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Pathways linking socioeconomic status to obesity through depression and lifestyle factors among young US adults

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Abstract

Obesity and depression are two diseases of major public health importance. While both correlate with each other, potential pathways involving depression that would link socioeconomic status (SES) to lifestyle factors and obesity have not been systematically examined using nationally representative data. Using rich data on 2,217 US young adults aged 20–39 years from the 1999–2004 National Health and Nutrition Surveys (NHANES) and multivariate linear and logistic regression models, we examined associations between major depressive disorder (MDD), dietary intake, physical activity (PA), and measured body mass index (BMI) controlling for socio-demographic factors. Further, structural equations models (SEM) were fit to test pathway explaining SES disparities in BMI through MDD and lifestyle factors. Recent prevalence of MDD was lower among young US men than women (6.4% vs 9.2%) although their prevalence of obesity was similar (21.2% vs 22.7%). Among women, MDD was associated with higher BMI and inversely associated with PA, but not among men. MDD was specifically associated with increased risk of morbid obesity (BMI≥40) among women (OR: 2.88 (1.32, 6.30)). Using SEM, a main pathway linking SES to BMI among women was that linking SES → food insecurity → MDD → PA → BMI. A main pathway linking MDD to BMI in both genders was that going through PA rather than overall dietary quality. Gender and ethnic differences existed underlying how MDD, SES and lifestyle factors were associated with adiposity. Future prospective studies are needed to examine potential mechanisms using physiological markers of depression, lifestyle and obesity.

Keywords

Depression; body mass index; obesity; socio-economic status; diet; physical activity

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CONTRIBUTORS

May A. Beydoun: Conceptualization, data management, statistical analysis, plan of analysis, write-up of the manuscript.

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CONFLICT OF INTEREST:

None declared.

INTRODUCTION

Major depressive disorder (MDD) is the most prevalent psychiatric illness in the United States, affecting over 16% of adults in their lifetime and around 6.6% over a period of twelve months (Kessler et al., 2003). The World Health Organization (WHO) (Murray, 1996) projected that, in 2020, MDD would account for approximately 11% of the world's total disease burden. Unipolar MDD is currently established to be a life-long illness (Keller et al., 1992) and among the most prevalent illnesses in the health care spectrum (Kessler et al., 1994). Depression has been associated with a variety of diseases, particularly with the development of cardiovascular disease (CVD) and all-cause mortality (Musselman et al., 1998; Wulsin et al., 1999).

The causal association between obesity and depression is still controversial and is possibly a bi-directional one. During the past three decades, the United States has witnessed a dramatic increase in the prevalence of obesity, which has become a public health crisis (Ogden et al., 2006; Wang and Beydoun, 2007; Wang et al., 2008). A growing body of evidence has reported large disparities between population groups (age, gender, SES and ethnicity) (Ogden et al., 2006; Wang and Zhang, 2006). MDD may be one of the risk factors for obesity, which remains poorly understood and potentially can be targeted in future intervention efforts. Previous studies have shown that depression can lead to eating disorders including over-consumption (Liu et al., 2007), poor quality of dietary intake (Guo et al., 2004) and reduced physical activity (PA) (Adams et al., 2007; Fox et al., 2007), which in turn may increase the risks of obesity (Ferro-Luzzi and Martino, 1996; Maskarinec et al., 2000). However, a number of studies tested the relationship in the opposite direction, i.e. whether obesity may cause depression, and some support the direction (Herva et al., 2006; Onyike et al., 2003; Roberts et al., 2003).

SES disparities in MDD has great policy implications and may be mediated by many factors including food insecurity, or the "limited or uncertain availability of nutritionally adequate and safe foods or limited or uncertain ability to acquire acceptable foods in socially acceptable ways" (1990). While previous research has focused on understanding the relationship between SES and BMI or lifestyle factors (Beydoun and Wang, 2008; Cerin and Leslie, 2008; Zhang and Wang, 2004) on one hand and depressive symptoms and BMI on the other (Carpenter et al., 2000; Dragan and Akhtar-Danesh, 2007; Heo et al., 2006; Herva et al., 2006; Istvan et al., 1992; Johnston et al., 2004; Onyike et al., 2003; Simon et al., 2006), to our knowledge, few have tried to uncover pathways linking SES to obesity through depression and lifestyle factors, beyond controlling for the confounding effects of SES in the depression-obesity association. In particular, no studies have implicated food insecurity as a potential mediator between SES and depression, given its previously shown association with SES (Hadley et al., 2007) and various outcomes of interest in our study, including obesity (Dinour et al., 2007; Gulliford et al., 2006), weight-control practices (Gulliford et al., 2006), and dietary quality (Ruel, 2003).

In the present study, we focused on the pathway in which depression may affect obesity risk through other lifestyle factors, using the rationale that depressed individuals are more self-neglecting than their non-depressed counterparts and may potentially lead less healthy lifestyles, including poorer dietary quality and lower physical activity. We tested these pathways (Figure 1 shows our conceptual framework) using a nationally representative sample of young US adults. Specifically, we examined the following: 1) The role of depression as a potential mediator between SES and obesity; 2) The main lifestyle mediators in the association between depression and obesity; and 3) The role of SES and gender as moderators in the depression-obesity association, and 4) Gender and ethnic differences in the overall pathway linking SES to obesity through food insecurity, MDD and lifestyle

factors (diet and physical activity). Note that we focused on the gender differences rather than ethnic-gender disparities, considering the constraints of our sample sizes.

