Socioeconomic Status, Dietary Patterns, and Cardiovascular Risk Factors in an Aging Population

by

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DEDICATION

I dedicate this dissertation to the people that have given me the greatest strength and courage to rise up stronger after falling.

First and foremost, to my parents, Xiaoling and Dehong – the people that know me better than anyone else in the world. No matter how far away I am from home, your love and support have always been my biggest source of strength. It is what I learned from your resilience in life that enabled me to navigate through the many ups and downs of this marathon.

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ABSTRACT

As the leading cause of death in the United States (U.S.), cardiovascular disease (CVD) is responsible for 17% of national health expenditures. The health and economic burden of CVD is especially high in older adults. As a consequence of population aging, it has been projected by the American Heart Association that by 2030, 40.5% of the U.S. population will have some form of CVD, assuming no change to current prevention strategies. In order to address this rising public health issue with more effective prevention strategies, a better understanding of CVD risk factors and their interrelationship is needed.

Obesity, type 2 diabetes (T2D), and hypertension (HTN) are each a leading and proximal CVD risk factor. In addition, they tend to coexist with each other and form multimorbidities, which are associated with substantially further elevated CVD risk. Using a life course approach, this dissertation examined the complex interrelationship among childhood socioeconomic status (SES), adulthood SES, adulthood dietary patterns, and the health outcomes of obesity, T2D, and HTN, individually and in the form of multimorbidities. Each of the three aims in this dissertation used a cross-sectional study design and was based on data from the Health and Retirement Study (HRS), a large nationally representative longitudinal study of U.S. adults aged over 50. Indicators of childhood SES included paternal education, maternal education and childhood financial strain. Indicators of adulthood SES included education and wealth. Two dietary patterns, prudent and Western, were initially identified using principal component analysis (PCA).

The first aim examined the associations of childhood SES with adulthood dietary patterns using linear regression models. Results of this aim showed that, in selected sociodemographic groups, higher paternal education is associated with healthier dietary habits in adulthood, independent of adulthood SES. The second aim examined the associations of adulthood dietary patterns with obesity, T2D, and HTN, separately, accounting for potential confounding by childhood and adulthood SES and using logistic regression models. Under the potential influence of reverse causation and survival bias, results of this aim showed positive associations of both the prudent and Western dietary patterns with the health outcomes. SES, whether in childhood or in adulthood, did not appear to be a strong confounder to these associations. The third aim used multinomial logistic regression models to explore the associations of childhood SES, adulthood SES, and adulthood dietary patterns with the multimorbidity of obesity, T2D, and HTN. Results of this aim showed that the multimorbidity of obesity, T2D, and HTN is highly prevalent among older U.S. adults, and under the potential influence of reverse causation and survival bias, it is independently associated with lower paternal education, lower participant's education and wealth, and greater adherence to the prudent and Western dietary patterns.

Overall, this dissertation provides further evidence that childhood environment and conditions may underlie adulthood dietary habits and proximal CVD risk factors (e.g. obesity, T2D, and HTN), individually and in the form of multimorbidity. More studies, especially prospective studies, are needed to further evaluate the potential impact of paternal education on later life CVD risk, as well as whether dietary behavior is an underlying pathway linking childhood SES to later life CVD risk.

CHAPTER I: Introduction

Overview

CVD is the leading cause of death in the U.S. and the most costly contributor to national health expenditures (1, 2). The health and economic burden of CVD is especially high in older adults (3). As the consequence of population aging, by 2030, 21% of the U.S. population will be 65 years and older and 40.5% of the U.S. population will have some form of CVD, which is associated with a three-fold increase in real total direct medical costs and a 61% increase in real indirect cost compared to 2010 (4, 5). In order to address this public health issue with more effective prevention strategies, a better understanding of the interrelationship among CVD risk factors is needed.

While the traditional adult lifestyle model of CVD risk hypothesizes that behaviors during adulthood cause CVD via proximal risk factors, the life course model of CVD risk further incorporates the contribution of early-life factors (6). Using a life course approach, this dissertation examined the complex interrelationship among childhood SES, adulthood SES, adulthood dietary patterns, and leading proximal CVD risk factors, including obesity, T2D, and HTN, both individually and in the form of multimorbidity (7).

This dissertation contains three specific aims, which are presented in the following section. Each aim was examined with a cross-sectional study design and based on data from the HRS, a large

nationally representative longitudinal study of U.S. adults aged over 50. All analyses took into account the complex survey design of HRS.

Research aims

In order to extend current understanding of life course CVD risk, especially the complex interrelationship among childhood SES, adulthood SES, adulthood dietary patterns, and leading CVD risk factors, the three aims of this dissertation are as follows:

Aim 1: To identify the main dietary patterns in older U.S. adults and estimate the associations of these dietary patterns with childhood SES, taking into consideration the potential mediation and/or effect modification by adulthood SES and effect modification and/or confounding by age, sex, and race.

Aim 2: To examine the associations of adulthood dietary patterns with obesity, T2D, and HTN, separately, in older U.S. adults, before and after adjusting for childhood SES and adulthood SES and confounding by age, sex, race, smoking, alcohol intake, and physical activity.

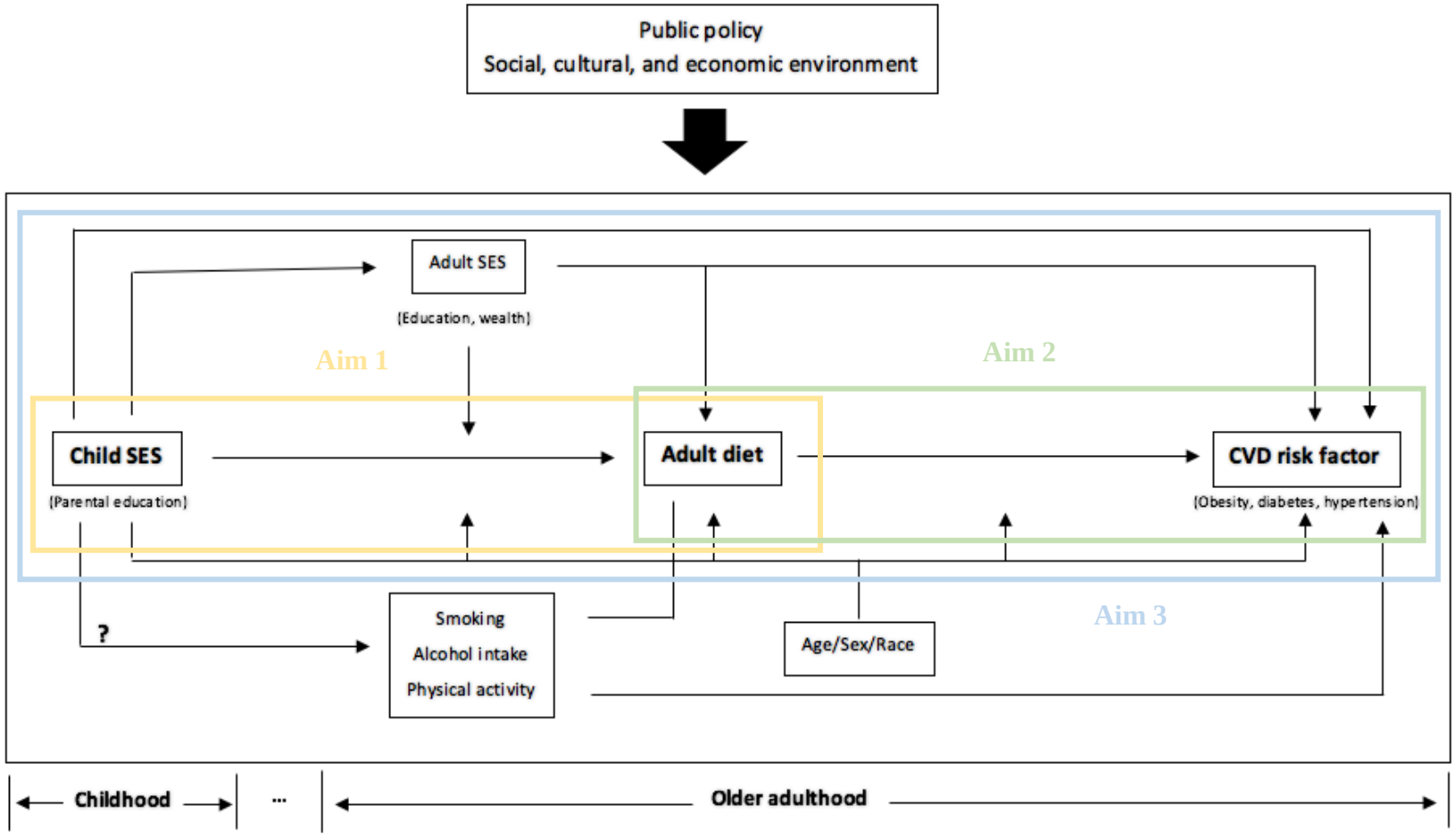
Aim 3: To explore the association of childhood SES, adulthood SES, and adulthood dietary patterns with the multimorbidity of obesity, T2D, and HTN in older U.S. adults, taking into consideration the potential confounding by age, sex, and race.

Conceptual framework

Prior knowledge related to the interrelationships among childhood SES, adulthood dietary patterns, and obesity, T2D, and HTN is illustrated in **Figure 1**. Briefly, childhood SES has been associated with obesity, T2D, and HTN (8-11); however, its associations with adulthood dietary patterns have not been well understood. If childhood SES is indeed associated with adulthood dietary patterns, it could serve as a confounder to the known associations of adulthood dietary patterns with obesity, T2D, and HTN, and therefore it would be worthwhile to reevaluate these known associations. Additionally, obesity, T2D, and HTN tend to coexist with each other (12). Risk factors of patterns of multimorbidity are largely unknown and, therefore, worth exploring.

There are three groups of important covariates in this conceptual model. First, adulthood SES can be influenced by childhood SES and has been shown to be a predictor of adulthood dietary patterns and CVD risk (8, 13-15). Therefore, it is a potential mediator for the childhood SES – adulthood dietary pattern association and confounder for the adulthood dietary pattern – CVD risk factor association. Additionally, adulthood SES is a potential effect modifier for the childhood SES – adulthood dietary pattern association (14, 15). Second, smoking, alcohol intake, and physical activity are known confounders for the associations between dietary patterns and CVD risk, as they are independent predictors of CVD and at the same time associated with dietary patterns (16, 17). Third, age, sex, and race are included as confounders, as SES, dietary patterns, and CVD risk all vary by social groups, and as effect modifiers, as SES can interact with different social groups (18-20).

Figure 1. CVD risk from a life-course perspective



Background

Life course conceptual models linking childhood SES to CVD risk

For decades, studies of chronic disease etiology have primarily identified CVD risk factors based on the adult lifestyle model and the fetal origins hypothesis (6). While the former approach focuses entirely on the effects of adult behaviors (e.g. diet, physical activity, smoking, alcohol intake, etc.), the latter approach focuses almost exclusively on the long-term effects of in utero biological programming (6, 21). The more recent life course approach to chronic disease bridges the adult lifestyle model and the fetal origins hypothesis by studying the contribution of early-life factors jointly with later-life factors to identify risk and protective processes across the life course (6).

Individuals with lower SES in childhood have consistently been found to have elevated risk of CVD or CVD risk factors (e.g. obesity, T2D, and HTN) in adulthood (8-11, 22). However, little is known about its underlying mechanism. Possible life course conceptual models that have been proposed include: 1) the latent effects model (hypothesizing that childhood is a critical period where exposure to low SES has lasting effect of elevated risk of CVD in adulthood, independent of intervening adulthood SES, lifestyle, or traditional CVD risk factors), 2) the pathway model (hypothesizing that low childhood SES leads to unfavorable developmental trajectories of physiologic and psychosocial processes and health-related behaviors, which then proceed through adulthood and lead to more proximal causes of CVD), 3) the social mobility model (hypothesizing that deprivation in early life followed by later affluence combine to produce elevated CVD risk in adulthood), and 4) the cumulative model (hypothesizing that a higher total dosage of adverse psychosocial/physiological experiences and environments related to low SES

in childhood increases adult CVD risk, regardless of timing of experience) (8, 13, 23). No single conceptual model has been strongly supported over the others, and more than one model could apply (8, 23).

Obesity, T2D, and HTN as leading CVD risk factors

CVD is the leading cause of death in the U.S., and obesity, T2D, and HTN are its leading risk factors (1, 7). Obesity, T2D, and HTN have each been associated with a significantly elevated risk of CVD in prior studies. For example, a 44-year follow-up cohort study among Framingham Heart Study participants (n=5209, male and female residents of the town of Framingham, Massachusetts, U.S.) aged 35 to 75 years showed that, compared to being normal weight (body mass index (BMI) 18.5 – 24.9 kg/m^2), being obese was associated with a 1.38 times higher risk of total CVD (95% confidence interval (CI): 1.12-1.69 for men, 1.14-1.68 for women), after adjustment for age, smoking, HTN, hypercholesterolemia, and T2D (24). A 20-year follow-up cohort study among the Framingham Heart Study participants aged 45 to 74 years showed that, compared to nondiabetics, diabetic men and women had 2.1 and 2.0 times, respectively, higher risk of CVD, after adjusting for age, systolic blood pressure, cigarettes per day, cholesterol, and left ventricular hypertrophy – electrocardiogram (25). A cohort study of 10.8 years of median follow-up of the Physicians' Health Study participants (n=11,150, US male physicians) aged 40 to 84 years showed that among men aged ≥60 years, those in the highest quartile (≥135 mm Hg) of average SBP had 1.69 times higher risk of CVD compared to those in the lowest quartile (<120 mm Hg) (95% CI: 1.21-2.38), after adjusting for age, BMI, HTN treatments, smoking, exercise, alcohol consumption, parental history of MI, and history of T2D (26). Additionally, obesity, T2D, and HTN are all highly prevalent chronic conditions. In 2015-2016, the prevalence

of obesity, T2D, and HTN were estimated to be 39.6%, 8.6%, and 29.8%, respectively, among U.S. adults and even higher among older adults (27-29).

Obesity is defined as excessive fat accumulation that may increase risk to health (30). BMI (weight in kilograms divided by the square of height in meters) is commonly used to classify obesity in adults, where a BMI greater than or equal to 30 is indicative of obesity status. Causes of obesity are complex. Generally, obesity is considered a result of the combination of inherited genes that confer susceptibility and a lifestyle consisting of low levels of physical activity and consumption of excess calories (31). Obesity may affect CVD risk via not only an altered metabolic profile (e.g. endothelial dysfunction, inflammation, insulin resistance, etc.) but also alterations in cardiac structure and function (31, 32). In addition, obesity in older adults could lead to respiratory and chronic musculoskeletal problems (33).

T2D accounts for ~90-95% of those with diabetes (i.e. type 1 diabetes accounts for the remaining 5-10%), and it is a chronic disease caused by a combination of resistance to insulin action and an inadequate compensatory insulin secretory response (34, 35). Common diagnostic criteria for T2D is 1) fasting plasma glucose $\geq$ 7.0 mmol/L (126 mg/dl), 2) 2-h plasma glucose $\geq$ 11.1 mmol/L (200 mg/dl), or 3) hemoglobin A1C (HbA1c) $\geq$ 6.5% (34, 35). Acting on a substrate of genetic susceptibility, insulin resistance develops from obesity, unhealthy diet, physical inactivity, and smoking; whereas insulin secretion declines with advancing age (35, 36). CVD accounts for the majority of the morbidity and mortality associated with T2D, for which the underlying mechanism is complex and multifactorial (37). In addition, T2D in older adults could lead to microvascular complications, depression, and dementia (38).

HTN, also known as high blood pressure, is a condition in which the blood vessels have persistently raised pressure (39). It is defined as a systolic blood pressure equal to or above 140 mmHg and/or diastolic blood pressure equal to or above 90 mmHg (39). HTN can be caused by behavioral risk factors (e.g. unhealthy diet, tobacco use, physical inactivity, poor stress management, and harmful use of alcohol) that are highly influenced by social determinants, such as income, education, and housing (39). Aging, genetic factors, or preeclampsia experience in women may also play a role in the development of HTN (39). HTN is the top contributor to CVD morbidity and mortality via functional and structural adaptations (40, 41). In addition, HTN in older adults could lead to stroke and dementia (42).

Adulthood dietary patterns as known risk factors for obesity, T2D, and HTN

Dietary intake of sodium, saturated fats, sugar-sweetened beverages and other high calorie processed foods has long been identified as a risk factor for obesity, T2D, and HTN (31, 35, 39). On the other hand, overall dietary behaviors and patterns have only recently been examined as chronic disease risk factors. The dietary patterns that have been commonly studied in relation to obesity, T2D, HTN, or CVD risk in general include the Mediterranean, prudent, and Western dietary patterns; or, with respect to interventions, such as the Dietary Approaches to Stop Hypertension (DASH).

The Mediterranean dietary pattern is a traditional dietary pattern found in olive-growing countries of the Mediterranean basin in the early 1960s (43). It is a dietary pattern that was originally invoked to explain the advantageous life expectancy in Mediterranean countries as

compared to Northern European countries or the U.S. (43, 44). Mediterranean diet is characterized by a high consumption of legumes, grains, fruits, vegetables, and complex carbohydrates, with a low-to-moderate consumption of meats and dairy products, the consumption of olive oil as the main source of fats, and a low-to-moderate amount of red wine during meals (44, 45). Multiple scoring schemes have been created to evaluate individual's adherence to the Mediterranean diet, among which the Mediterranean Diet Score developed by Trichopoulou et al. is the most important and frequently used (43, 45, 46). There has been consistent evidence from prospective studies of a protective effect of the Mediterranean diet on CVD risk and T2D; current literature also supports its protective effect on obesity and HTN (43, 45, 47-49).

Although the traditional Mediterranean diet has been shown to be beneficial to cardiovascular health in the U.S. population, it is fundamentally different than the regular eating patterns in the U.S. population (50, 51). For example, in the traditional Mediterranean diet, the primary source of monounsaturated fat is olive oil, rather than red meat in the U.S.; the main source of ethanol intake is wine, rather than beer and liquors in the U.S.; the main sources of micronutrients are fresh fruits and vegetables, rather than vitamin supplements in the U.S. (44).

The prudent and Western dietary patterns are two major dietary patterns that have been empirically identified in the U.S. population, using factor analysis or PCA. The earliest studies in which these dietary patterns were identified include the Health Professionals Follow-up Study (HPFS, a prospective cohort study among male U.S. health professionals 40 to 75 years of age at baseline), the Nurses' Health Study (NHS, a prospective cohort study among female U.S. nurses

30 to 55 years of age at baseline), and the Nurses' Health Study II (NHS II, a prospective cohort study among female U.S. nurses 24 to 44 years of age at baseline) (16, 52-54). The prudent dietary pattern is characterized by a higher intake of vegetables, fruits, legumes, whole grains, fish, and poultry; whereas the Western dietary pattern is characterized by a higher intake of processed meat, red meat, French fries, high-fat dairy products, eggs, butter, refined grains, and sweets and desserts (52, 55). The degree to which an individual conforms to each dietary pattern is measured by a score generated based on the underlying principal component (PC) (52, 53). In the past few decades, the prudent and Western dietary patterns have been increasingly investigated in relation to obesity, T2D, and HTN in the U.S. population.

Overall, existing evidence indicates a protective effect of the prudent dietary pattern and adverse effect of the Western dietary pattern on obesity and T2D. For example, the HPFS showed that the prudent dietary pattern was inversely associated with insulin concentration and risk of T2D in men aged 40-75 years; whereas the Western dietary pattern was positively associated with biomarkers of obesity (e.g. leptin) and T2D (e.g. insulin, C-peptide), as well as risk of T2D (16, 55). The NHS showed that the prudent dietary pattern was associated with decreased risk of T2D in women aged 38 to 63 years; whereas the Western dietary pattern was associated with increased risk of T2D (56). The NHS II showed that the prudent dietary pattern was associated with smaller weight gain in women aged 26 to 46 years; whereas the Western dietary pattern was associated with larger weight gain (57). Not much is known about the effect of the prudent and Western dietary patterns on HTN in the U.S. population, although a study based on the Mexican Teachers' Cohort showed that the Western dietary pattern was associated with increased risk of HTN in Mexican women aged 25 years and older (58).

It is worth noting that participants in the HPFS, NHS, and NHS II cohorts are of high SES and primarily white, which raises the question whether findings from these studies are generalizable to other SES/ethnicity groups or the general U.S. population. Similar examinations of the associations between dietary patterns and obesity, T2D, or HTN have been conducted in the Multi-Ethnic Study of Atherosclerosis (MESA) and the National Health and Nutrition Examination Survey (NHANES) populations.

The MESA study is a population-based cohort study of non-Hispanic white, African American, Hispanic, and Asian men and women aged 45 to 84 years, who were free of clinical CVD at baseline (59). In 2009, four dietary patterns ("fats and processed meat" "vegetables and fish" "beans, tomatoes, and refined grains" "whole grains and fruit") were identified in the MESA cohort by Nettleton, et al., using PCA (60). Although these dietary patterns are labeled differently, they have similar characteristics as the prudent and Western dietary patterns, and their findings are in general consistent (61).

The NHANES study is a nationally representative survey conducted by the National Center for Health Statistics to obtain information on the health and nutritional status of the U.S. population (17). In 2003, two predominant dietary patterns ("Western" and "American-healthy") were identified among healthy U.S. adults aged 20 years or older in NHANES III, using PCA (17). The "Western" dietary pattern here is very similar to the previously introduced Western dietary pattern; the "American-healthy" dietary pattern here is somewhat similar to the previously introduced prudent dietary pattern. In consistency with findings from the HPFS cohort, this

NHANES study showed that the Western dietary pattern was positively associated with serum C-peptide and fasting insulin levels (16, 17).

In general, all of the studies reviewed above that assessed the associations of dietary patterns with obesity, T2D, or HTN in the U.S. population tended to focus on the adult lifestyle model of CVD risk. That is, they adjusted for adulthood lifestyle factors (e.g. smoking, alcohol intake, physical activity, etc.) as potential confounders. Compared to the highly selective HPFS, NHS, and NHS II cohorts, the MESA and NHANES populations include both men and women, as well as multiple SES and ethnicity groups. This allows the latter two studies to further adjust for demographic indicators (e.g. sex, ethnicity) and adulthood SES (e.g. education, income) as potential confounders. Nevertheless, none of these studies incorporated the potential impact of childhood SES, from a life course perspective of CVD risk. Nor was the role of adulthood SES as a potential effect modifier considered.

Often times, people with common chronic conditions adopt special diets upon recommendation of their physicians. A good example of such diets is the DASH diet, which is an index-based dietary pattern promoted by the U.S.-based National Heart, Lung, and Blood Institute to prevent and control HTN. This dietary pattern is rich in fruits, vegetables, whole grains and low-fat dairy; includes poultry, fish, and nuts; and limits saturated fat, red meat, sweets, and sugar containing beverages (62). Multiple, consistent DASH diet indexes have been developed to measure individual's adherence to the dietary pattern (63). The DASH diet has consistently been shown in randomized controlled trials to reduce both systolic and diastolic blood pressure, as well as weight, in adults; current literature also supports its protective effect on T2D (62, 64-66).

Although, in reality, most patients are not fully following the recommended diets, there has been evidence of dietary changes in U.S. adults in response to obesity, T2D, or HTN diagnosis (67, 68). For example, between 2001 and 2006, 63% of obese adults reported trying to lose weight in the previous year, among whom 40% lost ≥5% weight and 20% lost ≥10% weight, both associated with eating less fat (69). Between 1999 and 2014, adults diagnosed with diabetes or prediabetes consumed 14.9 grams less sugar and 11.6 grams less carbohydrate per day, compared to those with similar demographic characteristics and HbA1c levels but without a diagnosis; these reductions were even greater among those whose diagnoses were recent (70). Between 2007 and 2010, adults who received a diagnosis of HTN tended to have lower odds of using added salt and consume less sodium, compared to those who were undiagnosed with HTN or at risk for developing HTN; a decrease in consumption of total energy, protein, fats, and saturated fats was observed among newly diagnosed hypertensives but not those who had been diagnosed for a prolonged period of time (71).

Stability of dietary patterns over the life course

In this dissertation, a one-time cross sectional measure of diet was used and related to childhood SES and proximal CVD risk factors (e.g. obesity, T2D, and HTN). Our interpretation of and inference from the observed relationships depends on what was captured in this one-time dietary measure. Especially, are the measured dietary patterns good representations of participants' long-term dietary habits (e.g. over decades or the life course), or can they only reflect participants' short-term dietary choices (e.g. over a few years)? In other words, to what extent do individuals' dietary patterns change over the life course?

Although this topic has not been very well understood, findings of prior longitudinal studies support the notion that dietary behaviors and choices are established early in life and track into later adulthood. For example, a 21-year follow-up study among 1037 Finnish children and adolescents (3-18 years old at baseline) who participated in the Cardiovascular Risk in Young Finns Study showed clear tracking in dietary patterns ("traditional pattern" and "health-conscious pattern"), especially from adolescence to adulthood (72). A 17-year follow-up study among 5362 adults (36 years old at baseline) in England, Scotland, and Wales who participated in the Medical Research Council National Survey of Health and Development showed fair-to-moderate stability in the "fruits, vegetables, and dairy" and "mixed" dietary patterns in women and men, but poor stability in the "meat, potatoes, and sweet foods" dietary pattern in women (73). Additionally, a 10-year follow-up study among 923 older adults (50-69 years old at baseline) in Ireland who participated in the Cork and Kerry Diabetes and Heart Disease Study showed that most participants remained stable in their dietary pattern ("Western" "Healthy" or "Low-Energy") and that most of those who changed dietary pattern moved to the "Health" pattern (74). However, there is also evidence of substantial decline with age in total energy intake and food quantity among older U.S. adults, which could be driven by changes along the aging process (e.g. reduced nutrient requirements, reduced physical abilities, and onset of chronic conditions) (18).

Childhood SES and adulthood dietary patterns

If, as indicated in prior studies, dietary behaviors and choices are established early in life and track into later adulthood, we would expect to observe an association between childhood

conditions (e.g. childhood SES) and adulthood dietary patterns. However, not much has been examined about this potential association. A 20- to 22-year longitudinal study among 1904 Danish teenagers identified two adulthood dietary patterns, namely "traditional-western food pattern" and "green food pattern," the latter of which was found to be associated with childhood SES (15). Specifically, this study found significant association between high (vs. low) childhood SES and greater adherence to the green food pattern (e.g. high intake of raw vegetables, cooked vegetables, fresh fruit, dried fruit and nuts, poultry, fish, eggs, cereal, desserts, and pasta, rice, and bulgur) in adulthood among women, regardless of adulthood SES; whereas among men, an association was found between high (vs. low) adulthood SES and greater adherence to the green food pattern in adulthood, regardless of childhood SES (15). However, it is unknown whether the conclusions from this study could be extended to dietary patterns in later adulthood or applied to the U.S. population, given that dietary behavior is culture specific.

Besides the longitudinal study in Denmark, there have been a few cross-sectional studies and cohort studies that examined the association of childhood SES with diet as measured by individual foods, food groups, or diet score but not the overall dietary pattern. For example, the cross-sectional studies found a positive association between childhood SES and consumption of fruits and vegetables among middle-aged and older adults in Finland (75), the United Kingdom (14), and Japan (76). Findings of the two cohort studies do not agree, although they both attempted to calculate an overall diet score to represent the relative healthiness of adulthood dietary intake (77, 78).

One potential mechanism for the associations between childhood SES and adulthood dietary patterns is that children with low SES have early unhealthy dietary exposure (e.g. more fast food restaurants and less fresh fruits/vegetables available in neighborhood, family cannot afford nutritious food or lack understanding of nutritional contents/dietary recommendations), from which initiates their poor dietary habits that persist throughout adulthood (23, 79). Children with low SES may also adopt unhealthy dietary behavior modeled by their caregivers, which place them on trajectories that remain stable into adulthood (23, 79). Alternatively, the effect of childhood SES on diet later in life may result from the taste development in childhood (14, 80).

