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Race/Ethnicity Moderates Associations between Depressive Symptoms and Diet Composition among U.S. Adults

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Abstract

Objective: Although depression is associated with poorer overall diet quality, few studies have examined its association with levels of particular macronutrients, and none has examined moderation by race/ethnicity. The present study examined (1) associations between depressive symptom severity and nine indices of diet composition and (2) whether race/ethnicity moderates these associations.

Methods: Participants were 28,940 adults (mean age=49 years, 52% female, 52% non-white) from NHANES 2005–2018. Depressive symptom severity was measured using the Patient Health Questionnaire-9 (PHQ-9). Nine diet composition indices were derived from the average of two 24-hour dietary recalls (e.g., total energy, total fat, saturated fat, total carbohydrate, sugar, fiber, and protein).

Results: Separate linear regression analyses revealed that PHQ-9 total was positively associated with saturated fat and sugar and negatively associated with protein and fiber. Moderation by race/ethnicity was observed (interaction *p*s<.05). Among non-Hispanic Whites, PHQ-9 total was positively associated with sugar and negatively associated with protein and fiber. Among non-Hispanic Blacks, PHQ-9 total was positively associated with total energy, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, total carbohydrate, and sugar. Among Mexican Americans, PHQ-9 was positively associated with saturated fat. Among other Hispanics,

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PHQ-9 total was negatively associated with fiber, protein, and total, monounsaturated, and polyunsaturated fat.

Conclusions: Findings from this large, nationally representative sample demonstrate that associations between depressive symptom severity and diet composition vary by race/ethnicity. Critically, an unhealthy diet composition pattern may be one mechanism explaining the excess risk of obesity and cardiometabolic diseases in individuals with depression, especially in non-Hispanic Blacks.

Keywords

depression; population groups; health status disparities; diet; food; nutrition

Introduction

Obesity is a significant public health concern affecting 42.4% of U.S. adults (Hales, Carroll, Fryar, & Ogden, 2020). Numerous factors have been identified as potential contributors to the development of obesity, including biological, behavioral, psychological, and environmental factors (Pi-Sunyer, 2002; Straub, 2014). Among the potential psychological contributors, meta-analytic findings demonstrate that people with elevated depressive symptoms have a 58% greater risk of developing obesity than people without elevated depressive symptoms (Luppino et al., 2010). Several candidate mechanisms underlying the depression-to-obesity relationship have been proposed, including biological (e.g., hypothalamic–pituitary–adrenal axis dysregulation, inflammation, and neurotransmitter dysregulation) and psychosocial mechanisms (e.g., diet, physical activity, alcohol use, and medication use) (Hawkins et al., 2015; Luppino et al., 2010; Markowitz, Friedman, & Arent, 2008). The focus of the present study is on one of the psychosocial candidate mechanisms – diet.

A growing body of research examines associations between depression and diet. Dietary patterns of Mediterranean and healthy diets – defined as diets with high intake of fruits and vegetables, fish, and whole grains – are associated with lower depression (Lai et al., 2014; Psaltopoulou et al., 2013; Quirk et al., 2013; Rahe, Unrath, & Berger, 2014), whereas Western diet is unrelated to depression (Lai et al., 2014; Psaltopoulou et al., 2013; Quirk et al., 2013; Rahe et al., 2014). Findings from cross-sectional studies of depression and diet composition are mixed. The most consistent evidence indicates that there is no cross-sectional association between depression and protein intake (Davison & Kaplan, 2012; Nanri et al., 2010; Oishi, Doi, & Kawakami, 2009; Woo et al., 2006). Evidence for fat, carbohydrate, and energy intake is limited and mixed (Anton & Miller, 2005; Dressler & Smith, 2015; Fulkerson, Sherwood, Perry, Neumark-Sztainer, & Story, 2004; Hyakutake et al., 2016; Mooreville et al., 2014; Nanri et al., 2014; Oishi et al., 2009; Simon et al., 2008; Umegaki et al., 2009; Whitaker, Sharpe, Wilcox, & Hutto, 2014; Woo et al., 2006). Several methodological factors may contribute to these mixed findings, including the use of small or heterogeneous samples and the use of varied approaches for measuring diet and depression. Moreover, few studies have examined the association of depression with both total levels (e.g., total carbohydrates) and subordinating levels (e.g., fiber and sugar) of nutrients. Depression may have different relationships with subordinating nutrients that

vary in nutritional quality. To illustrate, Whitaker et al. (2014) report that depression was not associated with total fat intake but was inversely associated with saturated fat intake.

To our knowledge, no study has examined the associations between depressive symptom severity and multiple indices of diet composition in a large, nationally representative sample of U.S. adults, and few studies have examined both total and subordinating levels of nutrients within the same study. Moreover, no investigation has examined moderation of these associations by race/ethnicity, which is critical because: obesity is more prevalent among non-Hispanic Black adults (49.6%) and Hispanic adults (44.8%) than non-Hispanic White adults (42.2%) in the U.S (Hales et al., 2020); there are racial/ethnic disparities in depression treatment (Simpson, Krishnan, Kunik, & Ruiz, 2007); and there are geographical differences in food availability between neighborhoods of differing racial/ethnic composition, likely affecting diet composition (Block, Scribner, & DeSalvo, 2004; Larson, Story, & Nelson, 2009; Powell, Slater, Mirtcheva, Bao, & Chaloupka, 2007). Accordingly, the objectives of the present study were (1) to examine the cross-sectional associations between depressive symptom severity and multiple indices of diet composition (i.e., total energy intake and nutrient intake) and (2) to test race/ethnicity as a moderator of the depressive symptom severity-diet composition associations in a large, representative sample of U.S. adults. Given the limited and mixed literature on the relationships between depression and diet composition factors, our analyses were largely exploratory.

