Role of depressive symptoms in explaining socioeconomic status disparities in dietary quality and central adiposity among US adults: a structural equation modeling approach[1–4]

May A Beydoun, Marie T Fanelli Kuczmarski, Marc A Mason, Shari M Ling, Michele K Evans, and Alan B Zonderman

ABSTRACT

Background: The link between socioeconomic status (SES), depression, dietary quality, and central adiposity remains unclear.

Objective: Pathways linking SES to dietary quality and central adiposity through depressive symptoms were examined across sex-ethnicity groups.

Design: Extensive data on US adults aged 30–64 y from the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study were used in multiple linear logistic regression models and structural equation models to test pathway associations. Measures included Center for Epidemiologic Studies–Depression (CES-D) scores, 2005 Healthy Eating Index (HEI) values, and dual-energy X-ray absorptiometry. Sample sizes for most analyses ranged between 1789 for anthropometric outcomes and 1227 for trunk fat outcomes.

Results: The CES-D score was associated with lower HEI scores in all sex-ethnicity groups, except in African American men, and with higher waist-to-hip ratios (WHRs) among African American women. A CES-D score ≥ 16 was positively associated with waist circumference (WC) and with trunk fat among white women and men, respectively. SES was positively related to central adiposity among African American men (central obesity and WC) and African American women (central obesity and percentage trunk fat) but was inversely related to central adiposity among white women. Among whites only, the total positive effect of SES on HEI was significantly mediated by CES-D score. Among white women, the total inverse effect of SES on WC and WHR was significantly explained by the CES-D score and HEI, whereas the CES-D score was positively associated with WHR among African American women, independently of SES.

Conclusion: Future mental health interventions targeted at reducing SES disparities in dietary quality and central adiposity may have different effects across sex-ethnicity groups.   *Am J Clin Nutr* 2009;90:1084–95.

INTRODUCTION

Obesity is a major public health problem in the United States, and its adverse health effects (1–3) as well as its related behavioral risk factors, specifically physical activity (4) and dietary patterns (5–8), have been well documented. Approximately 30% of the US adult population is currently obese (based on body mass index cutoffs) (9), and the prevalence of central obesity is ≈38.3% among men and ≈59.9% in women (10–13). Obesity and central obesity have recently been associated with an increased number of depressive symptoms and a higher prevalence of major depressive disorders (14–44). Whereas the obesity-depression association may

be bidirectional, most studies have concluded that obesity causes depression, even when the design was cross-sectional (14–29, 42, 43). However, a growing number of studies have investigated the opposite association (30–38, 44). It is still unknown, however, whether unhealthy eating is a mediating factor in this association and whether depression has an effect on dietary quality that is driven by socioeconomic status (SES) differences in depressive symptoms. SES is determined early in life (eg, educational attainment), and depression may well be a result of lower SES (45), whereas poor dietary quality related to binge eating or a generally neglected and unhealthy lifestyle may be the outcome of depression (46). Thus, in the present study, we hypothesized that a lower SES may cause a higher number of depressive symptoms, which in turn leads to a poorer quality of dietary intake and finally higher central adiposity.

To our knowledge, no large study has used dual-energy X-ray absorptiometry (DXA) to examine the depression-adiposity association. DXA provides quantitative body-composition data and allows estimation of the proportion of fat to lean mass of individuals as well as the distribution of fat within the body. Our present study used extensive data from adults participating in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study to examine *1)* the association between depressive symptoms and central adiposity measures and the moderation of this association by sex and race-ethnicity; *2)* the association between depressive symptoms and dietary quality and its moderation by sex and race-ethnicity; *3)* SES disparities in central adiposity measures, moderation by sex and race-ethnicity, and mediation by depressive symptoms and dietary quality; and *4)* SES disparities in dietary quality, moderation by sex and race-ethnicity, and medi-

ation by depressive symptoms. We hypothesized that the association between depressive symptoms and central adiposity is positive and stronger among women and that the pathway explaining SES disparities in central adiposity includes depression and dietary quality, although it differs significantly between each sex and race-ethnicity stratum. We also hypothesize that SES disparities in dietary quality are largely determined by depressive symptoms.

SUBJECTS AND METHODS

Database

The HANDLS study is a prospective longitudinal study of a baseline representative sample of African Americans and whites between 30 and 64 y of age. Participants were recruited as a fixed cohort by household screenings from an area probability sample of 12 census segments in Baltimore City. Data were collected in 2 separate phases. Phase 1 consisted of screening, recruitment, a household interview (assessing sociodemographic information and physiologic and psychological chronic exposure), and a first 24-h recall; phase 2 consisted of an in-depth examination in a mobile Medical Research Vehicles (MRV) and included a second dietary assessment with a 24-h recall, psychometric measures (eg, for depressive symptoms and cognitive function), and anthropometric and body-composition measurements.

Study population

Three-thousand seven hundred twenty-four selected subjects have participated in the household survey at phase 1 (sample 1) since initial recruitment on 4 November 2004. Of these patients, 2436 (65.4%) have had complete baseline phase 2 examinations thus far (sample 2a). However, our data uses a subset with 2 d of dietary recall, anthropometric measures, and CES-D data ($n = 1789$; sample 2b). In addition, part of the analysis relies on subjects with complete DXA scans as well as complete dietary and CES-D data ($n = 1583$; sample 3). Subjects in sample 2b differed from the remaining subjects in sample 1 by having a higher percentage of poor (48% compared with 35%; $P < 0.05$, chi-square test), a higher proportion of African Americans (61% compared with 57%; $P < 0.05$, chi-square test), and a higher proportion of females (57% compared with 52%; $P < 0.05$, chi-square test). Similarly, subjects in sample 3 differed from the remaining subjects in sample 1 by having a significantly higher proportion of poor (49% compared with 36%; $P < 0.05$, chi-square test) and a higher proportion of African Americans (61% compared with 57%; $P < 0.05$, chi-square test). Both samples 3 and 2b were used in our analyses, depending on the outcome of interest. The procedures followed were in accordance with the ethical standards of the institution or regional committee on human experimentation, and approval was obtained from the relevant committee on human subjects.

Central adiposity outcomes

Waist circumference (WC) was assessed by using a tape measure starting from the hip bone and wrapping around the waist at the level of the navel in such a way that it was kept parallel to the floor and was not wrapped too tight or too loose; the subjects did not hold their breath during the measurement. Central obesity was defined as a WC ≥ 102 cm (40 in) for men or ≥ 88 cm (35 in) for women (10, 47). In addition, waist-to-hip ratio (WHR) was used as a second measure for relative central obesity (48–52). To measure hip circumference, the anthropometric tape used to measure WC was placed on the widest part of the participant's hip and at the maximum extension of the buttocks. Both measures were made to the nearest 1/10th of a centimeter. WHR was the ratio of WC to hip circumference. DXA was performed by using a Lunar DPX-IQ (Lunar Corp, Madison, WI), producing scans that would measure total tissue mass, fat mass, lean mass, regional fat mass, etc. Our analysis focused on trunk fat (kg) and the ratio of trunk to total body fat (trunk fat as a percentage of total body fat; trunk fat%).

Depressive symptoms assessment

Extensively trained psychometricians administered, among others, a baseline battery of cognitive and neuropsychological tests (53), which included baseline depressive symptoms, using the Center for Epidemiologic Studies–Depression (CES-D) scale —a 20-item self-report symptom rating scale that emphasizes the affective depressed mood component (54). The invariant factor structure of the CES-D was shown by using confirmatory factor analysis that compared the first National Health and Nutrition Examination Survey (NHANES I) with HANDLS data (55). In most of our analysis, the CES-D was used in its continuous form. However, cutoffs of 16 and 20 were also used to assess symptoms of depression and severe depression, respectively.

Dietary intakes and dietary quality assessment

With the use of the US Department of Agriculture (USDA) Automated Multiple Pass Method (AMPM), two 24-h dietary recalls were administered by trained interviewers, usually 4–10 d apart. Approximately 65% of the recalls reflect weekday intakes (Monday to Thursday), and the remaining 35% reflect weekend consumption (Friday to Sunday). The AMPM provides an automated, standardized, 5-step process; engages the participants; maintains their interest through use of the Food Model Booklet; and prompts more complete recollection of consumed food and beverage. This collection method was validated for intakes of protein, carbohydrate, fat, and energy in obese and nonobese men and women (56, 57).

