fatty meats in the diabetes population.

Markers of Chronic Diseases by Level of Glycemic Control

Data show highest rates of normal weight in adults with normal glycemia (30.6%) and lowest rates in those with controlled diabetes (8.3%). Likewise, the normal glycemia group had greatest rates of overweight (36.7%), which were lower in prediabetes (32.8%) and diabetes groups (25.2% and 23.5%). Obesity rates were highest in the controlled (66.5%) and poorly controlled (65.7%) diabetes adults, and lower in the normal glycemia (31.2%) and prediabetes (48.4%) groups. Although lower when compared to the diabetes groups, obesity was still largely prevalent in adults with prediabetes.

Adults with normal glycemia had highest rates of normal blood pressure (47.1%), with lower rates in across prediabetes (32.0%) and diabetes groups (25.4% and 28.0%). Prevalence of elevated blood pressure and stage 1 and stage 2 HTN were greater in the diabetes and prediabetes groups compared to diabetes-free adults. Adults with controlled (30.8%) and poorly controlled diabetes (31.4%) had greatest rates of stage 2 HTN.

Contrary to the previous trends, adults with diabetes (controlled diabetes: 67.2%; poorly controlled diabetes: 60.4%) had greater rates of desirable cholesterol levels than those without diabetes (normal glycemia: 50.6%; prediabetes: 52.8%). Rates of moderate cholesterol levels were lowest in the diabetes groups (controlled diabetes: 22.8%; poorly controlled diabetes: 23.8%). High risk levels fluctuated between groups, with prediabetes adults claiming the greatest percentage (17.3%).

Diet Quality in Adults by Level of Glycemic Control and Markers of Chronic Diseases

Adults with normal glycemia had better diet quality than their prediabetes and diabetes counterparts with the exceptions of highest Total HEI-2015 scores in adults with controlled diabetes for the elevated cholesterol, normal blood pressure, and elevated BMI groups (Table 4). All of the lowest diet quality scores belonged to the diabetes or prediabetes groups. Overall, diet quality was poorer in adults with both poorly controlled diabetes and obesity (50.9 ± 0.4) as opposed to diabetes alone (50.7 ± 0.39).

Discussion

Medical nutrition therapy for adults with diabetes emphasizes overall diet quality, yet the lay public focuses on individual dietary components[95–98]. This study contributes to the body of evidence supporting the need for an emphasis on overall diet quality in adults with diabetes and in those with comorbidities. Diet quality and prevalence of markers of chronic diseases were examined in US adults with normal glycemia, prediabetes, controlled diabetes, and poorly controlled diabetes. This study found that adults with diabetes and prediabetes have significantly poorer diet quality than those without diabetes. Prevalence of obesity and stage 2 HTN is higher in adults with diabetes, yet lower for elevated cholesterol levels compared to those with normal glycemia and prediabetes. Lastly, diet quality is poorer in the presence of elevated markers for chronic diseases.

Assessing overall diet quality provides insight into the risk for disease development and progression[46,51]. The HEI indicates compliance to the DGA, which are aimed at the general population[46,52,53]. Core tenants of the DGA include emphasis on fruit and vegetable consumption, whole grains as opposed to refined grains, and lean proteins as opposed to fatty meats[66]. These same core principles are shared across multiple recommended dietary patterns, such as the Dietary Approaches to Stop Hypertension (DASH) eating plan and the Mediterranean diet, as well as in the prevention and treatment of diabetes and other chronic diseases[66,99–101]. Therefore, assessing the overall diet quality through these indices provides insight into the risk of chronic disease development in the general population as well as in adults with diabetes.

US adults have an overall poor diet quality. A previous study found a mean diet quality score (Total HEI-2015) of 58 out of 100 for adults in the general population[56]. Although slightly lower, our findings are in alignment, with a total score of 53 out of 100 for US adults greater than 30 years of age without diabetes. Our findings confirm poor overall diet quality in the general population of US adults.

Furthermore, we place particular interest in the population with diabetes. The presence of diabetes itself serves as a risk factor for morbidity and mortality[13,21,69,70]. In addition, previous studies have identified inverse associations between diet quality and CVD, cancer, and all-cause mortality[45,63,74,75,102]. The simultaneous presence of diabetes and poor diet quality is a cause for concern and warrants the evaluation of diet quality in this population. A prior study identified a Total HEI-2010 score of 57.7 for adults with diabetes, and report higher diet quality in those with the disease than those without[61]. However, the current study reports lower diet quality results of 50.8 in adults with diabetes, supporting our original hypothesis that adults with diabetes have poorer diet quality than their diabetes-free counterparts. In the current analysis, the range of total diet quality scores in all glycemic groups fits into the lowest quintile of diet quality from a previous study, which is associated with 12-28% increased risk for CVD, cancer, and all-cause mortality[74].

A benefit of the current analysis is the ability to examine which dietary elements are responsible for the overall poor diet quality of the diabetes population in addition to identifying which areas are the most successful. One area that the diabetes population reigns in is added sugar consumption. Previous studies have identified conflicting results regarding added sugar intake[10,12,63,67,103]. Our findings align with those reporting significantly lower intakes and moderate adherence to the DGA recommendation in adults with diabetes[12,63,67,103]. These lower intakes are likely due to greater exposure to nutrition education and awareness of dietary sugar's affect on blood glucose[67]. A cross-sectional analysis identified an inverse dose response between duration of diabetes as well as frequency of blood glucose self-monitoring and sugar-sweetened beverage consumpsion, supporting the prior hypotheses[103].

Although added sugar intake is reduced in adults with diabetes, our data inform us of the many remaining areas of concern in this target population that pose as threats to the exacerbation of diabetes, comorbidities, and mortality. Adults with diabetes consume significantly higher amounts of saturated fat than those without diabetes. Considering total protein intakes were fairly consistent across glycemic groups, we infer that adults with diabetes consume more fatty meats, including red meats and processed meats, and more added fats than their diabetes-free counterparts[11]. These results align with those of previous analyses, in which intakes of adults with diabetes surpass the recommended limit of saturated fat[6–9,11,12].