MATERIALS AND METHODS

Database and study population

The National Health and Nutrition Examination Surveys (NHANES) include a series of cross-sectional surveys that provided nationally representative information on the nutrition and health status of the U.S. civilian population. The National Center for Health Statistics (NCHS) conducted three waves of NHANES surveys (NHANES I, II, and III) in 1971–1975, 1976–1980, and 1988–1994, respectively. Since 1999, NHANES has been a continuous survey. The data were recently made available for the first 6 years of the period 1999–2004. Using a stratified, multistage probability cluster sampling design, the NHANES survey consists of an in-home interview for demographic and basic health information and a health examination in a mobile examination center (MEC). Household interviews were conducted by trained staff and the MEC consists of physicians, medical and health technicians, and dietary and health interviewers. Detailed descriptions of the sample design, interview procedures, and physical examinations conducted were published elsewhere (Center for Disease Control and Prevention (CDC), 2006a; Centers for Disease Control (CDC), 1988–94).

NHANES data was pooled for periods 1999–2000, 2001–2002 and 2003–2004. The target age group (18 years and over) was selected and yielded a sample of 17,061 (8,091 men and 8,970 women) that had complete demographic data. Out of those, only 15,009 had complete dietary data (7,149 men and 7,860 women) and 14,618 had both anthropometric and dietary data available (6,966 men and 7,652 women).

However, the instruments used to assess mental health conditions including MDD were administered only to a half-sample of examination participants, ages 20 – 39 years. Proxies were ineligible, as were persons who required interpreters (i.e., non-English or Spanish speakers). As a result, our final sample consisted of 2,217 young adults aged 20–39 years (49.7% were men). The sub-sample was weighted in the analysis accordingly to generate nationally representative estimates, particularly in the descriptive and regression analyses.

Measurements and key study variables

Weight status outcomes—Body weight and standing height were directly measured (Center for Disease Control and Prevention (CDC), 2006b, c; Centers for Disease Control (CDC), 1988–94). Body Mass Index ($BMI = \text{weight}/\text{height}^2$, kg/m^2) were calculated for each subject. Weight status was defined using BMI cut points as underweight ($<18.5 \text{ kg}/\text{m}^2$), normal weight ($18.5\text{--}24.9 \text{ kg}/\text{m}^2$), overweight ($25\text{--}29.9 \text{ kg}/\text{m}^2$), obesity ($\geq 30 \text{ kg}/\text{m}^2$), and morbid obesity ($\geq 40 \text{ kg}/\text{m}^2$) (WHO Expert Committee, 1995).

Major Depressive Disorder (MDD)—An NHANES version of three modules from the automated version of the WHO Composite International Diagnostic Interview, Version 2.1 (CIDI-Auto 2.1) was administered during the face-to-face MEC interview. The CIDI is a comprehensive, fully standardized interview that is used to assess mental disorders and provide diagnoses according to definitions and criteria of the tenth revision of the International Classification of Diseases (ICD-10) (World Health Organization, 1993) and the fourth edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994). The CIDI is especially suitable for large epidemiological studies because it can be administered by lay interviewers, does not require outside informants or medical records, and does not assume the presence of

a current disorder. It is used in both clinical and research settings. CIDI has been previously validated in the WHO world mental health surveys against standardized clinical assessment and receiver-operating curve analysis indicated a high level of concordance for any mood disorder including MDD as reflected by an area under the curve $AUC=0.83$ (Haro et al., 2006).

The NHANES CIDI, developed as a computer-administered version, consisted of three diagnostic modules that addressed diagnoses present in the past 12 months. These modules were (a) Panic Disorder, (b) Generalized Anxiety Disorder, and (c) Depressive Disorders. Additional questions were added to measure both the quality and quantity of impairment, and to assess risk factors such as physical illness and life events. This can be summarized by the following Diagnostic and Statistical Manual fourth revision (DSM IV) criteria: (A) Five (or more) of nine potential depressive symptoms (e.g. depressed mood, diminished interest, insomnia, fatigue etc.); (C) the symptoms cause clinically significant distress; (D) Symptoms are not due to the direct physiological effects of a substance or general medical condition; (E) Symptoms are not better accounted by bereavement. The final diagnosis of MDD is made only when criteria A, C, D and E are mutually met (Center for Disease Control and Prevention (CDC), 2006a).

Dietary intakes—NHANES 1999–00 and 2001–02 collected one 24-hr recall of dietary intake among all participants, while NHANES 2003–04 collected two recalls. To allow all eligible subjects to have equal opportunities in terms of dietary assessment, we only used the first day of recall for NHANES 2003–04. In our analysis we focused on dietary quality and assessed it using the 2005 United States Department of Agriculture (USDA) Health Eating Index (HEI) (U.S. Department of Agriculture (USDA), 2005), while so far in the related literature, this index has been used in its earlier 1995 version (McCullough et al., 2000).

The 1995 HEI was revised recently to reflect the 2005 Dietary Guidelines for Americans, had a number of improvements (U.S. Department of Agriculture (USDA), 2005) and is considered a valid tool to be used for monitoring overall dietary quality in US populations and sub-population as well as for research purposes (Guenther et al., 2008). The new HEI includes twelve components and is measured on a scale of 0 to 100, i.e., the higher the total HEI score, the better the diet. For many of the food group criteria, serving estimates rather than grams were used as made available by the USDA. A major difference between this new HEI and the old 1995 HEI is its inclusion of total energy intake.