Participants and Methods

Study Design and Sample

This study examined cross-sectional data from the 2005–2018 years of NHANES, a survey conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention examining the health and nutritional status of the U.S. population. Detailed descriptions of the survey design and procedures are available on the NHANES website (www.cdc.gov/nchs/nhanes.htm). Briefly, individuals were recruited from a nationally representative sample of the U.S. civilian, non-institutionalized population. Participants were asked to complete a household interview to assess demographic and health-related factors. Approximately 1–2 weeks later, participants were asked to visit a Mobile Examination Center (MEC) to undergo physical examinations, laboratory assessments, and additional interviews. Written informed consent was obtained from all study participants prior to the household interview and the MEC examination. The institutional review board at Indiana University-Purdue University Indianapolis (IUPUI) approved this archival study.

From the total sample of 70,190 participants, all participants 18 years and older were selected (n=42,143). To minimize the influence of potential confounders, the following participants were excluded: (1) those who were pregnant at the time of assessment due to the influence of pregnancy on depression and diet (n=737) (Vesga-Lopez et al., 2008) and (2) those who did not complete two days of the dietary assessment (n=9,253). Next, we excluded participants in the “other race including multi-racial” race/ethnicity category (n=1,241) because this group was small and was comprised of individuals with heterogeneous racial/ethnic backgrounds, which would make interpretation difficult. Finally, we excluded participants in the non-Hispanic Asian group (n=1,972), as this race/ethnicity

category was not assessed before the 2011–2012 survey cycle. Our final sample was comprised of 28,940 adults.

Measures

Depressive Symptom Severity.—The Patient Health Questionnaire-9 (PHQ-9) (Kroenke, Spitzer, & Williams, 2001) was administered by NHANES personnel during the MEC visit. Participants indicated the frequency with which they experienced each of nine symptoms of major depressive disorder over the past two weeks. Total scores range from 0 to 27; scores ≥10 represent clinically significant depressive symptoms (Kroenke & Spitzer, 2002). The PHQ-9 has high internal consistency and good sensitivity and specificity for identifying major depressive disorder in community samples (Manea, Gilbody, & McMillan, 2012; Patten & Schopflocher, 2009; Van der Kooy et al., 2007; Wittkampf, Naeije, Schene, Huyser, & van Weert, 2007).

Diet Composition.—Diet composition was assessed via two 24-hour dietary recalls using the interviewer-administered automated multiple-pass method (AMPM). The first assessment was conducted at the MEC, and the second was conducted by telephone 3–10 days later. The AMPM algorithm has been established as a valid measure of usual energy intake (Moshfegh et al., 2008). Data from the two dietary recalls were averaged to create estimates for total energy, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, total carbohydrate, sugar, fiber, and protein.

Other Factors.—Demographic information was collected during the household interview. Statistical models included the following demographic covariates: age (years), sex (0=male, 1=female), three dummy variables for race/ethnicity, education, and poverty-income ratio (PIR). NHANES personnel classified participates into five race/ethnicity categories (non-Hispanic White, non-Hispanic Black, Mexican American, other Hispanic, and other race including multi-racial) for the 2005–2010 years and six categories (non-Hispanic Asian was added) for the 2011–2018 years. Participants in the “other race including multi-racial” category were excluded due to the heterogeneity of this group, and participants in the non-Hispanic Asian category were excluded due to lack of consistent assessment over the survey years (see Study Design and Sample section).Thus, the race/ethnicity variable had four categories, which was used to compute three dummy variables comparing the non-Hispanic Black (RE1), Mexican American (RE2), and other Hispanic (RE3) groups to the non-Hispanic White reference group. Participants reported their highest level of education, from which a five-level categorical variable was created: less than 9th grade, $9–11^{th}$ grade with no diploma, high school diploma or GED, some college or associate degree, and college graduate or above. Participants reported their total annual family or individual income. From these data, a PIR variable (range: 0 to 5) was calculated by dividing annual income by the poverty level established by the Federal Register, specific to family size, survey year, and state. PIR values below 1.0 indicate living below the poverty level; PIR values above 1.0 indicate living above the poverty level. Body mass index (BMI; kg/m^2) was calculated using height/weight measured at the MEC and was modeled as a continuous variable. BMI values are classified into the following categories: < 18.5 kg/m^2 is considered underweight, 18.5–24.9 kg/m^2 is considered normal weight, 25.0–29.9 kg/m^2 is

considered overweight, and ≥ 30.0 kg/m^2 is considered obesity (Centers for Disease Control and Prevention, 2020). Cardiovascular disease (CVD), diabetes, kidney disease, liver disease were assessed during the household interview via a self-report item asking, "Has a doctor or other health professional ever told you that you had [medical condition]?" from which a dichotomous variable was created for each condition (0=no, 1=yes). A "yes" response to the diabetes item included individuals with type 1 or type 2 diabetes. Hypertension was assessed with the aforementioned item and a follow-up item asking, "Were you told on 2 or more different visits that you had hypertension, also called blood pressure?" Participants who responded "yes" to both items were coded as having reported hypertension (0=no, 1=yes).