Across all levels of glycemia in our sample, participants have especially poor intakes of whole grains, green vegetables, beans, fruit, seafood, and plant proteins, and excessive sodium intakes. Previously, it has been reported that adults with diabetes do not meet recommended fruit, vegetable, whole grain, and fiber intakes[6–9]. Additionally, their sodium consumption has increased all the while maintaining inadequate potassium intakes[7]. Our findings support the notion that US adults, with and without diabetes, do not meet recommendations for such dietary components. These inadequacies are problematic, as these food categories serve as the main pillars of the DGA, the DASH diet, and the Mediterranean diet, which have been linked to reduced risk for morbitiy and mortality[45,63,74,75]. Conversely, dietary patterns that do not comply with the recommendations may contribute to the development or progression of diabetes and common comorbidities, such as hypertension, hyperlipidemia, and obesity[13,15,68].

The current study found that more than 50% of adults with diabetes have HTN, 30% of which are classified as stage 2. Our rates are slightly lower than previous estimates that state 60-80% of adults with diabetes have concurrent hypertension[1,13,70,83]. Previous research also found that it is twice as common in the diabetes population than in the normal glycemia population[83]. Our findings support that adults with diabetes have double the rate of stage 2 HTN than normal glycemia adults. By following the 2017 ACC/AHA's lowered diagnostic thresholds for elevated blood pressure and both stages of HTN, our data nearly double the proportion of adults with markers of HTN. The benefit to utilizing the 2017 thresholds includes outcomes that reflect most widespread protocol and clinical practice guidelines. Along with prevalence, we found that diet quality worsened in the presence of HTN for all glycemic groups. Considering this study's data suggest noncompliance to key principles of the DASH diet, including emphasis on whole grains, fruits, vegetables, and lean proteins as well as limited sodium and sufficient potassium intake, it can be presumed that diet quality plays a role in the high rates of HTN in this target population[13,104,105].

Epidemiology data associate high intakes of saturated fat and added sugars with worsened total cholesterol values in adults with diabetes[10,12,72,73]. HLD has been reported in more than 70% of adults with diabetes[106]. However, results from the current study differ from previous prevalence estimations and show that only 33-40% of adults with diabetes have HLD, whereas nearly 50% of adults with prediabetes and normal glycemia have elevated cholesterol levels. These findings may be due to use of statin medications in the diabetes population. A national analysis identified that 58-67% of adults with diabetes were on lipid-lowering statin therapy[1]. Medication data were not included in this current analysis due to their unavailability from NHANES at the time of analysis. This absence of medication data inhibits our ability to determine whether cholesterol values are metabolic or pharmacologic. This notion may explain the absence of significant differences in diet quality seen among adults with normal versus elevated total cholesterol levels. However, even in the presence of pharmacologic blood lipid control, diet quality remains to be relevant in the prevention and management of comorbidities and mortality[10,12,72].

Obesity is highly prevalent among the diabetes population, and an increase in BMI is considered to be one of the strongest predictors of diabetes in both sexes[3,38]. In this study, more than 90% of adults with diabetes are either overweight or obese, mirroring previous prevalence estimates[1]. Although more prominent in the diabetes groups, nearly 50% of adults with prediabetes and more than 30% of those without diabetes are obese. These data reflect previous findings regarding weight trends in US adults[107]. Although multifaceted, one potential contributor to the higher overweight and obesity rates in the diabetes groups is use of insulin to manage blood glucose. Weight gain is a common side effect of insulin therapy, and therefore, may contribute to these outcomes[108]. Across all glycemic groups, diet quality was poorer when overweight or obesity was present. The high rates of overweight or obesity and reduced diet quality among all groups, including in normal glycemia and prediabetes adults, are alarming and may forebode disease development and exacerbation[3].

The current study has numerous strengths. Sampling weights produced nationally representative estimates for noninstitutionalized US citizens, increasing the external validity. Generalizability was further increased by the study design of NHANES and its multistage sampling process. Additionally, data from six NHANES cohorts were pooled to generate stronger results. One limitation is the use of data from only one day of intake per participant. However, the large sample size of the current analysis allows for cumulative results. Additionally, dietary data were gathered from 24-hour dietary recalls utilizing the Automated Multiple Pass Method, increasing the accuracy of the recall.

Also, the 2017 ACC/AHA guidelines recommend averaging blood pressure readings from two or more separate visits, whereas all three readings were taken in one single visit to the MEC in NHANES. Lastly, the absence of prescription medication data serves as a limitation in preventing us from determining which markers of chronic diseases are metabolic versus pharmacologic. Our data may underrepresent the proportion of adults with HLD that are masked by pharmacologic control.

Conclusions

Poorer diet quality is associated with greater risk for CVD, cancer, and all-cause mortality in the general population[45,63,74,75]. Presence of diabetes and the comorbidity triad of HTN, HLD, and obesity further increase these risks[3,4,20,106]. Our findings support the notion that adults with diabetes and additional chronic diseases have poorer diet quality than the general population. These simultaneous risk factors for morbidity and mortality create a sense of urgency for lifestyle interventions to target diet quality in adults with diabetes and comorbidities. Acknowledging the aspects of diet quality in which adults with diabetes are doing well and are doing poor allows for better targeted nutrition therapy, improving the overall diet quality, and in turn, reducing morbidity and mortality.

		Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)
		n (%)			
Gender	Male	7165 (47.1%)	2881 (46.5%)	484 (52.1%)	1071 (53.7%)
	Female	7676 (52.9%)	3042 (53.5%)	439 (47.9%)	950 (46.3%)
Weight Status	UW (<18.5)	234 (1.4%)	58 (0.9%)	0 (0%)	3 (0.2%)
	NW (18.5-24.9)	4447 (30.6%)	1018 (17.8%)	98 (8.3%)	248 (10.6%)
	OW (25-29.9)	5365 (36.7%)	1983 (32.8%)	253 (25.2%)	518 (23.5%)
	OB (≥30)	4655 (31.2%)	2796 (48.4%)	556 (66.5%)	1205 (65.7%)
Marital Status	Single/Widowed/ Divorced	5095 (30.7%)	2353 (35.7%)	359 (36%)	790 (35.5%)
	Married/Living as Married	9741 (69.3%)	3569 (64.3%)	564 (64%)	1228 (64.5%)
Education Level	<9th grade	1425 (4.5%)	880 (8.6%)	170 (11.4%)	424 (13.3%)
	9-11th grade	1976 (9.5%)	927 (13.3%)	172 (16.9%)	348 (14.1%)
	High School/GED	3233 (21.4%)	1467 (26.4%)	208 (22.9%)	461 (26%)
	Some college or AA degree	4156 (30.3%)	1541 (28.7%)	226 (29.8%)	528 (30.1%)
	College graduate	4042 (34.3%)	1101 (23.1%)	147 (19.1%)	255 (16.5%)
Total Cholesterol	Desirable (<200)	7849 (50.6%)	3171 (52.8%)	612 (67.2%)	1192 (60.4%)
	Moderate risk (200-239)	4705 (33.5%)	1778 (29.9%)	202 (22.8%)	477 (23.8%)
	High risk (≥240)	2150 (16%)	903 (17.3%)	88 (10%)	316 (15.8%)
Blood Pressure	Normal (<120/<80)	6414 (47.1%)	1709 (32%)	240 (25.4%)	494 (28%)
	Elevated (120-129/<80)	2336 (16.1%)	1027 (18.4%)	162 (19.2%)	350 (19.3%)
	Stage 1 (130-139 or 80-89)	2910 (21.3%)	1382 (24.8%)	196 (24.7%)	430 (21.3%)
	Stage 2 (≥ 140 or ≥ 90)	2479 (15.4%)	1485 (24.9%)	271 (30.8%)	626 (31.4%)
		Mean (SE)			
Age (years)		50.5 (0.2)	59.6 (0.3)	61.4 (0.6)	58.6 (0.4)

Unweighted counts and population percentages.

Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).

Table 11. Demographic Characteristics of Sample Population by Level of Glycemic Control

HEI-2015 Score (Score range)	Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)	P
	Adjusted means (SEM)				
Total Fruit (0-5)	2.3 (0.03)[a]	2.2 (0.05)[b]	2.0 (0.10) [b]	2.1 (0.07) [b]	0.011
Whole Fruit (0-5)	2.3 (0.03)	2.2 (0.05)	2.1 (0.10)	2.1 (0.08)	0.330
Total Vegetables (0-5)	3.2 (0.02)	3.2 (0.04)	3.2 (0.08)	3.2 (0.06)	1.000
Greens and Beans (0-5)	1.7 (0.04)	1.6 (0.04)	1.5 (0.10)	1.5 (0.07)	0.061
Whole Grains (0-10)	2.6 (0.04)	2.5 (0.08)	2.8 (0.16)	2.6 (0.12)	0.764
Dairy (0-10)	4.9 (0.05)[a]	4.8 (0.06)[a]	5.4 (0.13)[b]	4.9 (0.10)[a]	0.007
Total Protein Foods (0-5)	4.3 (0.01)[a]	4.3 (0.03)[ab]	4.3 (0.07)[ab]	4.4 (0.04)[b]	0.046
Seafood and Plant Proteins (0-5)	2.5 (0.03)[a]	2.4 (0.05)[b]	2.4 (0.12)[ab]	2.3 (0.08)[b]	0.018
Fatty Acids (0-10)	5.3 (0.06)	5.1 (0.07)	5.1 (0.15)	5.2 (0.13)	1.000
Refined Grains[1] (0-10)	6.4 (0.05)[a]	6.0 (0.08)[b]	5.5 (0.17)[b]	5.7 (0.12)[b]	<0.001
Sodium[1] (0-10)	4.6 (0.04)[a]	4.0 (0.08)[b]	3.4 (0.18)[c]	3.5 (0.12)[c]	<0.001
Added Sugars[1] (0-10)	6.8 (0.05)[a]	6.7 (0.08)[a]	7.5 (0.14)[b]	7.7 (0.12)[b]	<0.001
Saturated Fat[1] (0-10)	6.3 (0.05)[a]	5.9 (0.08)[b]	5.6 (0.14)[c]	5.6 (0.13)[c]	<0.001
Total HEI Score (0-100)	**53 (0.24)[a]**	**50.8 (0.29)[b]**	**50.8 (0.6)[b]**	**50.7 (0.39)[b]**	**<0.001**

Adjusted for age, race, ethnicity, sex, marital status, and percent of federal poverty rate.
Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).
[1]Moderation components; higher scores indicate lower consumption.

Table 12. Differences in Healthy Eating Index 2015 Scores by Level of Glycemic Control in US Adults

		Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)	
		n (%)				P
Cholesterol	Desirable (<200)	7849 (50.6%)	3171 (52.8%)	612 (67.2%)	1192 (60.4%)	<0.001
	Moderate (200-239)	4705 (33.5%)	1778 (29.9%)	202 (22.8%)	477 (23.8%)	
	High (≥240)	2150 (16.0%)	903 (17.3%)	88 (10.0%)	316 (15.8%)	
Blood pressure	Normal (<120/<80)	6414 (47.1%)	1709 (32.0%)	240 (25.4%)	494 (28.0%)	<0.001
	Elevated (120-129/<80)	2336 (16.1%)	1027 (18.4%)	162 (19.2%)	350 (19.3%)	
	Stage 1 (130-139 or 80-89)	2910 (21.3%)	1382 (24.8%)	196 (24.7%)	430 (21.3%)	
	Stage 2 (≥140 or ≥90)	2479 (15.4%)	1485 (24.9%)	271 (30.8%)	626 (31.4%)	
Obesity	UW (<18.5)	234 (1.4%)	58 (0.9%)	0 (0.0%)	3 (0.2%)	<0.001
	NW (18.5-24.9)	4447 (30.6%)	1018 (17.8%)	98 (8.3%)	248 (10.6%)	
	OW (25-29.9)	5365 (36.7%)	1983 (32.8%)	253 (25.2%)	518 (23.5%)	
	OB (≥30)	4655 (31.2%)	2796 (48.4%)	556 (66.5%)	1205 (65.7%)	

Adjusted for age, race, ethnicity, sex, marital status, and percent of federal poverty rate.
Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).