Physical activity (PA)—In the main part of the analysis, PA was assessed by a single question with responses measured on a 3-point likert scale, which asked whether the subject was less active (score=1), about the same (score=2) or more active (score=3) than his age peers. This single question was validated against a more objective measure which was $MET \times hr/week$ of PA (Cheng et al., 2007; Lagerros and Lagiou, 2007; McCullough et al., 2000). This measure was constructed based on individual leisure-time activities that were given an intensity score assessed by the metabolic equivalent or MET which was further multiplied by duration of this particular activity and frequency converted to per week unit. This $MET \times hr/week$ value was then summed up for each subject depending on the number of leisure-time physical activities elicited. Subjects that did not elicit any activity were considered sedentary and given a score of zero. The mean of this objective measure increased steadily and in a pseudo-linear fashion with increased level of self-reported PA. In fact, mean of METs (SE) were 9.1(1.2), 17.7(1.6) and 38.9(2.6) for those who reported having lower, similar or higher PA than their peers. A sensitivity analysis was also conducted using this measure instead of the self-report in all our analyses.

Socio-economic status (SES)—SES was measured by completed years of education and poverty income ratio. Education was categorized into <High school (0–8 years), High school (9–12 years) and >High school (13+ years), while poverty income ratio (PIR) was categorized as below the poverty line (PIR<100%), PIR between 101% and 200% and PIR above 201%. In addition, principal components analysis of categorized education and continuous PIR was conducted to obtain a single measure of SES measured on a standardized z-score scale.

Food Insecurity—Adult food insecurity in the household was measured on 4-point likert scale of: 1: adult fully food secure (“had no problems, or anxiety about, consistently accessing adequate food”); 2: adult marginally food secure (“had problems at times, or anxiety about, accessing adequate food, but the quality, variety, and quantity of their food intake were not substantially reduced”); 3: adult food insecure without hunger (“reduced the quality, variety, and desirability of their diets, but the quantity of food intake and normal eating patterns were not substantially disrupted”); 4: adult food insecure with hunger (“At times during the year, eating patterns of the adult were disrupted and food intake reduced because the household lacked money and other resources for food”). This item was elicited from respondents as part of a questionnaire on food security administered by study staff. Many of the items correlated strongly with that particular item which was thought to be specific to individuals in the age group under study (i.e. young adults either married or single, living within a larger household or living alone).

Other main covariates—Covariates were selected based on published literature and bivariate analyses were conducted to assess potential for confounding. They included race/ethnicity categorized as “Non-Hispanic (NH) white”, “NH black”, “Mexican-American (MA)” and “Others”, age, sex and current marital status (married vs. unmarried, including never married, divorced and widowed).

Statistical analysis

In all analyses, except for SEM, complex sampling design was taken into account using STATA release 9.0 (STATA, 2005), survey-related commands and specifying PSUs, strata and weights. Most analyses were stratified by gender. After conducting descriptive analyses, we ran several multivariate regression models for several purposes: (1) Identifying correlates of MDD, (2) Testing the independent effect of MDD on BMI, and lifestyle factors namely dietary quality and PA, controlling for covariates including socio-demographic factors; (3) Testing the independent effect of MDD on several binary measures of adiposity (overweight, obesity and morbid obesity); (4) Identifying potential lifestyle mediators in the association between MDD and obesity; and (5) Testing moderation by gender and SES of the MDD-binary BMI association.

Using SAS CALIS procedure (Hatcher, 1994), we fit structural equations models (SEM) to determine the most likely pathway explaining SES disparities in BMI through MDD and lifestyle factors. Path analysis was performed to test a theoretical model in which demographic factors (age, sex, marital status, race/ethnicity) were exogenous (i.e. were not predicted by other variables) while SES was an endogenous variable which was allowed to predict food insecurity and all other outcomes and food insecurity was allowed to predict MDD, PA and HEI. MDD was allowed to predict HEI, PA and BMI while HEI and PA were only allowed to predict BMI (See equations 1 through 6).

$$SES = \sum_{j=1}^k \alpha_{z_{j1}} Z_j + e_1$$

Eq. 1

$$Foodins = \alpha_{12}SES + \sum_{j=1}^k \alpha_{z_{j2}} Z_j + e_2$$

Eq. 2

$$MDD = \alpha_{13}SES + \alpha_{23}Foodins + \sum_{j=1}^k \alpha_{z_{j3}} Z_j + e_3$$

Eq. 3

$$HEI = \alpha_{14}SES + \alpha_{24}Foodins + \alpha_{34}MDD + \sum_{j=1}^k \alpha_{z_{j4}} Z_j + e_4$$

Eq. 4

$$PA = \alpha_{15}SES + \alpha_{25}Foodins + \alpha_{35}MDD + \sum_{j=1}^k \alpha_{z_{j5}} Z_j + e_5$$

Eq. 5

$$BMI = \alpha_{16}SES + \alpha_{26}Foodins + \alpha_{36}MDD + \alpha_{46}HEI + \alpha_{56}PA + \sum_{j=1}^k \alpha_{z_{j6}} Z_j + e_6$$