Dietary quality was assessed by using the 2005 USDA Healthy Eating Index (HEI), with the use of the average of the two 24-h recalls (58). The 1995 HEI version (59) was revised recently to reflect the 2005 Dietary Guidelines for Americans, and the standards for each component are now based on an energy density approach (58). The new HEI includes 12 components and is measured on a scale of 0–100, ie, the higher the total HEI score, the better the diet (for more details, *see* Appendix A under "Supplemental data" in the online issue).

Covariates

Other covariates were considered in various parts of our analyses as potential confounders, moderators, or mediators. Sociodemographics included age grouped into seven 5-y categories, sex, race-ethnicity (white compared with African American), and marital status (married compared with unmarried; based on the question, "Are you currently married or living with someone?"). SES was measured on the basis of the completed years of education and the poverty-income ratio (PIR). Education was

categorized into <high school (0–8 y), high school (9–12 y), and >high school (≥13 y), whereas the PIR was categorized as poor (PIR < 125%) and nonpoor (PIR ≥ 125%). In addition, principal components analysis of continuous education and PIR was conducted to obtain a single measure of SES measured on a standardized z score scale. Finally, smoking status was considered in all analyses as a potential confounder and was coded as 0 for never or former smoker and 1 for current smoker.

Statistical analysis

STATA release 10.0 (60) was used in all analyses, except for structural equation models for which SAS version 9.2 was used (61). Most analyses were stratified by sex and ethnic group. One-factor analysis of variance was conducted followed by Bonferroni-corrected multiple t tests to test differences between means across categorical variables. A chi-square test was conducted to test associations between categorical variables. After descriptive analyses and an exploratory correlation analysis using Pearson's correlations and associated P values were conducted, several multiple regression models and path analyses were run for 3 specific purposes: *1*) to identify the association between depression on one hand and dietary quality and adiposity measures on the other hand, stratified by sex and race-ethnicity; *2*) to test SES differences in dietary quality, depressive symptoms, and central adiposity measures, stratified by race-ethnicity and sex and to test the significance of interaction terms at a type I error of 0.05; and *3*) to identifying the main pathways explaining SES disparities in central adiposity measures through depressive symptoms and dietary quality and SES disparities in dietary quality through depressive symptoms, with sex-race groups as a potential moderator. Path analysis was performed to test a theoretical model in which demographic factors (age and marital status) and smoking status were exogenous (ie, were not predicted by other variables), whereas SES was an endogenous variable that was allowed to predict all other outcome variables (ie, depressive symptoms, HEI, and central adiposity measures). Moreover, the CES-D continuous score was allowed to be associated with HEI along with a direct effect on central adiposity. Finally, HEI was allowed to predict central adiposity (Equations *1–4*).

$$SES = \sum_{j=1}^{k} \alpha_{Z_{j1}} Z_j + e_1 \qquad (1)$$

$$CES-D = \alpha_{12}SES + \sum_{j=1}^{k} \alpha_{Z_{j2}} Z_j + e_2 \qquad (2)$$

$$HEI = \alpha_{13}SES + \alpha_{23}CES-D + \sum_{j=1}^{k} \alpha_{Z_{j3}} Z_j + e_3 \qquad (3)$$

$$Adip_{cent} = \alpha_{14}SES + \alpha_{24}HEI + \alpha_{34}CES-D$$
$$+ \sum_{j=1}^{k} \alpha_{Z_{j4}} Z_j + e_4 \qquad (4)$$

where Z_j in these equations is the vector of other exogenous variables (eg, sociodemographic and lifestyle factors) and e_1

through e_4 are the error terms. Correlations between all exogenous variables, except between error terms (assumed to be zero), were estimated.

The global Goodness of Fit Index for the structural equation model included the chi-square statistic, which tested the null hypothesis that the reproduced covariance matrix has the specified structure or that the model fits the data. Moreover, the Adjusted Goodness of Fit Index (AGFI; adjusted for df) was presented with specific cutoff criteria, as suggested elsewhere (62). Furthermore, the mediation proportion (MP; %) was computed to quantify the proportion of the total effect of a variable that is explained by a particular pathway (63, 64). For instance, if interested in the pathway SES→CES-D→HEI, the mediation proportion with Equations *2*, *3*, and *4* is MP = indirect × 100/ total = $(\alpha_{12}× \alpha_{23}) × 100/(\alpha_{12} × \alpha_{23}+ \alpha_{13})$, whereas that of SES→CES-D→HEI→Adip$_{cent}$ (central adiposity) is MP = $(\alpha_{12}× \alpha_{23}× \alpha_{34}) × 100/[(\alpha_{12}× \alpha_{23}× \alpha_{24}) + (\alpha_{12}× \alpha_{34}) + (\alpha_{13}× \alpha_{24}) + \alpha_{14}]$. In these 2 examples, the direct effects α_{13} and α_{14} should be in the same direction as the indirect effects to obtain a meaningful positive MP.

All structural equations model–stratified analyses were conducted by using the SAS CALIS procedure, whereas multigroup analyses to assess the significance of differences between groups of main path coefficients was done by using PROC TCALIS. In the latter analysis, we specifically examined the Lagrange multiplier statistic (chi-square test, 1 df) (65) to assess change in model fit when each path coefficient for each of the 4 groups was constrained to be equal to that path coefficient in other groups (66). To account for potential selection bias in all main analyses (ordinary least-squares logistic regression and structural equation models), a 2-stage Heckman selection model was constructed (67). A probit model was conducted as a first stage in which 2 alternative selection variables (ie, belonging to sample 2b or not or to sample 3 or not) were modeled against basic sociodemographic variables that were pseudocomplete for the total HANDLS sample ($n \approx 3724$). These variables included age, sex, race-ethnicity, marital status, and current smoking status. Missing data for those variables were included as a separate dummy variable. The probability of being selected was predicted, and an inverse mills ratio was derived from that probability and included as a second stage into the main model or equation in which ≥2 of depressive symptoms, dietary quality, SES, and anthropometric/DXA variables were included.

RESULTS

Characteristics of the study population: sex-ethnicity differences

Baseline characteristics of our study population are shown in **Table 1**. Whereas the age distributions across sex and race were similar, the 4 sex-ethnicity groups differed significantly in terms of marital status (the proportion married was lowest among African American women and highest among white men), educational attainment (>high school prevalence was lowest among African American men and highest among white men), and PIR (the proportion poor was lowest among white men and highest among African American women). More than one-half of African American men were current smokers (55.2%),

TABLE 1
Main characteristics of US adults aged 30–64 y in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study[1]

Characteristic	White (n = 691)		African American (n = 1080)	
	Men	Women	Men	Women
Sample 2b (n = 1789)[2]				
n	295	396	473	607
Age (%)				
30–34 y	10.1	12.0	10.6	9.5
35–39 y	13.5	10.3	11.9	10.9
40–44 y	10.4	16.8	14.0	14.0
45–49 y	17.8	16.5	19.2	20.6
50–54 y	17.8	17.8	16.7	16.8
55–59 y	16.5	14.3	16.7	15.7
60–64 y	13.8	12.3	11.0	12.6
Married (%)	56.2	49.4	49.0	37.2[3]
Education (%)				
<High school	10.1	7.3	5.4	4.4[3]
High school	44.8	49.4	65.6	59.4
>High school	38.4	34.6	24.4	32.3
Missing	6.7	8.8	4.6	3.9
Poverty-income ratio (%)				
<125%, poor	32.0	43.0	52.3	55.8
≥125%, not poor	68.0	57.1	47.7	44.2[3]
Current smoker (%)	48.8	42.1	55.2	39.1[3]
CES-D score	10.0 ± 7.7[4]	12.5 ± 8.9[5]	11.0 ± 6.86	12.0 ± 8.5[3,5]
CES-D score ≥16 (%)	20.0	35.1	22.1	29.5[3]
CES-D score ≥20 (%)	12.5	21.8	12.3	18.9[3]
WC (cm)	100.9 ± 19.8	99.3 ± 19.6	94.7 ± 16.7[5,6]	99.4 ± 20.1[3,7]
Central obesity (%)[8]	45.8	68.7	29.0	72.4[3]
Waist-to-hip ratio	0.98 ± 0.07	0.89 ± 0.08[5]	0.93 ± 0.07[5,6]	0.89 ± 0.08[3,5,7]
Healthy Eating Index[9]	48.5 ± 11.9	50.7 ± 13.2	46.6 ± 11.0[6]	50.0 ± 11.9[3,7]
Sample 3 (n = 1583)[10]				
n	263	354	425	541
Trunk fat (kg)[11]	12.9 ± 5.2	16.5 ± 6.4[5]	10.1 ± 5.0[5,6]	16.4 ± 6.5[3,5,7]
Trunk fat (% of total body fat)[11]	55.1 ± 5.3	49.8 ± 5.6[5]	51.5 ± 5.1[5,6]	47.8 ± 5.7[3,5–7]

[1] CES-D, Center for Epidemiologic Studies–Depression; WC, waist circumference.