Table 13. Prevalence of Markers of Chronic Diseases by Level of Glycemic Control in US Adults

Chronic Disease (Measure)		Normal Glycemia (n=14,481)	Prediabetes (n=5,923)	Controlled Diabetes (n=923)	Poorly Controlled Diabetes (n=2,021)
		Adjusted means (SEM)			
Hyperlipidemia (Total cholesterol)	Normal	51.9 (0.3)	51.4 (0.3)	50.4 (0.7)	50.8 (0.5)
	Elevated[a]	51.9 (0.3)	51.1 (0.4)	53.2 (1.2)	51.5 (0.6)
Hypertension (Blood pressure)	Normal	52.8 (0.3)*	52.0 (0.5)	53.4 (1.1)	51.3 (0.7)
	Elevated[b]	51.2 (0.3)	51.0 (0.3)	50.9 (0.8)	50.9 (0.5)
Overweight or Obesity (BMI)	Normal	53.9 (0.3)*	53.0 (0.7)*	52.7 (1.9)	51.5 (1.3)
	Elevated[c]	51.0 (0.2)	50.8 (0.3)	51.3 (0.6)	50.9 (0.4)

Adjusted for age, race, ethnicity, sex, marital status, and percent of federal poverty rate.
Glycemic control evaluated as: Normal glycemia (<5.7%); prediabetes (5.7-6.4%); controlled diabetes (6.5-6.9%); poorly controlled diabetes (≥7%).
[a]Elevated total cholesterol: ≥200 mg/dL.
[b]Elevated blood pressure: ≥120/<80 mm Hg, systolic blood pressure ≥130 mm Hg, or diastolic blood pressure >80 mm Hg.
[c]Elevated BMI: overweight/obesity categorized by BMI values of ≥25 kg/m^2.
*P<0.05

Table 14. Differences in Total Healthy Eating Index 2015 Scores across Glycemic Level by Markers of Chronic Diseases in US Adults

References

1. Centers for Disease Control and Prevention. National Diabetes Statistics Report, 2017. Centers for Disease Control and Prevention, U.S. Department of Health and Human Services. 2017:1-20.

2. Wild S, Roglic G, Green A, Sicree R, King H. Global Prevalence of Diabetes: Estimates for the year 2000 and projections for 2030. *Diabetes Care*. 2004;27(5):1047-1053. doi:10.2337/diacare.27.5.1047

3. Zimmet P, Alberti KGMM, Shaw J. Global and societal implications of the diabetes epidemic. *Nature*. 2001;414(6865):782-787. doi:10.1038/414782a

4. Wu Y, Ding Y, Tanaka Y, Zhang W. Risk Factors Contributing to Type 2 Diabetes and Recent Advances in the Treatment and Prevention. *Int J Med Sci*. 2014;11(11):1185-1200. doi:10.7150/ijms.10001

5. Tuomilehto J, Lindström J, Eriksson JG, et al. Prevention of Type 2 Diabetes Mellitus by Changes in Lifestyle among Subjects with Impaired Glucose Tolerance. *New England Journal of Medicine*. 2001;344(18):1343-1350. doi:10.1056/NEJM200105033441801

6. Nelson KM, Reiber G, Boyko EJ. Diet and Exercise Among Adults With Type 2 Diabetes: Findings from the Third National Health and Nutrition Examination Survey (NHANES III). *Diabetes Care*. 2002;25(10):1722-1728. doi:10.2337/diacare.25.10.1722

7. Casagrande SS, Cowie CC. Trends in dietary intake among adults with type 2 diabetes: NHANES 1988-2012. *Journal of Human Nutrition and Dietetics*. 2017;30(4):479-489. doi:10.1111/jhn.12443

8. Oza-Frank R, Cheng YJ, Narayan KMV, Gregg EW. Trends in Nutrient Intake among Adults with Diabetes in the United States: 1988-2004. *Journal of the American Dietetic Association*. 2009;109(7):1173-1178. doi:10.1016/j.jada.2009.04.007

9. Eilat-Adar S, Xu J, Zephier E, O'Leary V, Howard BV, Resnick HE. Adherence to Dietary Recommendations for Saturated Fat, Fiber, and Sodium Is Low in American Indians and Other U.S. Adults with Diabetes. *The Journal of Nutrition*. 2008;138(9):1699-1704. doi:10.1093/jn/138.9.1699

10. Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. Added Sugar Intake and Cardiovascular Diseases Mortality Among US Adults. *JAMA Internal Medicine*. 2014;174(4):516. doi:10.1001/jamainternmed.2013.13563

11. Castetbon K, Bonaldi C, Deschamps V, et al. Diet in 45- to 74-Year-Old Individuals with Diagnosed Diabetes: Comparison to Counterparts without Diabetes in a Nationally Representative Survey (Etude Nationale Nutrition Santé 2006-2007). *Journal of the Academy of Nutrition and Dietetics*. 2014;114(6):918-925. doi:10.1016/j.jand.2013.08.002

12. Vitale M, Masulli M, Cocozza S, et al. Sex differences in food choices, adherence to dietary recommendations and plasma lipid profile in type 2 diabetes – The TOSCA.IT study. *Nutrition, Metabolism and Cardiovascular Diseases*. 2016;26(10):879-885. doi:10.1016/j.numecd.2016.04.006

13. Provenzano LF, Stark S, Steenkiste A, Piraino B, Sevick MA. Dietary Sodium Intake in Type 2 Diabetes. *Clinical Diabetes*. 2014;32(3):106-112. doi:10.2337/diaclin.32.3.106

14. Bowman SA, Clemens JC, Martin CL, Anand J, Steinfeldt LC, Moshfegh AJ. Added Sugars Intake of Americans: What We Eat in America, NHANES 2013-2014. 2017;(18):9.