Eq. 6

Global goodness of SEM fit indices included the chi-square statistic that tested the null hypothesis that the reproduced covariance matrix has the specified structure, or the model fits the data. In addition, the Goodness of Fit Index adjusted for degrees of freedom (AGFI) is presented which ranges between 0 and 1 (model fits). Other goodness of fit statistics were also examined, though not presented and these included the Root Mean Square Error of Approximation (RMSEA) and the comparative fit index (CFI) with their specific cut-point criteria as suggested elsewhere (Hu and Bentler, 1999). While models with 1 degree of freedom are presented, additional more parsimonious models were tested for goodness of fit. In these models, only paths in the original model that are significant at a type I error of 0.10 were kept. When alternative models were fit using MET PA measures and, model fit using χ^2 test was not acceptable at a type I error of 0.05. Thus, only self-reported PA were presented. Further, the mediation proportion (MP, %) was computed to estimate the proportion of the total effect of a variable that is explained by a particular pathway (Beydoun and Wang, 2008; Ditlevsen et al., 2005). Stratified analysis of the SEM model was conducted by gender and selected ethnic groups, separately.

RESULTS

Characteristics of study population

Table 1 showed the main characteristics. While age, race/ethnicity and food security distributions were similar between men and women, they differed in other characteristics. In particular, women were more likely to be married (48.4% vs. 43.2%), better educated (>HS education: 60.1% vs. 51.0%) and poorer (PIR<100%: 18.9% vs. 13.7%); (χ^2 p<0.05). Moreover, more women than men were diagnosed with MDD (9.2% vs. 6.4%; p<0.05). Neither men nor women among US young adults had a good diet as their HEI score was

low, although women had a slightly better overall dietary quality based on mean HEI (49.2 vs. 46.3, $p<0.05$). However, these young US women had a lower PA (based on self-reported and METs $\times$ hrs/week measure) and a higher prevalence of morbid obesity (BMI $\geq$ 40; 8.1% vs. 5.5%, $p<0.05$).

Socio-demographic correlates of MDD

Among men, none of the variables were found significantly correlated with MDD (Table 2). However, among women, MDD was significantly less prevalent among NH blacks and Hispanics compared to NH whites, and was also less prevalent among married women and higher among those who were food insecure. SES factors were not significantly associated with MDD for both genders, particularly when controlling for food insecurity.

MDD's association with BMI, HEI and PA

After adjustment for potential confounders, namely age, ethnicity, marital status and food insecurity, among women, MDD was positively associated with BMI. MDD was not associated with BMI in men. Dietary quality as measured by HEI was not significantly associated with MDD. MDD had an inverse relationship with PA among women, but not in men. In addition, and among men only, being food insecure with hunger was associated with significantly lower level of self-reported PA. Using an objective measure of PA (METs $\times$ hr/week), MDD was inversely and significantly associated with physical activity among both men and women.

MDD's association with overweight, obesity and morbid obesity

Table 4 shows results of multivariate logistic regression models for the association between MDD and three binary adiposity outcomes (overweight, obesity and morbid obesity) as contrasted with healthy weight status ($18.5\leq\text{BMI}\leq 24.9$). The role of HEI and PA in explaining those associations was also investigated. Men with MDD were less likely to be overweight ($25<\text{BMI}\leq 30$) (Model 1, OR=0.42; 95% CI: 0.19, 0.92) or morbidly obese (Model 3, OR=0.22; 5% CI: 0.06, 0.88). In contrast, women with MDD were 2.88 times more likely to be morbidly obese. Addition of an interaction term (sex $\times$ MDD) indicated statistically significant effect modification of the MDD-“morbidly obese” association by sex ($p<0.05$). However, adding HEI or PA or both to the baseline model did not indicate significant mediation.

The moderating roles of SES and gender in the depression-obesity association

Further analyses looking at potential moderating roles of SES and gender are presented in Table 5. We found that there was significant effect modification of the SES-overweight association by gender, whereby the positive effect of SES among non-depressed men on overweight risk was reversed among non-depressed women. In the case of obesity as main outcome, an even stronger effect modification was found in the same direction. As for morbid obesity, in addition to the significant effect modification by sex of the SES-morbid obesity association, there was also significant effect modification by sex of the MDD-morbid obesity association. In particular, the effect of MDD on morbid obesity was positive among women and appears to be reversed among men with average SES level (SES=0 corresponded to an average SES level based on PIR and education). However, there was no significant SES moderation of the MDD-adiposity association; neither was there a simultaneous gender and SES moderation in those associations.

The role of MDD in the pathways between SES, dietary quality (HEI), PA and BMI: findings of structural equation models and gender and ethnic differences

Figure 2 evaluates a theoretical model with 1 degree of freedom in which SES, food insecurity, MDD, HEI, PA and BMI were allowed to predict each other in a structured manner while demographic variables (age, sex, race/ethnicity and marital status) were considered exogenous to the system and are allowed to predict all endogenous variables. Close fit was concluded based on several measures. Among the total population (Figure 2a), pathways linking SES to BMI include a pathway through food insecurity, MDD and low PA, in addition to a direct effect of SES on BMI. Pathways linking SES to HEI, included one through food insecurity and MDD in addition to a main direct effect. The same pathway linked SES to PA.