Multimorbidity of obesity, T2D, and HTN

Multimorbidity, the coexistence of multiple chronic conditions, is highly prevalent among older adults (81, 82). With the consideration of 11 chronic conditions (e.g. CVD, chronic obstructive pulmonary disease, chronic kidney disease, asthma, arthritis, cancer, stroke, HTN, hyperlipidemia, diabetes, and obesity), King et al. estimated that, between 2013 and 2014, 91.8% of U.S. adults aged 65 years or older were living with 2 or more chronic conditions (83). Multimorbidity in older adults can lead to disability, functional decline, poorer quality of life, as well as higher health care utilization and costs (81).

While some chronic conditions could coexist with each other simply by chance alone, a "metabolic" multimorbidity pattern, characterized by the specific coexistence of obesity, T2D, and HTN, has consistently been identified among adults older than 50 years in low-, middle-, and high-income countries (12). As reviewed in the previous sections, obesity, T2D, and HTN are each a highly prevalent chronic condition in older adults, as well as a leading risk factor of CVD.

Therefore not surprisingly, this "metabolic" multimorbidity pattern has been associated with substantially further elevated risk of CVD, worsen health-related quality of life, health services use, and mortality (84-88). For example, a pooled analysis of four cohort studies in the U.S. found that at age 45 years, the "metabolic" multimorbidity pattern, in comparison to the absence of obesity, T2D, and HTN, is associated with 3.70 to 6.67 times higher risk of incident heart failure over the remaining life course (87). A cross-sectional study among U.S. adults aged 51 to 61 years who participated in the 1992 HRS and those aged 70 years or older who participated in the 1993 Assets and Health Dynamics of the Oldest Old (AHEAD) study showed that the "metabolic" multimorbidity pattern, in comparison to the absence of obesity, T2D, and HTN, is associated with 2.13-2.99 times higher odds of hospital stay, 4.05-4.91 times higher odds of doctor visit, 3.96-5.98 times higher odds of mobility difficulty, and 1.27-3.26 times higher odds of mortality (88). However, the burden of the "metabolic" multimorbidity pattern in older U.S. adults has not been recorded in recent years. Nor is much known about its risk factors.

Studies on pair-wise associations among obesity, T2D, and HTN have provided some indications about their inter-relationships. First, obesity is a known risk factor of T2D (89, 90). It has been estimated that 60% to 90% of T2D is related to obesity (90). According to a cross-sectional study based on NHANES 1999-2006, the prevalences of T2D in U.S. adults with normal weight (BMI between 18.5 and 24.9 kg/m2), obesity class 1 (BMI between 30.0 and 34.9 kg/m2), class 2 (BMI between 35.0 and 39.9 kg/m2), and class 3 (BMI $\geq$40.0 kg/m2) were 8%, 23%, 33%, and 43%, respectively (91). Potential mechanisms linking obesity to T2D that have been proposed in prior studies include insulin resistance, endothelial dysfunction, and oxidative stress (92, 93). Second, obesity is a known risk factor of HTN (93, 94). A meta-analysis by Guh, et al. showed

that obese men and women in Western countries have 1.84 (95% CI: 1.51-2.24) and 2.42 (95% CI: 1.59-3.67) times higher risk of HTN, respectively, compared to their normal weight counterparts (89). Various mechanisms have been proposed to explain the obesity-HTN association, including increased sympathetic nervous system activity, increased activation of the renin-angiotensin-aldosterone system, endothelial dysfunction, structural or functional changes in the kidneys, increased production of leptin, and insulin resistance (95, 96). Lastly, T2D and HTN share other underlying risk factors. For example, both conditions are associated with increased low-grade inflammatory markers, elevated oxidative stress, and insulin resistance (97). Additionally, genetic factors and mental stress could also play a role (97).

Prior studies also proposed that environmental factors, together with genetic factors, can be driving the multimorbidity of obesity, T2D, and HTN (98, 99). While dietary behavior, a modifiable environmental factor, has long been identified as a risk factor for obesity, T2D, and HTN, individually, not much is known about its association with the multimorbidity of these three conditions (31, 35, 39). A cross-sectional study among adult participants (aged 20 years and older) of NHANES 1999-2002 found a stronger inverse association between dietary fiber intake and level of C-reactive protein (a biomarker of inflammation that is positively associated with obesity, T2D, and HTN statuses, as well as CVD risk in general) among those who had multimorbidity of obesity, T2D, and HTN (e.g. had 2 or 3 of the conditions) than those with a single condition (100). Empirically identified dietary patterns, as better representations of individuals' overall dietary behavior, however, have not been examined in relation to the multimorbidity of obesity, T2D, and HTN (52).

SES, an important social environment indicator, has also been consistently shown to be inversely associated with obesity, T2D, and HTN, individually, in high-income countries (101-103). In addition to SES in adulthood (e.g. education, occupation, income), there has been an increasing interest in childhood SES (e.g. parental education, paternal occupation) in relation to obesity, T2D, and HTN, individually, from a life-course perspective (104-107). Yet, the literature on the relationships of childhood SES and adulthood SES with the multimorbidity of obesity, T2D, and HTN is sparse. In Spanish adults aged ≥60 years, a report in 2004 showed that lower social class in adulthood, regardless of childhood social class, was associated with higher prevalence of the multimorbidity of abdominal obesity, T2D, and HTN among women (108). This topic has not been examined in older U.S. adults.

Finally, it is worth noting the similarities and distinctions between the "metabolic" multimorbidity pattern discussed above and the commonly known Metabolic Syndrome (also referred to as the "Syndrome X" or the "Insulin Resistance Syndrome") (109, 110). The Metabolic Syndrome represents the constellation of vascular risk factors including atherogenic dyslipidemia, elevated blood pressure, dysglycemia, a pro-thrombotic state, and a pro-inflammatory state (111). A clinical diagnosis of the Metabolic Syndrome is based on the presence of three or more of the following five conditions: 1) elevated waist circumference (≥102 cm in males; ≥88 cm in females), 2) elevated triglycerides (≥150 mg/dL or 1.7mmol/L), 3) reduced high-density lipoprotein cholesterol (HDL-C, <40 mg/dL or 1.0 mmol/L in males; <50 mg/dL or 1.3 mmol/L in females), 4) elevated blood pressure (Systolic ≥130 and/or diastolic ≥85 mmHg), and 5) elevated fasting glucose (≥100 mg/dL) (111). Essentially, the Metabolic Syndrome covers all the elements of the "metabolic" multimorbidity pattern (e.g. obesity, T2D,

and HTN), with the additional inclusion of elevated triglycerides and reduced HDL-C. However, the Metabolic Syndrome stresses the abdominal obesity type and does not require the coexistence of obesity, T2D, and HTN. Also, the cut points used in the diagnostic criteria of Metabolic Syndrome are not exactly the same as those used for T2D or HTN as an individual health outcome.

According to an NHANES analysis, the prevalences of the Metabolic Syndrome during 2007-2014 in U.S. adults aged 20–39 years, 40–59 years, and ≥60 years were 19.3%, 37.7%, and 54.9%, respectively (112). Similar to the "metabolic" multimorbidity pattern, the pathogenesis of the Metabolic Syndrome is still unclear. Insulin resistance and obesity has each been hypothesized as its cause (111). There has also been studies on the associations of the Metabolic Syndrome with dietary patterns and SES. For example, a systematic review and meta-analysis of 19 studies (cross-sectional, cohort, or longitudinal by design) in adults aged 18 to 60 years that identified dietary patterns using factor analysis or PCA showed that the "Healthy/Prudent" dietary pattern is inversely associated with the Metabolic Syndrome (odds ratio (OR)=0.89, 95% CI: 0.84-0.94); whereas the "Unhealthy/Western" dietary pattern is positively associated with the Metabolic Syndrome (OR=1.16, 95% CI 1.11-1.22) (113). A 12-year longitudinal study among white and black women (aged 42–52 years at baseline) in the U.S. who participated in the Study of Women's Health Across the Nation found an independent inverse association of both childhood SES and adulthood SES with the Metabolic Syndrome (114). There is also evidence that health behaviors (e.g. physical activity, diet) could be pathways from childhood SES to the Metabolic Syndrome in adulthood (114, 115). We did not investigate the Metabolic Syndrome

in this dissertation because the HRS did not have measures of dyslipidemia (i.e. measured levels of fasting plasma triglycerides).

Public health significance

CVD is the leading cause of death in the U.S. and it is responsible for 17% of national health expenditures (1, 2, 4). The health and economic burden of CVD is especially high in older adults (3). By 2030, as the consequence of population aging, 40.5% of the U.S. population will have some form of CVD, which is associated with a three-fold higher real total direct medical costs (e.g. from $273 billion to $818 billion) and a 61% increase of real indirect cost (e.g. from $172 billion to $276 billion) compared to 2010, assuming no change to current CVD prevention strategies (4, 5). In order to address this rising public health issue, more effective CVD prevention strategies are greatly needed, and it is the wider goal of this dissertation to promote the development of such prevention strategies.

Thinking upstream, this dissertation examined obesity, T2D, and HTN, three leading risk factors of CVD, as the health outcomes (7). Obesity, T2D, and HTN are each a highly prevalent chronic condition, with a prevalence of 39.6%, 8.6%, and 29.8%, respectively, in 2015-2016 among U.S. adults and even higher among older adults (27-29). Not only have obesity, T2D, and HTN each been associated with a significantly elevated risk of CVD, but also, they could lead to health problems that significantly reduce quality of life (24-26). For example, obesity could lead to respiratory and chronic musculoskeletal problems; T2D could lead to microvascular complications, depression, and dementia; and HTN could lead to stroke and dementia (33, 38, 42). By 2030, in the U.S., it is estimated that the obesity-related combined medical costs will

increase by $48–66 billion/year from 2010 (116); the T2D-related total annual medical and societal costs will increase by 53% from 2015 to more than $622 billion (117); the HTN-related direct medical and indirect costs will increase by 186% (from $69.9 billion to $200.3 billion) and 69% (from $23.6 billion to $39.8 billion) from 2010 (4). Therefore, targeting obesity, T2D, and HTN is beneficial for not only CVD prevention, but also reduction of the health and economic burden of these conditions themselves and other downstream health outcomes.

In addition to examining obesity, T2D, and HTN as individual CVD risk factors, this dissertation also examined the multimorbidity of these health outcomes in older U.S. adults. The multimorbidity of obesity, T2D, and HTN, also referred to as the "metabolic" multimorbidity pattern, has consistently been observed among older adults in low-, middle-, and high-income countries (12). Compared to obesity, T2D, or HTN as an individual health outcome, their multimorbidity has been associated with substantially further elevated risk of CVD, worse health-related quality of life, health services use, and mortality (84-88). Without a doubt, the multimorbidity of obesity, T2D, and HTN is also a public health concern.

CHAPTER II: Adulthood Dietary Patterns and Their Associations with Childhood SES

Abstract

Background: Dietary pattern is a comprehensive measure of dietary behavior, a known risk factor for numerous adverse health outcomes. However, not much is known about the association between adulthood dietary patterns and childhood SES.

Objective: To identify the main dietary patterns in older U.S. adults and examine the associations of these dietary patterns with childhood SES.

Design: This is a cross-sectional examination of a random subsample of 8,035 adults over age 50 who were enrolled in the nationally representative longitudinal HRS. Participants self-reported their dietary behavior and childhood SES. Dietary patterns were derived from PCA. Linear regression models were used to examine the associations of dietary pattern scores with childhood SES indicators, including paternal and maternal education levels and childhood financial strain score. All analyses took into account the complex survey design.

Results: We identified two main dietary patterns in older U.S. adults (prudent and Western). Having 8+ years (vs. <8 years) of paternal education was associated with a 0.32-unit (95% CI: 0.08-0.57, p=0.0117) higher prudent dietary pattern score among "other" race (not White, Black, or Hispanic) and a 0.16-unit (95% CI: 0.03-0.29, p=0.0147) lower Western dietary pattern score among males, independent of adulthood SES. Maternal education was not associated with either of the dietary patterns, and the associations between childhood financial strain and the dietary patterns appeared to be completely mediated through adulthood SES. In addition, participant's

education could also modify the association between paternal education and the prudent dietary pattern.

Conclusions: Paternal education, but not maternal education or childhood financial strain, is independently positively associated with the prudent dietary pattern among "other" race and inversely associated with the Western dietary pattern among males. The role of adulthood SES in dietary patterns is complex and needs further investigation.

Introduction

Diet is a crucial component of daily life that directly determines the composition of individuals' nutrient intake, which largely influences human body's normal function and well-being. Increasing evidence from recent epidemiologic studies has shown associations between dietary behavior and various adverse health outcomes, such as CVD, cancer, and neurodegenerative diseases (45, 118). Understanding dietary behavior in the population, therefore, is an important step towards a better understanding of disease risk at the population level and the potential of dietary interventions.

Prior longitudinal studies support the notion that dietary behaviors and patterns are established in early childhood or even infancy and track moderately into later adulthood (72-74, 119, 120). Although dietary patterns varied across study populations and it is still unclear as to what dietary patterns track more strongly, one important inference that we can make from these findings is that early life experience can not only shape one's dietary pattern early in life, but also, through the stability of dietary behavior, it has potentially a long-lasting effect on one's health status over

the life course. Identifying early life determinants of dietary patterns also opens the door to more efficient dietary interventions.

The association between childhood SES and adulthood dietary patterns has not been well studied. A 20- to 22-year longitudinal study among Danish teenagers showed significant association between high childhood SES and greater adherence to the "green food pattern" in adulthood among women, regardless of adulthood SES (15). However, it is unknown whether the conclusions from this study could be extended to dietary patterns in later adulthood or applied to the U.S. population, given that dietary behavior is culture specific. Other studies examined the association of childhood SES with diet as measured by individual foods, food groups, or a diet score, rather than the overall dietary patterns. Despite some inconsistency in findings, there is overall evidence of a positive association between childhood SES and consumption of fruits and vegetables among middle-aged and older adults (14, 75-78).

The purpose of the present study is to identify the main dietary patterns in older U.S. adults and examine their associations with childhood SES, using the nationally representative HRS. Potential confounding and effect modification to the associations by age, sex, and race, as well as potential mediation and effect modification by adulthood SES were also examined.

Methods

Study population

This study is a cross-sectional examination of HRS, a large longitudinal study of U.S. individuals over age 50. The design of HRS has been described in detail previously (121). Briefly, HRS was

launched in 1992, with core surveys fielded every 2 years and supplemental studies taking place in off-years between core surveys starting in 1999. Using a steady state design, HRS has covered seven birth cohorts so far, replenishing the sample with younger cohorts every 6 years. In any given wave, the combination of different birth cohorts generates a sample size of about 18,000-23,000. The core sample of HRS was selected based on a multi-stage area probability design. Additionally, HRS oversamples African-Americans, Hispanics and residents of the state of Florida. Sampling weights are derived to compensate for the unequal probabilities of selection between the core and oversample domains, as well as differential non-response in each wave (baseline response rate 69.9% - 81.6%, follow-up response rate 85.4% - 93.0%).

Participants of this study are comprised of a random subsample of HRS participants, who completed the supplemental off-year Health Care and Nutrition Survey (HCNS) in 2013. All living HRS respondents of all ages and their spouse/partners, who were not included in the other off-year survey (Consumption and Activities Mail Survey) in 2013, were eligible to participate in HCNS. Among those 12,418 who were eligible, 65% responded. We excluded 37 respondents who answered very few of the food consumption questions and 1 respondent who reported as using a feeding tube. The final sample size for this study was 8,035, including 449 participants who were assigned zero sampling weights (therefore not used in weighted analyses) due to: 1) in a nursing home (n=1), 2) not cohort-eligible (n=434, e.g. HRS recruits spouses without age restriction), and 3) incomplete interview in the 2012 wave (n=14). A total of 7,331 observations with non-zero sampling weights and no missing data for explanatory or outcome variables were used in the multivariate analyses.

Data collection

Dietary intake

Information on dietary intake was collected using an embedded 164-item food frequency questionnaire (FFQ) within the mailed HCNS. This self-administered FFQ was based on the Harvard FFQ, which has been validated against one-week diet records and 24-hour diet recalls over a 1-year period for intakes of individual nutrients (122-125) , individual foods (126, 127), food groups (128), and dietary patterns (129). For each food item in the FFQ, participants were asked to indicate how often on average they had used the amount specified over the past twelve months, ranging from never to multiple (e.g. ≥4, ≥5, or ≥6) times per day. In HCNS, 97% of respondents answered 90% or more of the food consumption questions. Missing data was imputed based on 5 respondent predictors from the core HRS survey, including age, sex, race, years of education, and BMI. Categorical response of consumption frequency for each food item was converted to number of servings per day, using Harvard University's food serving conversion guides.

Childhood SES

Childhood SES was measured by three self-reported indicators: 1) paternal education level, 2) maternal education level, and 3) a composite score (ranging from 0-4) representing the intensity of childhood financial strain. Parental education captures participants' access to health-related knowledge and material resources during childhood, which could have long-lasting impact on their health behavior and health conditions throughout life (130). Financial strain experienced in childhood is a stressor that could also have persisted effect on health behavior and health status

later in life (131, 132). In the absence of complete data across the life course, the quality of retrospectively reported childhood SES has been confirmed in prior studies (133, 134).

In HRS core surveys, information about parental education level was collected by asking "Did your father/mother attend 8 years or more of school?" with yes/no as possible answers or "What is the highest grade of school your father/mother completed?" with possible answers ranging from 0 to 17+. Summary measures of paternal and maternal education levels (8+ years vs. <8 years) were each generated from the first reported value across all waves, giving priority to the waves where the second set of questions were used. Credentials were used to measure education instead of years of schooling, assuming specific educational achievements are more important than time spent in education (135). Because our data indicated that missingness of paternal and maternal education (15.42% and 8.66%, respectively) was associated with dietary pattern scores and the other childhood SES indicators, we imputed paternal and maternal education levels as needed to minimize potential selection bias. This imputation was a multivariate, regression-based procedure using Imputation and Variance Estimation (IVEware) software (http://www.isr.umich.edu/src/smp/ive/). The basis of imputation was a combination of sociodemographic characteristics (including mother/father alive, mother/father age at death, respondent's birth year, gender, race, and Hispanic ethnicity) and SES measures (including parental education of the opposite parent and the respondent's own education).

Childhood financial strain score was calculated as the total number of affirmative responses to four questions regarding financial status during respondents' childhood (before age 16), including 1) "did financial difficulties ever cause you or your family to move to a different

place?" (1=yes and 0=no), 2) "was there a time when you or your family received help from relatives because of financial difficulties?" (1=yes and 0=no), 3) "was there a time of several months or more when your father had no job?" (1=yes and 0=no), and 4) "would you say your family during that time was pretty well off financially, about average, or poor?" ("poor" or "varied" were coded as 1, and "average" or "well-off" were coded as 0).

Adulthood SES

Indicators of adulthood SES included education and wealth. Education captures the knowledge-related assets of participants (e.g. general and health-related knowledge, access appropriate health services, problem solving, material resources), and it is a strong determinant of subsequent employment and income (130, 135). Participants' educational attainments were measured by asking "What is the highest grade of school or year of college you completed?" with possible answers ranging from 0 to 17+. Years of education was then converted into three categories, including 1) less than high school (<12 years), 2) high school (12 years), and 3) some college or above (>12 years).

Wealth captures the accumulation of total assets (including financial and physical assets) and income, and it is especially important for older adults due to accumulation of assets over time and impact of retirement on income (136, 137). During HRS core interviews, participants were asked a series of questions about assets and income (e.g. "Do you have any shares of stock or stock mutual funds?" "About how much did you receive from stocks or stock mutual funds in last year (before taxes and other deductions)?" "Did you do any work for pay last year?" "About how much wage and salary income did you receive in last year, before taxes and other

deductions?"). In cases where a participant was unable or unwilling to provide an exact amount of the value of assets or income, they were asked a series of follow-up questions to estimate a range (e.g. $0-2,500; $2,500-25,000; $25,000-125,000; $125,000-400,000; $400,000 or more). Starting from the year of 2002, discrepancies (e.g. >$50,000 difference) of asset values across waves have been corrected for with additional asset verification questions (138). Additionally, missing data of all asset and income types were imputed using a consistent method across waves (138). For each participant, a total wealth (including secondary residence) in nominal dollars was calculated as sum of all wealth components less all debt (**Supplementary Table 1**). In this study, wealth was measured by quintiles of this total wealth according to its weighted distribution in the population.

Assessment of dietary patterns using PCA

The 164 food items in the FFQ were first classified into 40 food groups, based on similar nutrient profiles or culinary usage (**Supplementary Table 2**). Daily consumption frequency for each food group was calculated as the sum of daily frequencies of all included food items, in terms of typical servings specified in the questionnaire. Daily frequencies of the 40 food groups were then adjusted for total energy intake using the residual method (139). This adjustment was performed on the log-scale, to improve normality, and without 63 participants who had extreme total energy intake (> 4 SD or < -4 SD on the original scale), so as to remove the influence of extreme values on regression results. Although these 63 participants had missing values for daily frequency residuals, they were kept in the analyses to maintain the integrity of sampling weights.

PCA was performed on daily frequency residuals of the 40 food groups, accounting for the complex survey design, so that participants' dietary behaviors were measured relative to the population distribution. To achieve this, we first created a weighted correlation matrix using the svycor function from the jtools package in R, accounting for the complex survey design (140, 141). Next, this weighted correlation matrix was used as the input for PCA, using the FACTOR PROCEDURE in SAS.

Two PCs were retained based on 1) percent variation explained by the component, 2) the Scree plot, and 3) interpretability. Since the un-rotated PC loadings did not follow simple structure (that is, a few close to ±1, the remainder close to 0), Varimax (e.g. orthogonal) rotation of PC loadings was performed to achieve better interpretability and independence between rotated PCs. Sensitivity analyses were performed on 1) retaining 3 PCs instead, 2) using Promax (e.g. oblique) rotation instead, 3) without removing outliers of total energy intake, 4) daily frequencies of food groups adjusted for total energy intake on the original scale, 5) daily frequencies of food groups without adjusting for total energy intake, 6) daily frequencies of food items without grouping or total energy intake adjustment.

Each PC (to be translated later into a dietary pattern) represents a linear combination of residuals of all 40 food groups and explains as much inter-individual variation as possible. Each participant will receive a score for each identified PC calculated by summing consumptions of food groups weighted by their PC loadings, with a higher score indicating a greater adherence to the corresponding dietary pattern.

Statistical analysis

Data preparation and statistical analyses were performed using SAS software, Version 9.4 of the SAS System for Windows (SAS Institute Inc., Cary, NC, USA). Statistical associations were examined using linear regression models, with score of each PC (PC1 and PC2) as a separate outcome variable. Main exposure variables under examination were childhood SES indicators, including paternal and maternal education levels and childhood financial strain score. Other covariates included age (<70 years vs. 70 years and older), sex (male vs. female), race (white vs. black vs. Hispanic vs. other), and adulthood SES indicators, including participant's education level and wealth.

For each PC, a series of models were run in a step-wise fashion to assess its associations with childhood SES indicators, as well as relevant roles of other covariates. First, we examined crude associations using univariate models. Second, we examined mutually adjusted associations using multivariate models including all three childhood SES indicators and adjusting for age, sex, and race as potential confounders. Third, potential effect modifications by age, sex, and race were assessed by further including in the models their interaction terms with each childhood SES indicator. Statistically significant interaction terms were identified with a P-value<0.05 of the corresponding t-test and were kept in the model moving forward. Fourth, potential mediation by adulthood SES was assessed by further including in the models participant's education and wealth, where an attenuation (e.g. >10% change towards 0) of the parameter estimates for childhood SES indicators from the previous model indicates existence of mediation. Lastly, potential effect modification by adulthood SES indicators was assessed by further including in the models their interaction terms with each childhood SES indicator. Statistically significant

interaction terms were identified with a P-value<0.05 of the corresponding t-test. The Type III test was used for assessing overall associations.

Results

Through weighted PCA, we have identified two major dietary patterns in older U.S. adults, which, in combination, explained 16.32% of the overall variation in dietary intake (**Table 1**). The first dietary pattern (PC1), namely prudent dietary pattern, is characterized by high (loading>0.3) intake of vegetables, tomatoes, garlic, legumes, fruit, olive oil, fish and other seafood, and salad dressing, as well as low (loading<-0.3) intake of sweets and desserts and high-energy drinks. The second dietary pattern (PC2), namely Western dietary pattern, is characterized by high intake of red meats, processed meats, condiments, French fries, and refined grains, as well as low intake of cold breakfast cereal, whole grains, low-fat dairy products, and fruit. These two major dietary patterns are robust to decisions on number of PCs to retain, rotation method used, outlier elimination, total energy intake adjustment, and food grouping, according to results of multiple sensitivity analyses on PCA.

As shown in **Table 2**, older U.S. adults who were <70 years, female, Hispanic, who had higher childhood SES (higher parental education, higher maternal education, fewer childhood financial strains) or higher adulthood SES (higher education, greater wealth) had significantly higher prudent dietary pattern scores; whereas those who were <70 years, male, who had lower adulthood SES (lower education, less wealth) had significantly higher Western dietary pattern scores. It is worth noticing that childhood SES indicators appeared to be significantly associated with the prudent, but not the Western dietary pattern.

A multivariate model with prudent dietary pattern score as the outcome identified significant paternal education by race and financial strain score by sex interactions (**Table 3**). Specifically, having 8+ (vs. <8) years of paternal education is associated with significantly higher prudent dietary pattern score among whites (mean difference=0.18, 95% CI: 0.09 to 0.27, p=0.0002) and "other" race group (mean difference=0.48, 95% CI: 0.23 to 0.74, p=0.0004), but not among blacks or Hispanics; having a 1-unit greater financial strain score is associated with significantly lower prudent dietary pattern score among females (mean difference= -0.06, 95% CI: -0.09 to -0.02, p=0.0022), but not among males. After including adulthood SES indicators in the multivariate model, paternal education was no longer significantly associated with prudent dietary pattern score among whites, whereas the significant association remained among "other" race group with a 33% attenuation in mean difference; financial strain score was no longer significantly associated with the prudent dietary pattern score among females. No significant association was observed between maternal education and prudent dietary pattern score.

A multivariate model with Western dietary pattern score as the outcome identified significant paternal education by sex and financial strain score by age interactions (**Table 4**). Specifically, having 8+ (vs. <8) years of paternal education is associated with significantly lower Western dietary pattern score among males (mean difference= -0.22, 95% CI: -0.35 to -0.09, p=0.0015), but not among females; having 1-unit greater financial strain score is associated with significantly higher Western dietary pattern score among participants <70 years old (mean difference=0.03, 95% CI: 0 to 0.07, p=0.0394), but not among those who were 70 years and older. After including adulthood SES indicators in the multivariate model, the significant

association between paternal education and Western dietary pattern score among males remained, with 27% attenuation in mean difference; whereas financial strain score was no longer significantly associated with Western dietary pattern score among participants in the younger age group. Similar to the analyses on prudent dietary pattern score, a total of 7,331 observations were used in the multivariate analyses on Western dietary pattern score, and no significant association was observed between maternal education and Western dietary pattern score.

In assessing potential interaction between childhood SES and adulthood SES, we have identified a significant interaction between paternal education and participant's education in relation to the prudent dietary pattern score (p for interaction=0.0004). Given the existing interaction between paternal education and race, we are presenting the paternal education - participant's education interaction separately for each race group (**Figure 2**). Overall, within each paternal education level, participants with higher education level had higher prudent dietary pattern score, regardless of race. However, the directionality of association between paternal education and prudent dietary pattern score varies by race and participants' education. Specifically, among whites, blacks, and Hispanics, for participants with less than high school or high school degree, higher paternal education is associated with lower prudent dietary pattern score; whereas for participants with college degree or above, higher paternal education is associated with higher prudent dietary pattern score. Within the "other" race group, higher paternal education is associated with higher prudent dietary pattern score, and this association is stronger among participants with higher education.