Data Analysis

Multiple imputation was used to handle missing data for PHQ-9 total (n=1,638), education (n=57), PIR (n=2,321), and BMI (n=350). SAS 9.4 was used to conduct the PROC MI Fully Conditional Specification method to obtain 20 imputed datasets with 200 burn-in iterations (Berglund, 2015). Variables that significantly predicted missingness of the incomplete variables were included in the imputation model. To account for stratum and cluster effects, the imputation model included dummy variables for stratum and cluster (Reiter, Raghunathan, & Kinney, 2006). After imputation, a series of logistic regression models were conducted within each of the imputed datasets using SAS PROC SURVEYREG. Finally, estimates for the 20 imputed datasets were combined using SAS PROC MIANALYZE (Berglund, 2015).

To determine the relationship between depressive symptom severity and diet composition, nine separate linear regression analyses were conducted with PHQ-9 total (z-scored) predicting each diet composition variable (z-scored). Models were adjusted for age, sex, race/ethnicity, PIR, education, BMI, CVD, diabetes, kidney disease, liver disease, hypertension, and NHANES sampling design.

To test whether race/ethnicity moderated the depressive symptom severity-diet composition relationship, three cross-product interaction terms were computed by multiplying PHQ-9 total (z-scored) by each race/ethnicity dummy variable. The interaction terms were entered into separate linear regression models with PHQ-9 total (z-scored) predicting each diet composition variable (z-scored) in fully adjusted models. Given the presence of significant interactions, the linear regression models were reanalyzed after stratifying by race/ethnicity.

Estimates from analytic models were weighted using the appropriate NHANES sampling design variables – strata, primary sampling unit, and dietary two-day sample weights. Applying NHANES sampling design variables accounts for survey design factors and provides estimates representative of the U.S. civilian, non-institutionalized population.

Results

Participant Characteristics

Table 1 shows participant characteristics. The mean age of the sample was 49 years. Fifty two percent were women, 52% were non-white, 25% reported an education level below a high school diploma or GED, and mean PIR was 2.5. Mean BMI fell in the overweight

range. Regarding comorbid medical conditions, 11% reported CVD, 13% reported diabetes, 3% reported kidney disease, 4% reported liver disease, and 30% reported hypertension. Table 2 displays descriptive statistics for the depressive symptoms and diet composition variables. PHQ-9 total fell in the minimal depression range. Mean energy intake was 2,036 kilocalories, which is consistent with USDA Dietary Guidelines recommendation of 1,600–3,000 calories per day depending on sex and body size (U.S. Department of Agriculture, 2015). Participant characteristics are reported by race/ethnicity. Tests for differences by race/ethnicity demonstrate differences for all characteristics.

Depressive Symptom Severity and Diet Composition Associations

As is shown in Figure 1, separate linear regression models revealed that PHQ-9 total was positively associated with saturated fat (p=.021) and sugar intake (p<.001) and negatively associated with fiber (p<.001) and protein intake (p<.001). A two-standard deviation (2-*SD*) increase in PHQ-9 total (8.4 points on a 0–27 scale) was associated with consuming 0.5 more grams of saturated fat, 5.8 more grams of sugar, 1.0 fewer grams of fiber, and 2.3 fewer grams of protein per day. PHQ-9 total was unrelated to total energy, total fat, monounsaturated fat, polyunsaturated fat, and total carbohydrate intake (all ps≥.098).

Race/Ethnicity as a Moderator of the Depressive Symptom Severity-Diet Composition Associations

To examine race/ethnicity as a moderator of the associations between depressive symptom severity and diet composition factors, interaction terms were tested for PHQ-9 total (z-scored) by the race/ethnicity dummy variables. See Table 3 for a full reporting of interaction analyses. Notably, the PHQ-9 totalxRE1 (non-Hispanic Black vs. non-Hispanic White) interaction term was significant for all diet composition factors. Given the presence of significant interaction terms, the primary analyses were re-conducted after stratifying by race/ethnicity.

As shown in Figure 2, among non-Hispanic Whites, PHQ-9 total was positively associated with sugar intake (p<.001) and negatively associated with fiber (p<.001) and protein intake (p<.001). A 2-*SD* increase in PHQ-9 total (8.4 points) was associated with consuming 5.3 more grams of sugar, 1.1 fewer grams of fiber, and 3.0 fewer grams of protein per day. PHQ-9 total was not associated with the remaining six diet composition factors among non-Hispanic Whites (ps≥.321).

Among non-Hispanic Blacks, a remarkably different pattern of associations was found. PHQ-9 total was positively associated with total energy (p<.001), total fat (p=.003), saturated fat (p=.002), monounsaturated fat (p=.005), polyunsaturated fat (p=.009), total carbohydrate (p<.001), and sugar intake (p<.001). A 2-*SD* increase in PHQ-9 total (8.6 points) was associated with consuming 116.0 more kilocalories, 4.5 more grams of total fat, 1.5 more grams of saturated fat, 1.6 more grams of monounsaturated fat, 1.0 more grams of polyunsaturated fat, 13.6 more grams of carbohydrates, and 8.9 more grams of sugar per day. PHQ-9 total was not associated with fiber (p=.251) or protein intake (p=.061) among non-Hispanic Blacks.