Further, stratified analysis by selected ethnicities (NH whites, NH blacks and MA), indicated disparities across groups. In particular, only among NH whites was MDD inversely related to HEI, independently of SES and food insecurity. Among NH blacks, SES was inversely related to MDD independently of food insecurity, and MDD was inversely related to PA which in turn was inversely associated with BMI. In contrast, among NH whites and MA, PA's inverse association with BMI did not involve any pathway with MDD, SES or food insecurity. Examining "other ethnicities" (data not shown), indicated that food insecurity mediated the SES-MDD inverse relationship only among this group.

Among men (Figure 2b), SES was associated with lower food insecurity, which in turn was inversely though marginally related to PA. PA was associated with a reduced BMI. Moreover, while SES did not have a direct effect on BMI, it had mainly a direct effect on HEI that was independent of food insecurity and MDD. However, SES was not associated with PA. MDD had a marginally significant inverse direct association with BMI. However, the indirect association between MDD and BMI was mainly through PA as a mediator and was indicative of an adverse effect of MDD on PA which increased the level of BMI.

Among women (Figure 2c), SES was inversely associated with BMI partly through lower food insecurity (which was positively associated with MDD) and reduced PA (which was associated with lower BMI). However, the direct effect of SES on BMI was the major pathway explaining its total effect. More importantly, the significant positive association between MDD and BMI was mostly (79%) explained by a pathway through PA (i.e., the estimated mediation proportion; $MP = \text{indirect effect through PA} / \text{total effect of MDD on BMI}$). Finally, the total effect of SES on HEI was mainly a direct effect, but not through MDD. Similar patterns of association were retained when parsimonious models were fit (retaining only paths that were significant at a type I error of 0.10) and goodness of fit based on χ^2 test was acceptable at a type I error level of 0.05.

DISCUSSION

Using nationally representative data collected in the United States, we examined the complex relationships between depression (MDD), weight status outcomes, SES, food insecurity, demographic and lifestyle factors including physical activity and dietary intakes using both linear regression and structural equation models. We observed several key findings: First, we found a different association between weight status outcomes and depression in young US women and men. BMI and depression were associated only among women but not among men, which is consistent with some previous studies (Carpenter et al., 2000; Dragan and Akhtar-Danesh, 2007; Heo et al., 2006; Istvan et al., 1992; Onyike et al., 2003), although some other studies found no gender difference in the BMI-depression association (Herva et al., 2006; Simon et al., 2006). In general, we found that MDD was associated with higher BMI and with morbid obesity (OR=2.2 to 2.9) among young U.S.

women compared to their non-depressed counterparts. Several recent studies have also replicated those findings (Carpenter et al., 2000; Dragan and Akhtar-Danesh, 2007; Istvan et al., 1992; Onyike et al., 2003; Simon et al., 2006), though others found no or an inverse significant association particularly among older adults (Kuriyama et al., 2006; Li et al., 2004). In contrast, we found an inverse association between MDD and overweight and morbid obesity among men (OR=0.2 to 0.4). Our findings and those of others should be carefully interpreted in light of the fact that loss of or enhanced appetite are possible symptoms inherent in the definition of depression in most of the scales and diagnostic tools used including CIDI (American Psychiatric Association, 1994; de Wit et al., 2009).

Second, our multivariate linear regression models indicated that BMI was inversely related to SES (mainly income) among women but not among men, independently of MDD. Moreover, MDD was associated with both higher BMI and lower PA, among women, independently of SES factors. This finding has been replicated elsewhere (Dragan and Akhtar-Danesh, 2007). Improved dietary quality, as measured by HEI, was not associated with MDD independently of SES among both men and women. In multivariate logistic regression models, MDD was not significantly associated with overweight or obesity but only with morbid obesity among women. Gender, but not SES, moderated the MDD-binary adiposity associations.

Third, our SEM models indicate that lower food insecurity, lack of MDD and higher PA were one mechanism by which higher SES was associated with lower BMI, particularly among women. The direct effect of MDD on BMI was not significant, indicating that PA is the main mediator that explained the association between MDD and adiposity among women as well as NH blacks. Among men, NH white and NH black subjects, SES had no direct association with BMI. However, MDD among men (as was the case among women and NH blacks) was related to lower PA which in turn was associated with reduced BMI. In addition, the direct association of MDD with BMI among men was a marginally inverse one ($p < 0.10$). For either gender, SEM models did not indicate that dietary quality was a significant mediator between MDD and BMI. Stratified analysis also indicated that MDD and HEI were inversely associated independently of SES and food insecurity among NH whites and among women.

Finally, another important finding is the lower prevalence of MDD among minority ethnic groups compared to NH White, despite the inverse association between SES and MDD and the lower SES experienced in minority ethnicities. This may suggest that NH whites are more likely to elicit depressive symptoms than other ethnic groups, independently of SES factors, though another possible explanation is that NH White may have better access to medical services increasing their chance for MDD diagnosis.

Several biological mechanisms have been suggested to explain the association between obesity and depression. For example, leptin resistance may contribute to alterations of 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 (Lu, 2007; Munzberg and Myers, 2005). This would give rise to a causal pathway in which depression is directly caused by leptin resistance which in turn alters appetite and in turn increasing the risk of obesity. Another suggested 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 (Vogelzangs et al., 2007; Weber-Hamann et al., 2002; Young, 2004). Finally, depressed subjects are often prescribed anti-depressant medication which enhances appetite and thus, overconsumption of food (Fava et al., 2005).