[2] Sample 2b included subjects with complete data for both 24-h dietary recalls, CES-D scores, and anthropometric measures (43% men).

[3] Significant differences between proportions or means across sex-ethnicity groups, $P < 0.05$ (chi-square test or ANOVA).

[4] Mean ± SD (all such values).

[5] Significantly different from white men, $P < 0.05$ (t test, Bonferroni corrected for multiple testing after ANOVA).

[6] Significantly different from white women, $P < 0.05$ (t test, Bonferroni corrected for multiple testing after ANOVA).

[7] Significantly different from African American men, $P < 0.05$ (t test, Bonferroni corrected for multiple testing after ANOVA).

[8] Defined as a WC ≥ 102 cm (40 in) for men or ≥88 cm (35 in) for women.

[9] Score can range between 0 and 100.

[10] Sample 3 included subjects with complete data for both 24-h dietary recalls, CES-D scores, and dual-energy X-ray absorptiometry scan measures (43% men).

[11] Analyses with these variables as outcomes used subjects from sample 3 (*see* Subjects and Methods for more details).

compared with 39–49% among other groups ($P < 0.05$, chi-square test). Mean CES-D scores differed significantly between sex-ethnicity groups, whereas the prevalence of elevated scores was consistently the highest among white women for the 16 and 20 cutoffs. In contrast, mean WC was highest among white men, though central obesity was highest among African American women followed closely by white women. The mean WHR was significantly more elevated among white men than among their female counterparts. Differences in the mean HEI were only significant between white women and African American men on one hand (mean HEI: 50.7 and 46.6, respectively) and between African American women and African American men on the other hand (mean HEI: 50.0 and 46.6, respectively). Absolute trunk fat was the highest among white and African American women, although trunk fat% was on average highest among white men.

Correlations between central adiposity, dietary quality, depressive symptoms, and SES across sex-ethnicity groups

The results of an exploratory analysis in which continuous central adiposity measures, dietary quality (HEI total score), depressive symptoms (CES-D score), and SES factor scores were correlated by using Pearson's correlation coefficients for the reduced sample size (complete case analysis) with associated P values are shown in **Table 2**. Results indicated that correlation coefficients between central adiposity measures ranged between 0.18 [WC and trunk fat% among white women] and 0.92 [WC vs trunk fat (in kg) among white men]; $P < 0.05$]. Moreover, CES-D was significantly associated with WHR only among both white and African American women within that sample ($r =$ 0.16 and 0.11, respectively; $P < 0.05$). Associations between SES, HEI, and CES-D varied across sex-ethnicity groups, with only whites having consistent significant and inverse SES–CES-D and HEI–CES-D associations as well as significant and positive SES-HEI associations. SES–central adiposity and HEI–

central adiposity associations were consistently inverse and significant only among white women.

Association of depressive symptoms with central adiposity and dietary quality within each sex-ethnic group

The results of a set of sex- and race-ethnicity-stratified multiple linear and logistic regression models with main exposure being depressive symptoms and main outcomes being central adiposity measures and dietary quality are shown in **Table 3**. For central adiposity indexes, among white women, a CES-D score ≥ 16 was positively associated with WC ($\beta = 4.17$, SE = 2.05, $P < 0.05$). The same pattern of association was found between a CES-D score ≥ 16 and trunk fat (kg) among white men ($\beta =$ 1.72, SE = 0.08, $P < 0.05$). This significant positive association was found between WHR and the continuous CES-D score and a CES-D score ≥ 16 among African American women. Moreover, in separate models with 2-factor and 3-factor interactions,

TABLE 2
Pearson's correlation coefficients between central adiposity indexes (continuous), the 2005 Healthy Eating Index (HEI), socioeconomic status (SES) factor scores (z scores), and Center for Epidemiologic Studies–Depression (CES-D) total score in the Healthy Aging in Neighborhoods of Diversity Across the Life Span (HANDLS) study ($n = 1208$)[1]

	WC	WHR	Trunk fat (kg)	Trunk fat (% of total body fat)	CES-D	HEI	SES
White men ($n = 198$)							
WC	1						
WHR	0.71[2]	1					
Trunk fat (kg)	0.92[2]	0.58[2]	1				
Trunk fat (% of total body fat)	0.33[2]	0.51[2]	0.33[2]	1			
CES-D	0.07	0.11	0.06	−0.02	1		
HEI	−0.05	−0.07	−0.02	−0.02	−0.26*	1	
SES	0.08	−0.02	0.11	0.08	−0.27[2]	0.29[2]	1
White women ($n = 272$)							
WC	1						
WHR	0.60[2]	1					
Trunk fat (kg)	0.87[2]	0.39[2]	1				
Trunk fat (% of total body fat)	0.18[2]	0.39[2]	0.28[2]	1			
CES-D	0.09	0.16[2]	0.03	0.06	1		
HEI	−0.28[2]	−0.21[2]	−0.22[2]	−0.11	−0.22[2]	1	
SES	−0.19[2]	−0.24[2]	−0.12[2]	−0.20[2]	−0.30[2]	0.33[2]	1
African American men ($n = 313$)							
WC	1						
WHR	0.80[2]	1					
Trunk fat (kg)	0.91[2]	0.68[2]	1				
Trunk fat (% of total body fat)	0.27[2]	0.34[2]	0.33	1			
CES-D	−0.08	−0.02	−0.06	−0.08	1		
HEI	0.16	0.07	0.14[2]	0.02	−0.03	1	
SES	0.09	0.05	0.08	0.07	−0.12[2]	0.08	1
African American women ($n = 425$)							
WC	1						
WHR	0.53[2]	1					
Trunk fat (kg)	0.89[2]	0.29[2]	1				
Trunk fat (% of total body fat)	0.26[2]	0.42[2]	0.30[2]	1			
CES-D	−0.01	0.11[2]	−0.04	0.03	1		
HEI	0.04	−0.02	0.07	−0.05	−0.13[2]	1	
SES	0.07	−0.04	0.11[2]	0.14[2]	−0.08	0.05	1

[1] WC, waist circumference; WHR, waist-to-hip ratio. *See* Subjects and Methods for definitions of WC, WHR, trunk fat (kg), trunk fat (% of total body fat), CES-D, HEI, and SES.

[2] $P < 0.05$ for null hypothesis that Pearson's correlation coefficient (r) = 0, on the basis of a complete case analysis. A pairwise correlation coefficient analysis yielded similar findings.

TABLE 3
Multiple ordinary least-squares linear and logistic regression models (Heckman selection) of the effect of depressive symptoms and depression on select adiposity measures and unhealthy eating in sample 2b and sample 3: moderation by sex and race-ethnicity among US adults aged 30–64 y in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study[1]

	Waist circumference (cm)		Central obesity[2]		Waist-to-hip ratio		Trunk fat (kg)[3]		Trunk fat (% of total body fat)[3]		HEI	
	β	SE	OR	95% CI	β	SE	β	SE	β	SE	β	SE
White men[4]												
Model 1, a–f: CES-D	0.28[5]	0.16	1.02	(0.99, 1.06)	0.0007	0.0005	0.08[5]	0.04	0.03	0.04	−0.25[6]	0.08
Model 2, a–f: CES-D ≥ 16	4.72	2.99	1.34	(0.72, 2.50)	0.0083	0.0102	1.72[6]	0.08	−0.26	0.85	−3.44[6]	1.62
Model 3, a–f: CES-D ≥ 20	4.17	3.66	1.19	(0.56, 2.55)	0.0127	0.0125	0.98	1.01	0.57	1.06	−2.82	1.99
White women												
Model 1, a–f: CES-D	0.18	0.11	1.02	(0.99, 1.05)	0.0004	0.0005	−0.02	0.04	−0.01	0.03	−0.19[6]	0.07
Model 2, a–f: CES-D ≥ 16	4.17[6]	2.05	1.63[5]	(0.98, 2.73)	0.0089	0.0089	−0.11	0.72	−0.00	0.64	−3.45[6]	1.26
Model 3, a–f: CES-D ≥ 20	−0.22	2.39	1.19	(0.66, 2.15)	−0.0073	0.0103	−1.49[5]	0.84	−0.52	0.74	−3.93[6]	1.46
African American men												
Model 1, a–f: CES-D	0.01	0.11	1.00	(0.97, 1.04)	0.0004	0.0005	−0.02	0.03	−0.03	0.04	−0.03	0.07
Model 2, a–f: CES-D ≥ 16	0.78	1.78	1.35	(0.81, 2.26)	0.0064	0.0079	0.28	0.56	−0.27	0.59	−0.08	1.22
Model 3, a–f: CES-D ≥ 20	0.87	2.25	1.58	(0.84, 2.97)	0.0082	0.0099	0.11	0.72	−0.64	0.75	−0.90	1.52
African American women												
Model 1, a–f: CES-D	0.08	0.10	1.00	(0.98, 1.02)	0.0090[6]	0.00042	0.02	0.03	0.06[5]	0.03	−0.10[5]	0.06
Model 2, a–f: CES-D ≥ 16	0.74	1.80	1.04	(0.69, 1.56)	0.0155[6]	0.00775	0.29	0.61	0.79	0.54	−1.24	1.04
Model 3, a–f: CES-D ≥ 20	−0.82	2.08	0.83	(0.52, 1.32)	0.0044	0.00897	0.10	0.71	0.34	0.62	−1.22	1.20