Discussion

Using nationally representative data, this study identified two main dietary patterns in older U.S. adults, namely the prudent and Western dietary patterns. Higher paternal education was associated with greater adherence to the prudent dietary pattern among "other" race and less adherence to the Western dietary pattern among males, independent of adulthood SES. The former association also tended to be stronger among participants with higher education. The associations of childhood financial strain with the dietary patterns appeared to be completely mediated through adulthood SES. Maternal education was not associated with either dietary pattern, once other two childhood SES indicators were accounted for.

The dietary patterns identified in the present study are consistent with those identified in prior studies among selected older U.S. adults or the entire U.S. adult population (17, 142). Despite cultural differences in dietary patterns, the present study also supports the prior finding in Danish population that higher childhood SES, as well as adulthood SES, is independently associated with healthier dietary habits in adulthood (15). In addition, both studies support the existence of variation in this association across sociodemographic (e.g., sex, race) subgroups.

As has been proposed in prior studies, one potential mechanism for the association between childhood SES and adulthood dietary habits is that children with low SES have early unhealthy dietary exposure (e.g. more fast food restaurants and less fresh fruits/vegetables available in neighborhood, family cannot afford nutritious food or lack understanding of nutritional contents/dietary recommendations), from which initiates their poor dietary habits that persist throughout adulthood (23, 79). Children with low SES may also adopt unhealthy dietary behavior modeled by their caregivers, which place them on trajectories that remain stable into

adulthood (23, 79). Alternatively, the effect of childhood SES on diet later in life may result from the taste development in childhood (14, 80).

One interesting finding of the present study is that paternal education, but not maternal education, is associated with dietary patterns in older adulthood. This resonates with an earlier finding of positive association between children's and their fathers', but not mothers', use of and time spent in fast-food and full-service restaurants, given that fathers' unhealthy dietary behavior is likely a result of lower paternal education level (143-145). However, not much is known about father's role in the development of children's dietary behavior. Unlike mothers who often determine how much food is offered to their children, fathers tend to be indulgent to a child's food request and exert less active control on food intake (146). Modeling of dietary behavior could be key influence of fathers (143).

On the contrary, more studies have been conducted on mothers' role in the development of children's dietary behavior. Higher maternal education has also been shown to be associated with healthier dietary intake in children, potentially through healthier diet during pregnancy and lactation, better feeding decisions, role modeling, or creating healthier food environment (146-150). There are some possible reasons why such association was not observed in the present study. First, maternal and paternal education levels are mutually adjusted in the present study, but not in prior studies. It is possible that the apparent association between maternal education and children's dietary behavior in prior studies was confounded by paternal education, a potentially stronger predictor of children's dietary behavior. Second, maternal education in the present study was only divided into two levels, which could have masked an association of more

refined maternal education levels with children's dietary behavior. Third, dietary behaviors were examined in older adulthood in the present study but in childhood or even infancy in prior studies. Unmeasured factors throughout the life course could have contributed to the difference in findings.

Another interesting finding of the present study is the racial difference in paternal education's association with the prudent dietary pattern and sex difference in its association with the Western dietary pattern. If role modeling is the mechanism for the association between paternal education and dietary patterns in older adulthood, as mentioned above, then the racial difference in association could be explained by racial difference in father's role within family (151); the sex difference in association could be explained by different dynamics between father-daughter and father-son relationships (152, 153).

A main strength of the present study is the examination of diet in the form of dietary patterns. Prior studies on the same topic largely focused on diet as measured by individual foods or food groups (14, 75, 76). Such approaches could shed light on potential underlying mechanisms, but only to a limited extend, due to the complicated interactions and intercorrelations among foods and nutrients (52). By examining the overall dietary patterns, the present study took into consideration the combination of nutrients and foods being consumed. Not only is dietary pattern a comprehensive target for dietary behavior change, but also, dietary recommendations in terms of dietary patterns are easily interpretable to the public (52). Moreover, in identification of dietary patterns using PCA, multiple sensitivity analyses were performed to assess the robustness of results to a series of decisions, ranging from number of PCs to retain to food grouping

strategy. Another strength of the present study is the examination of a large, nationally representative sample of older U.S. adults. By accounting for the complex survey design in both PCA and statistical modeling, the present study generated results that are reflective of the dietary patterns and their associations with childhood SES in the entire older U.S. adult population.

However, the present study also have some limitations. First, detailed dietary intake data of the participants was collected only once via FFQ, which might not have captured very well their long-term intake in comparison to repeated FFQs over the years. Although one FFQ is sufficient for ranking participants in terms of dietary intake, it is prone to measurement error that can bias towards the null the association under study. Second, the selected indicators used for childhood and adulthood SES might only have captured specific aspects of SES, instead of its whole construct. While, typically, causal inference cannot be made from a cross-sectional study design, it is less of a concern in the present study, as there is clear temporality among childhood SES, adulthood SES, and adulthood dietary intake measurements. Therefore, mediation assessment of adulthood SES is also justified, even though the lack of information regarding other important life course histories prevents the investigation of specific underlying mechanisms of the associations under study.

In conclusion, findings of the present study indicate that higher childhood SES, specifically paternal education, is associated with healthier dietary behavior in older adulthood, independent of adulthood SES. Dietary interventions early in life or targeting high-risk adults based on early-life experiences might be more efficient strategies for disease prevention. Future studies are

needed to better understand the impact of paternal education on the development of dietary behavior early in life and how this impact varies in different sociodemographic groups.

Table 1. Factor loadings for dietary patterns in older U.S. adults, 2013*

Food Groups	PC1 (Prudent)
Vegetables, other	**0.73**
Vegetables, green leafy	**0.69**
Vegetables, cruciferous	**0.62**
Vegetables, dark-yellow	**0.59**
Tomatoes	**0.53**
Garlic	**0.49**
Legumes	**0.41**
Fruit	**0.39**
Olive oil	**0.36**
Fish and other seafood	**0.35**
Salad dressing	**0.34**
Poultry	0.28
Drinks, low-energy	0.23
Wine	0.23
Tea	0.23
Eggs	0.21
Grains, whole	0.19
Sweeteners	0.13
Nuts	0.12
Condiments	0.06
Coffee	0.06
Liquor	0.04
Chowder or cream soup	0.03
Butter	-0.01
Dairy products, high-fat	-0.03
Organ meats	-0.03
Potatoes	-0.06
Snacks	-0.06
Red meats	-0.07
Beer	-0.07
Fruit juices	-0.07
Dairy products, low-fat	-0.10
Pizza	-0.11
Processed meats	-0.14
Non-dairy fat	-0.15
French fries	-0.17
Cold breakfast cereal	-0.20
Grains, refined	-0.22
Sweets and desserts	**-0.41**
Drinks, high-energy	**-0.41**
% variance explained by PC1 = 10.95%	

Food Groups	PC2 (Western)
Red meats	**0.50**
Processed meats	**0.50**
Condiments	**0.50**
French fries	**0.39**
Grains, refined	**0.39**
Beer	0.28
Drinks, high-energy	0.26
Butter	0.24
Pizza	0.23
Eggs	0.22
Potatoes	0.21
Liquor	0.18
Snacks	0.16
Dairy products, high-fat	0.16
Coffee	0.15
Garlic	0.13
Salad dressing	0.08
Non-dairy fat	0.08
Organ meats	0.08
Chowder or cream soup	0.06
Sweeteners	0.04
Tomatoes	0.02
Poultry	0.02
Sweets and desserts	0.02
Vegetables, other	-0.01
Wine	-0.02
Olive oil	-0.03
Drinks, low-energy	-0.03
Fish and other seafood	-0.08
Tea	-0.09
Vegetables, green leafy	-0.14
Vegetables, cruciferous	-0.16
Legumes	-0.19
Nuts	-0.22
Fruit juices	-0.27
Vegetables, dark-yellow	-0.28
Cold breakfast cereal	**-0.37**
Grains, whole	**-0.39**
Dairy products, low-fat	**-0.50**
Fruit	**-0.54**
% variance explained by PC2 = 5.37%	

* PCA accounted for the complex survey design. Observations used n=7,586; weighted population size n=90,698,784.

Table 2. Sociodemographic characteristics of older U.S. adults, 2013, and crude mean differences (95% CIs) of dietary pattern scores across levels of each characteristic[§]

Characteristics	Observed N	Weighted Prevalence (SE)	Prudent β	Prudent P-value*	Western β	Western P-value*
Age				<0.0001		<0.0001
<70 years	4260	68.45 (1.08)	Ref		Ref	
70 years and older	3326	31.55 (1.08)	-0.17 (-0.23, -0.12)		-0.38 (-0.44, -0.32)	
Sex				<0.0001		<0.0001
Male	3191	45.92 (0.45)	Ref		Ref	
Female	4395	54.08 (0.45)	0.34 (0.30, 0.39)		-0.32 (-0.37, -0.27)	
Race				<0.0001		0.3384
White	5230	77.84 (1.43)	Ref		Ref	
Black	1255	10.26 (0.65)	-0.11 (-0.19, -0.03)		-0.07 (-0.16, 0.02)	
Hispanic	858	8.40 (1.12)	0.26 (0.19, 0.33)		-0.09 (-0.22, 0.04)	
Other	243	3.50 (0.38)	0.15 (-0.09, 0.39)		0.01 (-0.18, 0.19)	
Paternal education				0.0003		0.4739
Less than 8 years	2000	22.09 (1.18)	Ref		Ref	
8+ years	5566	77.91 (1.18)	0.13 (0.06, 0.19)		-0.03 (-0.12, 0.05)	
Maternal education				0.0196		0.2110
Less than 8 years	1593	17.17 (1.04)	Ref		Ref	
8+ years	5984	82.83 (1.04)	0.08 (0.01, 0.15)		0.05 (-0.03, 0.13)	
Participant's education				<0.0001 (<0.0001)		0.0005 (0.0002)
Less than high school	1368	15.12 (0.75)	Ref		Ref	
High school	2497	31.88 (0.86)	0.08 (-0.02, 0.17)		-0.07 (-0.16, 0.02)	
Some college or above	3721	53.00 (1.08)	0.41 (0.32, 0.50)		-0.17 (-0.26, -0.07)	
Wealth quintile				<0.0001 (<0.0001)		<0.0001 (<0.0001)
Q1 (lowest)	1588	20.00 (0.90)	Ref		Ref	
Q2	1493	19.91 (0.79)	0.12 (0.04, 0.20)		0.03 (-0.09, 0.14)	
Q3	1521	20.08 (0.90)	0.15 (0.05, 0.26)		-0.03 (-0.13, 0.06)	
Q4	1437	20.01 (0.84)	0.35 (0.24, 0.46)		-0.16 (-0.26, -0.07)	
Q5 (highest)	1390	20.01 (1.05)	0.51 (0.40, 0.62)		-0.32 (-0.41, -0.23)	
Financial strain score	7574	0.77 (0.02)^	-0.03 (-0.06, 0)	0.0223	0.01 (-0.02, 0.04)	0.3889

[§]All analyses accounted for the complex survey design. Observations used n=7,586; weighted population size n=90,698,784.
^Weighted mean (SE)
*P-values are calculated from Type III tests; for ordinal variables, an additional P-value for linear trend is presented in parenthesis

Table 3. Multivariable-adjusted mean differences (95% CIs) of prudent dietary pattern score across levels of childhood and adulthood SES indicators in older U.S. adults, 2013[§]

	Model 1†		Model 2‡		Δβ^
	β	**P-value***	**β**	**P-value***	**(%)**
Age		<0.0001		<0.0001	
<70 years	Ref		Ref		
70 years and older	-0.16 (-0.21, -0.10)		-0.15 (-0.20, -0.09)		6
Sex		<0.0001		<0.0001	
Male	Ref		Ref		
Female	0.41 (0.35, 0.48)		0.43 (0.37, 0.50)		5
Race		<0.0001		<0.0001	
White	Ref		Ref		
Black	0.05 (-0.09, 0.18)		0.16 (0.02, 0.29)		220
Hispanic	0.44 (0.29, 0.59)		0.57 (0.42, 0.72)		30
Other	-0.02 (-0.34, 0.30)		0.03 (-0.26, 0.32)		250
Paternal education		0.0002		0.1429	
Less than 8 years	Ref		Ref		
8+ years	0.18 (0.09, 0.27)		0.06 (-0.03, 0.14)		**67**
Maternal education		0.0942		0.9194	
Less than 8 years	Ref		Ref		
8+ years	0.07 (-0.01, 0.16)		0 (-0.08, 0.08)		
Financial strain score	0.01 (-0.03, 0.05)	0.1148	0.03 (-0.01, 0.07)	0.8073	
Paternal education * race[a]	-	0.0067	-	0.0511	
Financial strain score * sex[a]	-	0.0089	-	0.0208	
Participant's education		-		<0.0001	
Less than high school	-		Ref		
High school	-		0.09 (0.01, 0.18)		
Some college or above	-		0.35 (0.26, 0.43)		
Wealth quintile		-		<0.0001	
Q1 (lowest)	-		Ref		
Q2	-		0.14 (0.06, 0.22)		
Q3	-		0.19 (0.08, 0.30)		
Q4	-		0.36 (0.24, 0.47)		
Q5 (highest)	-		0.50 (0.39, 0.62)		
[a]Group-specific associations, given the significant interactions above					
Paternal education (8+ vs. <8 years) among:					
White	*0.18 (0.09, 0.27)*	*0.0002*	*0.06 (-0.03, 0.14)*	*0.1822*	***67***
Black	*-0.01 (-0.13, 0.11)*	*0.8980*	*-0.05 (-0.17, 0.08)*	*0.4540*	
Hispanic	*0.06 (-0.13, 0.26)*	*0.5113*	*-0.06 (-0.26, 0.15)*	*0.5699*	
Other	*0.48 (0.23, 0.74)*	*0.0004*	*0.32 (0.08, 0.57)*	*0.0117*	***33***
Financial strain score (1-unit increment) among:					
Male	*0.01 (-0.03, 0.05)*	*0.5893*	*0.03 (-0.01, 0.07)*	*0.0896*	
Female	*-0.06 (-0.09, -0.02)*	*0.0022*	*-0.03 (-0.06, 0.01)*	*0.1347*	***50***

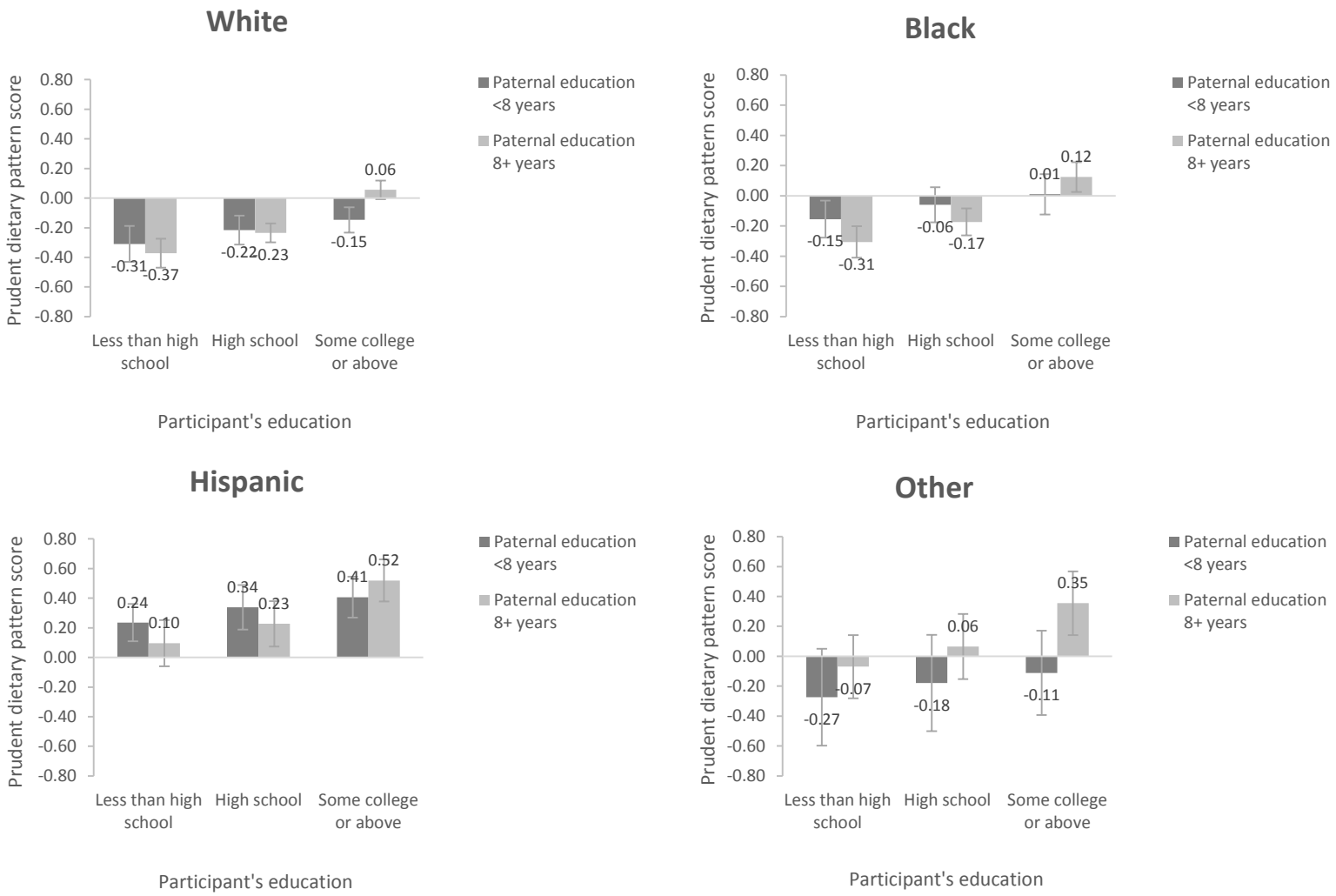
[§]All analyses accounted for the complex survey design. Observations used n=7,331; weighted population size n=87,438,319.
†Model 1: mutually adjusted, R^2=0.06
‡Model 2: Model 1 + further adjusting for participant's education and wealth quintile, R^2=0.11
*P-values are calculated from Type III tests
^For significant β's (p<0.05), Δβ is calculated as |β (Model 2) – β (Model 1) /β (Model 1)|*100%, bolded numbers indicate an attenuation of association of more than 10% in Model 2 as compared to Model 1

Table 4. Multivariable-adjusted mean differences (95% CIs) of Western dietary pattern score across levels of childhood and adulthood SES indicators in older U.S. adults, 2013[§]

	Model 1†		Model 2‡		Δβ^
	β	**P-value***	**β**	**P-value***	**(%)**
Age		<0.0001		<0.0001	
<70 years	Ref		Ref		
70 years and older	-0.35 (-0.42, -0.28)		-0.36 (-0.43, -0.29)		3
Sex		<0.0001		<0.0001	
Male	Ref		Ref		
Female	-0.43 (-0.55, -0.31)		-0.46 (-0.58, -0.34)		7
Race		0.0042		<0.0001	
White	Ref		Ref		
Black	-0.13 (-0.21, -0.05)		-0.23 (-0.31, -0.14)		77
Hispanic	-0.21 (-0.34, -0.07)		-0.30 (-0.44, -0.17)		43
Other	-0.07 (-0.25, 0.12)		-0.09 (-0.28, 0.09)		29
Paternal education		0.0037		0.1042	
Less than 8 years	Ref		Ref		
8+ years	-0.22 (-0.35, -0.09)		-0.16 (-0.29, -0.03)		**27**
Maternal education		0.9660		0.1901	
Less than 8 years	Ref		Ref		
8+ years	0 (-0.08, 0.08)		0.05 (-0.03, 0.13)		
Financial strain score	0.03 (0, 0.07)	0.4714	0.02 (-0.02, 0.05)	0.5537	
Paternal education * sex[a]	-	0.0240	-	0.0099	
Financial strain score * age[a]	-	0.0242	-	0.0392	
Participant's education		-		<0.0001	
Less than high school	-		Ref		
High school	-		-0.14 (-0.23, -0.04)		
Some college or above	-		-0.24 (-0.34, -0.14)		
Wealth quintile		-		<0.0001	
Q1 (lowest)	-		Ref		
Q2	-		0.02 (-0.09, 0.14)		
Q3	-		-0.02 (-0.12, 0.07)		
Q4	-		-0.14 (-0.25, -0.04)		
Q5 (highest)	-		-0.31 (-0.41, -0.20)		
[a]Group-specific associations, given the significant interactions above					
Paternal education (8+ vs. <8 years) among:					
Male	*-0.22 (-0.35, -0.09)*	*0.0015*	*-0.16 (-0.29, -0.03)*	*0.0147*	***27***
Female	*-0.07 (-0.16, 0.03)*	*0.1523*	*0.01 (-0.09, 0.10)*	*0.8801*	
Financial strain score (1-unit increment) among:					
<70 years	*0.03 (0, 0.07)*	*0.0394*	*0.02 (-0.02, 0.05)*	*0.3298*	***33***
70 years and older	*-0.02 (-0.05, 0.02)*	*0.3509*	*-0.03 (-0.06, 0)*	*0.0682*	

[§]All analyses accounted for the complex survey design. Observations used n=7,331; weighted population size n=87,438,319.
†Model 1: mutually adjusted, R^2=0.06
‡Model 2: Model 1 + further adjusting for participant's education and wealth quintile, R^2=0.08
*P-values are calculated from Type III tests
^For significant β's (p<0.05), Δβ is calculated as |β (Model 2) – β (Model 1)/β (Model 1)|*100%, bolded numbers indicate an attenuation of association of more than 10% in Model 2 as compared to Model 1

Figure 2. Average prudent dietary pattern score by paternal and participant's education levels, among whites, blacks, Hispanics, and other racial groups of older U.S. adults, 2013[§]



[§]Covariates in the model include: age, sex, race, paternal education, maternal education, financial strain score, participant's education, wealth quintile, as well as interaction terms of paternal education*race, paternal education*participant's education, and financial strain score*sex. All analyses accounted for the complex survey design. Observations used n=7,331; weighted population size n=87,438,319. R^2=0.11.

Supplementary Table 1. Components of total wealth (including secondary residence) calculation in HRS

Wealth Components	Debt
• value of primary residence, • value of secondary residence, • net value of real estate (not primary residence), • net value of vehicles, • net value of businesses, • net value of IRA/Keogh accounts, • net value of stocks/mutual funds/investment trusts, • value of checking/savings/money market accounts, • value of CD/government savings bonds/T-bills, • net value of bonds and bond funds, • net value of all other savings	• value of all mortgages/land contracts (primary residence), • value of other home loans (primary residence), • value of other debt, • value of all mortgages/land contracts (secondary residence)

Supplementary Table 2. Food groupings used in PCA

Food groups (n=40)	**Food items** (n=164)
Beer	Regular beer, light beer
Butter	Pure butter, spreadable butter
Chowder or cream soup	Chowder or cream soup
Coffee	Decaffeinated coffee, coffee with caffeine, dairy coffee drink
Cold breakfast cereal	Cold breakfast cereal
Condiments	Ketchup or red chili sauce, salt, low fat or fat free mayonnaise, regular mayonnaise
Dairy products, high-fat	Whole milk, cream, regular ice cream, cottage or ricotta cheese, cream cheese, other cheese
Dairy products, low-fat	Flavored yogurt, low carb yogurt, skim milk, 1% or 2% milk, frozen yogurt/sherbert/low-fat ice cream
Drinks, high-energy	Carbonated beverage with caffeine and sugar, other carbonated beverages with sugar, other sugared beverages
Drinks, low-energy	Low-calorie beverage with caffeine, other low-calorie beverages without caffeine, water (bottled, sparkling or tap)
Eggs	Egg Beaters or egg whites only, omega-3 fortified eggs (with yolk), regular eggs (with yolk)
Fish and other seafood	Canned tuna fish, breaded fish cakes/pieces/fish sticks, shrimp/lobster/scallops/clams as a main dish, dark meat fish, other fish
French fries	French fried potatoes
Fruit	Raisins or grapes, prunes or dried plums, melon, avocado, applesauce, fresh apples or pears, oranges, grapefruit, strawberries, other berries, peaches or plums, apricots, bananas
Fruit juices	Prune juice, apple juice or cider, fortified orange juice (with calcium or vitamin D), regular orange juice, grapefruit juice, other fruit juices
Garlic	Garlic
Grains, refined	White bread, bagels/English muffins/rolls, muffins or biscuits, white rice, pasta, tortillas, pancakes or waffles
Grains, whole	Cooked oatmeal/cooked oat bran, other cooked breakfast cereal, rye or Pumpernickel bread, whole wheat/oatmeal/other whole grain bread, brown rice, other grains, oat bran added to food, other bran added to food, wheat germ
Legumes	Soy milk, tofu/soy burgers/soybeans/miso/other soy protein, beans or lentils, peas or lima beans
Liquor	Liquor
Non-dairy fat	Margarine or spread, non-dairy coffee whitener
Nuts	Peanut butter, peanuts, walnuts, other nuts
Olive oil	Olive oil added to food or bread
Organ meats	Beef/calf/pork liver, chicken or turkey liver
Pizza	Pizza
Potatoes	Potatoes (baked/boiled/mashed)
Poultry	Chicken or turkey sandwich or frozen dinner, other chicken or turkey with skin, other chicken or turkey without skin
Processed meats	Bacon, beef or pork hot dogs, chicken or turkey hot dogs or sausage, salami/bologna/other processed meat sandwiches, other processed meats
Red meats	Lean or extra lean hamburger, regular hamburger, beef/pork/lamb as a sandwich or mixed dish, pork as a main dish, beef or lamb as a main dish
Salad dressing	Salad dressing
Snacks	Potato chips or corn/tortilla chips, whole wheat or whole grain crackers, regular or low fat crackers, other crackers, fat free or light popcorn, regular popcorn, pretzels
Sweeteners	Splenda, other artificial sweetener
Sweets and desserts	Cake*, pie*, sweet roll/coffee cake/other pastry†, cookies†, brownies, doughnuts, milk chocolate, dark chocolate, candy without chocolate, candy bars, energy bars, low carb bars, breakfast bars, sugar, jams/jellies/preserves/syrup/honey
Tea	Herbal tea or decaffeinated tea, tea with caffeine
Tomatoes	Tomatoes, tomato or V8 juice, tomato sauce, salsa/picante/taco sauce
Vegetables, cruciferous	Broccoli, cabbage or cole slaw, cauliflower, brussels sprouts
Vegetables, dark-yellow	Raw carrots, cooked carrots or carrot juice, dark orange (winter) squash, yams or sweet potatoes
Vegetables, green leafy	Cooked spinach, raw spinach as in salad, kale/mustard/chard greens, iceberg or head lettuce, romaine or leaf lettuce
Vegetables, other	Onions as a garnish or in salad, onions as cooked vegetable/rings/soup, corn, mixed or stir-fry vegetables/vegetable soup, eggplant/zucchini/other summer squash, celery, peppers (green/yellow/red), green beans or string beans
Wine	Red wine, white wine

*Combination of ready-made and home baked

†Combination of fat free or reduced fat, ready-made, and home baked

CHAPTER III: The Associations Between Adulthood Dietary Patterns and Prevalent Obesity, T2D, and HTN

Abstract

Background: In light of the recently proposed life course approach, the role of childhood SES on adulthood dietary behavior and CVD risk factors is starting to unfold. While dietary patterns have long been identified as a risk factor for obesity, T2D, and HTN, these examinations have not incorporated the life course approach.

Objective: Using a life course approach, this study examined the associations of adulthood dietary patterns with obesity, T2D, and HTN, separately, in older U.S. adults, taking into consideration the potential confounding by childhood SES.