Among Mexican Americans, PHQ-9 total was positively associated with saturated fat intake only (p=.030; all other ps≥.105). A 2-*SD* increase in PHQ-9 total (8.4 points) was associated with consumption of 1.0 more grams of saturated fat per day. Among other Hispanics, PHQ-9 total was negatively associated with total fat (p=.021), monounsaturated fat (p=.014), polyunsaturated fat (p=.028), fiber (p<.001), and protein intake (p=.040) and was unrelated to the remaining four diet composition factors (ps≥.130). A 2-*SD* increase in PHQ-9 total (9.8 points) was associated with consumption of 2.5 fewer grams of total fat, 1.0 fewer grams of monounsaturated fat, 1.2 fewer grams of polyunsaturated fat, 1.7 fewer grams of fiber, and 1.1 fewer grams of protein per day.

Discussion

In a large, diverse sample of adults representative of the U.S. population, greater depressive symptom severity was associated with increased intake of diet components to limit or consume in moderation (e.g., saturated fat and sugar) and reduced intake of diet components to consume abundantly (e.g., fiber and protein), based on guidance from the Dietary Guidelines for Americans (U.S. Department of Agriculture, 2015). Analyses stratified by race/ethnicity revealed that, particularly among non-Hispanic Blacks, greater depressive symptom severity was associated with increased intake of diet components that represent an unhealthy pattern of eating. Among non-Hispanic Blacks, a clinically significant increase in depressive symptom severity (i.e., a 2-*SD* increase in PHQ-9) was associated with consuming 116.0 more kilocalories, 4.5 more grams of total fat, 1.5 more grams of saturated fat, 1.6 more grams of monounsaturated fat, 1.0 more grams of polyunsaturated fat, 13.6 more grams of carbohydrates, and 8.9 more grams of sugar per day. To illustrate, this increased energy intake translates to a weight increase of over 12 pounds per year among non-Hispanic Blacks with elevated depressive symptom severity (Guth, 2014). In this group, depressive symptom severity was unrelated to fiber and protein intake, which is inconsistent with findings for the entire sample and may reflect an overall pattern of greater depressive symptoms being related increased intake across all nutrient types in non-Hispanic Blacks relative to non-Hispanic Whites. The pattern of results among non-Hispanic Whites was similar to that of the entire sample – greater depressive symptom severity was associated with increased intake of sugar and reduced intake of fiber and protein. Among Mexican Americans, greater depressive symptom severity was associated with increased saturated fat intake only. Among other Hispanics, greater depressive symptom severity was associated with reduced intake of several diet components (i.e., total fat, monounsaturated fat, polyunsaturated fat, fiber, and protein).

Prior studies of depression and diet composition are limited and generally yield mixed findings. The most consistent evidence has found no cross-sectional association between depression and protein intake (Davison & Kaplan, 2012; Nanri et al., 2010; Oishi et al., 2009; Woo et al., 2006). In contrast, the present study demonstrated a negative association between depressive symptom severity and protein intake. Prior studies of depression and other diet composition factors (i.e., total energy, carbohydrates, and fat) have reported inconsistent findings (Anton & Miller, 2005; Dressler & Smith, 2015; Hyakutake et al., 2016; Mooreville et al., 2014; Nanri et al., 2014; Oishi et al., 2009; Simon et al., 2008; Whitaker et al., 2014; Woo et al., 2006). Few of the available cross-sectional studies have

examined the associations of depression with both total levels (e.g., total carbohydrates) and subordinating levels (e.g., fiber and sugar) of nutrients to understand the differential associations of depression with nutrients of varying nutritional quality. One study reported that depression was not associated with total fat but was inversely associated with saturated fat (Whitaker et al., 2014). In the present study, although depressive symptom severity was not associated total carbohydrate intake, it was positively associated with sugar intake and negatively associated with fiber intake. Taken together, these findings suggest that examining only total levels of nutrients may mask and/or underestimate the relationships between depression and dietary intake in cases where subordinating levels of nutrients vary in nutritional quality (e.g., fiber versus sugar).To our knowledge, no study has examined the depression-diet composition associations within specific race/ethnicity groups. In one study among low-income predominantly African American (87%) women with overweight or obesity, greater depressive symptom severity was associated with increased sugar and saturated fat intake; depressive symptoms were not related to total fat intake (Whitaker et al., 2014). In the present study, findings for non-Hispanic Black adults were somewhat consistent with these prior findings – depressive symptom severity was associated with greater sugar and saturated fat intake; however, the present study additionally found that depressive symptom severity was associated with greater total fat intake.

There are several potential explanations for why greater depressive symptom severity was cross-sectionally associated with increased saturated fat and sugar intake and reduced fiber and protein intake in the entire sample. We first consider explanations for how depressive symptoms may contribute to this pattern of diet composition. One, the experience of negative affect in depression may lead to emotional eating (Konttinen, Silventoinen, Sarlio-Lahteenkorva, Mannisto, & Haukkala, 2010; van Strien, Konttinen, Homberg, Engels, & Winkens, 2016), which leads to increased consumption of energy-dense, highly palatable foods (Konttinen, Mannisto, Sarlio-Lahteenkorva, Silventoinen, & Haukkala, 2010) that are high in saturated fat and sugar and low in protein and fiber. Two, depression is associated with deficits in executive functioning (Snyder, 2013), which is a critical neurocognitive skill for planning. It is plausible that people with elevated depressive symptoms may consume foods that take less effort to access and prepare (e.g., fast foods or packaged foods), which are high in sugar, saturated fat, and energy and low in protein and fiber. Three, one symptom of depression – increased appetite – may lead to greater intake of foods, particularly those that provide comfort, such as high-sugar and high-fat foods. This mechanism may be particularly salient for individuals with atypical depression, which is characterized by increased appetite/weight gain and excessive sleep (American Psychiatric Association, 2013). Although these mechanisms may explain depression's association with higher saturated fat and sugar intake and lower fiber and protein intake, they are inconsistent with the finding that depressive symptom severity was unrelated to total energy intake.