[1] Sample 2b included subjects with complete data for both 24-h dietary recalls, Center for Epidemiologic Studies–Depression (CES-D) scores, and anthropometric measures (n = 1789; 43% men), whereas sample 3 included subjects with complete data for both 24-h dietary recalls, CES-D scores, and dual-energy X-ray absorptiometry scan measures (n = 1583; 43% men). Heckman selection models were used to adjust for potential selection bias due to missing data (*see* Subjects and Methods for more details). HEI, Healthy Eating Index; OR, odds ratio.

[2] Defined as a waist circumference ≥102 cm (40 in) for men or ≥88 cm (35 in) for women.

[3] Analyses with these variables as outcomes used subjects from sample 3 (*see* Subjects and Methods for more details).

[4] Each model was adjusted for age group (5-y categories), poverty status, educational attainment, marital status, and smoking status. Ordinary least-squares regression models were used for continuous central adiposity outcomes, whereas logistic regression models were used when the outcome was binary (ie, central obesity defined by waist circumference cutoffs).

[5,6] For a null hypothesis that β = 0 in the multiple linear or logistic regression model (Wald test): [5] $P < 0.10$, [6] $P < 0.05$.

significant effect modification of the CES-D–central adiposity association by sex and race-ethnicity was observed for most central adiposity measures.

In general, depressed white women had a lower HEI score (poorer quality diet) than did their nondepressed counterparts, especially in those with a CES-D ≥ 20 (β = −3.93, SE = 1.46, $P < 0.05$). Among white women, this significant inverse association with CES-D score pertained to components 3 (increased total vegetables intake), 4 (increased dark-green and orange vegetables and legume intake), 5 (increased total grain intake), and 12 (reduced discretionary calories) of the HEI, whereas among African American women, only component 8 (increased intakes of meats and beans) was inversely related to CES-D. Among white men, this inverse association with CES-D was observed for components 1 (increase in total fruit intake), 2 (increase in whole fruit intake), and 12 (reduced discretionary calories intake), whereas among African American men this was true only for component 4 (increased dark-green and orange vegetables and legume intake) (*see* Appendixes A and B under "Supplemental data" in the online issue).

Socioeconomic differences in central adiposity and dietary quality within each sex-ethnicity group

Socioeconomic disparities in adiposity and dietary quality are presented in **Table 4,** which shows findings from various mul-

tiple linear and logistic models. The SES factor score (which combines effects of education and PIR) was positively associated with WC among African American men ($P < 0.05$, Wald test) but not among African American women ($P > 0.05$). The SES factor score was inversely related to this central adiposity measure among white women. In fact, a 1-SD increase in SES was associated with a 25% reduced risk of central obesity among white women (odds ratio: 0.75; 95% CI: 0.59, 0.94), whereas the same increase in SES was associated with an increased risk in central obesity of 30% and 36% on average among African American men and women, respectively. Similarly, the SES in white women was inversely associated with WHR, trunk fat (in kg), and trunk fat% ($P < 0.05$), whereas among African American women, SES was positively associated with trunk fat%. For both men and women, SES was associated with an improved dietary quality, with significant relations observed only among whites.

In separate models in which interaction terms between SES, sex, and ethnic group were added in addition to the main effects, there was a significant effect modification of the SES-Adip$_{cent}$ and SES-HEI associations by both sex and race-ethnicity (data not shown). The components of HEI that were positively associated with SES were components 2 (increase whole fruit intake) and 3 (increased total vegetables intake) among men and components 1 through 4 (increased total fruit, whole fruit, total vegetables, dark-green and orange vegetables, and legumes), 6

TABLE 4
Multiple ordinary least-squares linear and logistic regression models (Heckman selection) for the effect of socioeconomic status factor score on select adiposity measures and dietary quality in sample 2b and sample 3: moderation by sex and race-ethnicity among US adults aged 30–64 y in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study[1]

	Waist circumference (cm)		Central obesity[2]		Waist-to-hip ratio		Trunk fat (kg)[3]		Trunk fat (% of total body fat)[3]		HEI	
	β	SE	OR	95% CI	β	SE	β	SE	β	SE	β	SE
White men[4]	1.70	1.23	1.06	(0.82, 1.38)	0.0004	0.0043	0.38	0.33	0.51	0.35	3.06[5]	0.68
White women	−3.38[5]	1.01	0.75[5]	(0.59, 0.94)	−0.0160[5]	0.0043	−0.71[5]	0.33	−0.66[5]	0.29	3.04[5]	0.610
African American men	2.00[5]	0.95	1.30[5]	(1.01, 1.67)	0.00662[6]	0.00376	0.24	0.29	0.18	0.30	0.58	0.62
African American women	1.02	1.02	1.36[5]	(1.07, 1.72)	−0.0007	0.0044	0.61[6]	0.33	0.99[5]	0.29	0.91	0.59

[1] Sample 2b included subjects with complete data for both 24-h dietary recalls, Center for Epidemiologic Studies–Depression (CES-D) scores, and anthropometric measures (*n* = 1325), whereas sample 3 included subjects with complete data for both 24-h dietary recalls, CES-D scores, and dual-energy X-ray absorptiometry scan measures (*n* = 1227). Missing data for the socioeconomic status factor score (education in particular) accounted for the smaller sample size in this analysis than that in Table 2. HEI, Healthy Eating Index; OR, odds ratio.

[2] Defined as a waist circumference ≥102 cm (40 in) for men or ≥88 cm (35 in) for women.

[3] Analyses with these variables as outcomes used only subjects from sample 3 (*see* Subjects and Methods for more details).

[4] Each model was adjusted for age group (5-y categories), marital status, and smoking status. Ordinary least-squares regression models were used for continuous central adiposity outcomes, whereas logistic regression models were used when the outcome was binary (ie, central obesity defined by waist circumference cutoffs).

[5,6] For a null hypothesis that β = 0 in the multiple linear or logistic regression model (Wald test): [5]$P < 0.05$, [6]$P < 0.10$.

(increased whole-grain intake), and 12 (reduced discretionary calories) among women with a number of significant sex-ethnicity interactions in the strength of the SES-HEI components associations, which indicate a weaker association among African American women but a stronger association among women overall (data not shown; *see* Appendix A under "Supplemental data" in the online issue for details on HEI-2005).

Findings from the structural equation model: pathways across sex-ethnicity groups

An evaluation of a theoretical structural equation model, with examination of pathways across sex-ethnicity groups, is shown in **Figure 1**. The findings of the structural equations model with the final outcome being WC are shown in Figure 1A. The results differed between sex-ethnicity groups. Among white men, SES was inversely related to CES-D scores, which in turn were inversely associated with HEI and positively associated with WC. Among this group, however, HEI had no significant association, whereas SES had a borderline significant direct positive association with WC. CES-D explained 11.5% of the total effect of SES on HEI. Among white women, in contrast, SES had an inverse borderline significant direct association with WC ($P <$ 0.05; *t* test 1 df) and an inverse association with CES-D score that, in turn, was inversely related to HEI. HEI was also inversely related to WC. Mediation of the total effect of SES on HEI through CES-D within this sex-ethnicity category (ie, white women) was also significant (MP = 15.5%), which indicated that ≈15% of the total SES effect on HEI was explained by CES-D. Moreover, given that, in this group, both direct and indirect effects between SES and WC had the same direction of association, a meaningful MP was possible to compute for alternative pathways. Taking 2 alternative pathways separately with CES-D as mediator (SES→CES-D→WC and SES→CES-D→HEI→WC) and assessing their contribution to the total effect of SES on WC, we found that their MPs were 15.2% and

4.5%, respectively. The pathway that involved only HEI (ie, SES→HEI→WC) contributed to 24.8% of the total SES-WC effect. Among African American men, however, SES and CES-D were not associated with HEI, although the SES–CES-D inverse association was statistically significant. Moreover, SES was positively associated with WC in this group (unlike among white women). Among African American women, none of the main associations were statistically significant. Many of the path coefficients were significantly different between groups based on multigroup analysis findings.