Design: This is a cross-sectional examination of a random subsample of 8,035 adults over age 50 who were enrolled in the nationally representative longitudinal HRS. Participants self-reported their dietary behavior, childhood SES, and statuses of T2D and HTN. Obesity status was determined based on measured height and weight. Dietary patterns were derived from PCA. Logistic regression models were used to examine the association of obesity, T2D, and HTN, separately, with dietary patterns, adjusting for childhood SES and other potential confounders. All analyses took into account the complex survey design.

Results: We identified two main dietary patterns in older U.S. adults, including the prudent and the Western dietary patterns. After adjusting for childhood SES and other potential confounders, obesity and T2D were each positively associated with both prudent and Western dietary patterns ($p=0.0007$ and 0.0005, respectively, for obesity; $p<0.0001$ and $=0.0080$, respectively, for T2D).

HTN was positively associated with the prudent dietary pattern (p=0.0226) but not the Western dietary pattern.

Conclusions: This study found positive associations of the Western dietary pattern with obesity, T2D, and HTN, in consistency with prior studies, and positive associations of the prudent dietary pattern with the health outcomes, in contrary to prior studies. The latter is possibly due to the influence of reverse causation and survival bias. While childhood SES did not strongly confound the associations in our study, its impact as a potential confounder needs to be further evaluated in cohort studies where the relationships between dietary patterns and development of health outcomes can be better established.

Introduction

CVD is the leading cause of death in the U.S., and obesity, T2D, and HTN are its leading risk factors (1, 7). In 2015-2016, the prevalences of obesity, T2D, and HTN among U.S. adults were estimated to be 39.6%, 8.6%, and 29.8%, respectively, and were even higher among older adults (27-29). Not only could obesity, T2D, and HTN in older adults lead to life-threatening illnesses, primarily CVD, but also, they could lead to health problems that significantly reduce quality of life (33, 38, 42).

As the U.S. population is aging dramatically, by 2030, the proportion of population that are 65 years and older is projected to reach 21% (5). This would be driving a further increase in the health and economic burden of obesity, T2D, and HTN in the upcoming decade (117, 154, 155). In order to address this more efficiently, better understanding of obesity, T2D, and HTN, as well as their correlates, in older U.S. adults is needed. While inevitable physiological changes along

the aging process might contribute to the high prevalence of obesity, T2D, and HTN in older adults, lifestyle, which is modifiable, serves as another important contributor (33, 38, 42, 156).

Dietary intake of sodium, saturated fats, sugar-sweetened beverages and other high calorie processed foods has long been identified as a risk factor for obesity, T2D, and HTN (31, 35, 39). On the other hand, overall dietary behaviors and patterns have only recently been examined as potential risk factors. Among the most studied dietary patterns are the prudent and Western dietary patterns, which are two major dietary patterns empirically identified among older male health professionals and female nurses in the U.S. (52). Variations of these dietary patterns have been identified and studied among older U.S. adults of multi-ethnic backgrounds and the general U.S. adult population (17, 157). In addition, the index-based DASH diet is promoted to prevent and control HTN, and, therefore, has been mostly studied in relation to HTN (62). Overall, prior findings support that a "healthy" dietary pattern (e.g. fruits and vegetables, whole grains, poultry, fish, nuts, and low-fat dairy) is associated with reduced risk of obesity, T2D, and HTN; whereas an "unhealthy" dietary pattern (e.g. red meat, processed meat, refined grains, high-fat dairy products, sugar containing beverages, sweets and desserts) is associated with elevated risk of obesity, T2D, and HTN (16, 17, 55-57, 61, 62, 64).

In examination of the associations between dietary patterns and the health conditions, other lifestyle factors (e.g. smoking, alcohol intake, and physical activity) were commonly adjusted for as potential confounders in prior studies. However, no study that we are aware of has incorporated the potential impact of childhood conditions on the associations under study, in light of the recently proposed life course approach (158). Individuals with lower SES during

childhood have consistently been found to have elevated risk of obesity, T2D, and HTN (10, 11, 159). Our Aim 1 also showed that childhood SES could be associated with adulthood dietary patterns, independent of adulthood SES. Therefore, childhood SES could serve as a confounder to the associations of adulthood dietary patterns with obesity, T2D, and HTN.

Using a life course approach, this study examined the associations of adulthood dietary patterns with obesity, T2D, and HTN, separately, in the U.S. aging population, taking into consideration the potential confounding by childhood SES. With the use of nationally representative data, we were also able to examine adulthood SES as a potential confounder.

Methods

Study population

This study is a cross-sectional examination of HRS, a large longitudinal study of U.S. individuals over age 50. The design of HRS has been introduced in detail previously (121). Briefly, HRS was launched in 1992, with core surveys fielded every 2 years and supplemental studies taking place in off-years between core surveys starting 1999. Using a steady state design, HRS has covered seven birth cohorts so far, replenishing the sample with younger cohorts every 6 years. In any given wave, the combination of different birth cohorts generates a sample size of about 18,000-23,000. The core sample of HRS was selected based on a multi-stage area probability design. Additionally, HRS oversamples African-Americans, Hispanics and residents of the state of Florida. Sampling weights are derived to compensate for the unequal probabilities of selection between the core and oversample domains, as well as differential non-response in each wave (baseline response rate 69.9% - 81.6%, follow-up response rate 85.4% - 93.0%).

Participants of this study is comprised of a random subsample of HRS participants, who completed the supplemental off-year HCNS in 2013. All living HRS respondents of all ages and their spouse/partners, who were not included in the other off-year survey (Consumption and Activities Mail Survey) in 2013, were eligible to participate in HCNS. Among those 12,418 who were eligible, 65% responded. We excluded 37 respondents who answered very few of the food consumption questions and 1 respondent who reported as using a feeding tube. The final sample size for this study was 8,035, including 449 participants who were assigned zero sampling weights (therefore not used in weighted analyses) due to: 1) in a nursing home (n=1), 2) not cohort-eligible (n=434, e.g. HRS recruits spouses without age restriction), and 3) incomplete interview in the 2012 wave (n=14).

Data collection

Dietary intake

Information on dietary intake was collected using an embedded 164-item FFQ within the mailed HCNS. This self-administered FFQ was based on the Harvard FFQ, which has been validated against one-week diet records and 24-hour diet recalls over a 1-year period for intakes of individual nutrients (122-125) , individual foods (126, 127), food groups (128), and dietary patterns (129). For each food item in the FFQ, participants were asked to indicate how often on average they had used the amount specified over the past twelve months, ranging from never to multiple (e.g. ≥4, ≥5, or ≥6) times per day. In HCNS, 97% of respondents answered 90% or more of the food consumption questions. Missing data was imputed based on 5 respondent predictors from the core HRS survey, including age, sex, race, years of education, and BMI. Categorical

response of consumption frequency for each food item was converted to number of servings per day, using Harvard University's food serving conversion guides.

Additionally, information about participants' special diet status was also collected in 2013 HCNS by asking "Do you currently follow a special diet?" (yes/no). Types of special diet that were reported include low calorie, low cholesterol, low sodium, diabetic, low fat, low triglyceride, ulcer, high potassium, heart healthy/high blood pressure, gluten free, high protein, low carbohydrate/paleo, vegetarian/vegan, and low sugar diets.

CVD risk factors

Obesity status of each participant in 2013 was determined based on BMI (e.g. $\geq 30 kg/m^2$ vs. not) calculated from height and weight data that was measured in recent core interviews (e.g. weight (kg)/height $(m)^2$). Height was tape measured by having the respondent stand against a wall, without shoes. A marker is made on a post-it on the wall by the interviewer, who then measure the distance from the floor to the mark. Weight was measured on a Healthometer 830KL scale. Measured data from 2012, 2014, or 2010 core surveys was available for 85.40% of participants. In cases where recent measured data was unavailable, recent self-reported data was used (12.67% of participants); in cases where recent data was unavailable, we searched further back to 2004 and used in place the most recent measured or self-reported data available; in cases where BMI cannot be calculated in any of the waves (n=58), a mean BMI of the sample was assigned by gender (29.7 kg/m2 for men and 30.1 kg/m2 for women).

During the core interview when participants first join HRS, they were asked "Has a doctor ever told you that you have diabetes or high blood sugar?" and "Has a doctor ever told you that you have high blood pressure or HTN?" (yes/no), with follow-up questions about T2D and HTN statuses asked in each following core interviews. T2D and HTN statuses of each participant in 2014 were determined based on self-reported information in 2014 and all prior core interviews. Specifically, participants' T2D/HTN status in 2014 was determined to be "Yes" if they reported having been told by a doctor to have the condition in 2014 or any prior wave and to be "No" if they reported not having been told by a doctor to have the condition in 2014 and not otherwise in any of the prior waves. The majority of affirmative T2D and HTN statuses (83.28% and 92.70%, respectively) were based on participants' affirmative reporting in at least two waves of the core interview.

Childhood SES

Childhood SES was measured by three self-reported indicators: 1) paternal education level, 2) maternal education level, and 3) a composite score (ranging from 0-4) representing the intensity of childhood financial strain. Parental education captures participants' access to health-related knowledge and material resources during childhood, which could have long-lasting impact on their health behavior and health conditions throughout life (130). Financial strain experienced in childhood is a stressor that could also have persisted effect on health behavior and health status later in life (131, 132). In the absence of complete data across the life course, the quality of retrospectively reported childhood SES has been confirmed in prior studies (133, 134).

In HRS core surveys, information about parental education level was collected by asking "Did your father/mother attend 8 years or more of school?" with yes/no as possible answers or "What is the highest grade of school your father/mother completed?" with possible answers ranging from 0 to 17+. Summary measures of paternal and maternal education levels (8+ years vs. <8 years) were each generated from the first reported value across all waves, giving priority to the waves where the second set of questions were used. Credentials were used to measure education instead of years of schooling, assuming specific educational achievements are more important than time spent in education (135). To minimize potential selection bias, missing data for paternal and maternal education levels (15.42% and 8.66%, respectively) were imputed as needed. This imputation was a multivariate, regression-based procedure using Imputation and Variance Estimation (IVEware) software (http://www.isr.umich.edu/src/smp/ive/). The basis of imputation was a combination of sociodemographic characteristics (including mother/father alive, mother/father age at death, respondent's birth year, gender, race, and Hispanic ethnicity) and SES measures (including parental education of the opposite parent and the respondent's own education).

Childhood financial strain score was calculated as the total number of affirmative responses to four questions regarding financial status during respondents' childhood (before age 16), including 1) "did financial difficulties ever cause you or your family to move to a different place?" (1=yes and 0=no), 2) "was there a time when you or your family received help from relatives because of financial difficulties?" (1=yes and 0=no), 3) "was there a time of several months or more when your father had no job?" (1=yes and 0=no), and 4) "would you say your

family during that time was pretty well off financially, about average, or poor?" ("poor" or "varied" were coded as 1, and "average" or "well-off" were coded as 0).

Adulthood SES

Indicators of adulthood SES included education and wealth. Education captures the knowledge-related assets of participants (e.g. general and health-related knowledge, access appropriate health services, problem solving, material resources), and it is a strong determinant of subsequent employment and income (130, 135). Participants' educational attainments were measured by asking "What is the highest grade of school or year of college you completed?" with possible answers ranging from 0 to 17+. Years of education was then converted into three categories, including 1) less than high school (<12 years), 2) high school (12 years), and 3) some college or above (>12 years).

Wealth captures the accumulation of total assets (including financial and physical assets) and income, and it is especially important for older adults due to accumulation of assets over time and impact of retirement on income (136, 137). During HRS core interviews, participants were asked a series of questions about assets and income (e.g. "Do you have any shares of stock or stock mutual funds?" "About how much did you receive from stocks or stock mutual funds in last year (before taxes and other deductions)?" "Did you do any work for pay last year?" "About how much wage and salary income did you receive in last year, before taxes and other deductions?"). In cases where a participant was unable or unwilling to provide an exact amount of the value of assets or income, they were asked a series of follow-up questions to estimate a range (e.g. $0-2,500; $2,500-25,000; $25,000-125,000; $125,000-400,000; $400,000 or more).

Starting from the year of 2002, discrepancies (e.g. >$50,000 difference) of asset values across waves have been corrected for with additional asset verification questions (138). Additionally, missing data of all asset and income types were imputed using a consistent method across waves (138). For each participant, a total wealth (including secondary residence) in nominal dollars was calculated as sum of all wealth components less all debt (**Supplementary Table 1**). In this study, wealth was measured by quintiles of this total wealth according to its weighted distribution in the population.

Other health behaviors

Smoking status of each participant was determined in 2012 by asking "Have you ever smoked cigarettes?" and "Do you smoke cigarettes now?" (yes/no as possible answers to both questions). Participants who answered "no" to both questions were defined as "never smokers"; those who answered "yes" to the first question and "no" to the second question were defined as "former smokers"; and those who answered "yes" to both questions were defined as "current smokers."

Alcohol intake status of each participant was determined in 2012 by asking "Do you ever drink any alcoholic beverages, such as beer, wine, or liquor?" (yes/no). If a participant answered "yes," two follow-up questions were asked about his/her drinking behavior in the last three months, including 1) "on average, how many days per week have you had any alcohol to drink? (For example, beer, wine, or any drink containing liquor.)" (none or less than once a week and 1-7 as possible answers), 2) "on the days you drink, about how many drinks do you have?" If the answer to the first follow-up question was "none or less than once a week", then the second follow-up question is skipped. Participants who answered "no" to the initial question were

defined as “never drinkers”; for those who answered “yes” to the initial question, an average number of drinks per week was calculated as the product of the inputs from the two follow-up questions, which was then converted to average number of drinks per day. Referring to the 2015-2020 Dietary Guidelines for Americans, female participants who reported to have up to one drink per day on average and male participants who reported to have up to two drinks per day on average were defined as “moderate drinkers” (160); whereas female and male participants who reported to have more drinks per day on average than their corresponding cut points were defined as “heavy drinkers.”

Physical activity was measured by an index calculated as the weighted sum of daily frequencies for light (e.g. vacuuming, laundry, home repairs), moderate (e.g. gardening, cleaning the car, walking at a moderate pace, dancing, floor or stretching exercises), and vigorous (such as running or jogging, swimming, cycling, aerobics or gym workout, tennis, or digging with a spade or shovel) physical activity in 2012. Participants were asked about how often they took part in each level of physical activity, with every day, more than once a week, once a week, one to three times a month, or hardly ever or never as possible answers. These frequencies were first converted to daily frequencies (e.g. 1/day, 0.57/day, 0.14/day, 0.07/day, 0/day, respectively). Then, a weight of 2, 4, and 6 was assigned to the daily frequency of light, moderate, and vigorous physical activity, respectively, in calculating the overall index, referring to the intensity of effort required in metabolic equivalent for task (MET) scores at each activity level (161).

Assessment of dietary patterns using PCA

The 164 food items in the FFQ were first classified into 40 food groups, based on similar nutrient profiles or culinary usage (**Supplementary Table 2**). Daily consumption frequency for each food group was calculated as the sum of daily frequencies of all included food items, in terms of typical servings specified in the questionnaire. Daily frequencies of the 40 food groups were then adjusted for total energy intake using the residual method (139). This adjustment was performed on the log-scale, to improve normality, and without 63 participants who had extreme total energy intake (> 4 SD or < -4 SD on the original scale), so as to remove the influence of extreme values on regression results. Although these 63 participants had missing value for daily frequency residuals, they were kept in the analyses to maintain the integrity of sampling weights.

PCA was performed on daily frequency residuals of the 40 food groups, accounting for the complex survey design, so that participants' dietary behaviors were measured relative to the population distribution. To achieve this, we first created a weighted correlation matrix using the svycor function from the jtools package in R, accounting for the complex survey design (140, 141). Next, this weighted correlation matrix was used as the input for PCA, using the FACTOR PROCEDURE in SAS.

Two PCs were retained based on 1) percent variation explained by the component, 2) the Scree plot, and 3) interpretability. Since the un-rotated PC loadings did not follow simple structure (that is, a few close to ±1, the remainder close to 0), Varimax (e.g. orthogonal) rotation of PC loadings was performed to achieve better interpretability and independence between rotated PCs. Sensitivity analyses were performed on 1) retaining 3 PCs instead, 2) using Promax (e.g.

oblique) rotation instead, 3) without removing outliers of total energy intake, 4) daily frequencies of food groups adjusted for total energy intake on the original scale, 5) daily frequencies of food groups without adjusting for total energy intake, 6) daily frequencies of food items without grouping or total energy intake adjustment.

Each PC (to be translated later into a dietary pattern) represents a linear combination of residuals of all 40 food groups and explains as much inter-individual variation as possible. Each participant received a score for each identified PC calculated by summing consumptions of food groups weighted by their PC loadings, with a higher score indicating a greater adherence to the corresponding dietary pattern. PC scores were categorized into tertiles, according to its weighted distribution in the population, for better interpretation.

Statistical analysis

Data preparation and statistical analyses were performed using SAS software, Version 9.4 of the SAS System for Windows (SAS Institute Inc., Cary, NC, USA). Statistical associations were examined using logistic regression models, with obesity, T2D, and HTN statuses as separate outcome variables. Main exposure variables under examination were tertiles of prudent and Western dietary pattern scores. Other covariates included age (<70 years vs. 70 years and older), sex (male vs. female), race (white vs. black vs. Hispanic vs. other), smoking, alcohol intake, special diet status, physical activity index, childhood SES indicators (including paternal and maternal education levels and childhood financial strain score), and adulthood SES indicators (including participant's education level and wealth).

For each CVD risk factor (obesity, T2D, and HTN), a series of models were run in a step-wise fashion to assess its associations with the dietary patterns. First, we examined crude associations using univariate models. Second, we ran multivariate models with the adjustment of potential confounders, including age, sex, race, smoking, alcohol intake, special diet status, and physical activity. Results were compared before and after further adjusting for adulthood SES indicators (participant's education and wealth quintile) and childhood SES indicators (paternal education, maternal education, financial strain score) as potential confounders, so as to assess the impact of SES on the main associations of interest. Third, we estimated stratum-specific, fully adjusted (including SES indicators) associations for each health outcome by age, sex, race, participant's education and wealth quintile, with the use of interaction terms between dietary pattern tertiles and the corresponding stratifying variable. A P-value<0.05 for an interaction term indicates statistically significant difference across strata. Lastly, univariate log-binomial regression models were used to explore the association between dietary patterns (assuming stable over time) and incident T2D or HTN in 2012, 2014 and 2016. This analysis was not performed for obesity, considering the nature of obesity measurement in this study and height shrinkage in the aging process (162-164). The Wald Chi-Square test was used to test for the statistical significance of all covariate and interaction terms.

Results

Through weighted PCA, two major dietary patterns were identified in older U.S. adults, which, in combination, explained 16.32% of the overall variation in dietary intake (**Table 1**). The first dietary pattern (PC1), namely prudent dietary pattern, is characterized by high (loading>0.3) intake of vegetables, tomatoes, garlic, legumes, fruit, olive oil, fish and other seafood, and salad

dressing, as well as low (loading<-0.3) intake of sweets and desserts and high-energy drinks. The second dietary pattern (PC2), namely Western dietary pattern, is characterized by high intake of red meats, processed meats, condiments, French fries, and refined grains, as well as low intake of cold breakfast cereal, whole grains, low-fat dairy products, and fruit. These dietary patterns are robust to decisions on number of PCs to retain, rotation method used, outlier elimination, total energy intake adjustment, and food grouping, according to results of multiple sensitivity analyses on PCA.

A total of 6,784 observations with non-zero sampling weights and no missing data for explanatory or outcome variables were used in the statistical analysis. Of the weighted U.S. aging population, nearly 70% were under the age of 70 years, 54% were females, and 79% were whites (**Table 5**). Around half of the population had some college education or above, and the majority of them had at least one parent with high school education. Overall, around 44% of the U.S. aging population were obese, 26% were diabetic, and 63% were hypertensive. There appeared to be a crude association of the prudent dietary pattern with T2D (p=0.0001) and HTN (p=0.0042), and of the Western dietary pattern with obesity (p<0.0001) and T2D (p=0.0064). The prevalence of HTN tended to be lower in the 3rd tertile of the prudent dietary pattern and the 1st tertile of the Western dietary pattern. However, the prevalence of obesity and T2D tended to be lower in the 1st tertile of both dietary patterns, in comparison to the corresponding higher tertiles.

<u>Obesity</u>

Consistent with the crude associations, obesity was positively associated with both prudent and Western dietary patterns, after adjusting for age, sex, race, smoking, alcohol intake, special diet status, and physical activity index (p-value =0.0401 and 0.0001, respectively) (**Table 6**). Specifically, tertiles 2 and 3 of prudent dietary pattern were associated with 1.15 (95% CI: 0.99-1.34) and 1.21 (95% CI: 1.03-1.42) times higher odds of obesity, respectively, as compared to the 1st tertile; whereas tertiles 2 and 3 of Western dietary pattern were associated with 1.30 (95% CI: 1.14-1.50) and 1.39 (95% CI: 1.17-1.66) times higher odds of obesity, respectively, as compared to the 1st tertile. Further adjustment of SES indicators only slightly strengthened the ORs for the prudent dietary pattern and slightly attenuated those for the Western dietary pattern, without changing the directionality of the associations. Interestingly, race was no longer statistically significantly associated with obesity after adjustment of SES indicators. Among the childhood and adulthood SES indicators, only wealth was statistically significantly associated with obesity (p-value<0.0001).

The fully adjusted association between obesity and prudent dietary pattern significantly differed by age (p-value<0.0001) and participant's education (p-value=0.0013) (**Table 9**). Participants who were 70 years old and older appeared to have lower odds of having obesity compared to those younger than 70 years old, regardless of prudent diet status. Within each age group, the association between obesity and prudent dietary pattern appeared to be positive. A positive association between obesity and prudent dietary pattern was also observed among participants who had less than high school or high school education, but this directionality was less clear among those with some college education or above. Additionally, the fully adjusted association between obesity and Western dietary pattern significantly differed by race (p-value=0.0001). The

odds of having obesity appeared to be lower among other race compared to whites, blacks, and Hispanics, regardless of Western diet status. A positive association between obesity and Western dietary pattern was observed among whites and other race, but the directionality was less clear among Hispanics and was not observed among blacks.

T2D

Consistent with the crude associations, T2D was positively associated with both prudent and Western dietary patterns, after adjusting for age, sex, race, smoking, alcohol intake, special diet status, and physical activity index, although a dose-response relationship was not obvious for the Western dietary pattern (p-value <0.0001 and =0.0024, respectively) (**Table 7**). Specifically, tertiles 2 and 3 of prudent dietary pattern were associated with 1.47 (95% CI: 1.19-1.82) and 1.59 (95% CI: 1.35-1.88) times higher odds of T2D, respectively, as compared to the 1st tertile; whereas tertiles 2 and 3 of Western dietary pattern were associated with 1.31 (95% CI: 1.10-1.56) and 1.30 (95% CI: 1.11-1.53) times higher odds of T2D, respectively, as compared to the 1st tertile. Further adjustment of SES indicators only slightly strengthened the ORs for the prudent dietary pattern and slightly attenuated those for the Western dietary pattern, without changing the directionality of the associations. Among the childhood and adulthood SES indicators, only paternal education and wealth were statistically significantly associated with T2D (p-value=0.0116 and <0.0001, respectively).

The fully adjusted association between T2D and prudent dietary pattern significantly differed by participant's education (p-value=0.0461) (**Table 10**). Similar to what was observed for obesity, a positive association between T2D and prudent dietary pattern was observed among participants

who had less than high school or high school education, but this directionality was less clear among those with some college education or above. Additionally, the fully adjusted association between T2D and Western dietary pattern significantly differed by wealth (p-value=0.0223). A positive association between T2D and Western dietary pattern was observed among participants at the lowest wealth quintile, but this directionality was less clear among those in other wealth groups.

In exploration of the association between dietary patterns in 2013 and incident T2D, no statistically significant association was observed (**Table 12**). Between 2010-2012, there appeared to be a positive association between the prudent dietary pattern and incident T2D. This directionality became unclear in the 2012-2014 and 2014-2016 periods. The association between the Western dietary pattern and incident T2D was mostly positive across the three waves, but a dose-response relationship was not obvious.

HTN

In contrast to the crude associations, HTN was not statistically significantly associated with either dietary pattern, after adjusting for age, sex, race, smoking, alcohol intake, special diet status, and physical activity index (**Table 8**). It became statistically significantly positively associated with the prudent dietary pattern with the further adjustment of SES indicators (p-value=0.0226), although a dose-response relationship was not obvious. Among the childhood and adulthood SES indicators, only participant's education and wealth were statistically significantly associated with HTN (p-value=0.0039 and <0.0001, respectively).

The fully adjusted association between HTN and prudent dietary pattern significantly differed by race (p-value=0.0212) (**Table 11**). There appeared to be much higher odds of having HTN among blacks compared to whites, Hispanics, and other race, regardless of prudent diet status. While the association between the prudent dietary pattern and HTN appeared to be positive among the Hispanics, no clear directionality was observed for the other race groups. Additionally, the fully adjusted association between HTN and Western dietary pattern significantly differed by sex (p-value=0.0261). Females appeared to have lower odds of having HTN compared to males, regardless of Western diet status. There did not seem to be a clear directionality of the association between Western dietary pattern and HTN within each sex group.

In exploration of the association between dietary patterns in 2013 and incident HTN, a statistically significant association was observed for prudent diet in the 2010-2012 period (p-value=0.0392) (**Table 12**). While the directionality was not clear for the association between the prudent dietary pattern and incident HTN in the 2010-2012 and 2012-2014 periods, it appeared to be inverse between 2014-2016. In all three waves, we observed a positive association between the Western dietary pattern and incident HTN.

Discussion

Using nationally representative data, this study identified two main dietary patterns in older U.S. adults. As these dietary patterns are very similar to the prudent and Western dietary patterns identified in HPFS, NHS, and NHS II, we labeled them consistently. Compared to the first tertile, greater adherence to prudent dietary pattern, as well as greater adherence to Western

dietary pattern, was associated with higher odds of having obesity, T2D, and HTN, after adjusting for potential confounders. SES did not seem to be a strong confounder. However, this does not necessarily reflect the relationships among dietary patterns, SES, and risks of developing the health outcomes.

The positive associations between the Western dietary pattern and health outcomes that were observed in this study are consistent with findings from prior prospective studies. For example, the HPFS showed that the Western dietary pattern was positively associated with biomarkers of obesity (e.g. leptin) and T2D (e.g. insulin, C-peptide), as well as risk of T2D, in men aged 40-75 years (16, 55). The NHS and NHS II showed that the Western dietary pattern was associated with increased risk of T2D in women aged 38 to 63 years and larger weight gain in women aged 26 to 46 years, respectively (56, 57). Although not much is known about the effect of the Western dietary pattern on HTN in the U.S. population, per se, a study based on the Mexican Teachers' Cohort showed that this dietary pattern was associated with increased risk of HTN in Mexican women aged 25 years and older (58).

However, the positive associations between the prudent dietary pattern and health outcomes that were observed in this study are counterintuitive in comparison to findings from prior prospective studies. For example, the HPFS showed that the prudent dietary pattern was inversely associated with insulin concentration and risk of T2D in men aged 40-75 years (16, 55). The NHS and NHS II showed that the prudent dietary pattern was associated with decreased risk of T2D in women aged 38 to 63 years and smaller weight gain in women aged 26 to 46 years, respectively (56, 57). Although not much is known about the association between prudent dietary pattern, per se, and

HTN in the U.S. population, the DASH diet, which shares many elements with the prudent diet, has consistently been shown in randomized controlled trials to reduce both systolic and diastolic blood pressure in adults (62, 64).