These alternative mechanisms might explain at least part of the associations we detected between morbid obesity and MDD among women in logistic regression models and the inverse relationship between MDD and HEI observed in Whites and in women in SEM models. However, PA had a major mediating role in the positive association between MDD and BMI among women and NH blacks, indicating that sedentary behaviors resulting from elevated depressive symptoms may be explaining higher adiposity in those two groups. It is worth noting that the association between mood and obesity may apply to other disorders particularly bipolar depression (Alciati et al., 2007; Hasler et al., 2004; McElroy et al., 2004; Pickering et al., 2007).

The present study has several strengths. First, the sample was nationally representative of young adults in the United States. Second, DSM-IV criteria were applied to obtain diagnosis of major depressive disorder which is the gold standard and allows for better comparability with previous literature. Third, stratified analysis was carried out to identify gender and ethnic differences in patterns of association. Fourth, this is one of the very few attempts to study the association between BMI and depressive symptoms using SEM models (Dragan and Akhtar-Danesh, 2007; Stunkard et al., 2003) and the first to test pathways linking depression to SES, lifestyle factors and BMI outcomes. Finally, we used a recently released USDA overall dietary quality measure that reflects several aspects of healthy eating based on the new 2005 Dietary Guidelines (U.S. Department of Agriculture (USDA), 2005). In addition, NHANES data allowed us to study PA as well as dietary intake and quality, in particular, using self-reported measures both simple ones and ones computed using METs×hrs/week for PA (Cheng et al., 2007; Lagerros and Ligiou, 2007).

Limitations of our study included its cross-sectional nature, which impedes ascertainment of causality. Second, depression data was only collected for a relatively small sample of young US adults aged 20–39 years. This limited stratified analyses by ethnicity, age, or SES groups to detect effect modification by these variables. Third, measurement error in study participants' self-reported dietary intake and PA would be of concern. Random errors may bias the findings toward the null while systematic measurement errors could bias the results toward either direction depending on the nature of the errors. Fourth, the HEI is a reflection of overall dietary quality though some of its components and related dietary/nutrient intake patterns may have a greater weight than others in explaining the MDD-HEI association. In fact, a sensitivity analysis correlating HEI with various food groups and nutrients showed that HEI was most strongly associated with higher total fruit ($r=0.47$), lower added sugar ($r=-0.36$), fiber ($r=0.36$), and vitamin C ($r=0.31$) intakes. Fifth, many of the SEM path coefficients were between non-continuous variables and thus interpretability of their values was less evident. However, their statistical significance and the direction of the association were of main interest in this study. Sixth, some of the findings using our regression analysis models were discrepant from those in SEM models. This may be due to adjustment for sampling design complexity in the former but not in the latter models. Finally, while CIDI is an improvement over other measures of depression, it has its limitations and validity of lay interviews may be constrained by the amount of probing.

In conclusion, our study suggested major gender differences in the pathways linking SES, MDD (indicates depression) and lifestyle factors to body weight outcomes. Hence, SES, mental health and lifestyle interventions among men and women may have different effects on their adiposity. Among both genders and among NH blacks, however, PA seems to play a major role in explaining the relationship between MDD and BMI, which indicate the potential effectiveness of PA interventions among depressed subjects in reducing the risk of obesity. Moreover, our finding that a large part of the effect of SES on MDD is mediated by food insecurity (specifically among women and among “other ethnic groups”) suggests that availability and access to food assistance programs may help reduce MDD risk that is

associated with low SES. More research is needed to clarify the causal relationship between MDD and lifestyle factors, especially PA, and how they interact with SES and other demographic factors in affecting obesity. The mechanism by which depression and adiposity may affect each other must be studied in depth using advanced statistical models and physiological markers that would help explain reduced PA and the tendency to have poorer quality diets – which may be direct outcomes of self-neglect and fatigue associated with depression -- among depressed women with morbid obesity. A good understanding of the pathways between MDD and adiposity will help develop effective future interventions.

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Figure 1.
Conceptual framework: Pathways/Associations between depression, adiposity, socioeconomic status, and lifestyle factors

Exogenous variables in total population:
Age, sex, ethnicity, marital status

Model fit (total population; N=1,857):
 $\chi^2=3.06$, d.f.=1, $p=0.0800$; AGFI=0.9786
Significant exogenous predictors (total population) of:
SES: black(-), ma(-), other(-), age(+), married(+)
Food insecurity: none.
MDD: black(-), ma(-), age(+), married(-)
HEI: ma(+), other(+), female(+)
PA: female(-)
BMI: black(+), age(+), married(+)
* $p<0.10$; ** $p<0.05$; +: positive association; -:negative association; Numbers stand for path coefficients.
ma: Mexican American; MDD: Major depressive disorder.

a) Total population & by selected ethnicities

b) Men

Exogenous variables:
Age, ethnicity, marital status

Model fit (Men; N=815):
 $\chi^2=1.0316$, d.f.=1, $p=0.3098$; AGFI=0.9848
Significant exogenous predictors among men of:
SES: black(-), ma(-), other(-), age(+)
Food insecurity: none.
MDD: none.
HEI: ma(+), other(+)
PA: none.
BMI: age(+)
* $p<0.10$; ** $p<0.05$; Numbers stand for path coefficients;
+: positive association; -:negative association; ma: Mexican American; MDD: Major depressive disorder.

c) Women

Figure 2.
Findings of Structural Equations Models: mechanisms explaining Socio-economic (SES) disparity in body mass index (BMI) through major depressive disorder (MDD), food insecurity, healthy eating index (HEI) and physical activity (PA)