15. McCulloch DK, Robertson RP. Pathogeneses of type 2 diabetes mellitus. UpToDate. https://www-uptodate-com.proxy.lib.ohio-state.edu/contents/pathogenesis-of-type-2-diabetes-mellitus?search=type%202%20diabetes%20pathophysiology&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1. Published 2016. Accessed August 3, 2018.

16. IDF Diabetes Atlas Eighth Edition 2017. *International Diabetes Federation*.

17. Cornell S. Continual evolution of type 2 diabetes: an update on pathophysiology and emerging treatment options. *Therapeutics and Clinical Risk Management*. April 2015:621. doi:10.2147/TCRM.S67387

18. American Diabetes Association. 2. Classification and Diagnosis of Diabetes: *Standards of Medical Care in Diabetes—2018*. *Diabetes Care*. 2018;41(Supplement 1):S13-S27. doi:10.2337/dc18-S002

19. International Diabetes Federation. *Diabetes Atlas*. Brussels: IDF Executive Office; 2000.

20. Chatterjee S, Khunti K, Davies MJ. Type 2 diabetes. *The Lancet*. 2017;389(10085):2239-2251. doi:10.1016/S0140-6736(17)30058-2

21. Garber AJ, Abrahamson MJ, Barzilay JI, et al. CONSENSUS STATEMENT BY THE AMERICAN ASSOCIATION OF CLINICAL ENDOCRINOLOGISTS AND AMERICAN COLLEGE OF ENDOCRINOLOGY ON THE COMPREHENSIVE TYPE 2 DIABETES MANAGEMENT ALGORITHM – *2017 EXECUTIVE SUMMARY*. *Endocrine Practice*. 2017;23(2):207-238. doi:10.4158/EP161682.CS

22. Gastaldelli A. Role of beta-cell dysfunction, ectopic fat accumulation and insulin resistance in the pathogenesis of type 2 diabetes mellitus. *Diabetes Research and Clinical Practice*. 2011;93:S60-S65. doi:10.1016/S0168-8227(11)70015-8

23. Kahn SE, Cooper ME, Del Prato S. Pathophysiology and treatment of type 2 diabetes: perspectives on the past, present, and future. *The Lancet*. 2014;383(9922):1068-1083. doi:10.1016/S0140-6736(13)62154-6

24. International Diabetes Federation. *IDF Diabetes Atlas*. Brussels: International Diabetes Federation, Executive Office; 2011.

25. Giovannucci E, Harlan DM, Archer MC, et al. Diabetes and Cancer: A Consensus Report. *CA: A Cancer Journal for Clinicians*. 2010;60(4):207-221. doi:10.3322/caac.20078

26. Brownlee M. The Pathobiology of Diabetic Complications: A Unifying Mechanism. *Diabetes*. 2005;54(6):1615-1625. doi:10.2337/diabetes.54.6.1615

27. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. *Nature*. 2001;414(6865):813-820. doi:10.1038/414813a

28. The InterAct Consortium. The link between family history and risk of type 2 diabetes is not explained by anthropometric, lifestyle or genetic risk factors: the EPIC-InterAct study. *Diabetologia*. 2013;56(1):60-69. doi:10.1007/s00125-012-2715-x

29. Meigs JB, Cupples LA, Wilson PW. Parental transmission of type 2 diabetes: the Framingham Offspring Study. *Diabetes*. 2000;49(12):2201-2207. doi:10.2337/diabetes.49.12.2201

30. Nolan CJ, Damm P, Prentki M. Type 2 diabetes across generations: from pathophysiology to prevention and management. *The Lancet*. 2011;378(9786):169-181. doi:10.1016/S0140-6736(11)60614-4

31. Harris MI, Flegal KM, Cowie CC, et al. Prevalence of Diabetes, Impaired Fasting Glucose, and Impaired Glucose Tolerance in U.S. Adults: The Third National Health and Nutrition Examination Survey, 1988-1994. *Diabetes Care*. 1998;21(4):518-524. doi:10.2337/diacare.21.4.518

32. Mokdad AH, Ford ES, Bowman BA, et al. Prevalence of Obesity, Diabetes, and Obesity-Related Health Risk Factors, 2001. *JAMA*. 2003;289(1). doi:10.1001/jama.289.1.76

33. Mokdad AH, Ford ES, Bowman BA, et al. Diabetes trends in the U.S.: 1990-1998. *Diabetes Care*. 2000;23(9):1278-1283. doi:10.2337/diacare.23.9.1278

34. Sim SY, Chin SL, Tan JL, Brown SJ, Cussons AJ, Stuckey BG. Polycystic ovary syndrome in type 2 diabetes: does it predict a more severe phenotype? *Fertility and sterility*. 2016;106(5):1258-1263.

35. Shai I, Jiang R, Manson JE, et al. Ethnicity, Obesity, and Risk of Type 2 Diabetes in Women. *Diabetes Care*. 2006;29(7):1585. doi:10.2337/dc06-0057

36. Menke A, Casagrande S, Geiss L, Cowie CC. Prevalence of and Trends in Diabetes Among Adults in the United States, 1988-2012. *JAMA*. 2015;314(10):1021. doi:10.1001/jama.2015.10029

37. Bancks MP, Kershaw K, Carson AP, Gordon-Larsen P, Schreiner PJ, Carnethon MR. Association of Modifiable Risk Factors in Young Adulthood With Racial Disparity in Incident Type 2 Diabetes During Middle Adulthood. *JAMA*. 2017;318(24):2457. doi:10.1001/jama.2017.19546

38. Menke A, Rust K, Fradkin J, Cheng Y, Cowie C. Associations Between Trends in Race/Ethnicity, Aging, and Body Mass Index With Diabetes Prevalence in the United States: A Series of Cross-sectional Studies. *Annals of Internal Medicine*. 2014;161(5):328-335. doi:10.7326/M14-0286

39. McCulloch DK, Robertson RP. Risk factors for type 2 diabetes mellitus. UpToDate. https://www-uptodate-com.proxy.lib.ohio-state.edu/contents/risk-factors-for-type-2-diabetes-mellitus/print?search=type%202%20diabetes%20risk%20factors&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1. Published 2018. Accessed August 5, 2018.