In order to verify that the observed counterintuitive positive associations between the prudent dietary pattern and obesity, T2D, and HTN were not due to errors in the data or analysis, we performed a series of additional analyses and checks. First, we re-validated our initial dietary pattern analysis, with particular focuses on 1) checking the effectiveness of total energy intake adjustment before PCA, 2) checking the correlations between dietary pattern scores and daily frequency of food groups, and 3) examining the associations of the health outcomes (e.g. obesity, T2D, and HTN) with individual food groups. Second, we verified that the counterintuitive findings on the prudent dietary pattern were robust to its operationalization choices (e.g. continuous, binary, in quintiles). Third, we verified that the counterintuitive findings persisted under alternative model specifications (e.g. adjusting for total energy intake as a covariate, adjusting for BMI, without adjusting for special diet status, allowing for interactions). Lastly, we verified that the counterintuitive findings persisted using alternative analytical approaches (e.g. comparing mean dietary pattern scores by health outcome statuses). The persistence of conclusion after all the additional analyses and checks indicates the possible existence of other non-causal explanations.

One important factor that could have contributed to the discrepancy in findings on the prudent dietary pattern is reverse causation. As this study is cross-sectional by design, the temporality between dietary pattern measurements and onset of obesity, T2D, or HTN was not clearly

defined. In fact, based on self-reported T2D and HTN statuses across waves since 1992, we determined that among the prevalent cases of T2D and HTN in 2014, only less than 10% and less than 5%, respectively, were newly identified in the 2014 wave. In other words, the majority of prevalent T2D and HTN cases in this study were diagnosed before dietary patterns were measured in 2013. Given the nature of BMI measurement in this study, however, we were unable to perform similar analysis for onset of obesity. Since dietary change is a crucial component for managing obesity, T2D, and HTN, the measured dietary patterns of patients diagnosed in the 2012 wave and before could have been their modified (healthier) diet in response to disease diagnoses, rather than their original habitual diet (165-167).

Dietary changes among U.S. adults in response to obesity, T2D, or HTN diagnosis has been reported based on the NHANES data. Between 2001 and 2006, 63% of obese adults reported trying to lose weight in the previous year, among whom 40% lost ≥5% weight and 20% lost ≥10% weight, both associated with eating less fat (69). Between 1999 and 2014, adults diagnosed with diabetes or prediabetes consumed 14.9 grams less sugar and 11.6 grams less carbohydrate per day, compared to those with similar demographic characteristics and HbA1c levels but without a diagnosis; these reductions were even greater among those whose diagnoses were recent (70). Between 2007 and 2010, adults who received a diagnosis of HTN tended to have lower odds of using added salt and consume less sodium, compared to those who were undiagnosed with HTN or at risk for developing HTN; a decrease in consumption of total energy, protein, fats, and saturated fats was observed among newly diagnosed hypertensives but not those who had been diagnosed for a prolonged period of time (71). Yet, only a small

proportion (e.g. ~30% or less) of T2D or HTN patients were reported to be fully following the recommended diets (67, 68).

Our data also supports the existence of dietary change among the obese, diabetic, and hypertensive. First, participants who had the health outcomes were more likely to be on special diet (17.34%, 26.66%, and 17.06% for obese, diabetic, and hypertensive, respectively) than those who did not have the health outcomes (14.00%, 11.30%, and 12.43% for non-obese, non-diabetic, and non-hypertensive, respectively), although not 100% of the cases were diagnosed before dietary pattern measurements. Secondly, our sensitivity analysis without adjusting for special diet status in the models showed exaggerated parameter estimates for the prudent dietary pattern and attenuated parameter estimates for the Western dietary pattern. This indicates that dietary changes explained, at least partly, the observed positive associations between prudent dietary pattern and the health outcomes. It is worth noticing that not all dietary changes were necessarily captured by the special diet status. Had dietary changes been more properly adjusted for, we could have observed even further attenuated parameter estimates for the prudent dietary pattern that better line up with prior findings.

In further exploration of the potential existence of reverse causation in our data, we examined the crude associations of dietary patterns measured in 2013 with incident T2D and HTN cases captured between waves 2010-2012 (completely susceptible to reverse causation), 2012-2014 (partly susceptible to reverse causation), and 2014-2016 (not susceptible to reverse causation) (**Table 12**). Most of the parameter estimates did not reach statistical significance, likely due to small numbers of new cases captured over the two-year intervals. Still, our results showed that,

as the data became less susceptible to reverse causation, a positive association of the prudent dietary pattern with T2D or HTN became less supported or even reversed. On the contrary, the positive associations of the Western dietary pattern with T2D and HTN mostly remained, especially for HTN.

Survival bias is another potential contributor to the discrepancy in findings on the prudent dietary pattern between this study and prior studies. Cross-sectional by design, our study sample could only include adults who managed to live at the time when the survey was conducted. Since surviving becomes more and more challenging as people age, the potential for survival bias is especially high in older age groups of this aging population. Studies have shown that obesity, T2D, and HTN are each associated with elevated risk of mortality and contributing to a large proportion of deaths in U.S. adults (168-171). This makes survival, thus being eligible for the survey, even more challenging for those with the health conditions compared to those without. Among the obese, diabetic, or hypertensive, there is also evidence that healthier diet is associated with better survival (172-174). Therefore, participants in our study who were living with obesity, T2D, or HTN might have had healthier diet compared to those who had the condition but failed to survive, thus biasing our results towards a positive association between prudent dietary pattern and the health outcomes.

The main strength of this study is the life course perspective. Both childhood and adulthood SES indicators were included as potential confounders in the examination of the associations of adulthood dietary patterns with obesity, T2D, and HTN. Although these SES indicators might only have captured certain aspects of SES, this approach opens the door to more thorough

examination of CVD risk. Another strength of this study is the use of detailed dietary intake information collected from a large, nationally representative sample of older U.S. adults. By accounting for the complex survey design in the PCA process, we have identified two dietary patterns that could represent the overall dietary behavior in the U.S. aging population. However, because dietary intake information of the participants was collected only once, it might not have captured very well their long-term intake in comparison to repeated measures over the years. Although one dietary measure is sufficient for ranking participants in terms of dietary intake, it is prone to measurement error that can bias towards the null the association under study. As discussed above, the present study is also limited by its cross-sectional design, which makes our results susceptible to the influence of reverse causation and survival bias. Therefore, the observations in the present study should serve as the basis for hypotheses to be examined in future longitudinal studies, rather than causal inferences.

In conclusion, this cross-sectional examination of the U.S. aging population identified two main dietary patterns, namely prudent and Western dietary patterns. Under the potential influence of reverse causation and survival bias, positive associations of the Western dietary pattern with obesity, T2D, and HTN were observed, in consistency with prior studies; whereas positive associations of the prudent dietary pattern with the health outcomes were also observed, in contrary to prior studies. While SES did not strongly confound the associations in our study, its impact as a potential confounder needs to be further evaluated in cohort studies where the relationships between dietary patterns and development of health outcomes can be better established.

Table 5. Sociodemographic characteristics, lifestyle, and disease status by tertiles of dietary pattern scores in older U.S. adults, 2014[^§]

		Prudent dietary pattern				Western dietary pattern			
	Overall	**T1 (lowest) (n=2201)**	**T2 (n=2289)**	**T3 (highest) (n=2294)**	**P-value***	**T1 (lowest) (n=2377)**	**T2 (n=2263)**	**T3 (highest) (n=2144)**	**P-value***
Age					<.0001				<.0001
<70 years	3868 (69.63)	1175 (67.08)	1272 (68.18)	1421 (73.61)		1145 (60.41)	1293 (69.80)	1430 (78.45)	
70 years and older	2916 (30.37)	1026 (32.92)	1017 (31.82)	873 (26.39)		1232 (39.59)	970 (30.20)	714 (21.55)	
Sex					<.0001				<.0001
Male	2858 (46.07)	1105 (55.19)	996 (47.48)	757 (35.60)		846 (39.60)	924 (43.17)	1088 (55.22)	
Female	3926 (53.93)	1096 (44.81)	1293 (52.52)	1537 (64.40)		1531 (60.40)	1339 (56.83)	1056 (44.78)	
Race					<.0001				0.8333
White	4741 (78.74)	1574 (80.63)	1602 (78.08)	1565 (77.52)		1653 (78.80)	1561 (77.9)	1527 (79.50)	
Black	1105 (9.99)	402 (11.32)	377 (10.27)	326 (8.38)		369 (9.99)	396 (10.71)	340 (9.28)	
Hispanic	726 (7.86)	176 (5.39)	238 (8.31)	312 (9.85)		273 (7.67)	241 (8.29)	212 (7.62)	
Other	212 (3.42)	49 (2.66)	72 (3.35)	91 (4.26)		82 (3.55)	65 (3.11)	65 (3.61)	
Smoking					<.0001				<.0001
Never	3048 (45.14)	948 (42.11)	1014 (44.55)	1086 (48.75)		1288 (56.15)	1017 (44.77)	743 (34.76)	
Former	2933 (41.20)	875 (37.67)	1010 (41.84)	1048 (44.06)		957 (37.69)	1012 (43.56)	964 (42.31)	
Current	803 (13.66)	378 (20.23)	265 (13.61)	160 (7.19)		132 (6.16)	234 (11.67)	437 (22.93)	
Alcohol intake					<.0001				<.0001
Never	3025 (41.76)	1154 (49.78)	995 (40.94)	876 (34.64)		1138 (46.55)	1000 (39.96)	887 (38.86)	
Moderate	3342 (51.18)	943 (44.62)	1157 (52.19)	1242 (56.67)		1170 (50.44)	1139 (53.92)	1033 (49.21)	
Heavy	417 (7.06)	104 (5.60)	137 (6.87)	176 (8.69)		69 (3.01)	124 (6.13)	224 (11.93)	
Special diet					<.0001				<.0001
No	5727 (84.37)	2000 (91.18)	1969 (86.24)	1758 (75.75)		1904 (80.61)	1936 (84.40)	1887 (88.02)	
Yes	1057 (15.63)	201 (8.82)	320 (13.76)	536 (24.25)		473 (19.39)	327 (15.60)	257 (11.98)	
Physical activity index	3.47 (0.06)	2.90 (0.06)	3.32 (0.08)	4.20 (0.09)	<.0001	3.91 (0.08)	3.45 (0.08)	3.07 (0.07)	<.0001
Financial strain score	0.77 (0.02)	0.77 (0.03)	0.83 (0.03)	0.70 (0.03)	0.0080	0.72 (0.03)	0.79 (0.03)	0.78 (0.03)	0.1444
Paternal education					<.0001				0.2772
Less than 8 years	1732 (21.16)	578 (21.15)	640 (24.59)	514 (17.74)		599 (19.75)	594 (21.51)	539 (22.20)	
8+ years	5052 (78.84)	1623 (78.85)	1649 (75.41)	1780 (82.26)		1778 (80.25)	1669 (78.49)	1605 (77.80)	
Maternal education					0.2153				0.9583
Less than 8 years	1360 (16.30)	442 (16.44)	481 (17.53)	437 (14.91)		506 (16.51)	463 (16.24)	391 (16.14)	
8+ years	5424 (83.70)	1759 (83.56)	1808 (82.47)	1857 (85.09)		1871 (83.49)	1800 (83.76)	1753 (83.86)	
Participant's education					<.0001				0.0001
Less than high school	1157 (14.27)	474 (17.79)	388 (14.62)	295 (10.42)		377 (11.82)	376 (14.41)	404 (16.51)	
High school	2242 (31.95)	824 (37.90)	780 (32.71)	638 (25.29)		766 (31.92)	746 (30.70)	730 (33.21)	
Some college or above	3385 (53.78)	903 (44.31)	1121 (52.66)	1361 (64.30)		1234 (56.27)	1141 (54.89)	1010 (50.28)	
Wealth quintile					<.0001				<.0001
Q1 (lowest)	1410 (19.53)	538 (24.97)	460 (19.19)	412 (14.47)		447 (17.49)	462 (19.27)	501 (21.76)	
Q2	1336 (19.48)	487 (22.22)	445 (18.80)	404 (17.43)		405 (16.34)	445 (19.39)	486 (22.63)	
Q3	1392 (20.14)	502 (22.02)	494 (21.58)	396 (16.84)		463 (19.41)	456 (19.29)	473 (21.69)	
Q4	1342 (20.50)	361 (17.01)	466 (20.60)	515 (23.86)		512 (21.79)	467 (21.39)	363 (18.36)	
Q5 (highest)	1304 (20.36)	313 (13.79)	424 (19.83)	567 (27.41)		550 (24.97)	433 (20.66)	321 (15.57)	
Obesity					0.2273				<.0001
No	3765 (55.97)	1283 (57.78)	1240 (54.91)	1242 (55.25)		1460 (61.56)	1190 (53.86)	1115 (52.60)	
Yes	3019 (44.03)	918 (42.22)	1049 (45.09)	1052 (44.75)		917 (38.44)	1073 (46.14)	1029 (47.40)	
T2D					0.0001				0.0064
No	4842 (73.81)	1635 (77.86)	1614 (71.68)	1593 (71.93)		1762 (76.33)	1564 (72.10)	1516 (73.04)	
Yes	1942 (26.19)	566 (22.14)	675 (28.32)	701 (28.07)		615 (23.67)	699 (27.90)	628 (26.96)	
HTN					0.0042				0.2211
No	2192 (36.92)	685 (36.24)	706 (34.44)	801 (40.07)		806 (38.30)	729 (37.38)	657 (35.11)	
Yes	4592 (63.08)	1516 (63.76)	1583 (65.56)	1493 (59.93)		1571 (61.70)	1534 (62.62)	1487 (64.89)	

[^]Dietary pattern data was from 2013 FFQ

[§]All analyses accounted for the complex survey design. Observations used n=6,784; weighted population size n=81,125,269. For physical activity index and financial strain score, mean (SE) is reported; for all other variables, n (weighted %) is reported.

*P-values are calculated from F tests for physical activity index and financial strain score; Rao-Scott chi-square tests for all other variables.

Table 6. Multivariable-adjusted ORs for obesity by tertiles of dietary pattern scores before and after adjusting for SES indicators in older U.S. adults, 2014^§

	Model 1		Model 2	
	OR	**P-value***	**OR**	**P-value***
Prudent dietary pattern score		0.0401		0.0007
T1 (lowest)	Ref		Ref	
T2	1.15 (0.99, 1.34)		1.21 (1.04, 1.40)	
T3 (highest)	1.21 (1.03, 1.42)		1.34 (1.14, 1.57)	
Western dietary pattern score		0.0001		0.0005
T1 (lowest)	Ref		Ref	
T2	1.30 (1.14, 1.50)		1.28 (1.12, 1.48)	
T3 (highest)	1.39 (1.17, 1.66)		1.33 (1.12, 1.58)	
Age		<.0001		<.0001
<70 years	Ref		Ref	
70 years and older	0.53 (0.46, 0.60)		0.55 (0.48, 0.63)	
Sex		0.4661		0.2083
Male	Ref		Ref	
Female	0.94 (0.81, 1.10)		0.91 (0.78, 1.06)	
Race		0.0027		0.1108
White	Ref		Ref	
Black	1.25 (1.08, 1.45)		1.06 (0.88, 1.26)	
Hispanic	1.15 (0.90, 1.47)		1.00 (0.78, 1.29)	
Other	0.68 (0.45, 1.04)		0.65 (0.43, 0.98)	
Smoking		<.0001		<.0001
Never	Ref		Ref	
Former	1.08 (0.95, 1.22)		1.03 (0.90, 1.17)	
Current	0.48 (0.40, 0.58)		0.43 (0.35, 0.52)	
Alcohol intake		<.0001		<.0001
Never	Ref		Ref	
Moderate	0.80 (0.69, 0.93)		0.86 (0.74, 1.00)	
Heavy	0.49 (0.36, 0.65)		0.53 (0.40, 0.70)	
Special diet		0.0554		0.0804
No	Ref		Ref	
Yes	1.21 (1.00, 1.46)		1.19 (0.98, 1.43)	
Physical activity index	0.87 (0.84, 0.89)	<.0001	0.88 (0.86, 0.90)	<.0001
Financial strain score	-	-	1.03 (0.97, 1.09)	0.2955
Paternal education				0.1799
Less than 8 years	-	-	Ref	
8+ years	-	-	0.92 (0.80, 1.04)	
Maternal education				0.0724
Less than 8 years	-	-	Ref	
8+ years	-	-	1.15 (0.99, 1.34)	
Participant's education				0.3856
Less than high school	-	-	Ref	
High school	-	-	1.13 (0.95, 1.34)	
Some college or above	-	-	1.11 (0.89, 1.39)	
Wealth quintile				<.0001
Q1 (lowest)	-	-	Ref	
Q2	-	-	0.97 (0.81, 1.17)	
Q3	-	-	0.87 (0.70, 1.08)	
Q4	-	-	0.64 (0.52, 0.80)	
Q5 (highest)	-	-	0.46 (0.36, 0.59)	

^Dietary pattern data was from 2013 FFQ

§All the covariates were mutually adjusted. All analyses accounted for the complex survey design. Observations used n=6,784; weighted population size n=81,125,269.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Table 7. Multivariable-adjusted ORs for T2d by tertiles of dietary pattern scores before and after adjusting for SES indicators in older U.S. adults, 2014^[^§]

	Model 1		Model 2	
	OR	**P-value***	**OR**	**P-value***
Prudent dietary pattern score		<.0001		<.0001
T1 (lowest)	Ref		Ref	
T2	1.47 (1.19, 1.82)		1.52 (1.22, 1.89)	
T3 (highest)	1.59 (1.35, 1.88)		1.74 (1.47, 2.06)	
Western dietary pattern score		0.0024		0.0080
T1 (lowest)	Ref		Ref	
T2	1.31 (1.10, 1.56)		1.29 (1.08, 1.53)	
T3 (highest)	1.30 (1.11, 1.53)		1.25 (1.06, 1.47)	
Age		<.0001		0.0001
<70 years	Ref		Ref	
70 years and older	1.38 (1.19, 1.60)		1.35 (1.16, 1.58)	
Sex		<.0001		<.0001
Male	Ref		Ref	
Female	0.67 (0.59, 0.77)		0.65 (0.57, 0.74)	
Race		<.0001		<.0001
White	Ref		Ref	
Black	1.93 (1.65, 2.27)		1.63 (1.35, 1.96)	
Hispanic	1.79 (1.31, 2.46)		1.38 (1.00, 1.92)	
Other	1.73 (0.98, 3.05)		1.59 (0.89, 2.84)	
Smoking		0.0110		0.0006
Never	Ref		Ref	
Former	1.13 (0.99, 1.30)		1.08 (0.93, 1.25)	
Current	0.82 (0.69, 0.99)		0.74 (0.62, 0.88)	
Alcohol intake		<.0001		<.0001
Never	Ref		Ref	
Moderate	0.65 (0.56, 0.76)		0.71 (0.61, 0.83)	
Heavy	0.35 (0.25, 0.49)		0.38 (0.28, 0.53)	
Special diet		<.0001		<.0001
No	Ref		Ref	
Yes	3.05 (2.60, 3.58)		3.07 (2.63, 3.58)	
Physical activity index	0.86 (0.84, 0.88)	<.0001	0.87 (0.85, 0.90)	<.0001
Financial strain score	-	-	1.00 (0.94, 1.07)	0.9197
Paternal education				0.0116
Less than 8 years	-	-	Ref	
8+ years	-	-	0.79 (0.66, 0.95)	
Maternal education				0.5684
Less than 8 years	-	-	Ref	
8+ years	-	-	1.06 (0.87, 1.30)	
Participant's education				0.0611
Less than high school	-	-	Ref	
High school	-	-	0.86 (0.72, 1.03)	
Some college or above	-	-	0.79 (0.65, 0.96)	
Wealth quintile				<.0001
Q1 (lowest)	-	-	Ref	
Q2	-	-	0.81 (0.66, 1.00)	
Q3	-	-	0.97 (0.81, 1.17)	
Q4	-	-	0.73 (0.60, 0.89)	
Q5 (highest)	-	-	0.60 (0.49, 0.74)	

^Dietary pattern data was from 2013 FFQ

§All the covariates were mutually adjusted. All analyses accounted for the complex survey design. Observations used n=6,784; weighted population size n=81,125,269.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Table 8. Multivariable-adjusted ORs for HTN by tertiles of dietary pattern scores before and after adjusting for SES indicators in older U.S. adults, 2014^§

	Model 1		Model 2	
	OR	**P-value***	**OR**	**P-value***
Prudent dietary pattern score		0.0769		0.0226
T1 (lowest)	Ref		Ref	
T2	1.18 (1.02, 1.37)		1.24 (1.06, 1.45)	
T3 (highest)	1.07 (0.91, 1.27)		1.18 (1.01, 1.39)	
Western dietary pattern score		0.1955		0.5085
T1 (lowest)	Ref		Ref	
T2	1.05 (0.91, 1.22)		1.03 (0.89, 1.18)	
T3 (highest)	1.19 (0.98, 1.43)		1.12 (0.92, 1.36)	
Age		<.0001		<.0001
<70 years	Ref		Ref	
70 years and older	2.33 (1.98, 2.74)		2.30 (1.95, 2.70)	
Sex		0.0026		0.0003
Male	Ref		Ref	
Female	0.80 (0.69, 0.92)		0.76 (0.66, 0.88)	
Race		<.0001		<.0001
White	Ref		Ref	
Black	2.64 (2.24, 3.12)		2.12 (1.78, 2.52)	
Hispanic	1.42 (1.15, 1.74)		0.98 (0.78, 1.24)	
Other	1.07 (0.74, 1.54)		0.94 (0.65, 1.35)	
Smoking		0.0270		0.0203
Never	Ref		Ref	
Former	1.17 (1.03, 1.33)		1.11 (0.98, 1.26)	
Current	0.89 (0.70, 1.14)		0.77 (0.60, 0.98)	
Alcohol intake		<.0001		0.0019
Never	Ref		Ref	
Moderate	0.74 (0.64, 0.86)		0.83 (0.71, 0.96)	
Heavy	1.06 (0.81, 1.40)		1.20 (0.91, 1.59)	
Special diet		<.0001		<.0001
No	Ref		Ref	
Yes	1.65 (1.37, 1.99)		1.63 (1.36, 1.97)	
Physical activity index	0.86 (0.84, 0.88)	<.0001	0.87 (0.85, 0.90)	<.0001
Financial strain score	-	-	1.04 (0.97, 1.11)	0.2699
Paternal education				0.0673
Less than 8 years	-	-	Ref	
8+ years	-	-	0.84 (0.70, 1.01)	
Maternal education				0.7555
Less than 8 years	-	-	Ref	
8+ years	-	-	0.96 (0.75, 1.24)	
Participant's education				0.0039
Less than high school	-	-	Ref	
High school	-	-	0.87 (0.68, 1.09)	
Some college or above	-	-	0.74 (0.61, 0.90)	
Wealth quintile				<.0001
Q1 (lowest)	-	-	Ref	
Q2	-	-	0.93 (0.74, 1.17)	
Q3	-	-	0.72 (0.57, 0.90)	
Q4	-	-	0.67 (0.54, 0.84)	
Q5 (highest)	-	-	0.57 (0.46, 0.71)	

^Dietary pattern data was from 2013 FFQ

§All the covariates were mutually adjusted. All analyses accounted for the complex survey design. Observations used n=6,784; weighted population size n=81,125,269.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Table 9. Stratum-specific multivariable-adjusted ORs for obesity by tertiles of dietary pattern scores in older U.S. adults, 2014[^§]

	Prudent dietary pattern				Western dietary pattern			
	T1 (lowest)	**T2**	**T3 (highest)**	**P-value***	**T1 (lowest)**	**T2**	**T3 (highest)**	**P-value***
By age				<.0001				0.5587
<70 years	Ref	1.06 (0.89, 1.27)	1.13 (0.93, 1.37)		Ref	1.25 (1.03, 1.52)	1.28 (1.03, 1.58)	
70 years and older	0.39 (0.33, 0.48)	0.63 (0.50, 0.79)	0.80 (0.65, 0.99)		0.51 (0.42, 0.63)	0.69 (0.55, 0.86)	0.76 (0.57, 1.01)	
By sex				0.1328				0.7012
Male	Ref	1.16 (0.88, 1.52)	1.51 (1.17, 1.96)		Ref	1.16 (0.85, 1.57)	1.24 (0.89, 1.72)	
Female	0.94 (0.70, 1.28)	1.17 (0.94, 1.46)	1.16 (0.94, 1.44)		0.82 (0.60, 1.11)	1.13 (0.90, 1.42)	1.14 (0.84, 1.56)	
By race				0.6194				0.0001
White	Ref	1.17 (0.95, 1.44)	1.34 (1.13, 1.59)		Ref	1.31 (1.11, 1.55)	1.44 (1.17, 1.76)	
Black	0.93 (0.68, 1.27)	1.40 (1.00, 1.95)	1.46 (1.03, 2.05)		1.48 (1.13, 1.94)	1.47 (1.13, 1.91)	0.99 (0.72, 1.37)	
Hispanic	1.12 (0.73, 1.72)	1.12 (0.70, 1.80)	1.31 (0.95, 1.81)		0.97 (0.64, 1.47)	1.44 (1.00, 2.07)	1.35 (0.84, 2.15)	
Other	0.63 (0.32, 1.23)	1.09 (0.66, 1.79)	0.67 (0.36, 1.23)		0.64 (0.36, 1.16)	0.86 (0.44, 1.70)	0.93 (0.49, 1.76)	
By participant's education				0.0013				0.9698
Less than high school	Ref	1.78 (1.23, 2.59)	1.96 (1.31, 2.94)		Ref	1.16 (0.77, 1.76)	1.17 (0.74, 1.85)	
High school	1.24 (0.92, 1.66)	1.77 (1.30, 2.40)	2.14 (1.49, 3.08)		1.04 (0.74, 1.46)	1.30 (0.94, 1.81)	1.41 (0.98, 2.02)	
Some college or above	1.68 (1.17, 2.43)	1.55 (1.11, 2.17)	1.71 (1.24, 2.37)		1.01 (0.70, 1.45)	1.34 (0.93, 1.93)	1.36 (0.98, 1.89)	
By wealth quintile				0.3677				0.0813
Q1 (lowest)	Ref	1.34 (1.00, 1.80)	1.13 (0.81, 1.60)		Ref	0.84 (0.60, 1.17)	1.03 (0.71, 1.50)	
Q2	0.86 (0.62, 1.18)	1.18 (0.87, 1.59)	1.49 (1.12, 1.98)		0.88 (0.66, 1.16)	0.99 (0.68, 1.44)	0.93 (0.63, 1.39)	
Q3	0.87 (0.64, 1.17)	0.99 (0.72, 1.35)	1.23 (0.81, 1.87)		0.57 (0.40, 0.82)	0.97 (0.70, 1.33)	1.00 (0.71, 1.41)	
Q4	0.70 (0.49, 1.01)	0.71 (0.53, 0.94)	0.85 (0.59, 1.23)		0.51 (0.35, 0.74)	0.66 (0.49, 0.90)	0.67 (0.47, 0.95)	
Q5 (highest)	0.49 (0.32, 0.73)	0.56 (0.38, 0.82)	0.58 (0.42, 0.80)		0.32 (0.22, 0.47)	0.52 (0.38, 0.71)	0.51 (0.33, 0.79)	

[^]Dietary pattern data was from 2013 FFQ

[§]Models adjusted for age, sex, race, smoking, alcohol intake, physical activity index, special diet status, participant's education, wealth quintile, paternal education, maternal education, and childhood financial strain score. All analyses accounted for the complex survey design. Observations used n=6,784; weighted population size n=81,125,269.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Table 10. Stratum-specific multivariable-adjusted ORs for T2d by tertiles of dietary pattern scores in older U.S. adults, 2014[^§]