TABLE 1

Main characteristics of young US adults (20–39 years) with complete data on Major Depressive Disorder (MDD) and other key covariates; NHANES, 1999–2004

Characteristic	Men (n=977)		Women (n=1240)	
	%	SE	%	SE
Gender	49.7	1.1	50.2	1.1
Age (years)				
20–29	47.5	1.9	48.0	2.0
30–39	52.5	1.9	52.0	2.0
Race/ethnicity				
NH White	67.2	2.0	64.0	2.1
NH Black	11.4	1.4	13.8	1.3
Mexican American	11.7	1.1	10.3	1.2
Other	9.6	1.3	11.7	1.6
Married	43.2	2.2	48.4	2.0*
Education				
<High School	19.8	1.2	16.2	1.3*
High School	29.1	1.8	23.7	1.7
> High School	51.0	1.9	60.1	2.1
Poverty income ratio				
0–100%: Poor	13.7	1.2	18.9	1.5*
101%–200%: Near poor	21.9	1.7	20.6	1.4
>200% : Non-poor	64.5	2.2	60.4	1.5
Food insecurity				
Fully food secure	79.6	1.9	80.2	1.3
Marginally food secure	6.8	8.2	6.9	0.8
Insecure without hunger	8.3	1.4	7.1	0.9
Insecure with hunger	5.2	0.8	5.6	0.7
Major Depressive disorder (MDD)	6.4	0.9	9.2	1.0*
Healthy Eating Index; HEI ^a (revised, 2005) score: Mean and SE	46.3	0.5	49.2	0.6*
Physical activity (compared to others)				
Less active	17.1	1.1	28.4	1.7*
The same	48.5	1.9	46.9	1.7
More active	34.4	1.7	24.7	1.3
Physical activity (METs × hrs/week)	28.2	2.0	16.0	1.1*
0: Sedentary	27.7	1.7	31.0	1.7*
0.1–14.99 : Moderate activity	31.7	2.1	36.8	1.9
≥15 : Active	40.6	2.0	32.2	2.5
Weight status				

Characteristic	Men (n=977)		Women (n=1240)	
	%	SE	%	SE
Underweight (BMI<18.5)	1.9	0.5	4.2	0.8 [*]
Normal weight (BMI 18.5–24.9)	37.1	1.3	41.4	1.8
Overweight (BMI 25.0–29.9)	34.2	1.8	23.5	1.7
Obese (BMI≥30)	21.2	1.8	22.7	1.4
Morbidly obese (BMI≥40)	5.5	0.7	8.1	0.9
Mean BMI (kg/m ²)	27.5	0.2	27.5	0.3

Abbreviations: BMI=Body Mass Index; METs= metabolic equivalent; NH=Non-Hispanic; NHANES=National Health and Nutrition Examination Surveys; SE=Standard Error.

^{*} p<0.05 (design-based χ^2 or *t*-test) for gender difference.

^a HEI score may range between 0 and 100.

Socio-demographic correlates of major depressive disorder (MDD) among young US adults (20–39 years): Multivariate logistic regression models by gender, NHANES 1999–04

TABLE 2

Characteristics	Prevalence of MDD, %(SE)		Logistic regression models			
	Men	Women	Men (n=977)	OR	95% CI	Women (n=1240)
Age (years)						
20–29 (ref)	4.4 (1.1)	8.8 (1.6)				
30–39	8.2 (1.7)	9.5 (1.7)	1.78	(0.84, 3.79)	1.39	(0.70, 2.76)
Race/ethnicity						
NH white (ref)	7.2 (1.4)	10.8 (1.4)				
NH black	6.2 (1.7)	6.1 (1.9)	0.82	(0.40, 1.66)	0.42*	(0.19, 0.92)
Mexican-American	2.5 (1.3)	4.8 (1.4)	0.35	(0.12, 1.02)	0.32*	(0.14, 0.74)
Other	5.9 (2.9)	7.8 (2.8)	0.85	(0.25, 2.95)	0.67	(0.30, 1.52)
Marital status						
Unmarried (ref)	6.6 (1.3)	11.1 (1.3)				
Married	4.3 (1.1)	7.4 (1.3)	0.53	(0.27, 1.05)	0.59*	(0.35, 0.99)
Education						
<High School (ref)	6.9 (2.2)	9.0 (2.0)				
High School	5.7 (1.7)	13.0 (2.5)	0.69	(0.24, 1.95)	1.51	(0.69, 3.30)
> High School	6.7 (1.6)	7.7 (1.3)	0.64	(0.24, 1.64)	0.75	(0.34, 1.61)
Poverty income ratio						
0–100%: poor (ref)	5.4 (2.2)	13.4 (2.6)				
101%–200%: near poor	6.1 (1.6)	6.1 (1.8)	1.25	(0.47, 3.33)	0.48	(0.19, 1.21)
>200%: non-poor	6.7 (1.4)	8.9 (1.4)	1.15	(0.39, 3.36)	0.80	(0.36, 1.78)
Food insecurity						
Fully food secure (ref.)	6.8 (1.1)	7.8 (1.2)				
Marginally food secure	3.5 (2.6)	10.0 (4.3)	0.75	(0.13, 4.46)	1.33	(0.50, 3.55)
Insecure without hunger	9.1 (3.3)	16.0 (5.1)	1.75	(0.63, 4.86)	2.76*	(1.00, 7.65)
Insecure with hunger	6.6 (4.2)	16.9 (4.7)	1.19	(0.25, 5.58)	2.13	(0.83, 5.47)

* p<0.05 for null hypothesis that $\beta = \text{Loge}(\text{OR}) = 0$. Logistic regression models were conducted within men and women separately and controlled for all correlated presented in the table, simultaneously. See Table 1 for abbreviations.