We next consider how this pattern of diet composition may contribute to depressive symptoms. One, dietary patterns notable for high protein and fiber intake and low sugar intake (e.g., Mediterranean and healthy diets) are associated with a lower risk of depression (Lai et al., 2014; Psaltopoulou et al., 2013; Quirk et al., 2013; Rahe et al., 2014). Two, chronic high intake of foods high in energy and sugar may lead to weight gain, which could contribute to depression through mechanisms of body image dissatisfaction and low

self-esteem (Gavin, Simon, & Ludman, 2010; Wardle & Cooke, 2005). Three, biological mechanisms of inflammation (Berk et al., 2013) and gut microbiota (Berk et al., 2013; Dash, Clarke, Berk, & Jacka, 2015) may link diets that are high in sugar and fat and in low fiber to future depression.

A critical finding in the present study was that, among non-Hispanic Blacks, greater depressive symptom severity was associated with increased intake of diet components that represent an unhealthy pattern of eating, and the magnitudes of those relationships were larger than those of non-Hispanic Whites. These findings may constitute a disparity. Among non-Hispanic Blacks, social determinants of health, including neighborhood environment, health literacy, and health care access may contribute to disparities in the availability of healthy foods, access to supermarkets, convenience and cost of healthy foods, knowledge of nutrition, and knowledge of the link between diet and disease (Satia, 2009). Moreover, non-Hispanic Blacks are less likely to receive treatment for depression than non-Hispanic Whites, potentially due to preferences, limited access, and costs of treatment (Simpson et al., 2007). Cultural factors may also explain the observed relationship. Greater acceptance of larger shape and weight among some non-Hispanic Black women (Befort, Thomas, Daley, Rhode, & Ahluwalia, 2008; Flynn & Fitzgibbon, 1998) may lead to less concern over making healthy food choices, particularly in the presence of depression. Although not well understood, cultural aspects of eating behavior – such as eating food for comfort, the types of foods prepared/consumed, and the social context of eating – may serve as mechanisms between depression and diet.

Although Mexican Americans and other Hispanics experience high rates of obesity (Hales et al., 2020) and many of the same social determinants of health that affect non-Hispanic Blacks (Velasco-Mondragon, Jimenez, Palladino-Davis, Davis, & Escamilla-Cejudo, 2016), the patterns of associations between depressive symptoms and diet composition factors were quite different across these groups. Among Mexican Americans, greater depressive symptom severity was associated with increased saturated fat intake only. Among other Hispanics, greater depressive symptom severity was associated with reduced intake of several diet composition factors (i.e., total fat, monounsaturated fat, polyunsaturated fat, fiber, and protein). This contrast suggests that different social, behavioral, cultural processes may be involved. First, acculturation factors, such as acculturative stress and dietary acculturation, are complex and multifactorial processes that have the potential to impact psychological wellbeing (Koneru, Weisman de Mamani, Flynn, & Betancourt, 2007) and dietary intake (Perez-Escamilla, 2011; Satia, 2009). Second, Hispanics experience generally better health and lower mortality than non-Hispanic Whites, a phenomenon called the Hispanic health paradox (Franzini, Ribble, & Keddie, 2001). Sociocultural factors, such as social integration and social connection, have been proposed as resilience factors among Hispanics that may account for improved health and longevity (Ruiz, Hamann, Mehl, & O'Connor, 2016). Similarly, it is plausible that social integration, social connection, and communal coping have the potential to buffer the association between depressive symptoms and intake of unhealthful diet components. Notably, it is unclear why the pattern of associations in the present study varied between Mexican Americans and other Hispanics, suggesting the need for further study of Hispanic subgroups. Overall, additional research is needed to better understand the social, behavioral, and cultural mechanisms that may contribute to

differences between racial/ethnic groups in the associations between depression and diet composition.

Findings from this study have several clinical implications. Given the association of depression with generally unhealthy diet composition factors (i.e., reduced fiber and protein intake; increased saturated fat and sugar intake), there is a need to develop and evaluate screening and intervention approaches to identify and address both diet-related and depression-related risk factors for obesity and cardiometabolic diseases in individuals with depression. To illustrate, screening for dietary intake in individuals with depression may help to identify those in need of intervention and link them with appropriate mental health, behavioral health, and/or nutrition services. Given the finding that the relationships between depression and diet composition factors varied by race/ethnicity, intervention services should be delivered in a culturally-sensitive manner that takes into account an individual's beliefs, preferences, and practices related to mental health, body image, and diet. Furthermore, there is a need for community-based and health-related policy interventions and programs to address social determinants of health that impact access to high quality, healthy foods and mental health services to address diet-related and depression-related risk for obesity and cardiometabolic diseases.

This study has limitations that warrant mention. First, directionality cannot be determined in this cross-sectional study. Future prospective studies are needed to better understand directionality. Second, NHANES utilized a self-reported dietary recall measure, which is subject to response bias, social desirability, and forgotten foods. In the present study, diet was measured using two days of 24-hour dietary recalls using the interviewer-administered U.S. Department of Agriculture Automated Multiple-Pass Method, which is deemed a robust measure of dietary intake (Moshfegh et al., 2008). Third, depression was assessed as self-reported depressive symptom severity and not a clinical diagnosis of a depressive disorder. Fourth, lower power and wider confidence intervals for certain race/ethnicity groups could have resulted in type II errors. Finally, the analyses did not examine moderation of the depression-diet relationship by age or sex, and they did not examine moderation of the depression-obesity association by diet. Future studies should examine such interaction effects to understand the complex relationships between depression, diet, obesity, and demographic characteristics.