	Prudent dietary pattern				Western dietary pattern			
	T1 (lowest)	**T2**	**T3 (highest)**	**P-value***	**T1 (lowest)**	**T2**	**T3 (highest)**	**P-value***
By age				0.3878				0.1169
<70 years	Ref	1.62 (1.27, 2.08)	1.87 (1.56, 2.25)		Ref	1.31 (1.02, 1.69)	1.38 (1.08, 1.76)	
70 years and older	1.54 (1.17, 2.04)	2.10 (1.63, 2.71)	2.35 (1.91, 2.89)		1.50 (1.18, 1.90)	1.92 (1.43, 2.57)	1.51 (1.18, 1.92)	
By sex				0.0856				0.8387
Male	Ref	1.66 (1.29, 2.14)	2.10 (1.64, 2.70)		Ref	1.21 (0.90, 1.63)	1.22 (0.94, 1.57)	
Female	0.79 (0.63, 0.99)	1.07 (0.82, 1.40)	1.15 (0.93, 1.42)		0.62 (0.49, 0.77)	0.83 (0.65, 1.05)	0.78 (0.61, 1.01)	
By race				0.6189				0.0724
White	Ref	1.60 (1.26, 2.02)	1.82 (1.49, 2.23)		Ref	1.27 (1.04, 1.55)	1.24 (1.02, 1.50)	
Black	1.92 (1.44, 2.57)	2.21 (1.57, 3.13)	2.97 (2.12, 4.16)		1.78 (1.29, 2.44)	2.15 (1.53, 3.01)	1.76 (1.23, 2.50)	
Hispanic	1.24 (0.75, 2.07)	2.26 (1.37, 3.73)	2.59 (1.73, 3.88)		0.97 (0.66, 1.43)	1.92 (1.22, 3.02)	2.09 (1.29, 3.41)	
Other	2.45 (1.00, 6.00)	2.63 (1.20, 5.74)	2.11 (1.08, 4.11)		2.15 (1.04, 4.44)	1.51 (0.74, 3.10)	1.87 (0.85, 4.10)	
By participant's education				0.0461				0.4657
Less than high school	Ref	1.64 (1.13, 2.40)	2.60 (1.71, 3.96)		Ref	1.02 (0.66, 1.57)	0.99 (0.61, 1.63)	
High school	0.96 (0.67, 1.38)	1.37 (0.94, 2.00)	1.86 (1.36, 2.55)		0.66 (0.46, 0.95)	0.97 (0.65, 1.44)	0.94 (0.61, 1.43)	
Some college or above	0.96 (0.67, 1.37)	1.46 (1.00, 2.13)	1.40 (0.99, 1.98)		0.67 (0.44, 1.02)	0.86 (0.57, 1.27)	0.84 (0.59, 1.19)	
By wealth quintile				0.1244				0.0223
Q1 (lowest)	Ref	1.47 (1.00, 2.16)	1.41 (1.00, 1.99)		Ref	1.62 (1.17, 2.24)	1.73 (1.18, 2.53)	
Q2	0.69 (0.50, 0.94)	1.00 (0.65, 1.54)	1.70 (1.21, 2.39)		1.15 (0.75, 1.74)	1.19 (0.81, 1.75)	1.18 (0.80, 1.75)	
Q3	0.90 (0.64, 1.28)	1.38 (0.96, 1.99)	1.62 (1.13, 2.31)		1.07 (0.73, 1.56)	1.77 (1.23, 2.54)	1.42 (0.99, 2.03)	
Q4	0.65 (0.46, 0.91)	1.28 (0.85, 1.91)	1.01 (0.72, 1.42)		1.00 (0.70, 1.44)	1.18 (0.78, 1.80)	0.93 (0.66, 1.31)	
Q5 (highest)	0.64 (0.40, 1.02)	0.77 (0.52, 1.14)	1.01 (0.73, 1.41)		0.78 (0.57, 1.06)	0.78 (0.53, 1.14)	1.07 (0.70, 1.62)	

[^]Dietary pattern data was from 2013 FFQ

[§]Models adjusted for age, sex, race, smoking, alcohol intake, physical activity index, special diet status, participant's education, wealth quintile, paternal education, maternal education, and childhood financial strain score. All analyses accounted for the complex survey design. Observations used n=6,784; weighted population size n=81,125,269.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Table 11. Stratum-specific multivariable-adjusted ORs for HTN by tertiles of dietary pattern scores in older U.S. adults, 2014[^§]

	Prudent dietary pattern				Western dietary pattern			
	T1 (lowest)	**T2**	**T3 (highest)**	**P-value***	**T1 (lowest)**	**T2**	**T3 (highest)**	**P-value***
By age				0.2079				0.7973
<70 years	Ref	1.26 (1.04, 1.52)	1.12 (0.92, 1.36)		Ref	1.04 (0.85, 1.26)	1.11 (0.88, 1.39)	
70 years and older	2.17 (1.70, 2.77)	2.55 (1.98, 3.28)	3.11 (2.38, 4.08)		2.29 (1.73, 3.05)	2.27 (1.75, 2.95)	2.70 (1.99, 3.67)	
By sex				0.1061				0.0261
Male	Ref	1.32 (1.10, 1.60)	1.46 (1.11, 1.92)		Ref	0.78 (0.60, 1.02)	0.97 (0.75, 1.26)	
Female	0.91 (0.73, 1.14)	1.03 (0.84, 1.26)	0.91 (0.75, 1.10)		0.60 (0.47, 0.77)	0.75 (0.58, 0.96)	0.74 (0.54, 1.01)	
By race				0.0212				0.7261
White	Ref	1.28 (1.05, 1.55)	1.23 (1.03, 1.45)		Ref	1.02 (0.87, 1.18)	1.11 (0.90, 1.36)	
Black	2.59 (1.98, 3.38)	2.08 (1.55, 2.80)	2.75 (1.86, 4.06)		2.14 (1.56, 2.93)	2.23 (1.54, 3.23)	2.22 (1.48, 3.33)	
Hispanic	1.10 (0.77, 1.58)	1.12 (0.77, 1.64)	1.23 (0.91, 1.66)		0.81 (0.57, 1.15)	1.12 (0.77, 1.61)	1.16 (0.72, 1.86)	
Other	0.85 (0.43, 1.71)	2.15 (1.06, 4.38)	0.80 (0.52, 1.23)		1.12 (0.66, 1.93)	0.70 (0.34, 1.42)	1.13 (0.61, 2.10)	
By participant's education				0.8881				0.0995
Less than high school	Ref	1.09 (0.80, 1.48)	0.99 (0.66, 1.50)		Ref	1.44 (0.92, 2.25)	1.23 (0.75, 2.03)	
High school	0.80 (0.57, 1.13)	0.99 (0.69, 1.42)	0.92 (0.66, 1.28)		0.98 (0.66, 1.45)	1.14 (0.79, 1.63)	1.05 (0.69, 1.60)	
Some college or above	0.66 (0.49, 0.90)	0.85 (0.64, 1.13)	0.82 (0.63, 1.08)		0.89 (0.63, 1.26)	0.81 (0.57, 1.16)	1.01 (0.70, 1.48)	
By wealth quintile				0.4648				0.0625
Q1 (lowest)	Ref	1.10 (0.71, 1.69)	1.06 (0.73, 1.52)		Ref	1.31 (0.94, 1.83)	1.01 (0.63, 1.62)	
Q2	0.80 (0.57, 1.12)	0.95 (0.67, 1.34)	1.31 (0.99, 1.72)		0.81 (0.53, 1.24)	0.85 (0.65, 1.11)	1.40 (1.01, 1.92)	
Q3	0.66 (0.47, 0.92)	0.91 (0.67, 1.25)	0.72 (0.47, 1.11)		0.75 (0.56, 1.01)	0.80 (0.54, 1.19)	0.78 (0.57, 1.08)	
Q4	0.60 (0.44, 0.83)	0.79 (0.57, 1.08)	0.76 (0.55, 1.06)		0.77 (0.53, 1.10)	0.66 (0.45, 0.98)	0.75 (0.50, 1.13)	
Q5 (highest)	0.60 (0.40, 0.88)	0.68 (0.47, 1.00)	0.59 (0.43, 0.81)		0.60 (0.41, 0.87)	0.60 (0.42, 0.86)	0.65 (0.41, 1.03)	

[^]Dietary pattern data was from 2013 FFQ

[§]Models adjusted for age, sex, race, smoking, alcohol intake, physical activity index, special diet status, participant's education, wealth quintile, paternal education, maternal education, and childhood financial strain score. All analyses accounted for the complex survey design. Observations used n=6,784; weighted population size n=81,125,269.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Table 12. Crude RRs for incident T2D and HTN by tertiles of dietary pattern scores, HRS[^§]

	2012			2014			2016		
	$N_{cases}/N_{at\ risk}$[†]	RR	P-value*	$N_{cases}/N_{at\ risk}$[†]	RR	P-value*	$N_{cases}/N_{at\ risk}$[†]	RR	P-value*
T2D									
Prudent dietary pattern			0.0599			0.0849			0.8123
T1 (lowest)	57/1951	Ref		57/1853	Ref		55/1523	Ref	
T2	62/1922	1.10 (0.77, 1.57)		53/1813	0.95 (0.66, 1.37)		70/1575	1.23 (0.87, 1.74)	
T3 (highest)	79/1970	1.37 (0.98, 1.92)		77/1882	1.33 (0.95, 1.86)		57/1642	0.96 (0.67, 1.38)	
Western dietary pattern			0.6271			0.1777			0.2486
T1 (lowest)	66/2086	Ref		57/1997	Ref		64/1711	Ref	
T2	68/1893	1.14 (0.81, 1.58)		66/1789	1.29 (0.91, 1.83)		49/1515	0.86 (0.60, 1.25)	
T3 (highest)	64/1864	1.09 (0.77, 1.52)		64/1762	1.27 (0.90, 1.81)		69/1514	1.22 (0.87, 1.70)	
HTN									
Prudent dietary pattern			0.0392			0.4450			0.0545
T1 (lowest)	92/957	Ref		72/855	Ref		67/697	Ref	
T2	95/972	1.02 (0.77, 1.34)		73/854	1.02 (0.74, 1.39)		64/706	0.94 (0.68, 1.31)	
T3 (highest)	79/1112	0.74 (0.55, 0.99)		77/1028	0.89 (0.65, 1.21)		61/877	0.72 (0.52, 1.01)	
Western dietary pattern			0.3471			0.0589			0.3936
T1 (lowest)	90/1103	Ref		72/1000	Ref		66/849	Ref	
T2	88/995	1.08 (0.82, 1.44)		69/898	1.07 (0.78, 1.47)		65/751	1.11 (0.80, 1.55)	
T3 (highest)	88/943	1.14 (0.86, 1.51)		81/839	1.34 (0.99, 1.82)		61/680	1.15 (0.83, 1.61)	

[^]Dietary pattern data was from 2013 FFQ

[§]Analyses did not account for the complex survey design.

[†]$N_{at\ risk}$=number of participants at risk of developing the outcome at the end of previous waive; N_{cases}=number of participants newly developed the outcome between the previous and current waves.

*P values for trend are calculated from Wald Chi-square tests, with corresponding dietary pattern tertiles coded as continuous variable.

CHAPTER IV: The Associations of SES and Dietary Patterns with the Multimorbidity of Obesity, T2D, and HTN

Abstract

Background: Multimorbidity is highly prevalent among older adults. The coexistence of obesity, T2D, and HTN is a consistent multimorbidity pattern across populations. However, very little is known about this specific multimorbidity pattern and its risk factors.

Objective: This study aimed to explore the associations of the multimorbidity of obesity, T2D, and HTN with adulthood dietary patterns, adulthood SES, and childhood SES in older U.S. adults.

Design: This is a cross-sectional examination of a random subsample of 8,035 adults over age 50 who were enrolled in the nationally representative longitudinal HRS. Participants self-reported their dietary behavior, childhood and adulthood SES, and statuses of T2D and HTN. Obesity status was determined primarily based on measured height and weight. An indicator of multimorbidity status of obesity, T2D, and HTN was generated based on the combination of statuses of individual health outcomes. PCA was used to derive dietary patterns, and multinomial logistic regression models were used to assess the associations of the multimorbidity of obesity, T2D, and HTN with adulthood dietary patterns, adulthood SES, and childhood SES. All analyses took into account the complex survey design.

Results: Nearly 80% of older U.S. adults had obesity, T2D, or HTN, among whom more than half were living with multimorbidity of these conditions. The majority (~86%) of those with T2D were also living with HTN. Childhood SES (primarily paternal education) and adulthood

SES (education and wealth) were both independently inversely associated with the multimorbidity statuses, with wealth being the most strongly and robustly associated SES indicator (P<0.0001). Both the prudent and Western dietary patterns were positively associated with the multimorbidity statuses (P's<0.0001) but without clear dose-response relationships across tertiles. A higher number of coexisting conditions were not necessarily more strongly associated with the exposures under study.

Conclusions: The multimorbidity of obesity, T2D, and HTN is highly prevalent among older U.S. adults, and it is independently associated with lower childhood SES (primarily paternal education), lower adulthood SES (especially wealth), and greater adherence to both prudent and Western dietary patterns in adulthood. The role of paternal education and wealth in the development and management of the multimorbidity of obesity, T2D, and HTN needs to be further investigated; whereas the roles of dietary patterns need to be re-evaluated in prospective studies.

Introduction

Multimorbidity, the coexistence of multiple chronic conditions, is highly prevalent among older adults (81, 82). In the U.S., 91.8% of adults aged 65 years or older were living with 2 or more chronic conditions (e.g. CVD, chronic obstructive pulmonary disease, chronic kidney disease, asthma, arthritis, cancer, stroke, HTN, hyperlipidemia, diabetes, and obesity) between 2013 and 2014 (83). Not only is multimorbidity in older adults associated with disability, functional decline, and poorer quality of life, but also, it can lead to higher health care utilization and costs (81). Yet, very little is known about risk factors for multimorbidity and their corresponding underlying mechanisms.

A “metabolic” multimorbidity pattern, characterized by the coexistence of obesity, T2D, and HTN, has consistently been identified among adults older than 50 years in low-, middle-, and high-income countries (12). Obesity, T2D, and HTN are highly prevalent chronic conditions in older adults, and they all serve as leading independent risk factors of CVD, the leading cause of death in the U.S. (1, 7, 27-29). Not surprisingly, the coexistence of obesity, T2D, and HTN in older adults is associated with substantially further elevated risk of CVD, worsen health-related quality of life, health services use, and mortality (84-88). A pooled analysis of four cohort studies in the U.S. found that at age 45 years, the absence of obesity, T2D, and HTN, in comparison to the coexistence of all three conditions, is associated with 73% to 85% lower risk of incident heart failure over the remaining life course (87). As the U.S. population continues to age, it is of great public health importance to examine the presence of this “metabolic” multimorbidity pattern in the population and identify its risk factors for more efficient interventions to reduce the health and economic burden of obesity, T2D, and HTN (5).

Environmental factors, together with genetic factors, have been indicated to be driving the multimorbidity of obesity, T2D, and HTN (98, 99). While dietary behavior, a modifiable environmental factor, has long been identified as a risk factor for obesity, T2D, and HTN individually, not much is known about its association with the multimorbidity of these three conditions (31, 35, 39). A cross-sectional study among U.S. adults aged 20 years and older in NHANES 1999-2002 found that high fiber intake was associated with lower level of C-reactive protein, which was positively associated with multimorbidity of obesity, T2D, and HTN, compared to no or single condition (100). Empirically identified dietary patterns, as better

representations of individuals' overall dietary behavior, however, have not been examined in relation to the multimorbidity of obesity, T2D, and HTN (52).

SES, an important social environment indicator, has also been consistently shown to be inversely associated with obesity, T2D, and HTN individually in high-income countries (101-103). In addition to SES in adulthood (e.g. education, occupation, income), there has been an increasing interest in childhood SES (e.g. parental education, paternal occupation) in relation to obesity, T2D, and HTN, individually, from a life-course perspective (104-107). Yet, the literature on the relationships of childhood SES and adulthood SES with the multimorbidity of obesity, T2D, and HTN is sparse. In Spanish adults aged ≥60 years, a report in 2004 showed that lower social class in adulthood, regardless of childhood social class, was associated with higher prevalence of the multimorbidity of abdominal obesity, T2D, and HTN among women (108). This topic has not been examined in older U.S. adults.

The objective of this study was to explore the associations of multimorbidity of obesity, T2D, and HTN with adulthood dietary patterns, adulthood SES, and childhood SES in older U.S. adults, using nationally representative data of the HRS. Rather than focusing on the number of coexisting chronic conditions or the general contrasts between multimorbidity and individual conditions, as was a common approach in prior studies, this study examined all specific combinations of obesity, T2D, and HTN statuses, as each specific combination (e.g. obesity only vs. obesity + T2D vs. obesity + T2D + HTN) might indicate a unique underlying mechanism.

Methods

Study population

This study is a cross-sectional examination of HRS, a large longitudinal study of U.S. individuals over age 50. The design of HRS has been introduced in detail previously (121). Briefly, HRS was launched in 1992, with core surveys fielded every 2 years and supplemental studies taking place in off-years between core surveys starting 1999. Using a steady state design, HRS has covered seven birth cohorts so far, replenishing the sample with younger cohorts every 6 years. In any given wave, the combination of different birth cohorts generates a sample size of about 18,000-23,000. The core sample of HRS was selected based on a multi-stage area probability design. Additionally, HRS oversamples African-Americans, Hispanics and residents of the state of Florida. Sampling weights are derived to compensate for the unequal probabilities of selection between the core and oversample domains, as well as differential non-response in each wave (baseline response rate 69.9% - 81.6%, follow-up response rate 85.4% - 93.0%).

Participants of this study is comprised of a random subsample of HRS participants, who completed the supplemental off-year HCNS in 2013. All living HRS respondents of all ages and their spouse/partners, who were not included in the other off-year survey (Consumption and Activities Mail Survey) in 2013, were eligible to participate in HCNS. Among those 12,418 who were eligible, 65% responded. We excluded 37 respondents who answered very few of the food consumption questions and 1 respondent who reported as using a feeding tube. The final sample size for this study was 8,035, including 449 participants who were assigned zero sampling weights (therefore not used in weighted analyses) due to: 1) in a nursing home (n=1), 2) not cohort-eligible (n=434, e.g. HRS recruits spouses without age restriction), and 3) incomplete interview in the 2012 wave (n=14).

Data collection

CVD risk factors

Obesity status of each participant in 2013 was determined based on BMI (e.g. ≥30kg/m^2 vs. not) calculated from height and weight data that was measured in recent core interviews (e.g. weight (kg)/height $(m)^2$). Height was tape measured by having the respondent stand against a wall, without shoes. A marker is made on a post-it on the wall by the interviewer, who then measure the distance from the floor to the mark. Weight was measured on a Healthometer 830KL scale. Measured data from 2012, 2014, or 2010 core surveys was available for 85.40% of participants. In cases where recent measured data was unavailable, recent self-reported data was used (12.67% of participants); in cases where recent data was unavailable, we searched further back to 2004 and used in place the most recent measured or self-reported data available; in cases where BMI cannot be calculated in any of the waves (n=58), a mean BMI of the sample was assigned by gender (29.7 kg/m2 for men and 30.1 kg/m2 for women).

During the core interview when participants first join HRS, they were asked "Has a doctor ever told you that you have diabetes or high blood sugar?" and "Has a doctor ever told you that you have high blood pressure or HTN?" (yes/no), with follow-up questions about T2D and HTN statuses asked in each following core interviews. T2D and HTN statuses of each participant in 2014 were determined based on self-reported information in 2014 and all prior core interviews. Specifically, participants' T2D/HTN status in 2014 was determined to be "Yes" if they reported having been told by a doctor to have the condition in 2014 or any prior wave and to be "No" if they reported not having been told by a doctor to have the condition in 2014 and not otherwise in

any of the prior waves. The majority of affirmative T2D and HTN statuses (83.28% and 92.70%, respectively) were based on participants' affirmative reporting in at least two waves of the core interview.

For each participant, an indicator of multimorbidity status of obesity, T2D, and HTN was generated based on the combination of his/her statuses of individual health outcomes. Since each of the three health outcomes was measured as binary (yes/no), there were a total of eight possible multimorbidity statuses: 1) none, 2) obesity only, 3) T2D only, 4) HTN only, 5) obesity + T2D, 6) obesity + HTN, 7) T2D + HTN, and 8) obesity + T2D + HTN.

Dietary intake

Information on dietary intake was collected using an embedded 164-item FFQ within the mailed HCNS. This self-administered FFQ was based on the Harvard FFQ, which has been validated against one-week diet records and 24-hour diet recalls over a 1-year period for intakes of individual nutrients (122-125) , individual foods (126, 127), food groups (128), and dietary patterns (129). For each food item in the FFQ, participants were asked to indicate how often on average they had used the amount specified over the past twelve months, ranging from never to multiple (e.g. ≥4, ≥5, or ≥6) times per day. In HCNS, 97% of respondents answered 90% or more of the food consumption questions. Missing data was imputed based on 5 respondent predictors from the core HRS survey, including age, sex, race, years of education, and BMI. Categorical response of consumption frequency for each food item was converted to number of servings per day, using Harvard University's food serving conversion guides.

Additionally, information about participants' special diet status was also collected in 2013 HCNS by asking "Do you currently follow a special diet?" (yes/no). Types of special diet that were reported include low calorie, low cholesterol, low sodium, diabetic, low fat, low triglyceride, ulcer, high potassium, heart healthy/high blood pressure, gluten free, high protein, low carbohydrate/paleo, vegetarian/vegan, and low sugar diets.

Childhood SES

Childhood SES was measured by three self-reported indicators: 1) paternal education level, 2) maternal education level, and 3) a composite score (ranging from 0-4) representing the intensity of childhood financial strain. Parental education captures participants' access to health-related knowledge and material resources during childhood, which could have long-lasting impact on their health behavior and health conditions throughout life (130). Financial strain experienced in childhood is a stressor that could also have persisted effect on health behavior and health status later in life (131, 132). In the absence of complete data across the life course, the quality of retrospectively reported childhood SES has been confirmed in prior studies (133, 134).

In HRS core surveys, information about parental education level was collected by asking "Did your father/mother attend 8 years or more of school?" with yes/no as possible answers or "What is the highest grade of school your father/mother completed?" with possible answers ranging from 0 to 17+. Summary measures of paternal and maternal education levels (8+ years vs. <8 years) were each generated from the first reported value across all waves, giving priority to the waves where the second set of questions were used. Credentials were used to measure education instead of years of schooling, assuming specific educational achievements are more important

than time spent in education (135). To minimize potential selection bias, missing data for paternal and maternal education levels (15.42% and 8.66%, respectively) were imputed as needed. This imputation was a multivariate, regression-based procedure using Imputation and Variance Estimation (IVEware) software (http://www.isr.umich.edu/src/smp/ive/). The basis of imputation was a combination of sociodemographic characteristics (including mother/father alive, mother/father age at death, respondent's birth year, gender, race, and Hispanic ethnicity) and SES measures (including parental education of the opposite parent and the respondent's own education).

Childhood financial strain score was calculated as the total number of affirmative responses to four questions regarding financial status during respondents' childhood (before age 16), including 1) "did financial difficulties ever cause you or your family to move to a different place?" (1=yes and 0=no), 2) "was there a time when you or your family received help from relatives because of financial difficulties?" (1=yes and 0=no), 3) "was there a time of several months or more when your father had no job?" (1=yes and 0=no), and 4) "would you say your family during that time was pretty well off financially, about average, or poor?" ("poor" or "varied" were coded as 1, and "average" or "well-off" were coded as 0).

Adulthood SES

Indicators of adulthood SES included education and wealth. Education captures the knowledge-related assets of participants (e.g. general and health-related knowledge, access appropriate health services, problem solving, material resources), and it is a strong determinant of subsequent employment and income (130, 135). Participants' educational attainments were measured by

asking “What is the highest grade of school or year of college you completed?” with possible answers ranging from 0 to 17+. Years of education was then converted into three categories, including 1) less than high school (<12 years), 2) high school (12 years), and 3) some college or above (>12 years).

Wealth captures the accumulation of total assets (including financial and physical assets) and income, and it is especially important for older adults due to accumulation of assets over time and impact of retirement on income (136, 137). During HRS core interviews, participants were asked a series of questions about assets and income (e.g. “Do you have any shares of stock or stock mutual funds?” “About how much did you receive from stocks or stock mutual funds in last year (before taxes and other deductions)?” “Did you do any work for pay last year?” “About how much wage and salary income did you receive in last year, before taxes and other deductions?”). In cases where a participant was unable or unwilling to provide an exact amount of the value of assets or income, they were asked a series of follow-up questions to estimate a range (e.g. $0-2,500; $2,500-25,000; $25,000-125,000; $125,000-400,000; $400,000 or more). Starting from the year of 2002, discrepancies (e.g. >$50,000 difference) of asset values across waves have been corrected for with additional asset verification questions (138). Additionally, missing data of all asset and income types were imputed using a consistent method across waves (138). For each participant, a total wealth (including secondary residence) in nominal dollars was calculated as sum of all wealth components less all debt (**Supplementary Table 1**). In this study, wealth was measured by quintiles of this total wealth according to its weighted distribution in the population.

Assessment of dietary patterns using PCA

The 164 food items in the FFQ were first classified into 40 food groups, based on similar nutrient profiles or culinary usage (**Supplementary Table 2**). Daily consumption frequency for each food group was calculated as the sum of daily frequencies of all included food items, in terms of typical servings specified in the questionnaire. Daily frequencies of the 40 food groups were then adjusted for total energy intake using the residual method (139). This adjustment was performed on the log-scale, to improve normality, and without 63 participants who had extreme total energy intake (> 4 SD or < -4 SD on the original scale), so as to remove the influence of extreme values on regression results. Although these 63 participants had missing value for daily frequency residuals, they were kept in the analyses to maintain the integrity of sampling weights.

PCA was performed on daily frequency residuals of the 40 food groups, accounting for the complex survey design, so that participants' dietary behaviors were measured relative to the population distribution. To achieve this, we first created a weighted correlation matrix using the svycor function from jtools package in R, accounting for the complex survey design (140, 141). Next, this weighted correlation matrix was used as the input for PCA, using the FACTOR PROCEDURE in SAS.

Two PCs were retained based on 1) percent variation explained by the component, 2) the Scree plot, and 3) interpretability. Since the un-rotated PC loadings did not follow simple structure (that is, a few close to ±1, the remainder close to 0), Varimax (e.g. orthogonal) rotation of PC loadings was performed to achieve better interpretability and independence between rotated PCs. Sensitivity analyses were performed on 1) retaining 3 PCs instead, 2) using Promax (e.g.

oblique) rotation instead, 3) without removing outliers of total energy intake, 4) daily frequencies of food groups adjusted for total energy intake on the original scale, 5) daily frequencies of food groups without adjusting for total energy intake, 6) daily frequencies of food items without grouping or total energy intake adjustment.

Each PC (to be translated later into a dietary pattern) represents a linear combination of residuals of all 40 food groups and explains as much inter-individual variation as possible. Each participant will receive a score for each identified PC calculated by summing consumptions of food groups weighted by their PC loadings, with a higher score indicating a greater adherence to the corresponding dietary pattern. PC scores were categorized into tertiles, according to its weighted distribution in the population, for better interpretation.

Statistical analysis

Data preparation and statistical analyses were performed using SAS software, Version 9.4 of the SAS System for Windows (SAS Institute Inc., Cary, NC, USA). Statistical associations were examined using multinomial logistic regression models, with multimorbidity status of obesity, T2D, and HTN as the outcome variable. Main exposure variables under examination included tertiles of Prudent and Western dietary pattern scores, childhood SES indicators (including paternal and maternal education levels and childhood financial strain score), and adulthood SES indicators (including participant's education level and wealth). Other covariates included age (<70 years vs. 70 years and older), sex (male vs. female), and race (white vs. black vs. Hispanic vs. other).

The associations between the joint frequency distribution of health outcomes and the exposure variables were assessed in two steps. First, we examined crude association for each exposure variable using a univariate model. Then, we ran multivariate models with the mutual adjustment of all exposure variables under study, as well as the further adjustment of age, sex, and race as potential confounders. Statistical significance of all associations were tested using the Type 3 Wald Chi-Square test.