40. Roglic G, World Health Organization, eds. *Global Report on Diabetes*. Geneva, Switzerland: World Health Organization; 2016.

41. Crump C, Sundquist J, Winkleby MA, Sieh W, Sundquist K. Physical Fitness Among Swedish Military Conscripts and Long-Term Risk for Type 2 Diabetes Mellitus: A Cohort Study. *Annals of Internal Medicine*. 2016;164(9):577-584. doi:10.7326/M15-2002

42. McEvoy CT, Cardwell CR, Woodside JV, Young IS, Hunter SJ, McKinley MC. A Posteriori Dietary Patterns Are Related to Risk of Type 2 Diabetes: Findings from a Systematic Review and Meta-Analysis. *Journal of the Academy of Nutrition and Dietetics*. 2014;114(11):1759-1775.e4. doi:10.1016/j.jand.2014.05.001

43. Nathan DM, Davidson MB, DeFronzo RA, et al. Impaired Fasting Glucose and Impaired Glucose Tolerance. *Diabetes Care*. 2007;30(3):753. doi:10.2337/dc07-9920

44. Shan Z, Ma H, Xie M, et al. Sleep Duration and Risk of Type 2 Diabetes: A Meta-analysis of Prospective Studies. *Diabetes Care*. 2015;38(3):529-537. doi:10.2337/dc14-2073

45. Heidemann C, Hoffmann K, Spranger J, et al. A dietary pattern protective against type 2 diabetes in the European Prospective Investigation into Cancer and Nutrition (EPIC)—Potsdam Study cohort. *Diabetologia*. 2005;48(6):1126-1134. doi:10.1007/s00125-005-1743-1

46. Schwingshackl L, Hoffmann G. Diet Quality as Assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension Score, and Health Outcomes: A Systematic Review and Meta-Analysis of Cohort Studies. *Journal of the Academy of Nutrition and Dietetics*. 2015;115(5):780-800.e5. doi:10.1016/j.jand.2014.12.009

47. Jannasch F, Kröger J, Schulze MB. Dietary Patterns and Type 2 Diabetes: A Systematic Literature Review and Meta-Analysis of Prospective Studies. *The Journal of Nutrition*. 2017;147(6):1174-1182. doi:10.3945/jn.116.242552

48. Ley SH, Hamdy O, Mohan V, Hu FB. Prevention and management of type 2 diabetes: dietary components and nutritional strategies. *The Lancet*. 2014;383(9933):1999-2007. doi:10.1016/S0140-6736(14)60613-9

49. Ley SH, Pan A, Li Y, et al. Changes in Overall Diet Quality and Subsequent Type 2 Diabetes Risk: Three U.S. Prospective Cohorts. *Diabetes Care*. 2016;39(11):2011-2018. doi:10.2337/dc16-0574

50. Mazidi M, Katsiki N, Kengne AP, Mikhailidis DP, Banach M. Adiposity mediates the association between whole grain consumption, glucose homeostasis and insulin resistance: findings from the US NHANES. *Lipids in Health and Disease*. 2018;17(1). doi:10.1186/s12944-018-0805-6

51. Fung TT, McCullough ML, Newby P, et al. Diet-quality scores and plasma concentrations of markers of inflammation and endothelial dysfunction1–3. 2018:11.

52. Reedy J, Lerman JL, Krebs-Smith SM, Kirkpatrick SI. Evaluation of the Healthy Eating Index-2015. *Journal of the Academy of Nutrition and Dietetics*. 2018;118(9):1622-1633. doi:10.1016/j.jand.2018.05.019

53. Krebs-Smith SM, Pannucci TE, Subar AF, et al. Update of the Healthy Eating Index: HEI-2015. *Journal of the Academy of Nutrition and Dietetics*. 2018;118(9):1591-1602. doi:10.1016/j.jand.2018.05.021

54. Banfield EC, Liu Y, Davis JS, Chang S, Frazier-Wood AC. Poor Adherence to US Dietary Guidelines for Children and Adolescents in the National Health and Nutrition Examination Survey Population. *Journal of the Academy of Nutrition and Dietetics*. 2016;116(1):21-27. doi:10.1016/j.jand.2015.08.010

55. Bowman SA, Lino M, Gerrior SA, Basiotis PP. *The Healthy Eating Index: 1994-96*. United States Department of Agriculture, Center for Nutrition Policy and Promotion; 1998.

56. USDA. Healthy Eating Index. United States Department of Agriculture: Center for Nutrition Policy and Promotion. https://www.cnpp.usda.gov/hei-scores-americans.

57. Guo X, Warden BA, Paeratakul S, Bray GA. Healthy Eating Index and obesity. *European Journal of Clinical Nutrition*. 2004;58(12):1580-1586. doi:10.1038/sj.ejcn.1601989

58. Hiza HAB, Casavale KO, Guenther PM, Davis CA. Diet Quality of Americans Differs by Age, Sex, Race/Ethnicity, Income, and Education Level. *Journal of the Academy of Nutrition and Dietetics*. 2013;113(2):297-306. doi:10.1016/j.jand.2012.08.011

59. Wilson MM, Reedy J, Krebs-Smith SM. American Diet Quality: Where It Is, Where It Is Heading, and What It Could Be. *Journal of the Academy of Nutrition and Dietetics*. 2016;116(2):302-310.e1. doi:10.1016/j.jand.2015.09.020

60. Rehm CD, Peñalvo JL, Afshin A, Mozaffarian D. Dietary Intake Among US Adults, 1999-2012. *JAMA*. 2016;315(23):2542. doi:10.1001/jama.2016.7491