Several key strengths also warrant mention. This study utilized data from NHANES, which offers a large sample representative of the U.S. population and provides nationally representative estimates and opportunities to examine differences by race/ethnicity. In addition, NHANES used strong assessments of depressive symptom severity and diet, which allowed for simultaneous examination of multiple diet composition factors in one study.

Conclusions

In a large sample of adults representative of the U.S. population, greater depressive symptom severity was associated with increased intake of saturated fat and sugar and reduced intake of fiber and protein. Relationships between depression and diet composition varied by race/ethnicity. Critically, depressive symptom severity was associated with intake of diet components that represent an unhealthy pattern of eating, particularly among non-Hispanic

Blacks. Poor diet may be one mechanism explaining the excess risk of obesity in individuals with depression. There is a need for future studies to examine prospective associations and mechanistic pathways linking depression, diet, and obesity. Such studies will inform the design and evaluation of interventions at the individual, community, and public policy levels to address risk factors for and mechanisms implicated in the development of obesity and cardiometabolic diseases. Depression and dietary behaviors may serve as targets for such interventions and health policy efforts.

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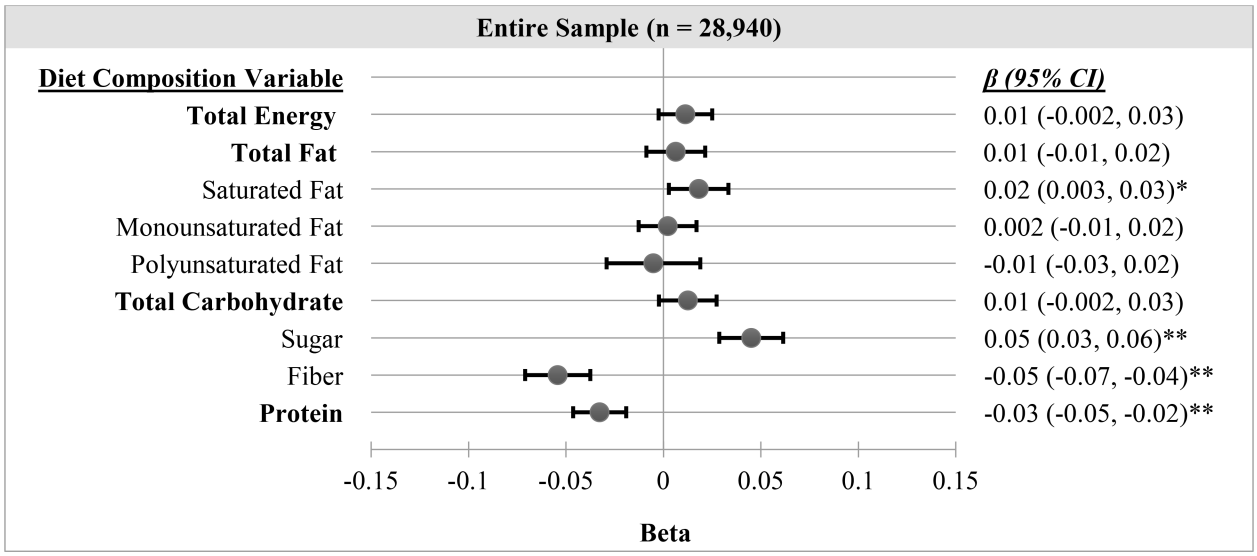

Figure 1.

Forest plot depicting results of linear regression analyses examining associations between depressive symptom severity (PHQ-9 total score) and each of the nine diet composition variables in separate models. Models are adjusted for age, sex, race/ethnicity, education, poverty-income ratio, body mass index, cardiovascular disease, diabetes, kidney disease, liver disease, hypertension, and NHANES sampling design. Bolded dietary factors are total levels of nutrients, and unbolded factors are subordinating levels of nutrients. ***p*<.01