Results

Through weighted PCA, two major dietary patterns were identified in older U.S. adults, which, in combination, explained 16.32% of the overall variation in dietary intake (**Table 1**). The first dietary pattern (PC1), namely Prudent dietary pattern, is characterized by high (loading>0.3) intake of vegetables, tomatoes, garlic, legumes, fruit, olive oil, fish and other seafood, and salad dressing, as well as low (loading<-0.3) intake of sweets and desserts and high-energy drinks. The second dietary pattern (PC2), namely Western dietary pattern, is characterized by high intake of red meats, processed meats, condiments, French fries, and refined grains, as well as low intake of cold breakfast cereal, whole grains, low-fat dairy products, and fruit. These dietary patterns are robust to decisions on number of PCs to retain, rotation method used, outlier elimination, total energy intake adjustment, and food grouping, according to results of multiple sensitivity analyses on PCA.

A total of 7,076 observations with non-zero sampling weights and no missing data for explanatory or outcome variables were used in the statistical analysis. Of the weighted U.S. aging population, nearly 70% were under the age of 70 years, 54% were females, and 78% were

whites (**Table 13**). Around half of the population had some college education or above, and the majority of them had at least one parent with high school education. Overall, nearly 80% of older U.S. adults had at least one of the health outcomes, among whom more than half were living with more than one condition. The most prevalent multimorbidity statuses were "HTN only" (22.96%), followed by "None" (22.89%), "Obesity + HTN" (17.62%), and "All" (14.45%). The least prevalent multimorbidity statuses were "T2D Only" (1.75%) and "Obesity + T2D" (2.08%), indicating the majority (~86%) of older adults living with T2D were also living with HTN. Compared to participants with at least one health condition under study, those with no health conditions tended to be younger, mostly white, more educated, wealthier, have lower childhood financial strain score and more educated parents, and adhere less to the Western diet. Among those with one or more health conditions, the distributions of sociodemographic characteristics and dietary patterns varied across multimorbidity statuses without obvious trend.

Our data showed that the overall prevalence of obesity, T2D, and HTN, individually, in older U.S. adults was 43.99%, 26.69%, and 63.44%, respectively. If the three chronic conditions coexist with each other due to chance alone, the expected prevalence of each multimorbidity status can be calculated as the product of the overall prevalences of the underlying conditions, assuming independence among the three conditions. (**Supplementary Table 3**). Compared to the expected prevalence, the corresponding observed prevalence was higher for "None" and "Obesity + T2D + HTN" but lower for all other multimorbidity statuses. Additionally, our Rao-Scott Chi-Square test showed significant association between obesity and T2D statuses ($P < 0.0001$), obesity and HTN statuses ($P < 0.0001$), as well as T2D and HTN statuses ($P < 0.0001$).

Overall, there appeared to be a crude association of the joint frequency distribution of obesity, T2D, and HTN with all the SES indicators and both dietary patterns (all P-values <0.0001) (**Table 14**). After adjusting for each other, as well as age, sex, and race, maternal education was no longer statistically significantly associated with the outcome (P-value = 0.1688), and childhood financial strain score became less statistically significantly associated with the outcome (P-value = 0.0372) (**Table 15**). In fact, 1-unit increment of childhood financial strain score was only borderline significantly associated with HTN only (OR=1.10, 95% CI: 1.02-1.18), obesity + HTN (OR=1.13, 95% CI: 1.04-1.22), and T2D + HTN (OR=1.11, 95% CI: 1.00-1.23). While the joint frequency distribution of the health outcomes remained highly statistically significantly associated with the other SES indicators after the adjustment, the magnitude of associations were greatly attenuated for paternal education and participant's education but only slightly attenuated for wealth quintiles. Changes of parameter estimates for the dietary patterns after adjusting for the covariates appeared to be in varied directions.

To facilitate the reading of the multiple ORs across multimorbidity statuses, we plotted the crude ORs in **Figure 3** and multivariable-adjusted ORs in **Figure 4**, converting the ORs that were less than 1 (indicating protective effects) to their reciprocals so that their magnitudes relative to the null (OR=1) were more visually comparable to the ORs that were greater than 1 (indicating risk effects). As shown in Figure 4, the ORs for each exposure variable or confounder across multimorbidity statuses did not follow a simple pattern (e.g. monotonically stronger or weaker association with larger number of comorbidities). For example, while multimorbidity of all three health conditions (e.g. Obesity + T2D + HTN) was most strongly, amongst all the multimorbidity statuses, associated with tertiles 2 (OR=1.56) and 3 (OR=1.55) of the Western

dietary pattern and quintiles 2 (OR=1.34), 4 (OR=3.09), and 5 (OR=5.26) of wealth, it was not the most strongly associated with tertiles of the Prudent dietary pattern, participant's education, or paternal education.

According to Table 15 and **Figure 4**, being aged 70 years and older was associated with 1.49 times lower odds of being only obese but elevated odds of being diabetic (OR=2.43), hypertensive (OR=3.10), or both (OR=4.08). Compared to older adults with less than high school education, those with high school degrees or had at least some college education had lower odds of being diabetic, hypertensive, or both but not obese. This protective association tended to be stronger with higher educational attainment. Similarly, female sex (vs. male) and having 8+ years of paternal education (vs. <8 years) appeared to be more strongly associated with lower odds of T2D and HTN, either alone or in combination with the other conditions; black race (vs. white) appeared to be more strongly associated with elevated odds of T2D and HTN, either alone or in combination with the other conditions. Yet, none of these covariates appeared to be associated with obesity alone. Wealth was associated with lower odds of living with all multimorbidity statuses (including "obesity only"), and this protective association tended to be stronger in higher wealth quintiles. Except for "obesity and T2D," there was also a tendency that the protective association of wealth was stronger for greater number of comorbidities (e.g. all three health outcomes, two health outcomes). Interestingly, while tertile 2 (vs. tertile 1) of the Prudent dietary pattern appeared to be associated with elevated odds of all multimorbidity statuses, most of the ORs shifted towards protective association from tertile 2 to tertile 3 with the exception of "obesity + T2D" and "T2D + HTN." On the contrary, the Western dietary pattern appeared to be associated with elevated odds of all multimorbidity statuses, especially obesity,

alone or in combination of other health conditions, in both tertiles 2 and 3 in comparison to tertile 1. From tertile 2 to tertile 3, the ORs shifted towards higher odds for "HTN only" and "obesity + HTN," remained almost the same for "T2D + HTN" "obesity + T2D" and "obesity + T2D + HTN," and shifted towards protective association for "obesity only" and "T2D only." It is worth mentioning that the parameter estimates for the "T2D only" and "obesity and T2D" groups had wider confidence intervals, in general, due to small sample sizes.

Discussion

This study showed that more than 40% of older U.S. adults were living with multimorbidity of obesity, T2D, and HTN (e.g. coexistence of at least two conditions) in 2014. Among the eight possible multimorbidity statuses, "None" and "Obesity + T2D + HTN" were more prevalent than expected by chance alone. Childhood SES (primarily paternal education) and adulthood SES (education and wealth) were both independently associated with the multimorbidity statuses. Interestingly, the wealth component of SES appeared to be inversely associated with all eight multimorbidity statuses; whereas the education component of SES appeared to be inversely associated with seven multimorbidity statuses but not associated with "Obesity Only." Both the Prudent and Western dietary patterns appeared to be positively associated with the multimorbidity of obesity, T2D, and HTN, although no clear dose-response relationship was observed across the dietary pattern tertiles. Under our specific scales of measures for the exposure variables, wealth appeared to be the exposure that was the most strongly and robustly associated with the multimorbidity of obesity, T2D, and HTN. The strength of association with a covariate across the multimorbidity statuses varied by specific disease combinations and was not necessarily greater as the number of coexisting conditions went up.

Our results showed that obesity, T2D, and HTN are highly prevalent in older U.S. adults, not only individually, but also in the form of multimorbidity. According to a cross-sectional study among U.S. adults aged 51 to 61 years who participated in the 1992 HRS and those aged 70 years or older who participated in the 1993 AHEAD study, the prevalence of the multimorbidity of obesity, T2D, and HTN was less than 3.6% among those aged 51 to 61 years in 1992 and less than 6.7% among those aged 70 years and older in 1993 (88). In comparison to this report, our results indicate a more than two-fold increase in the prevalence of the multimorbidity of obesity, T2D, and HTN among older U.S. adults two decades later. Additionally, we observed that these three chronic conditions especially tend to be all present or none present in the same individual. This finding, together with prior findings that 1) obesity, T2D, and HTN share epidemiological features (e.g. disease of civilization, very gradual onset, familial) and 2) clustering of obesity, T2D, and HTN has consistently been observed across multiple countries, supports the existence of common underlying etiopathogenic factors of obesity, T2D, and HTN (12, 99). Proposed mechanisms in prior studies include insulin resistance, inflammation, mental stress, and others (97, 175-177).

The inverse associations of participant's education and wealth with the multimorbidity statuses of obesity, T2D, and HTN observed in this study are consistent with prior findings that multimorbidity, in general, is more prevalent among individuals at lower SES (81, 178, 179). According to a recent systematic review and meta-analysis, education was the most commonly examined adulthood SES indicator in prior studies in relation to multimorbidity in general, and low versus high education level was associated with 1.64 times higher odds of living with

multimorbidity in general in adults (95% CI: 1.41-1.91) (179). Wealth as an adulthood SES indicator, on the other hand, has seldom been examined in relation to multimorbidity. Yet, our results showed that it is more strongly and robustly associated with the prevalence of the multimorbidity of obesity, T2D, and HTN. Similarly, a recent examination of adults aged 50 years and older in the English Longitudinal Study of Aging showed that lower wealth, but not education, was a risk factor of developing multimorbidity in general (180). These indicate that financial and physical assets might be more relevant than knowledge-related assets when it comes to multimorbidity risk in older adults.

The inverse association between paternal education and the multimorbidity of obesity, T2D, and HTN observed in this study is consistent with prior findings based on HRS data that childhood SES is inversely associated with multimorbidity in general in older U.S. adults (181, 182). However, results varied across studies in terms of the role of adulthood SES in the association between childhood SES and multimorbidity. For example, in the longitudinal analysis by Pavela and Latham, childhood SES was no longer associated with multimorbidity in general after adjustment for adulthood SES (182); whereas in the present study, paternal education remained significantly associated with the multimorbidity of obesity, T2D, and HTN after adjustment for adulthood SES. Such discrepancy could be due to differences in the study design, definition of multimorbidity, as well as selection of SES indicators. Among all the childhood SES indicators (e.g. paternal education, maternal education, and childhood financial strain) examined in the present study, only paternal education remained highly significantly associated with the multimorbidity of obesity, T2D, and HTN with the mutual adjustment of childhood SES indicators and adjustment of adulthood SES (e.g. participant's education and wealth). This

indicates that paternal education is the potential driving factor for the association between childhood SES and the multimorbidity of obesity, T2D, and HTN that is independent of adulthood SES. Although the impact of paternal education on health in later adulthood in largely unknown, it has been speculated that as an indicator of childhood intellectual environment, paternal education might reflect more of father's cultural or social capital; whereas maternal education might reflect more of mother's parenting skills (134, 183, 184).

An interesting finding of the present study is that "Obesity Only" was associated (inversely) with wealth but not with the education component of SES; whereas other multimorbidity statuses of obesity, T2D, and HTN were associated (inversely) with both wealth and the education components of SES. This indicates that the underlying etiopathogenesis of SES in relation to "Obesity Only" might be different from that to the other multimorbidity statuses, including obesity with comorbid T2D and/or HTN. Obesity patients with and without comorbid T2D or HTN have been shown to have different biomarker profiles (185-187). However, there is very limited literature on biomarkers related to multimorbidity in general, not to mention the specific multimorbidity of obesity, T2D, and HTN (188). Alternatively, the observed associations could have been biased due to measurement error in height and weight that were used to determine obesity status. One source of measurement error was the pooling of measured height and weight data from different waves, since recent measures would more accurately reflect participant's true height and weight in 2013 than distant measures. Another source of measurement error was the use of self-reported height and weight data in place of measured data when the latter was unavailable. It has been reported that self-reported height and weight tend to lead to underestimated BMI, especially for older adults (189).

Although the associations of dietary patterns with the multimorbidity of obesity, T2D, and HTN have not been examined previously, their observed associations in the present study are not entirely consistent with what would be expected given prior findings on the associations of dietary patterns with obesity, T2D, or HTN individually. The Western dietary pattern has consistently been found to be associated with increased risk of obesity, T2D, and HTN individually in prior cohort studies among adults (16, 55-58). In the present study, while this dietary pattern is, consistent with prior findings, positively associated with the multimorbidity statuses, the strength of these associations does not appear to increase from tertile 2 to tertile 3. This is contradictive to what would be expected (e.g. a dose-response relationship) under the assumptions that 1) the Western dietary pattern is causally associated with each individual chronic condition and 2) the three chronic conditions share common causal pathways. In prior cohort studies, the prudent dietary pattern has consistently been found to be associated with reduced risk of obesity and T2D in adults (16, 55-57); in randomized controlled trials, a similar dietary pattern named the DASH diet has consistently been found to reduce both systolic and diastolic blood pressure in adults (62, 64). However, the present study showed mostly positive associations between the Prudent dietary pattern and the multimorbidity statuses of obesity, T2D, and HTN. Additionally, moving from tertile 2 to tertile 3, the strength of association become greater for "Obesity + T2D" and "T2D + HTN" but weaker for the other five multimorbidity statuses. Both phenomena mentioned above are contradictive to what would be expected under the assumption of causal protective effect of the Prudent dietary pattern. Not surprisingly, our Aim 2 based on the same dataset examining the associations of dietary patterns and obesity, T2D, and HTN individually also showed contradictive results compared to prior findings.

As discussed in detail in Aim 2, one possible explanation to the contradictive findings in the present study is reverse causation. Given the fact that a large percentage of participants with T2D and HTN (and presumably obesity) had the condition before the dietary measurement, it is possible that the dietary patterns of these participants were actually reflecting their modified (healthier) diet in response to the development of the chronic conditions, rather than their original habitual diet. Assuming the Western diet is a true risk factor and the Prudent diet is a true protective factor for the multimorbidity statuses examined in the present study, as would be consistent with prior findings, an intentionally modified healthier diet (e.g. reduced Western diet and increased Prudent diet) could have led to attenuated or even reversed associations for both dietary patterns. As for the present study, the true association might have been attenuated for the Western dietary pattern and reversed for the Prudent dietary pattern due to reverse causation, so that lack of dose-response relationship was observed for the former and an association in the opposite direction was observed for the latter.

Nonetheless, a prior cross-sectional study among U.S. adults aged 20 years and older indicated an inverse association between fiber intake and the multimorbidity of obesity, T2D, and HTN, which was not contradictive to prior findings with individual chronic conditions (100). A possible explanation for the discrepancy in findings between this prior study and the present study is that the former included adults younger than 50 years old but the latter focused only on older adults. Cross-sectional studies are subject to survival bias, especially when it is conducted among older adults and the disease under study is associated with mortality. As discussed in detail in Aim 2, older adults who were living with obesity, T2D, and HTN and therefore able to

be included in the present study might have had healthier dietary behavior compared to those who had the conditions but failed to survive. Similar to the impact of potential reverse causation, this potential survival bias could also have attenuated or reversed the true associations of dietary patterns with the multimorbidity of obesity, T2D, and HTN in the present study.

In prior studies, multimorbidity has commonly been measured by the total number of coexisting chronic conditions or its dichotomized version using 2 or 3 as the cut point (81, 178). This approach treats all chronic conditions as equals, meaning a higher number of coexisting conditions would represent a worse or more severe multimorbidity condition (81). However, this is not supported by findings of the present study. In fact, none of the covariates examined in the present study was found to be more strongly associated with the multimorbidity statuses exactly as the number of coexisting chronic conditions went up. Even for wealth and the Western dietary pattern that were observed to be more strongly associated with the coexistence of all three conditions, their associations with one or two coexisting conditions varied by specific multimorbidity status rather than following a simply pattern based on the number of coexisting conditions. Of course, it is also worth acknowledging that each point estimate observed in the present study was followed with an imperfect degree of certainty, as reflected in its 95% CI.

The main strength of the present study is the examination of specific multimorbidity statuses of obesity, T2D, and HTN. In comparison to simply using the number of coexisting conditions as the measure of mutimorbidity, our approach provides more details about how different combinations of even the same number of coexisting conditions vary in terms of prevalence in the population and relationships with the covariates. Different relationships of the multimorbidity

statuses with the same covariate also indicate the possible existence of different underlying etiopathogenic factors. Additionally, the use of nationally representative data allowed us to show, for the first time to our knowledge, the population prevalence of each specific multimorbidity status of obesity, T2D, and HTN in older U.S. adults.

The main limitation of the present study is its cross-sectional design, under which our results are subject to reverse causation and survival bias and causal inference cannot be made. Also, the pooling of information from multiple waves while determining participants' obesity status could potentially have introduced measurement error. However, this measurement error should not have affected the conclusions about obesity in the present study. Firstly, on average, adults' BMI does not change drastically over time and they mostly remain within the same weight category (e.g. overweight, obese) (190). Secondly, there is a tendency for older adults to lose weight and BMI over time (190). In this case, our use of obesity status data before 2014 would have misclassified some participants who were non-obese in 2014 as obese, but only non-differentially across levels of the covariates, thus biasing our findings about obesity towards no association.

In conclusion, the multimorbidity of obesity, T2D, and HTN is highly prevalent among older U.S. adults and a better understanding of the common etiopathogenic mechanisms among the three conditions is necessary. The prevalence of the multimorbidity statuses is associated independently with both childhood SES (primarily paternal education) and adulthood SES. Among the SES indicators examined, wealth appears to be playing the most important role and its underlying mechanism needs to be investigated in future studies. The associations of dietary

patterns with the multimorbidity of obesity, T2D, and HTN need to be further examined in prospective studies where reverse causation is unlikely. Finally, the present study demonstrated that the commonly used number of coexisting conditions is not an ideal measure of multimorbidity. Better measures need to be developed that further incorporate the differences across disease combinations.

Table 13. Sociodemographic characteristics and dietary patterns by multimorbidity of obesity, T2D, and HTN in older U.S. adults, 2014[^§]

	Overall	Multimorbidity Status								P-value*
		None	Obesity Only	T2D Only	HTN Only	Obesity + T2D	Obesity + HTN	T2D + HTN	Obesity + T2D + HTN	
Total	7076 (100)	1367 (22.89)	612 (9.84)	138 (1.75)	1733 (22.96)	141 (2.08)	1288 (17.62)	690 (8.41)	1107 (14.45)	
Age										<.0001
<70 years	4010 (69.19)	930 (79.13)	476 (86.05)	73 (62.96)	761 (55.86)	96 (79.80)	777 (73.29)	282 (51.25)	615 (67.84)	
70 years and older	3066 (30.81)	437 (20.87)	136 (13.95)	65 (37.04)	972 (44.14)	45 (20.20)	511 (26.71)	408 (48.75)	492 (32.16)	
Sex										0.1318
Male	2976 (45.99)	550 (43.18)	242 (44.54)	66 (49.51)	748 (46.87)	70 (55.80)	509 (44.83)	314 (48.81)	477 (48.01)	
Female	4100 (54.01)	817 (56.82)	370 (55.46)	72 (50.49)	985 (53.13)	71 (44.20)	779 (55.17)	376 (51.19)	630 (51.99)	
Race										<.0001
White	4882 (77.99)	1078 (86.00)	444 (83.73)	89 (68.89)	1268 (79.56)	88 (70.71)	865 (76.64)	404 (66.22)	646 (69.53)	
Black	1170 (10.13)	110 (4.27)	83 (6.50)	20 (11.97)	264 (9.93)	17 (6.31)	262 (12.61)	151 (16.82)	263 (15.59)	
Hispanic	802 (8.41)	122 (6.04)	70 (6.98)	21 (13.30)	150 (7.47)	30 (17.75)	137 (8.23)	107 (11.52)	165 (11.13)	
Other	222 (3.47)	57 (3.69)	15 (2.79)	8 (5.83)	51 (3.04)	6 (5.23)	24 (2.51)	28 (5.44)	33 (3.75)	
Financial strain score	0.77 (0.02)	0.60 (0.03)	0.74 (0.05)	0.95 (0.11)	0.81 (0.03)	0.91 (0.12)	0.83 (0.03)	0.88 (0.05)	0.82 (0.04)	<.0001
Paternal education										<.0001
Less than 8 years	1855 (21.82)	223 (12.83)	114 (14.23)	48 (36.35)	461 (23.19)	46 (28.02)	347 (23.11)	241 (32.27)	375 (28.71)	
8+ years	5221 (78.19)	1144 (87.17)	498 (85.77)	90 (63.65)	1272 (76.81)	95 (71.98)	941 (76.89)	449 (67.73)	732 (71.29)	
Maternal education										<.0001
Less than 8 years	1470 (16.93)	190 (10.62)	86 (11.78)	33 (19.87)	364 (18.78)	37 (21.12)	252 (15.60)	205 (25.81)	303 (23.03)	
8+ years	5606 (83.07)	1177 (89.38)	526 (88.22)	105 (80.13)	1369 (81.22)	104 (78.88)	1036 (84.4)	485 (74.19)	804 (76.97)	
Participant's education										<.0001
Less than high school	1262 (14.95)	133 (7.69)	66 (9.71)	31 (21.61)	329 (16.79)	29 (16.51)	230 (14.35)	184 (25.41)	260 (20.68)	
High school	2332 (31.86)	397 (27.60)	195 (30.16)	37 (28.05)	584 (34.31)	44 (27.88)	457 (33.47)	216 (29.84)	402 (36.15)	
Some college or above	3482 (53.19)	837 (64.71)	351 (60.13)	70 (50.35)	820 (48.90)	68 (55.61)	601 (52.18)	290 (44.75)	445 (43.17)	
Wealth quintile										<.0001
Q1 (lowest)	1496 (19.78)	184 (11.57)	136 (18.98)	27 (21.72)	318 (19.04)	29 (17.94)	293 (22.00)	179 (26.09)	330 (28.19)	
Q2	1415 (19.72)	182 (13.05)	133 (21.44)	27 (19.25)	327 (19.89)	36 (21.15)	317 (23.93)	145 (21.03)	248 (22.77)	
Q3	1451 (20.13)	276 (20.13)	127 (20.50)	29 (20.59)	321 (17.45)	29 (19.29)	262 (20.94)	149 (20.14)	258 (23.20)	
Q4	1376 (20.16)	323 (23.70)	116 (23.11)	27 (17.73)	386 (22.00)	21 (20.54)	218 (17.04)	120 (18.01)	165 (14.92)	
Q5 (highest)	1338 (20.21)	402 (31.55)	100 (15.97)	28 (20.71)	381 (21.62)	26 (21.07)	198 (16.09)	97 (14.73)	106 (10.92)	
Prudent dietary pattern										<.0001
T1 (lowest)	2302 (33.21)	437 (33.08)	193 (33.84)	48 (31.68)	660 (38.08)	35 (22.01)	405 (33.92)	206 (28.81)	318 (28.76)	
T2	2364 (33.18)	424 (29.63)	202 (32.03)	46 (36.27)	576 (34.18)	46 (35.54)	448 (32.89)	225 (33.92)	397 (37.22)	
T3 (highest)	2410 (33.60)	506 (37.29)	217 (34.14)	44 (32.05)	497 (27.73)	60 (42.45)	435 (33.19)	259 (37.27)	392 (34.02)	
Western dietary pattern										0.0003
T1 (lowest)	2500 (33.10)	546 (37.19)	196 (29.48)	48 (33.73)	680 (35.67)	48 (27.62)	404 (30.74)	262 (37.06)	316 (26.32)	
T2	2358 (33.31)	435 (32.65)	212 (35.45)	52 (35.77)	527 (31.09)	49 (36.07)	441 (32.50)	227 (32.71)	415 (37.10)	
T3 (highest)	2218 (33.58)	386 (30.16)	204 (35.07)	38 (30.50)	526 (33.24)	44 (36.32)	443 (36.76)	201 (30.23)	376 (36.58)	

[^]Dietary pattern data was from 2013 FFQ

[§]Mean (SE) for financial strain score; N (%) for total and all other covariates. All analyses accounted for the complex survey design. Observations used n=7,076; weighted population size n=84,019,396.

*P-values are calculated from Rao-Scott Chi-Square tests for all variables, except that for financial strain score it is from the F-test.