In contrast with WC, a higher WHR (Figure 1B) among African American women was associated with a higher CES-D score ($P <$ 0.05), independently of SES. However, multigroup analysis did not indicate significant differences in that path coefficient when compared with the other 3 groups. More importantly, the total effect of SES on WHR among white women was appreciably mediated by pathways involving CES-D (MP = 11.6%) and HEI (MP = 12.3%), although the pathway involving both mediating factors yielded a nonsignificant MP (MP = 2.2%).

Both white men and African American women had a positive direct association between SES and trunk fat% ($P <$ 0.05; Figure 1C), whereas an inverse association between SES and trunk fat % was found among white women. None of the CES-D–trunk fat% or HEI-trunk fat% paths were significant for any of the sex-ethnicity groups. Constraining path coefficients to be equal indicated that there was significant heterogeneity in the associations between sex-ethnicity groups.

DISCUSSION

This was the first study, to our knowledge, to examine a comprehensive mechanism by which SES differences in adiposity may be mediated by depressive symptoms and unhealthy eating behavior by using data on adults with DXA measurements of body fat among others. There were several key findings. First, elevated CES-D scores were associated with a poorer-quality diet
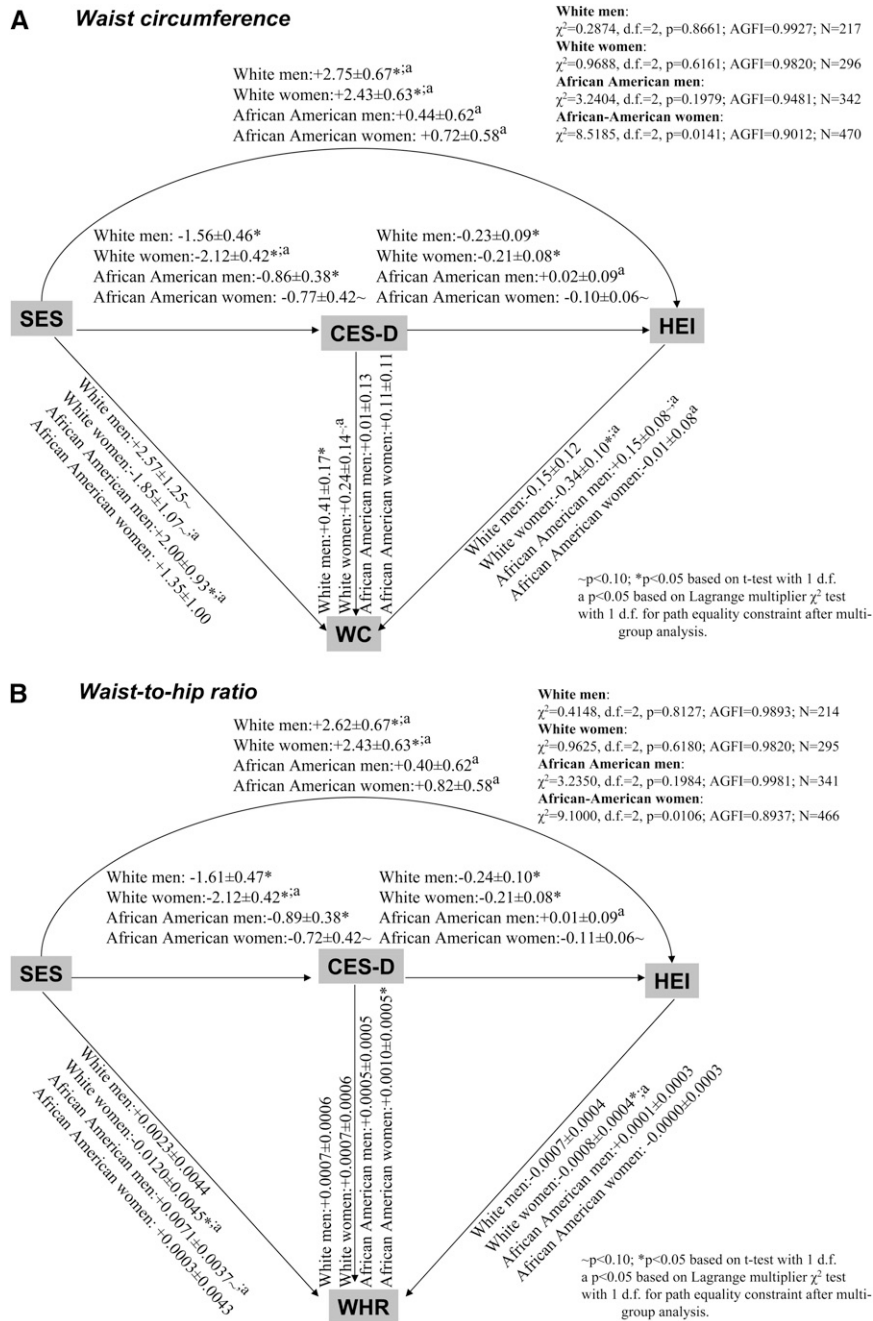
A   Waist circumference

White men:+2.75±0.67*;a
White women:+2.43±0.63*;a
African American men:+0.44±0.62a
African American women: +0.72±0.58a

White men:
χ^2=0.2874, d.f.=2, p=0.8661; AGFI=0.9927; N=217
White women:
χ^2=0.9688, d.f.=2, p=0.6161; AGFI=0.9820; N=296
African American men:
χ^2=3.2404, d.f.=2, p=0.1979; AGFI=0.9481; N=342
African-American women:
χ^2=8.5185, d.f.=2, p=0.0141; AGFI=0.9012; N=470

White men: -1.56±0.46*
White women:-2.12±0.42*;a
African American men:-0.86±0.38*
African American women: -0.77±0.42~

White men:-0.23±0.09*
White women:-0.21±0.08*
African American men:+0.02±0.09a
African American women: -0.10±0.06~

SES → CES-D → HEI

White men:+2.57±1.25~
White women:-1.85±1.07~;a
African American men:+2.00±0.93*;a
African American women: +1.35±1.00

White men:+0.41±0.17*
White women:+0.24±0.14~;a
African American men:+0.01±0.13
African American women:+0.11±0.11

White men:-0.15±0.12
White women:-0.34±0.10*;a
African American men:+0.15±0.08~;a
African American women:-0.01±0.08a

WC

~p<0.10; *p<0.05 based on t-test with 1 d.f.
a p<0.05 based on Lagrange multiplier χ^2 test
with 1 d.f. for path equality constraint after multi-
group analysis.

B   Waist-to-hip ratio

White men:+2.62±0.67*;a
White women:+2.43±0.63*;a
African American men:+0.40±0.62a
African American women:+0.82±0.58a

White men:
χ^2=0.4148, d.f.=2, p=0.8127; AGFI=0.9893; N=214
White women:
χ^2=0.9625, d.f.=2, p=0.6180; AGFI=0.9820; N=295
African American men:
χ^2=3.2350, d.f.=2, p=0.1984; AGFI=0.9981; N=341
African-American women:
χ^2=9.1000, d.f.=2, p=0.0106; AGFI=0.8937; N=466

White men: -1.61±0.47*
White women:-2.12±0.42*;a
African American men:-0.89±0.38*
African American women:-0.72±0.42~

White men:-0.24±0.10*
White women:-0.21±0.08*
African American men:+0.01±0.09a
African American women:-0.11±0.06~

SES → CES-D → HEI

White men:+0.0023±0.0044
White women:-0.0120±0.0045*;a
African American men:+0.0071±0.0037~;a
African American women:+0.0003±0.0043

White men:+0.0007±0.0006
White women:+0.0007±0.0006
African American men:+0.0005±0.0005
African American women:+0.0010±0.0005*

White men:-0.0007±0.0004
White women:-0.0008±0.0004*;a
African American men:+0.0001±0.0003
African American women:-0.0000±0.0003

WHR

~p<0.10; *p<0.05 based on t-test with 1 d.f.
a p<0.05 based on Lagrange multiplier χ^2 test
with 1 d.f. for path equality constraint after multi-
group analysis.