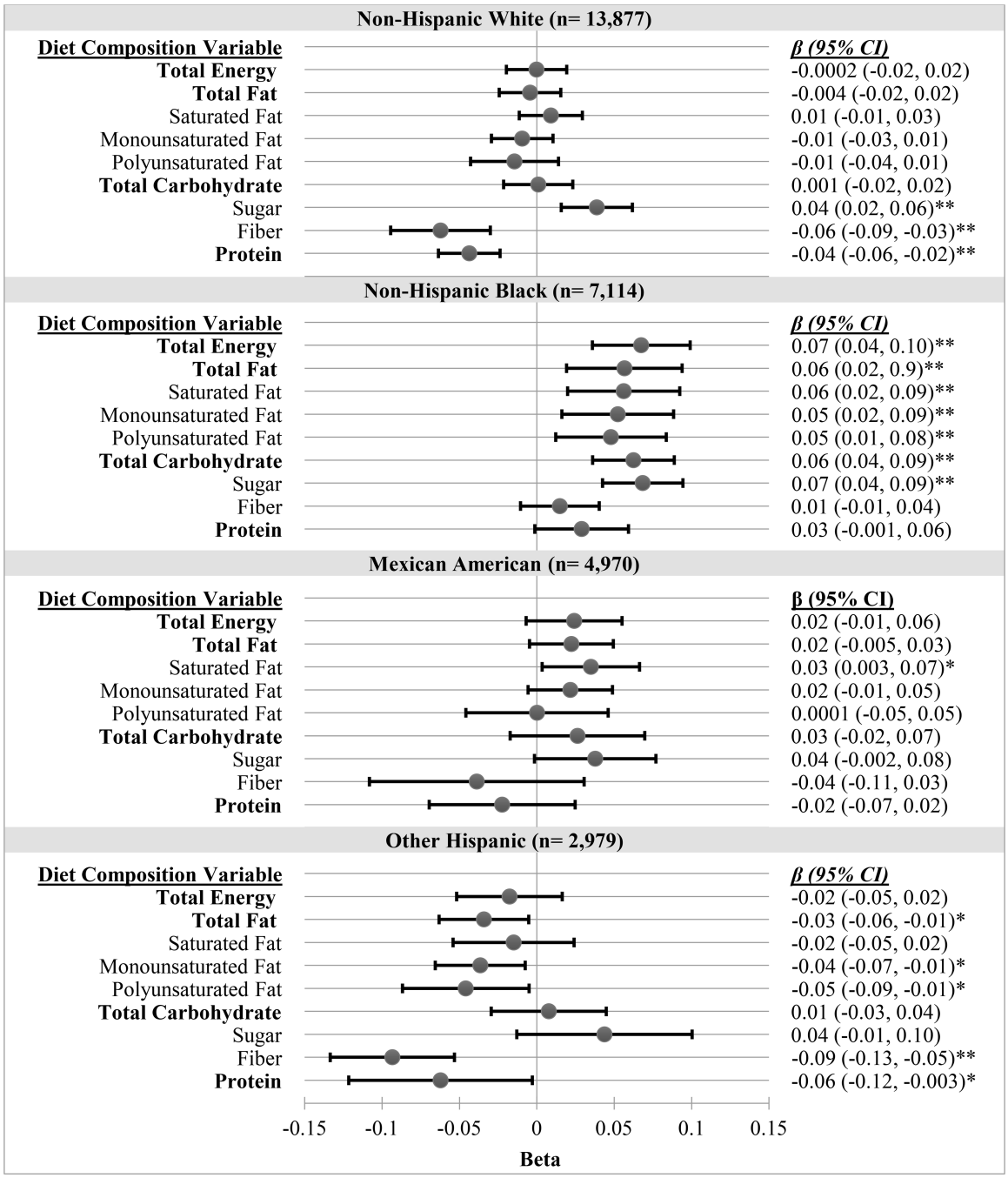

Figure 2.

Forest plot depicting results of linear regression analyses examining associations between depressive symptom severity (PHQ-9 total score) and each of nine diet composition variables, stratified by race/ethnicity. Models are adjusted for age, sex, education, poverty-income ratio, body mass index, cardiovascular disease, diabetes, kidney disease, liver disease, hypertension, and NHANES sampling design. Bolded dietary factors are total levels of nutrients, and unbolded factors are subordinating levels of nutrients. *p<.05; **p<.01

Table 1.

Characteristics of participants in the entire sample and by race/ethnicity group

	Entire Sample (*N* = 28,940)	Non-Hispanic White (*n* = 13,877)	Non-Hispanic Black (*n* = 7,114)	Mexican American (*n* = 4,970)	Other Hispanic (*n* = 2,979)	*p*-value
Age, years, *M (SD)*	49.1 (18.8)	52.1 (19.3) [b,c,d]	47.4 (18.1) [a,c]	44.0 (17.4) [a,b,d]	47.5 (17.4) [a,c]	<.001
Female, *%*	51.6	50.3 [b,d]	52.4 [a,d]	51.8 [d]	55.9 [a,b,c]	<.001
Education, less high school or GED, %[e]	25.3	15.5 [b,c,d]	22.6 [a,c,d]	50.4 [a,b,d]	35.5 [a,b,c]	<.001
Poverty-income ratio, *M (SD)*[f]	2.5 (1.6)	2.8 (1.6) [b,c,d]	2.3 (1.6) [a,c,d]	1.9 (1.4) [a,b,d]	2.1 (1.5) [a,b,c]	<.001
Body mass index, kg/m^2, *M (SD)*[g]	29.5 (7.0)	28.8 (6.9) [b,c]	30.6 (8.0) [a,c,d]	29.8 (6.3) [a,b,d]	29.1 (6.2) [b,c]	<.001
Cardiovascular disease, *%*	11.2	13.8 [b,c,d]	11.0 [a,c,d]	6.2 [a,b,d]	8.0 [a,b,c]	<.001
Diabetes, *%*	12.8	10.7 [b,c,d]	15.9 [a,c,d]	14.1 [a,b]	13.0 [a,b]	<.001
Kidney disease, *%*	3.3	3.2 [b]	3.7 [a,c]	2.8 [b]	3.3	.035
Liver disease, *%*	3.8	4.0 [b,d]	2.3 [a,c,d]	4.2 [b,d]	6.1 [a,b,c]	<.001
Hypertension, *%*	29.5	30.8 [b,c,d]	36.2 [a,c,d]	18.9 [a,b,d]	24.8 [a,b,c]	<.001

Note. To test for group differences across the race/ethnicity groups, one-way analyses of variance were used for continuous variables and chi-square tests were used for categorical variables. Post hoc tests were performed for all significant differences. GED = Generalized Educational Development Test, M = mean, PHQ-9 = Patient Health Questionnaire-9, and SD = standard deviation.