Table 14. Crude ORs for multimorbidity of obesity, T2D, and HTN by sociodemographic characteristics and dietary patterns in older U.S. adults, 2014[^§]

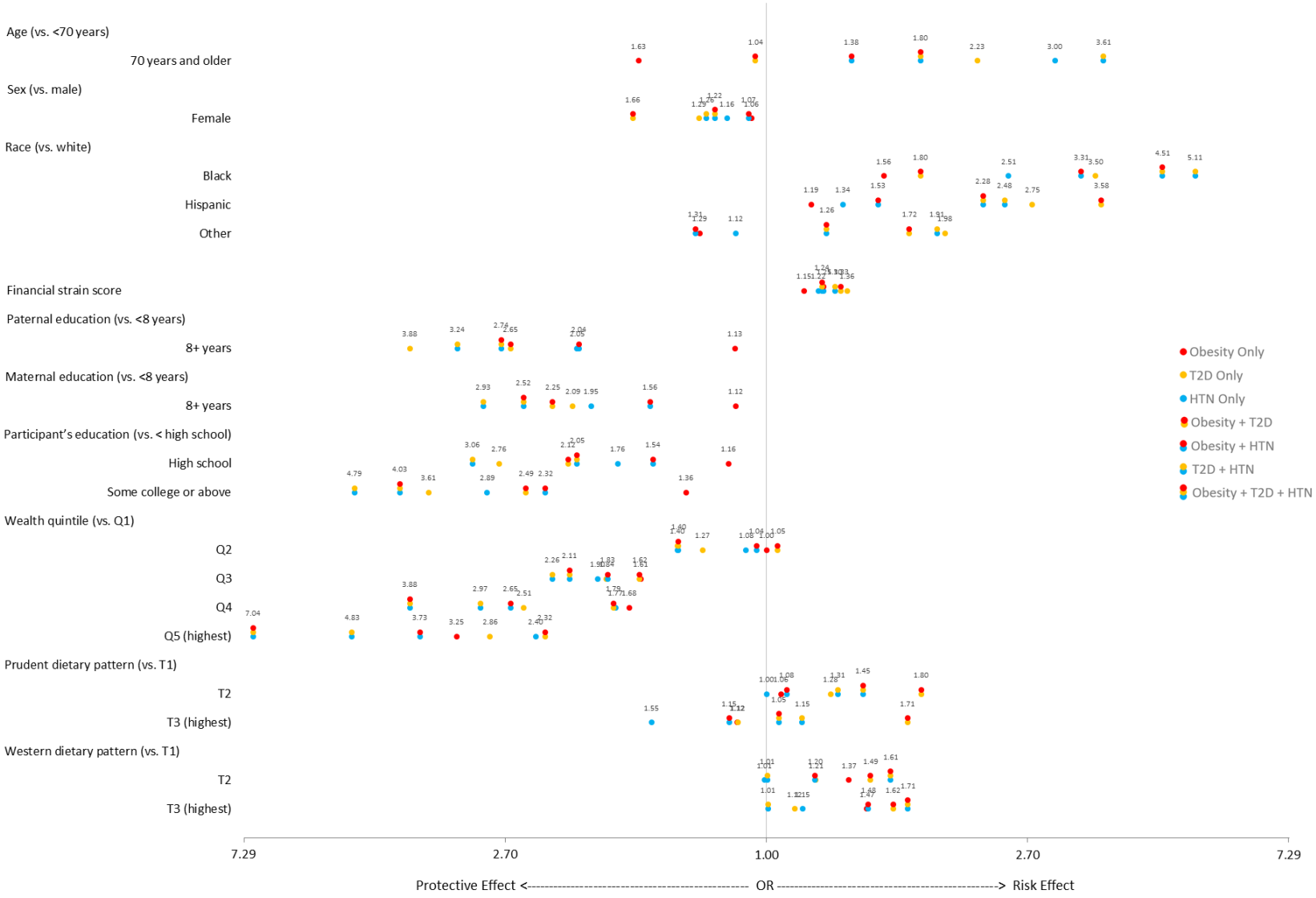
	Multimorbidity Status *(vs. None)*							P-value*
	Obesity Only	**T2D Only**	**HTN Only**	**Obesity + T2D**	**Obesity + HTN**	**T2D + HTN**	**Obesity + T2D + HTN**	
Age *(vs. <70 years)*								<.0001
70 years and older	0.62 (0.48, 0.79)	2.23 (1.41, 3.53)	3.00 (2.47, 3.63)	0.96 (0.59, 1.55)	1.38 (1.16, 1.65)	3.61 (2.79, 4.66)	1.80 (1.42, 2.28)	
Sex *(vs. male)*								0.0207
Female	0.95 (0.74, 1.21)	0.78 (0.51, 1.17)	0.86 (0.71, 1.04)	0.60 (0.42, 0.87)	0.94 (0.77, 1.13)	0.80 (0.64, 1.00)	0.82 (0.68, 1.00)	
Race *(vs. white)*								<.0001
Black	1.56 (1.05, 2.32)	3.50 (2.17, 5.63)	2.51 (1.97, 3.21)	1.80 (0.89, 3.63)	3.31 (2.54, 4.33)	5.11 (3.78, 6.92)	4.51 (3.58, 5.70)	
Hispanic	1.19 (0.77, 1.84)	2.75 (1.37, 5.51)	1.34 (0.98, 1.82)	3.58 (2.06, 6.21)	1.53 (1.13, 2.07)	2.48 (1.77, 3.48)	2.28 (1.64, 3.18)	
Other	0.78 (0.39, 1.55)	1.98 (0.73, 5.37)	0.89 (0.58, 1.36)	1.72 (0.64, 4.63)	0.76 (0.48, 1.22)	1.91 (0.89, 4.12)	1.26 (0.60, 2.63)	
Financial strain score	1.15 (1.05, 1.27)	1.36 (1.12, 1.65)	1.22 (1.13, 1.32)	1.33 (1.10, 1.61)	1.25 (1.15, 1.35)	1.30 (1.17, 1.45)	1.24 (1.12, 1.36)	<.0001
Paternal education *(vs. <8 years)*								<.0001
8+ years	0.89 (0.69, 1.14)	0.26 (0.17, 0.40)	0.49 (0.41, 0.59)	0.38 (0.23, 0.61)	0.49 (0.41, 0.59)	0.31 (0.24, 0.39)	0.37 (0.31, 0.44)	
Maternal education *(vs. <8 years)*								<.0001
8+ years	0.89 (0.65, 1.21)	0.48 (0.28, 0.81)	0.51 (0.40, 0.66)	0.44 (0.28, 0.71)	0.64 (0.50, 0.83)	0.34 (0.26, 0.45)	0.40 (0.31, 0.51)	
Participant's education *(vs. < high school)*								<.0001
High school	0.87 (0.61, 1.23)	0.36 (0.22, 0.60)	0.57 (0.43, 0.75)	0.47 (0.22, 1.00)	0.65 (0.48, 0.88)	0.33 (0.24, 0.45)	0.49 (0.38, 0.63)	
Some college or above	0.74 (0.51, 1.06)	0.28 (0.17, 0.45)	0.35 (0.27, 0.45)	0.40 (0.19, 0.84)	0.43 (0.33, 0.57)	0.21 (0.16, 0.28)	0.25 (0.18, 0.34)	
Wealth quintile *(vs. Q1)*								<.0001
Q2	1.00 (0.66, 1.51)	0.79 (0.38, 1.64)	0.93 (0.68, 1.26)	1.05 (0.52, 2.11)	0.96 (0.66, 1.41)	0.71 (0.51, 1.01)	0.72 (0.5, 1.02)	
Q3	0.62 (0.40, 0.97)	0.55 (0.28, 1.07)	0.53 (0.40, 0.70)	0.62 (0.29, 1.30)	0.55 (0.37, 0.80)	0.44 (0.31, 0.63)	0.47 (0.36, 0.63)	
Q4	0.59 (0.41, 0.87)	0.40 (0.21, 0.74)	0.56 (0.44, 0.73)	0.56 (0.26, 1.22)	0.38 (0.26, 0.56)	0.34 (0.23, 0.50)	0.26 (0.20, 0.33)	
Q5 (highest)	0.31 (0.21, 0.46)	0.35 (0.20, 0.60)	0.42 (0.32, 0.54)	0.43 (0.21, 0.88)	0.27 (0.19, 0.38)	0.21 (0.15, 0.29)	0.14 (0.10, 0.20)	
Prudent dietary pattern *(vs. T1)*								<.0001
T2	1.06 (0.80, 1.40)	1.28 (0.80, 2.04)	1.00 (0.80, 1.26)	1.80 (1.11, 2.93)	1.08 (0.89, 1.32)	1.31 (0.96, 1.81)	1.45 (1.13, 1.85)	
T3 (highest)	0.90 (0.70, 1.15)	0.90 (0.63, 1.27)	0.65 (0.54, 0.78)	1.71 (1.04, 2.82)	0.87 (0.70, 1.08)	1.15 (0.89, 1.49)	1.05 (0.82, 1.34)	
Western dietary pattern *(vs. T1)*								<.0001
T2	1.37 (1.03, 1.81)	1.21 (0.68, 2.16)	0.99 (0.80, 1.24)	1.49 (0.95, 2.33)	1.20 (0.98, 1.48)	1.01 (0.75, 1.35)	1.61 (1.26, 2.05)	
T3 (highest)	1.47 (1.08, 2.00)	1.12 (0.72, 1.74)	1.15 (0.92, 1.43)	1.62 (0.91, 2.89)	1.48 (1.18, 1.85)	1.01 (0.80, 1.27)	1.71 (1.33, 2.21)	

[^]Dietary pattern data was from 2013 FFQ

[§]All analyses accounted for the complex survey design. Observations used n=7,076; weighted population size n=84,019,396.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Figure 3. Crude ORs for multimorbidity of obesity, T2D, and HTN by sociodemographic characteristics and dietary patterns in older U.S. adults, 2014^§



^Dietary pattern data was from 2013 FFQ

§All analyses accounted for the complex survey design. Observations used n=7,076; weighted population size n=84,019,396.

Table 15. Multivariable-adjusted ORs for multimorbidity of obesity, T2D, and HTN by sociodemographic characteristics and dietary patterns in older U.S. adults, 2014[^§]

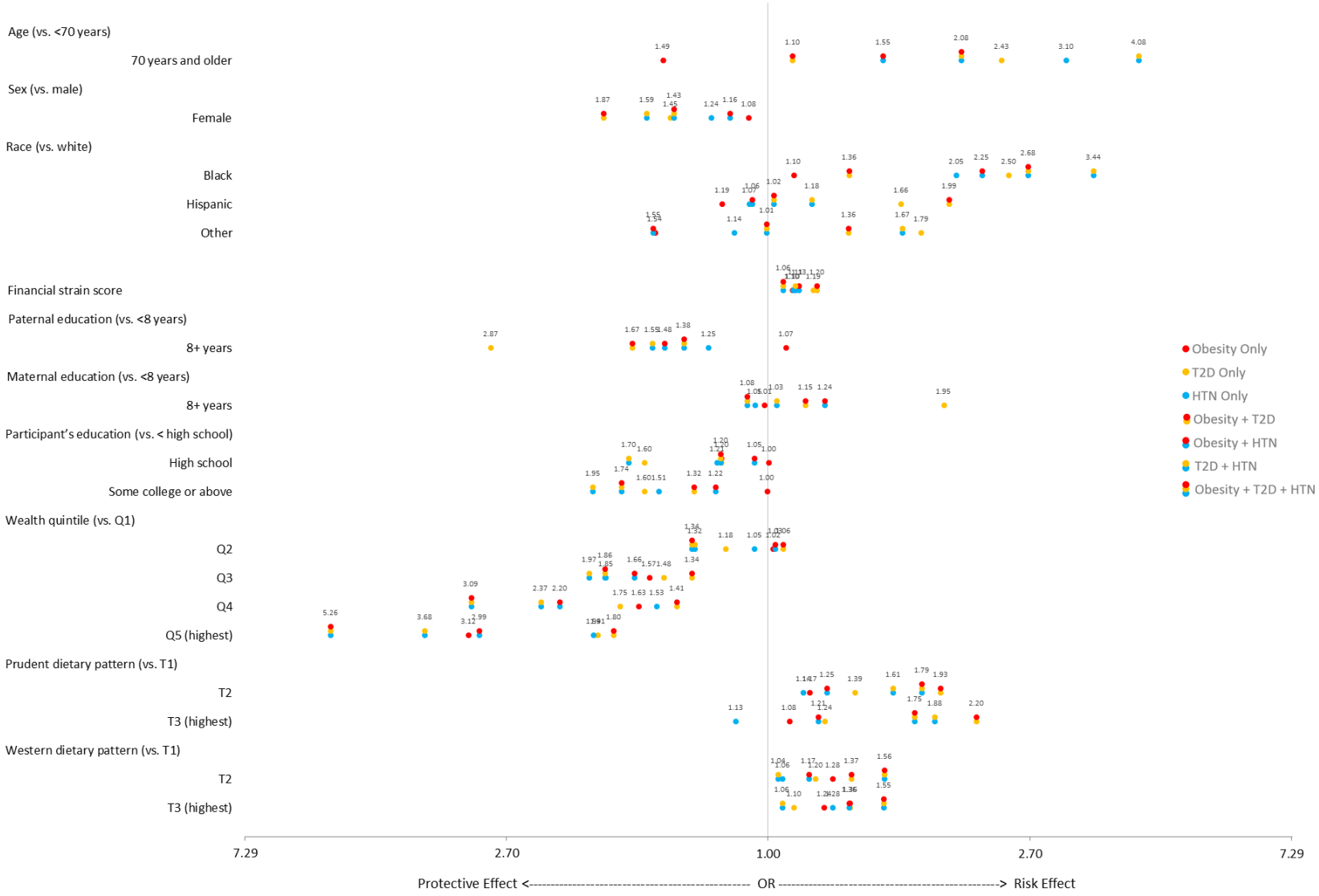
	Multimorbidity Status *(vs. None)*							P-value*
	Obesity Only	**T2D Only**	**HTN Only**	**Obesity + T2D**	**Obesity + HTN**	**T2D + HTN**	**Obesity + T2D + HTN**	
Age *(vs. <70 years)*								<.0001
70 years and older	0.67 (0.52, 0.87)	2.43 (1.47, 4.01)	3.10 (2.59, 3.72)	1.10 (0.66, 1.82)	1.55 (1.29, 1.86)	4.08 (3.20, 5.21)	2.08 (1.63, 2.67)	
Sex *(vs. male)*								<.0001
Female	0.93 (0.71, 1.22)	0.69 (0.45, 1.07)	0.81 (0.66, 0.99)	0.54 (0.38, 0.76)	0.87 (0.70, 1.07)	0.63 (0.49, 0.82)	0.70 (0.55, 0.89)	
Race *(vs. white)*								<.0001
Black	1.10 (0.77, 1.58)	2.50 (1.43, 4.36)	2.05 (1.58, 2.65)	1.36 (0.66, 2.81)	2.25 (1.68, 3.02)	3.44 (2.46, 4.83)	2.68 (2.05, 3.51)	
Hispanic	0.84 (0.48, 1.46)	1.66 (0.69, 3.99)	0.93 (0.65, 1.34)	1.99 (1.02, 3.89)	0.94 (0.63, 1.42)	1.18 (0.73, 1.90)	1.02 (0.64, 1.64)	
Other	0.65 (0.33, 1.29)	1.79 (0.66, 4.87)	0.88 (0.55, 1.40)	1.36 (0.51, 3.65)	0.65 (0.40, 1.04)	1.67 (0.71, 3.89)	0.99 (0.47, 2.1)	
Financial strain score	1.10 (0.99, 1.21)	1.19 (0.97, 1.45)	1.10 (1.02, 1.18)	1.20 (0.98, 1.48)	1.13 (1.04, 1.22)	1.11 (1.00, 1.23)	1.06 (0.97, 1.16)	0.0372
Paternal education *(vs. <8 years)*								<.0001
8+ years	1.07 (0.75, 1.53)	0.35 (0.20, 0.60)	0.80 (0.62, 1.03)	0.60 (0.33, 1.08)	0.68 (0.54, 0.84)	0.64 (0.49, 0.85)	0.73 (0.55, 0.96)	
Maternal education *(vs. <8 years)*								0.1688
8+ years	0.99 (0.65, 1.50)	1.95 (0.99, 3.82)	0.95 (0.68, 1.33)	1.15 (0.69, 1.93)	1.24 (0.91, 1.69)	1.03 (0.70, 1.53)	0.93 (0.66, 1.29)	
Participant's education *(vs. < high school)*								<.0001
High school	1.00 (0.66, 1.54)	0.63 (0.35, 1.11)	0.82 (0.61, 1.12)	0.84 (0.40, 1.74)	0.95 (0.68, 1.32)	0.59 (0.39, 0.89)	0.84 (0.62, 1.13)	
Some college or above	1.00 (0.58, 1.70)	0.63 (0.35, 1.11)	0.66 (0.49, 0.89)	0.76 (0.34, 1.69)	0.82 (0.61, 1.11)	0.51 (0.35, 0.75)	0.57 (0.40, 0.82)	
Wealth quintile *(vs. Q1)*								<.0001
Q2	1.02 (0.67, 1.56)	0.85 (0.40, 1.82)	0.95 (0.69, 1.30)	1.06 (0.52, 2.14)	1.03 (0.69, 1.53)	0.76 (0.55, 1.05)	0.75 (0.53, 1.07)	
Q3	0.64 (0.40, 1.03)	0.67 (0.34, 1.34)	0.54 (0.41, 0.70)	0.75 (0.35, 1.62)	0.60 (0.41, 0.89)	0.51 (0.36, 0.71)	0.54 (0.40, 0.73)	
Q4	0.61 (0.41, 0.92)	0.57 (0.30, 1.07)	0.66 (0.49, 0.87)	0.71 (0.30, 1.66)	0.45 (0.30, 0.69)	0.42 (0.28, 0.65)	0.32 (0.24, 0.43)	
Q5 (highest)	0.32 (0.21, 0.50)	0.52 (0.28, 0.97)	0.52 (0.39, 0.68)	0.56 (0.26, 1.21)	0.33 (0.23, 0.50)	0.27 (0.19, 0.39)	0.19 (0.14, 0.27)	
Prudent dietary pattern *(vs. T1)*								<.0001
T2	1.17 (0.87, 1.57)	1.39 (0.82, 2.36)	1.14 (0.92, 1.43)	1.93 (1.18, 3.14)	1.25 (1.01, 1.55)	1.61 (1.14, 2.27)	1.79 (1.37, 2.34)	
T3 (highest)	1.08 (0.83, 1.42)	1.24 (0.82, 1.87)	0.89 (0.74, 1.07)	2.20 (1.27, 3.82)	1.21 (0.98, 1.50)	1.88 (1.42, 2.50)	1.75 (1.32, 2.30)	
Western dietary pattern *(vs. T1)*								<.0001
T2	1.28 (0.96, 1.70)	1.20 (0.66, 2.19)	1.06 (0.86, 1.30)	1.37 (0.88, 2.15)	1.17 (0.94, 1.44)	1.04 (0.77, 1.41)	1.56 (1.20, 2.02)	
T3 (highest)	1.24 (0.90, 1.71)	1.10 (0.70, 1.74)	1.28 (1.00, 1.63)	1.36 (0.74, 2.51)	1.36 (1.05, 1.78)	1.06 (0.79, 1.40)	1.55 (1.15, 2.10)	

[^]Dietary pattern data was from 2013 FFQ

[§]All the covariates were mutually adjusted in the model. All analyses accounted for the complex survey design. Observations used n=7,076; weighted population size n=84,019,396.

*P-values are calculated from Type 3 Wald Chi-Square tests.

Figure 4. Multivariable-adjusted ORs for multimorbidity of obesity, T2D, and HTN by sociodemographic characteristics and dietary patterns in older U.S. adults, 2014[^§]



[^]Dietary pattern data was from 2013 FFQ
[§]All the covariates were mutually adjusted in the model. All analyses accounted for the complex survey design. Observations used n=7,076; weighted population size n=84,019,396.

Supplementary Table 3. Contrast between observed and expected prevalence (%) of multimorbidity statuses among obesity, T2D, and HTN

	None	Obesity Only	T2D Only	HTN Only	Obesity + T2D	Obesity + HTN	T2D + HTN	Obesity + T2D + HTN
Observed	22.89	9.84	1.75	22.96	2.08	17.62	8.41	14.45
Expected	15.01	11.79	5.47	26.05	4.29	20.46	9.48	7.45

CHAPTER V: Conclusions

Summary of findings

Using nationally representative data from HRS, this dissertation identified two main dietary patterns in older U.S. adults, namely the prudent and Western dietary patterns. The prudent dietary pattern is characterized by high intake of vegetables, tomatoes, garlic, legumes, fruit, olive oil, fish and other seafood, and salad dressing, as well as low intake of sweets and desserts and high-energy drinks; the Western dietary pattern is characterized by high intake of red meats, processed meats, condiments, French fries, and refined grains, as well as low intake of cold breakfast cereal, whole grains, low-fat dairy products, and fruit. These dietary patterns are consistent with those identified in prior studies among selected older U.S. adults or the entire U.S. adult population (17, 52).

Findings of the first aim of this dissertation support the notion that higher childhood SES (specifically paternal education) is associated with healthier dietary habits in adulthood, independent of adulthood SES. However, this association might only exist in selected sociodemographic groups. There could also be interaction between paternal education and participant's education in relation to the prudent dietary pattern.

Findings of the second aim, limited by its cross-sectional study design, agreed with prior studies on a positive association of the Western dietary pattern with obesity, T2D, and HTN; whereas an inverse association of the prudent dietary pattern with each of the health outcomes, as indicated

in prior studies, was not observed. SES, whether in childhood or in adulthood, did not appear to be a strong confounder to these associations.

Findings of the third aim highlight that obesity, T2D, and HTN are highly prevalent among older U.S. adults not only as individual health outcomes, but also, in the form of multimorbidity. While there are many possible combinations of the three health outcomes, the prevalence of having all three chronic conditions or having none of the three conditions was significantly higher than expected by chance alone, suggesting the existence of potential common underlying etiopathogenic factors. Under the limited cross-sectional study design, both childhood SES (primarily paternal education) and adulthood SES (education and wealth, especially the latter) are independently, inversely associated with variation in individual's multimorbidity status; both the prudent and Western dietary patterns appeared to be positively associated with multimorbidity status but without clear dose-response relationship. Additionally, findings of this aim demonstrated the importance of identifying predictors of specific disease combinations rather than only focusing on the number of coexisting conditions as has been primarily done in prior multimorbidity studies.

Public health implications

CVD is highly prevalent among older U.S. adults, and better understanding of its risk factors and their interrelationship is needed to address its increasing burden under population aging (4, 5, 191). Extending from the traditional adult lifestyle model, this dissertation took a life course approach and further accounted for the impact of childhood SES, an important indicator of childhood environment and conditions, on dietary behavior and proximal CVD risk factors (e.g.

obesity, T2D, and HTN) in older adulthood (134, 183, 184). The understanding of early-life contributors, jointly with later-life contributors, to disease risk enables us to identify, based on early-life experiences, high-risk groups to be targeted in later-life interventions (e.g. changing behaviors); also, it opens the door to early-life and more effective interventions.

Early studies on dietary behavior as a risk factor for obesity, T2D, or HTN typically focused only on the consumption of a single or a group of nutrients or foods. While such approaches could shed light on underlying mechanisms, their assessment of nutrients' independent effects is highly challenged due to the complicated interactions and intercorrelations among nutrients (52). Dietary patterns, as the measure of dietary behavior used in this dissertation, on the other hand, take into consideration the combination of nutrients and foods being consumed and examine the overall dietary behavior. Not only is dietary pattern a comprehensive target for dietary behavior change, but also, dietary recommendations in terms of dietary patterns are easily interpretable to the public (52). Given that dietary behavior is culture specific and each society has its unique social context, the examinations in this dissertation provide particular insights on adulthood dietary patterns and their relationships with childhood SES and obesity, T2D, or HTN in older adults across the U.S.

While the public health impact of multimorbidity in general among older adults has been increasingly recognized in recent years, not much has been studied specifically about the multimorbidity of obesity, T2D, and HTN (81). The detailed prevalence estimates for the multimorbidity statuses of obesity, T2D, and HTN in this dissertation provides key information about their burden in older U.S. adults nowadays, especially under the recent trends in the

prevalence of individual health outcomes (192-194). For example, the prevalence of the multimorbidity of obesity, T2D, and HTN (e.g. obesity + T2D + HTN) between 1992-1993 among older U.S. adults was estimated to be under 7% (88); whereas, according to this dissertation, its prevalence in 2014 was higher than 14%, indicating a more than two-fold increase from two decades ago.

Given that obesity, T2D, and HTN also share many epidemiological features (e.g. disease of civilization, older age of onset, familial, etc.), the existence of common underlying etiopathogenic factors is likely (99). Targeting common risk factors, if there is any, can greatly enhance the comprehensiveness and efficiency of prevention strategies for obesity, T2D, and HTN, altogether. Yet, not much is known about what the risk factors are for the multimorbidity of obesity, T2D, and HTN or their corresponding underlying mechanisms. By exploring childhood SES, adulthood SES, and adulthood dietary patterns as potential risk factors, this dissertation contributes to the filling of this gap of knowledge.

Strengths and limitations

A main strength of this dissertation is that it was based on a large, nationally representative probability-based sample of older U.S. adults, whose dietary intake information was collected in detail. By accounting for the complex survey design, not only did this dissertation identify dietary patterns that represent the overall dietary behavior of the entire older U.S. adult population; but also, it estimated the prevalence of obesity, T2D, and HTN, each as an individual health outcome or in the form of multimorbidity, in the entire older U.S. adult population. Another main strength of this dissertation is the use of a life course approach, which

comprehensively examined the interrelationships among childhood SES, adulthood dietary patterns, and the health outcomes (individually or in combinations). In the dietary pattern analysis using PCA, multiple sensitivity analyses were performed to assess the robustness of results to a series of decisions, ranging from number of PCs to retain to food grouping strategy. In the observation of counterintuitive associations of the Prudent dietary pattern and the health outcomes, a large series of additional analyses (details not included in this report) were performed to confirm the validity of study results and test the potential of alternative explanations. Moreover, in the examination of the multimorbidity of obesity, T2D, and HTN, this dissertation allowed for each disease combination as a distinct outcome category, which provides details about how different combinations of coexisting conditions vary in terms of prevalence in the population and relationships with potential risk factors.

A main limitation of this dissertation is its cross-sectional study design. While the temporality was clear for childhood SES relative to adulthood dietary patterns and the health outcomes, it was unclear for dietary patterns relative to the diagnoses of the health outcomes. Consequently, results on the associations of dietary patterns with obesity, T2D, and HTN, whether individually or in combinations, were subject to the influence of reverse causation and survival bias. There are also a few other limitations of this dissertation. For example, detailed dietary intake data of the participants was collected only once via FFQ, which might not have captured very well their long-term intake in comparison to repeated FFQs over the years. Although one FFQ is sufficient for ranking participants in terms of dietary intake, it is prone to measurement error that can bias towards the null the associations of dietary patterns with childhood SES and the health outcomes. A limited number of childhood SES and adulthood SES indicators have been examined in this

dissertation, which might not have captured the entire SES construct. Both dietary intake and childhood SES information was recalled, which is subject to measurement error. The use of multiple sources of data in determining participants' obesity statuses could potentially have misclassified some non-obese participants as obese, but only non-differentially across levels of the covariates, thus biasing findings about obesity towards no association. Lastly, the examination of multiple exposure and/or outcome variables in each aim might be subject to the multiple testing issue, although Bonferroni correction did not change the main conclusions of this dissertation.

Future directions

Although causal inferences cannot be made from this dissertation, the complex interrelationship among childhood SES, adulthood SES, adulthood dietary patterns, and the health outcomes of obesity, T2D, and HTN observed has generated important ideas and hypotheses to be examined in future studies.

First, a valuable, immediate next step would be to use alternative measures of dietary pattern to re-examine the associations of dietary pattern with obesity, T2D, and HTN, separately and in the form of multimorbidities. Good candidate measures include the Mediterranean-Diet Score and the Healthy Eating Index, both of which are index-based dietary patterns. The former, as described previously, is an index indicating the degree of adherence to the traditional Mediterranean diet, which is known to be healthy (46). The latter uses a total score to measure the overall diet quality in comparison to the Dietary Guidelines for Americans (195). Both indices account for not only healthy but also unhealthy diet components, which is helpful for

understanding the overall diet healthiness of participants with high Western diet intake who are also adopting healthier dietary habits to manage their CVD risk.

Given the suspected dietary change in older U.S. adults to manage their CVD risk, it would also be interesting to examine dietary patterns in older adults after developing obesity, T2D, or HTN, as well as associated factors. For example, taking advantage of the longitudinal nature of HRS design with repeated measures of health outcome statuses in each wave, it would be valuable to investigate how the diagnosis of a chronic condition, in combination with SES and other factors, affects the adoption of different dietary patterns in older adults.

In the long term, it would be important to replicate findings from this dissertation in prospective studies, without the influence of reverse causation or survival bias. Considering the dietary intake information in HRS was collected fairly recently (in 2013), it would be more practical to search for other data sources where data on health outcomes have been collected years or even decades after the initial collection of dietary intake information (e.g. Framingham Heart Study). A prospective study would also be a great opportunity to formally test for mediation by dietary patterns in the association between childhood SES and adulthood obesity, T2D, and HTN.

One area where further studies are especially needed is the impact of childhood SES and adulthood SES on obesity, T2D, and HTN, separately or in the form of multimorbidity. Studies are needed not only on potentially a wider range of SES indicators in comparison to what was done in this dissertation, but also, to acquire a deeper understanding of what each SES indicator represents and how it affects the health outcomes. For childhood SES, it would be especially

interesting to further assess the unique influence of paternal education on individuals' later life CVD risk. This should be done across multiple birth cohorts, in order to reveal whether the importance of paternal education to later life CVD risk is universal or unique to the experiences of certain birth cohorts. For adulthood SES, it would be interesting to further evaluate the relative contributions of education and wealth to later life CVD risk and the underlying mechanisms. Additionally, further examination is needed to better understand the complex interaction between childhood SES and adulthood SES in relation to CVD risk, and how it varies in different cultural and social contexts.

Another area where further studies are especially needed is the exploration of risk factors of the multimorbidities of obesity, T2D, and HTN. Examples of important research questions include: Is dietary behavior a mediator for the association between childhood SES and multimorbidities of obesity, T2D, and HTN? What are the distinctions between different disease combinations (e.g. "Obesity Only vs. Obesity + T2D vs. Obesity + T2D + HTN")? More broadly, it is crucial for future multimorbidity studies to take into account the differences across disease combinations, rather than only focusing on the number of coexisting conditions.

In conclusion, this dissertation provides further evidence that childhood environment and conditions may underlie adulthood dietary habits and multimorbidities of obesity, T2D, and HTN. The unexpected positive association of the prudent, in addition to the Western, dietary pattern with the prevalent health outcomes observed may indicate that older U.S. adults are adopting healthier dietary habits to prevent or manage CVD but unable to fully follow the recommended healthy diets. Much more work needs to be done to understand the complex

interrelationship among childhood SES, adulthood SES, and adulthood dietary patterns, as well as its impact on CVD risk later in life.

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