FIGURE 1. (Continued)

in most sex-ethnicity groups, except for African American men. Second, elevated CES-D scores (≥ 16) were associated with higher WC among white women, higher WHR among African American women, and higher trunk fat (kg) among white men. Third, the association of SES with central adiposity was positive among African American men (for WC and central obesity) and African American women (for central obesity and trunk fat%), whereas SES was inversely related to all central adiposity measures among white women. Fourth, the total positive association of SES with dietary quality was significantly mediated by depressive symptoms among whites but not among African Americans. Similarly, the total effect of SES on WC was significantly explained by pathways involving depressive symptoms and HEI only among white women. The same was also

found for the SES-WHR association among that sex-ethnicity group. Whereas WC, WHR, trunk fat (in kg), and trunk fat% are indeed alternative measures of central adiposity, their correlation with each others varied between 0.18 and 0.91, depending on the pair being compared and the sex-ethnicity group. This in part explains differential findings in terms of their associations with CES-D, SES, and HEI.

In most previous studies, the association between adiposity and depression was positive and stronger among women (14, 18, 19, 21, 25, 26, 31, 34, 35, 39, 41, 43), whereas in other studies there were no distinctive sex differences in the association (15, 20, 22, 23, 26–29, 33, 38, 40, 44). Central adiposity measured by non-DXA methods was particularly found to be associated with depression in a small number of studies (68–70).
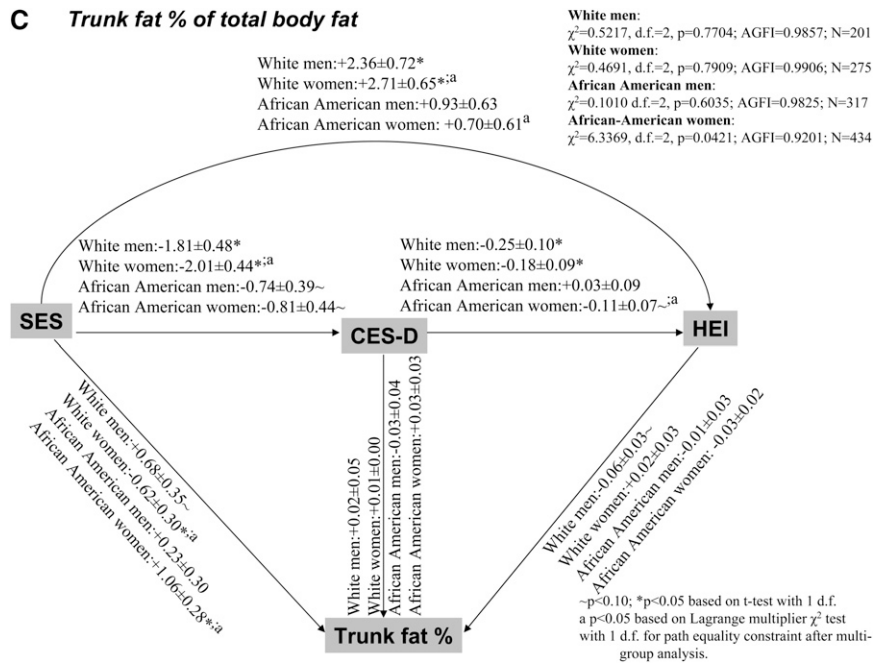
FIGURE 1. Findings from the use of structural equation models: mechanisms explaining socioeconomic status (SES) disparities in central adiposity through Center for Epidemiologic Studies–Depression (CES-D) score and the Healthy Eating Index (HEI) with waist circumference (WC; A), waist-to-hip ratio (WHR; B), and trunk fat as a percentage of body fat (C) as final outcomes. Age groups, marital status, and smoking status were treated as exogenous variables, which affected all other endogenous variables. One of the exogenous variables was dropped from each model with SES, CES-D, HEI, or central adiposity as outcomes depending on their significance to gain 1 df. Numbers on the arrows represent path coefficients ± SE. Fit statistics and sample sizes are presented for each stratum-specific model. Multigroup analysis was conducted to assess significant differences between groups for each path coefficient.

In terms of age, sex, and racial moderation of the association, one study found that depressive symptoms during adolescence were predictive of an elevated BMI in early adulthood for both black and white girls (32), whereas another found that young obese Hispanic women were at highest risk of depressive symptoms than were other groups. This study also suggested that older subjects did not present a clear association between depression and overweight or obesity (25). In a third study, extreme obesity was associated with depression across all sex-racial groups, even after potential confounding factors were controlled for (22). In a few studies, there was an inverse relation between depression and adiposity among men (16, 21, 27, 39), among postmenopausal or older women (71, 72), or among both sexes combined (73), which supports the "jolly fat hypothesis." For example, a recent cross-sectional study conducted in a large sample of Chinese elderly subjects concluded that obesity in old age is associated with a lower prevalence of depressive symptoms than is normal weight (73). In addition, unhealthy eating or poor dietary quality has been shown to be associated with depression (74–78; MF Kuczmarski et al, unpublished data, 2009) and obesity (5–8).

Several biological mechanisms have been suggested to explain the association between obesity and depression. Leptin resistance may contribute to alterations in affective status. Leptin resistance could occur at several levels, including impaired transport of leptin across the blood-brain barrier, reduced function of the leptin receptor, and defects in leptin signal transduction (79, 80). This would give rise to a causal pathway in which depression is directly caused by leptin resistance, which in turn alters appetite and possibly reduces dietary quality and in turn increases the risk

of obesity. Another possible mechanism is hypercortisolemia, which is associated with stress and depression and in turn was shown to be associated with greater fat deposits, particularly in the abdominal region, and with the metabolic syndrome (81–83). Finally, depressed subjects are often prescribed antidepressant medication, which enhances appetite (84). This might explain at least part of the associations found in our study.

Moreover, our findings indicate that the CES-D score, which is inversely related to both SES and HEI, is implicated in the positive association between those 2 variables, respectively, among white men and women, whereas both the CES-D score and HEI are implicated in mediating the SES-WC and SES-WHR associations among white women. In contrast, the positive relation of CES-D with WHR among African American women was independent of SES and was not mediated by HEI. Physiologically speaking, sweet fatty foods low in protein have been suggested to alleviate stress in vulnerable people via enhanced function of the serotonergic system (74). This mechanism may be at play more among whites than African Americans. Among African American women, stress may act on abdominal fat deposition (eg, WHR findings) independently of food intake, which suggests the possibility that hypercortisolemia is a pathway, among others (81–83).

In addition, our finding of a positive total effect of SES on central obesity (Table 3) among African American men and women may be attributed to differential ideal body image and body dissatisfaction between these 2 ethnic groups, as shown in previous studies (85), with a gap in values that may become more apparent with increased wealth. Moreover, the finding of a positive direct effect of SES on central adiposity (eg, WC and trunk

fat%) among white men in particular, independently of the pathways through depressive symptoms and dietary quality, may be indicative of reduced physical activity with increased wealth among this sex-ethnicity group. Finally, it is worth noting that a major part of the total SES-HEI positive association observed among whites was not explained by the CES-D score. Some of the factors explaining the direct SES-HEI association may include food insecurity (86–89), low access to a healthy food environment in poor neighborhoods (90–93), concerns about and actual food prices (64, 94, 95), as well as knowledge about healthy dietary habits (64, 95, 96).

Despite its many strengths, which include the use of DXA measures and structural equation modeling, our study had a few limitations. First, the cross-sectional nature of the data may have precluded causality. Whereas we did not study the effect of central adiposity on depression (ie, the other direction), comparison of these 2 competing pathways will only be possible with longitudinal data. Second, the HANDLS study lacked a reliable measure for physical activity, which might be involved in the residual or direct effect of SES on depression and obesity. Third, dietary intake was self-reported and based on two 24-h dietary recalls, which may lead to both random and systematic errors in the assessment. Whereas random errors with respect to outcomes (eg, depression and obesity measures) may bias the effect toward the null, systematic errors can produce bias in either direction.

In conclusion, our study suggests major sex and ethnic differences in the pathways linking SES, depressive symptoms, and lifestyle factors to central adiposity measures. Hence, future interventions related to mental health targeted at reducing SES disparities in dietary quality and central adiposity may potentially have different effects across sex-ethnicity groups. In particular, whereas in some groups and for particular adiposity measures (eg, African American women and WHR), unhealthy eating may not be the factor responsible for the association between depression and central adiposity, depressive symptoms and unhealthy eating may both contribute to SES disparities in central adiposity for other groups and adiposity measures (eg, white women, WC, and WHR).