[a]Significantly different from the non-Hispanic White group (p < .05)

[b]Significantly different from the non-Hispanic Black group (p < .05)

[c]Significantly different from the Mexican American group (p < .05)

[d]Significantly different from other Hispanic group (p < .05)

[e]Based on a reduced sample of 28,883 participants with complete data on education

[f]Based on a reduced sample of 26,619 participants with complete data on poverty-income ratio

[g]Based on a reduced sample of 28,590 participants with complete data on body mass index

Table 2.

Descriptive statistics for depression and diet composition variables in the entire sample and by race/ethnicity group

	Entire Sample (*N* = 32,153)	Non-Hispanic White (*n* = 13,877)	Non-Hispanic Black (*n* = 7,114)	Mexican American (*n* = 4,970)	Other Hispanic (*n* = 2,979)	*p*-value
PHQ-9 Total (0–27), *M (SD)*[e]	3.2 (4.2)	3.2 (4.2) [d]	3.2 (4.3) [d]	3.1 (4.2) [d]	3.7 (4.9) [a,b,c]	<.001
Total energy intake, kcal, *M (SD)*	2,035.5 (835.4)	2,069.3 (816.3) [b,d]	2,000.7 (859.1) [a,c,d]	2,063.3 (853.1) [b,d]	1,915.0 (822.2) [a,b,c]	<.001
Total fat, g, *M (SD)*	77.8 (38.3)	80.5 (37.8) [b,c,d]	78.0 (39.5) [a,c,d]	75.9 (38.4) [a,b,d]	68.4 (36.3) [a,b,c]	<.001
Saturated fat, g, *M (SD)*	25.3 (13.7)	26.8 (13.8) [b,c,d]	24.4 (13.5) [a,d]	24.5 (13.6) [a,d]	21.9 (12.7) [a,b,c]	<.001
Monounsaturated fat, g, *M (SD)*	27.8 (14.4)	28.6 (14.3) [c,d]	28.2 (14.9) [c,d]	27.4 (14.4) [a,b,d]	24.4 (13.6) [a,b,c]	<.001
Polyunsaturated fat, g, *M (SD)*	17.6 (9.8)	17.9 (9.7) [b,c,d]	18.4 (10.5) [a,c,d]	17.0 (9.4) [a,b,d]	15.6 (9.2) [a,b,c]	<.001
Total carbohydrate, g, *M (SD)*	274.3 (107.4)	247.9 (106.6) [b,c,d]	241.2 (109.2) [a,c]	257.7 (107.4) [a,b,d]	241.7 (105.2) [a,c]	<.001
Sugar, g, *M (SD)*	110.7 (64.8)	112.9 (68.1) [c,d]	111.4 (65.0) [c,d]	107.6 (58.0) [a,b,d]	103.3 (59.3) [a,b,c]	<.001
Fiber, g, *M (SD)*	16.3 (8.8)	16.4 (8.5) [b,c]	14.0 (7.7) [a,c,d]	19.4 (10.0) [a,b,d]	16.5 (8.8) [b,c]	<.001
Protein, g, *M (SD)*	79.4 (35.2)	80.0 (34.2) [b,c]	76.4 (34.9) [a,c,d]	83.0 (37.2) [a,b,d]	78.5 (36.2) [b,c]	<.001

Note. To test for group differences across the race/ethnicity groups, one-way analyses of variance were used for continuous variables and chi-square tests were used for categorical variables. Post hoc tests were performed for all significant differences. M = mean, PHQ-9 = Patient Health Questionnaire-9, SD = standard deviation.

[a]Significantly different from the non-Hispanic White group (p < .05)

[b]Significantly different from the non-Hispanic Black group (p < .05)

[c]Significantly different from the Mexican American group (p < .05)

[d]Significantly different from other Hispanic group (p < .05)

[e]Based on a reduced sample of 27,302 participants with complete data on PHQ-9 total

Table 3.

Linear regression analyses examining the interaction terms for PHQ-9 Total (z-scored) by race ethnicity as predictors of diet composition variables (*z*-scored)

	PHQ-9 total x RE1 (NHB vs. NHW)		PHQ-9 total x RE2 (MA vs. NHW)		PHQ-9 total x RE3 (OH vs. NHW)	
	β	***p*-value**	***β***	***p*-value**	***β***	***p*-value**
Total Energy	0.08**	<.001	0.02	.198	−0.02	.198
Total Fat	0.07**	<.001	0.01	.282	−0.04**	.005
Saturated fat	0.07**	.002	0.03	.104	−0.02	.273
Monounsaturated fat	0.06**	<.001	0.02	.268	−0.04**	.002
Polyunsaturated fat	0.06**	.001	−0.01	.782	−0.04**	.004
Total Carbohydrate	0.06**	<.001	0.0	.221	0.00	.992
Sugar	0.06**	<.001	0.03	.057	0.02	.490
Fiber	0.03**	.002	−0.04	.296	−0.06**	<.001
Protein	0.05**	<.001	−0.03	.294	−0.06*	.015

N = 28,940. Analyses are adjusted for age, sex, race/ethnicity, education, income, body mass index, diabetes, cardiovascular disease, kidney disease, liver disease, hypertension, and NHANES sampling design. OH = other Hispanic; MA = Mexican American; NHB = non-Hispanic Black; NHW = non-Hispanic White

* $p < .05$

** $p < .01$