61. Nowlin SY, Cleland CM, Vadiveloo M, D’Eramo Melkus G, Parekh N, Hagan H. Explaining Racial/Ethnic Dietary Patterns in Relation to Type 2 Diabetes: An Analysis of NHANES 2007-2012. *Ethnicity & Disease*. 2016;26(4):529. doi:10.18865/ed.26.4.529

62. Avedzi HM, Mathe N, Storey K, Johnson JA, Johnson ST. Examining sex differences in glycemic index knowledge and intake among individuals with type 2 diabetes. *Primary Care Diabetes*. 2018;12(1):71-79. doi:10.1016/j.pcd.2017.07.005

63. Panizza C, Shvetsov Y, Harmon B, et al. Testing the Predictive Validity of the Healthy Eating Index-2015 in the Multiethnic Cohort: Is the Score Associated with a Reduced Risk of All-Cause and Cause-Specific Mortality? *Nutrients*. 2018;10(4):452. doi:10.3390/nu10040452

64. Mathe N, Pisa PT, Johnson JA, Johnson ST. Dietary Patterns in Adults with Type 2 Diabetes Predict Cardiometabolic Risk Factors. *Canadian Journal of Diabetes*. 2016;40(4):296-303. doi:10.1016/j.jcjd.2015.11.006

65. ChooseMyPlate.gov. United States Department of Agriculture: ChooseMyPlate.gov. https://www.choosemyplate.gov/. Published January 2018.

66. U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015-2020 Dietary Guidelines for Americans. 2015;8th Edition:144.

67. Petersen KS, Blanch N, Wepener RH, Clifton PM, Keogh JB. Dietary quality in people with type 1 and type 2 diabetes compared to age, sex and BMI matched controls. *Diabetes Research and Clinical Practice*. 2015;107(2):e7-e10. doi:10.1016/j.diabres.2014.12.001

68. Taskinen M-R, Borén J. New insights into the pathophysiology of dyslipidemia in type 2 diabetes. *Atherosclerosis*. 2015;239(2):483-495. doi:10.1016/j.atherosclerosis.2015.01.039

69. Fox CS, Coady S, Sorlie PD, et al. Increasing Cardiovascular Disease Burden Due to Diabetes Mellitus: The Framingham Heart Study. *Circulation*. 2007;115(12):1544-1550. doi:10.1161/CIRCULATIONAHA.106.658948

70. Martín-Timón I. Type 2 diabetes and cardiovascular disease: Have all risk factors the same strength? *World Journal of Diabetes*. 2014;5(4):444. doi:10.4239/wjd.v5.i4.444

71. Mahan LK, Escot-Stump S, Raymond JL. Chapter 31: Medical Nutrition Therapy for Diabetes Mellitus and Hypoglycemia of Nondiabetic Origin. In: *Krause's Food and the Nutrition Care Process*. 13th ed. Elsevier; 2012:675-710.

72. Vitale M, Masulli M, Rivellese AA, et al. Influence of dietary fat and carbohydrates proportions on plasma lipids, glucose control and low-grade inflammation in patients with type 2 diabetes—The TOSCA.IT Study. *European Journal of Nutrition*. 2016;55(4):1645-1651. doi:10.1007/s00394-015-0983-1

73. Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. *The American Journal of Clinical Nutrition*. 2013;97(3):505-516. doi:10.3945/ajcn.112.042457

74. Reedy J, Krebs-Smith SM, Miller PE, et al. Higher Diet Quality Is Associated with Decreased Risk of All-Cause, Cardiovascular Disease, and Cancer Mortality among Older Adults. *The Journal of Nutrition*. 2014;144(6):881-889. doi:10.3945/jn.113.189407

75. de Koning L, Chiuve SE, Fung TT, Willett WC, Rimm EB, Hu FB. Diet-Quality Scores and the Risk of Type 2 Diabetes in Men. *Diabetes Care*. 2011;34(5):1150-1156. doi:10.2337/dc10-2352

76. Johnson C, Dohrmann S, Burt V, Mohadjer L. National Center for Health Statistics: Sample Design, 2011-2014. *National Center for Health Statistics*. Vital Health Stat 2(162):1-33.

77. National Center for Health Statistics. About the National Health and Nutrition Examination Survey. Centers for Disease Control and Prevention. https://www.cdc.gov/nchs/nhanes/about_nhanes.htm. Published 2017. Accessed August 26, 2018.

78. CDC. National Health and Nutrition Examination Survey: NCHS Research Ethics Review Board (ERB) Approval. Centers for Disease Control and Prevention. https://www.cdc.gov/nchs/nhanes/irba98.htm. Published November 29, 2017.

79. National Center for Health Statistics. National Health and Nutrition Examination Survey: Welcome NHANES Participants. https://www.cdc.gov/nchs/nhanes/participant.htm. Published June 2016.

80. Anderson G, Horvath J. The Growing Burden of Chronic Disease in America. *Public Health Reports*. 2004;119(3):263-270. doi:10.1016/j.phr.2004.04.005

81. Wolff JL, Starfield B, Anderson G. Prevalence, Expenditures, and Complications of Multiple Chronic Conditions in the Elderly. *Archives of Internal Medicine*. 2002;162(20):2269. doi:10.1001/archinte.162.20.2269

82. CDC. National Health and Nutrition Examination Survey: Physician Examination Procedures Manual. January 2007.

83. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. *Journal of the American College of Cardiology*. 2018;71(19):e127-e248. doi:10.1016/j.jacc.2017.11.006

84. James PA, Oparil S, Carter BL, et al. 2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report From the Panel Members Appointed to the Eighth Joint National Committee (JNC 8). *JAMA*. 2014;311(5):507. doi:10.1001/jama.2013.284427

85. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: (442802008-001). 2004. doi:10.1037/e442802008-001

86. Paul Muntner, Robert M Carey, Samuel Gidding, et al. Potential US Population Impact of the 2017 ACC/AHA High Blood Pressure Guideline. 2018:10.

87. The A1C Test & Diabetes. National Institute of Diabetes and Digestive and Kidney Diseases. https://www.niddk.nih.gov/health-information/diabetes/overview/tests-diagnosis/a1c-test. Published 2018. Accessed August 9, 2018.