REFERENCES

1. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. JAMA 2006;295:1549–55.
2. Whitlock G, Lewington S, Mhurchu CN. Coronary heart disease and body mass index: a systematic review of the evidence from larger prospective cohort studies. Semin Vasc Med 2002;2:369–81.
3. Wolin KY, Colditz GA. Can weight loss prevent cancer? Br J Cancer 2008;99:995–9.
4. Ferro-Luzzi A, Martino L. Obesity and physical activity. Ciba Found Symp 1996;201:207–21; discussion 221–7.
5. Maskarinec G, Novotny R, Tasaki K. Dietary patterns are associated with body mass index in multiethnic women. J Nutr 2000;130:3068–72.
6. Murtaugh MA, Herrick JS, Sweeney C, et al. Diet composition and risk of overweight and obesity in women living in the southwestern United States. J Am Diet Assoc 2007;107:1311–21.
7. Panagiotakos DB, Chrysohoou C, Pitsavos C, Stefanadis C. Association between the prevalence of obesity and adherence to the Mediterranean diet: the ATTICA study. Nutrition 2006;22:449–56.
8. Sichieri R. Dietary patterns and their associations with obesity in the Brazilian city of Rio de Janeiro. Obes Res 2002;10:42–8.
9. Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. Epidemiol Rev 2007;29:6–28.
10. National Institutes of Health, National Heart, Lung, and Blood Institute, North American Association for the Study of Obesity. The practical guide: identification, evaluation, and treatment of overweight and obesity in adults. Bethesda, MD: NIH, 2000.
11. Zhu S, Heymsfield SB, Toyoshima H, Wang Z, Pietrobelli A, Heshka S. Race-ethnicity-specific waist circumference cutoffs for identifying cardiovascular disease risk factors. Am J Clin Nutr 2005;81:409–15.
12. Wang Y, Rimm EB, Stampfer MJ, Willett WC, Hu FB. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. Am J Clin Nutr 2005;81:555–63.
13. Ford ES, Mokdad AH, Giles WH. Trends in waist circumference among U.S. adults. Obes Res 2003;11:1223–31.
14. Istvan J, Zavela K, Weidner G. Body weight and psychological distress in NHANES I. Int J Obes Relat Metab Disord 1992;16:999–1003.
15. Ross CE. Overweight and depression. J Health Soc Behav 1994;35:63–79.
16. Palinkas LA, Wingard DL, Barrett-Connor E. Depressive symptoms in overweight and obese older adults: a test of the "jolly fat" hypothesis. J Psychosom Res 1996;40:59–66.
17. Roberts RE, Kaplan GA, Shema SJ, Strawbridge WJ. Are the obese at greater risk for depression? Am J Epidemiol 2000;152:163–70.
18. Jorm AF, Korten AE, Christensen H, Jacomb PA, Rodgers B, Parslow RA. Association of obesity with anxiety, depression and emotional well-being: a community survey. Aust N Z J Public Health 2003;27:434–40.
19. Onyike CU, Crum RM, Lee HB, Lyketsos CG, Eaton WW. Is obesity associated with major depression? Results from the Third National Health and Nutrition Examination Survey. Am J Epidemiol 2003;158:1139–47.
20. Roberts RE, Deleger S, Strawbridge WJ, Kaplan GA. Prospective association between obesity and depression: evidence from the Alameda County Study. Int J Obes Relat Metab Disord 2003;27:514–21.
21. Stunkard AJ, Faith MS, Allison KC. Depression and obesity. Biol Psychiatry 2003;54:330–7.
22. Dong C, Sanchez LE, Price RA. Relationship of obesity to depression: a family-based study. Int J Obes Relat Metab Disord 2004;28:790–5.
23. Johnston E, Johnson S, McLeod P, Johnston M. The relation of body mass index to depressive symptoms. Can J Public Health 2004;95:179–83.
24. Gil K, Radzillowicz P, Zdrojewski T, et al. Relationship between the prevalence of depressive symptoms and metabolic syndrome. Results of the SOPKARD Project. Kardiol Pol 2006;64:464–9.
25. Heo M, Pietrobelli A, Fontaine KR, Sirey JA, Faith MS. Depressive mood and obesity in US adults: comparison and moderation by sex, age, and race. Int J Obes (Lond) 2006;30:513–9.
26. Herva A, Laitinen J, Miettunen J, et al. Obesity and depression: results from the longitudinal Northern Finland 1966 Birth Cohort Study. Int J Obes (Lond) 2006;30:520–7.
27. Kress AM, Peterson MR, Hartzell MC. Association between obesity and depressive symptoms among U.S. Military active duty service personnel, 2002. J Psychosom Res 2006;60:263–71.
28. Sachs-Ericsson N, Burns AB, Gordon KH, et al. Body mass index and depressive symptoms in older adults: the moderating roles of race, sex, and socioeconomic status. Am J Geriatr Psychiatry 2007;15:815–25.
29. Bjerkeset O, Romundstad P, Evans J, Gunnell D. Association of adult body mass index and height with anxiety, depression, and suicide in the general population: the HUNT study. Am J Epidemiol 2008;167:193–202.

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30. Miller GE, Freedland KE, Carney RM, Stetler CA, Banks WA. Pathways linking depression, adiposity, and inflammatory markers in healthy young adults. Brain Behav Immun 2003;17:276–85.

31. Richardson LP, Davis R, Poulton R, et al. A longitudinal evaluation of adolescent depression and adult obesity. Arch Pediatr Adolesc Med 2003;157:739–45.

32. Franko DL, Striegel-Moore RH, Thompson D, Schreiber GB, Daniels SR. Does adolescent depression predict obesity in black and white young adult women? Psychol Med 2005;35:1505–13.

33. Hasler G, Pine DS, Kleinbaum DG, et al. Depressive symptoms during childhood and adult obesity: the Zurich Cohort Study. Mol Psychiatry 2005;10:842–50.

34. Anderson SE, Cohen P, Naumova EN, Must A. Association of depression and anxiety disorders with weight change in a prospective community-based study of children followed up into adulthood. Arch Pediatr Adolesc Med 2006;160:285–91.

35. McIntyre RS, Konarski JZ, Wilkins K, Soczynska JK, Kennedy SH. Obesity in bipolar disorder and major depressive disorder: results from a national community health survey on mental health and well-being. Can J Psychiatry 2006;51:274–80.

36. Raikkonen K, Matthews KA, Kuller LH. Depressive symptoms and stressful life events predict metabolic syndrome among middle-aged women: a comparison of World Health Organization, Adult Treatment Panel III, and International Diabetes Foundation definitions. Diabetes Care 2007;30:872–7.

37. Simon GE, Ludman EJ, Linde JA, et al. Association between obesity and depression in middle-aged women. Gen Hosp Psychiatry 2008;30:32–9.

38. Strine TW, Mokdad AH, Dube SR, et al. The association of depression and anxiety with obesity and unhealthy behaviors among community-dwelling US adults. Gen Hosp Psychiatry 2008;30:127–37.

39. Carpenter KM, Hasin DS, Allison DB, Faith MS. Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: results from a general population study. Am J Public Health 2000;90:251–7.

40. Simon GE, Von Korff M, Saunders K, et al. Association between obesity and psychiatric disorders in the US adult population. Arch Gen Psychiatry 2006;63:824–30.

41. Dragan A, Akhtar-Danesh N. Relation between body mass index and depression: a structural equation modeling approach. BMC Med Res Methodol 2007;7:17.

42. Siegel JM, Yancey AK, McCarthy WJ. Overweight and depressive symptoms among African-American women. Prev Med 2000;31:232–40.

43. Blazer DG, Moody-Ayers S, Craft-Morgan J, Burchett B. Depression in diabetes and obesity: racial/ethnic/gender issues in older adults. J Psychosom Res 2002;53:913–6.

44. Scott KM, McGee MA, Wells JE, Oakley Browne MA. Obesity and mental disorders in the adult general population. J Psychosom Res 2008;64:97–105.

45. Lorant V, Deliege D, Eaton W, Robert A, Philippot P, Ansseau M. Socioeconomic inequalities in depression: a meta-analysis. Am J Epidemiol 2003;157:98–112.

46. Colles SL, Dixon JB, O'Brien PE. Loss of control is central to psychological disturbance associated with binge eating disorder. Obesity (Silver Spring) 2008;16:608–14.

47. Okosun IS, Choi ST, Boltri JM, et al. Trends of abdominal adiposity in white, black, and Mexican-American adults, 1988 to 2000. Obes Res 2003;11:1010–7.

48. Gupta R, Rastogi P, Sarna M, Gupta VP, Sharma SK, Kothari K. Body-mass index, waist-size, waist-hip ratio and cardiovascular risk factors in urban subjcts. J Assoc Physicians India 2007;55:621–7.

49. Al-Lawati JA, Jousilahti P. Body mass index, waist circumference and waist-to-hip ratio cut-off points for categorisation of obesity among Omani Arabs. Public Health Nutr 2008;11:102–8.

50. De Nicola L, Conte G. Waist:hip ratio is a better predictor of cardiovascular risk than BMI in patients with moderate CKD. Nat Clin Pract Nephrol 2008;4:592–3.

51. Hong YJ, Park HS, Lee ES, Suh YJ. Anthropometric analysis of waist-to-hip ratio in Asian women. Aesthetic Plast Surg 2009;33:185–90.

52. Shahraki M, Shahraki T, Ansari H. The effects of socio-economic status on BMI, waist:hip ratio and waist circumference in a group of Iranian women. Public Health Nutr 2008;11:757–61.

53. Lezak M, Lezak M, eds. Neuropsychological assessment, 4th ed. New York, NY: Oxford University Press, 2004.

54. Radloff L. The CES-D scale: a self-report depression scale for research in the general population. Appl Psychol Meas 1977;1:385–401.

55. Nguyen HT, Kitner-Triolo M, Evans MK, Zonderman AB. Factorial invariance of the CES-D in low socioeconomic status African Americans compared with a nationally representative sample. Psychiatry Res 2004;126:177–87.

56. Conway JM, Ingwersen LA, Moshfegh AJ. Accuracy of dietary recall using the USDA five-step multiple-pass method in men: an observational validation study. J Am Diet Assoc 2004;104:595–603.

57. Moshfegh AJ, Rhodes DG, Baer DJ, et al. The US Department of Agriculture Automated Multiple-Pass Method reduces bias in the collection of energy intakes. Am J Clin Nutr 2008;88:324–32.

58. Guenther PM, Reedy J, Krebs-Smith SM. Development of the Healthy Eating Index—2005. J Am Diet Assoc 2008;108:1896–901.

59. McCullough ML, Feskanich D, Rimm EB, et al. Adherence to the Dietary Guidelines for Americans and risk of major chronic disease in men. Am J Clin Nutr 2000;72:1223–31.

60. STATA. Statistics/data analysis: release 9. 9.0 ed. College Station, TX: Stata Corporation, 2005.

61. SAS Institute Inc. SAS/STAT 9.2 ed. Cary, NC: SAS Institute Inc, 2009.

62. Hu L, Bentler PM. Cutoff criteria in fix indexes in covariance structure analysis: conventional criteria versus new alternatives. Struct Equ Modeling 1999;6:1–55.

63. Ditlevsen S, Christensen U, Lynch J, Damsgaard MT, Keiding N. The mediation proportion: a structural equation approach for estimating the proportion of exposure effect on outcome explained by an intermediate variable. Epidemiology 2005;16:114–20.

64. Beydoun MA, Wang Y. How do socio-economic status, perceived economic barriers and nutritional benefits affect quality of dietary intake among US adults? Eur J Clin Nutr 2008;62:303–13.

65. Byrne B. Structural equation modeling with eqs: basic concepts, applications, and programming (multivariate applications). Mahwah, NJ: Lawrence Erlbaum Associates, 2006.

66. Hatcher L. A step-by-step approach to using the SAS system for factor analysis ad structural equation modeling. Cary, NC: SAS Institute, 1994.

67. Heckman JJ. Sample selection bias as a specification error. Econometrica 1979;47:153–61.

68. Lee ES, Kim YH, Beck SH, Lee S, Oh SW. Depressive mood and abdominal fat distribution in overweight premenopausal women. Obes Res 2005;13:320–5.

69. Rosmond R, Lapidus L, Marin P, Bjorntorp P. Mental distress, obesity and body fat distribution in middle-aged men. Obes Res 1996;4:245–52.

70. Raikkonen K, Matthews KA, Kuller LH. Anthropometric and psychosocial determinants of visceral obesity in healthy postmenopausal women. Int J Obes Relat Metab Disord 1999;23:775–82.

71. Jasienska G, Ziomkiewicz A, Gorkiewicz M, Pajak A. Body mass, depressive symptoms and menopausal status: an examination of the "jolly fat" hypothesis. Womens Health Issues 2005;15:145–51.

72. Kuriyama S, Koizumi Y, Matsuda-Ohmori K, et al. Obesity and depressive symptoms in elderly Japanese: the Tsurugaya Project. J Psychosom Res 2006;60:229–35.

73. Li ZB, Ho SY, Chan WM, et al. Obesity and depressive symptoms in Chinese elderly. Int J Geriatr Psychiatry 2004;19:68–74.

74. Gibson EL. Emotional influences on food choice: sensory, physiological and psychological pathways. Physiol Behav 2006;89:53–61.

75. Hibbeln JR. Depression, suicide and deficiencies of omega-3 essential fatty acids in modern diets. World Rev Nutr Diet 2009;99:17–30.

76. Kamphuis MH, Geerlings MI, Grobbee DE, Kromhout D. Dietary intake of B(6-9-12) vitamins, serum homocysteine levels and their association with depressive symptoms: the Zutphen Elderly Study. Eur J Clin Nutr 2008;62:939–45.

77. Kimura Y, Wada T, Ishine M, et al. Food diversity is closely associated with activities of daily living, depression, and quality of life in community-dwelling elderly people. J Am Geriatr Soc 2009;57:922–4.

78. Oishi J, Doi H, Kawakami N. Nutrition and depressive symptoms in community-dwelling elderly persons in Japan. Acta Med Okayama 2009;63:9–17.

79. Munzberg H, Myers MG Jr. Molecular and anatomical determinants of central leptin resistance. Nat Neurosci 2005;8:566–70.

80. Lu XY. The leptin hypothesis of depression: a potential link between mood disorders and obesity? Curr Opin Pharmacol 2007;7:648–52.

81. Vogelzangs N, Suthers K, Ferrucci L, et al. Hypercortisolemic depression is associated with the metabolic syndrome in late-life. Psychoneuroendocrinology 2007;32:151–9.

82. Weber-Hamann B, Hentschel F, Kniest A, et al. Hypercortisolemic depression is associated with increased intra-abdominal fat. Psychosom Med 2002;64:274–7.

83. Young AH. Cortisol in mood disorders. Stress 2004;7:205–8.

84. Fava M, Rush AJ, Thase ME, et al. 15 years of clinical experience with bupropion HCl: from bupropion to bupropion SR to bupropion XL. Prim Care Companion J Clin Psychiatry 2005;7:106–13.

85. Wildes JE, Emery RE, Simons AD. The roles of ethnicity and culture in the development of eating disturbance and body dissatisfaction: a meta-analytic review. Clin Psychol Rev 2001;21:521–51.

86. Hadley C, Zodhiates A, Sellen DW. Acculturation, economics and food insecurity among refugees resettled in the USA: a case study of West African refugees. Public Health Nutr 2007;10:405–12.

87. Ruel MT. Is dietary diversity an indicator of food security or dietary quality? A review of measurement issues and research needs. Food Nutr Bull 2003;24:231–2.

88. Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. Am J Clin Nutr 2004;79:6–16.

89. Monsivais P, Drewnowski A. The rising cost of low-energy-density foods. J Am Diet Assoc 2007;107:2071–6.

90. Cheadle A, Psaty BM, Curry S, et al. Community-level comparisons between the grocery store environment and individual dietary practices. Prev Med 1991;20:250–61.

91. Murakami K, Sasaki S, Takahashi Y, Uenishi K. Neighborhood food store availability in relation to food intake in young Japanese women. Nutrition 2009;25:640–6.

92. Zenk SN, Schulz AJ, Israel BA, James SA, Bao S, Wilson ML. Fruit and vegetable access differs by community racial composition and socio-economic position in Detroit, Michigan. Ethn Dis 2006;16:275–80.

93. Black JL, Macinko J. Neighborhoods and obesity. Nutr Rev 2008;66:2–20.

94. Beydoun MA, Powell LM, Wang Y. The association of fast food, fruit and vegetable prices with dietary intakes among US adults: is there modification by family income? Soc Sci Med 2008;66:2218–29.

95. Turrell G, Kavanagh AM. Socio-economic pathways to diet: modelling the association between socio-economic position and food purchasing behaviour. Public Health Nutr 2006;9:375–83.

96. Beydoun MA, Wang Y. Do nutrition knowledge and beliefs modify the association of socio-economic factors and diet quality among US adults? Prev Med 2008;46:145–53.