88. Rosenson RS, Freeman MW, Saperia GM. Measurement of blood lipids and lipoproteins. UpToDate. https://www-uptodate-com.proxy.lib.ohio-state.edu/contents/measurement-of-blood-lipids-and-lipoproteins?search=lipid%20panel&source=search_result&selectedTitle=2~150&usage_type=default&display_rank=2. Published January 2018.

89. CDC. National Health and Nutrition Examination Survey (NHANES) MEC In-Person Dietary Interviewers Procedures Manual. January 2016.

90. Moshfegh AJ, Rhodes DG, Baer DJ, et al. The US Department of Agriculture Automated Multiple-Pass Method reduces bias in the collection of energy intakes. *The American Journal of Clinical Nutrition*. 2008;88(2):324-332. doi:10.1093/ajcn/88.2.324

91. Rhodes DG, Murayi T, Clemens JC, Baer DJ, Sebastian RS, Moshfegh AJ. The USDA Automated Multiple-Pass Method accurately assesses population sodium intakes. *The American Journal of Clinical Nutrition*. 2013;97(5):958-964. doi:10.3945/ajcn.112.044982

92. Food Surveys Research Group, Beltsville Human Nutrition Research Center, USDA. Food Patterns Equivalents Database 2015-2016. September 2018:2.

93. Kennedy ET, Ohls J, Carlson S, Fleming K. The Healthy Eating Index: Design and applications. *Journal of the American Dietetic Association*. 1995;95(10):1103-1108. doi:https://doi.org/10.1016/S0002-8223(95)00300-2

94. National Institute of Health: National Heart, Lung, and Blood Institute. High Blood Cholesterol. NIH. https://www.nhlbi.nih.gov/health-topics/high-blood-cholesterol.

95. American Diabetes Association. 3. Prevention or Delay of Type 2 Diabetes: *Standards of Medical Care in Diabetes—2019*. *Diabetes Care*. 2019;42(Supplement 1):S29-S33. doi:10.2337/dc19-S003

96. Mottalib A, Salsberg V, Mohd-Yusof B-N, et al. Effects of nutrition therapy on HbA1c and cardiovascular disease risk factors in overweight and obese patients with type 2 diabetes. *Nutrition Journal*. 2018;17(1). doi:10.1186/s12937-018-0351-0

97. Briggs Early K, Stanley K. Position of the Academy of Nutrition and Dietetics: The Role of Medical Nutrition Therapy and Registered Dietitian Nutritionists in the Prevention and Treatment of Prediabetes and Type 2 Diabetes. *Journal of the Academy of Nutrition and Dietetics*. 2018;118(2):343-353. doi:10.1016/j.jand.2017.11.021

98. Pastors JG, Warshaw H, Daly A, Franz M, Kulkarni K. The Evidence for the Effectiveness of Medical Nutrition Therapy in Diabetes Management. *Diabetes Care*. 2002;25(3):608-613. doi:10.2337/diacare.25.3.608

99. Sacks FM, Bray GA, Iii ERM. Effects on Blood Pressure of Reduced Dietary Sodium and the Dietary Approaches to Stop Hypertension (DASH) Diet. *The New England Journal of Medicine*. 2001:8.

100. Mediterranean Dietary Pattern and Prediction of All-Cause Mortality in a US Population: Results From the NIH-AARP Diet and Health Study. *ARCH INTERN MED*. 2007;167(22):8.

101. American Diabetes Association. 5. Lifestyle Management: *Standards of Medical Care in Diabetes—2019*. *Diabetes Care*. 2019;42(Supplement 1):S46-S60. doi:10.2337/dc19-S005

102. Liese AD, Krebs-Smith SM, Subar AF, et al. The Dietary Patterns Methods Project: Synthesis of Findings across Cohorts and Relevance to Dietary Guidance. *The Journal of Nutrition*. 2015;145(3):393-402. doi:10.3945/jn.114.205336

103. Xu F, Park S, Siegel KR. Factors Associated With Frequency of Sugar-Sweetened Beverage Consumption Among US Adults With Diabetes or Prediabetes. *American Journal of Health Promotion*. 2018;32(7):1489-1497. doi:10.1177/0890117117746187

104. Maddock J, Ziauddeen N, Ambrosini GL, Wong A, Hardy R, Ray S. Adherence to a Dietary Approaches to Stop Hypertension (DASH)-type diet over the life course and associated vascular function: a study based on the MRC 1946 British birth cohort. *British Journal of Nutrition*. 2018;119(05):581-589. doi:10.1017/S0007114517003877

105. Azadbakht L, Fard NRP, Karimi M, et al. Effects of the Dietary Approaches to Stop Hypertension (DASH) Eating Plan on Cardiovascular Risks Among Type 2 Diabetic Patients: A randomized crossover clinical trial. *Diabetes Care*. 2011;34(1):55-57. doi:10.2337/dc10-0676

106. Lin P-J, Kent DM, Winn AN, Cohen JT, Neumann PJ. Multiple Chronic Conditions in Type 2 Diabetes Mellitus: Prevalence and Consequences. *THE AMERICAN JOURNAL OF MANAGED CARE*. 2015;21(1):13.

107. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in Obesity Among Adults in the United States, 2005 to 2014. *JAMA*. 2016;315(21):2284. doi:10.1001/jama.2016.6458

108. Russell-Jones D, Khan R. Insulin-associated weight gain in diabetes – causes, effects and coping strategies. *Diabetes, Obesity and Metabolism*. 2007;9(6):799-812. doi:10.1111/j.1463-1326.2006.00686.x

109. Parker AR, Byham-Gray L, Denmark R, Winkle PJ. The Effect of Medical Nutrition Therapy by a Registered Dietitian Nutritionist in Patients with Prediabetes Participating in a Randomized Controlled Clinical Research Trial. *Journal of the Academy of Nutrition and Dietetics*. 2014;114(11):1739-1748. doi:10.1016/j.jand.2014.07.020