TABLE 3

Association^a between major depressive disorder (MDD) and outcomes of interest (BMI, healthy eating index score and physical activity): Multivariate linear regression models stratified by gender, NHANES 1999–2004

	Men		Women	
	β	SEE ^b	β	SEE
Model 1				
Y=Body mass index				
MDD	-1.36	0.68	2.20	0.99*†
Education				
<High School (ref)				
High School	1.42	0.72*	0.45	0.84
> High School	0.48	0.69	0.10	0.70
Poverty income ratio				
0–100% (ref)				
101%–199%	1.86	0.71*	-0.76	0.81
200% or more	1.07	0.70	-2.15	0.89*
Model 2				
Y= Healthy Eating Index				
MDD	-3.29	2.12	-2.63	1.96
Education				
<High School (ref)				
High School	0.47	1.04	-2.09	1.69
> High School	3.87	0.93*	3.29	1.54
Poverty income ratio				
0–100% (ref)				
101%–199%	0.16	1.44	-0.89	0.38
200% or more	0.04	1.57	2.48	1.24
Model 3				
Y=self-reported physical activity (PA)				
MDD	-0.21	0.11	-0.19	0.08*
Education				
<High School (ref)				
High School	-0.02	0.06	-0.00	0.10
> High School	-0.01	0.07	-0.01	0.09
Poverty income ratio				
0–100% (ref)				
101%–199%	-0.11	0.06	0.04	0.08
200% or more	-0.00	0.07	0.14	0.10
Model 4				
Y=MET×hrs/week PA				

	Men		Women	
	β	SEE ^b	β	SEE
MDD	-11.30	5.54*	-6.88	2.92*
Education				
<High School (ref)				
High School	6.23	5.10	7.08	3.63
> High School	14.73	4.88*	6.48	2.81*
Poverty income ratio				
0–100% (ref)				
101%–199%	7.35	6.06	-2.83	3.03
200% or more	0.24	3.78	3.85	2.99

* $P < 0.05$ for null hypothesis that $\beta = 0$.

^a Multivariate linear regression adjusted for design complexity, using PSU, stratum and weight variables. All models were adjusted for age, race/ethnicity, marital status and food insecurity.

^b SEE: Standard Error of the Estimate. See Table 1 for other abbreviations.

TABLE 4

Association^a between major depressive disorder (MDD) and adiposity outcomes (overweight, obesity, morbid obesity): Multivariate logistic regression models stratified by gender, NHANES 1999–2004

	Men		Women	
	OR	95% CI	OR	95% CI
Overweight (25≤BMI<30):				
Model 1 ^b	0.42*	(0.19, 0.92)	0.85	(0.34, 2.13)
Model 2	0.39*	(0.17, 0.90)	0.91	(0.35, 2.35)
Model 3	0.41*	(0.19, 0.88)	0.74	(0.29, 1.87)
Model 4	0.39*	(0.17, 0.87)	0.80	(0.30, 2.08)
Obesity (BMI≥30):				
Model 1	0.71	(0.35, 1.43)	1.53	(0.90, 2.61)
Model 2	0.57	(0.26, 1.27)	1.42	(0.87, 2.34)
Model 3	0.62	(0.31, 1.27)	1.38	(0.77, 2.48)
Model 4	0.51	(0.23, 1.17)	1.29	(0.75, 2.23)
Morbid obesity (BMI≥40):				
Model 1	0.25	(0.05, 1.29)	2.88*	(1.32, 6.30)
Model 2	0.16	(0.02, 1.05)	2.87*	(1.28, 6.38)
Model 3	0.22*	(0.06, 0.88)	2.80*	(1.15, 6.78)
Model 4	0.15*	(0.03, 0.75)	2.88*	(1.16, 7.11)

* p<0.05 for null hypothesis that the 95% of OR include 1.

^a Multivariate logistic regression models were carried out with each outcome having the referent BMI in the healthy range of 18.5–24.99 kg/m².

^b Model 1 adjusted for socio-demographic factors (age, race/ethnicity, marital status, education, poverty income ratio, food insecurity); Model 2 adjusted for socio-demographics and HEI; Model 3 adjusted for socio-demographics and PA; and Model 4 adjusted for socio-demographics, HEI and PA.

TABLE 5

Association^{a,b} between major depressive disorder (MDD) and adiposity outcomes (overweight, obesity, morbid obesity) and moderating effects of SES and gender, NHANES 1999–2004: Multivariate logistic regression models

	Overweight (25≤BMI<30) vs. Normal		Obesity (BMI≥30) vs. Normal		Morbid obesity (BMI≥40) vs. Normal	
	β	SEE ^c	β	SEE	β	SEE
MDD=1; no MDD=0	0.88	0.41 *	-0.36	0.40	-1.45	0.79 *
Female=1; Male=0	-0.33	0.16 *	0.09	0.12	0.24	0.24
SES (Mean=0)	0.18	0.08 *	0.08	0.09	0.06	0.16
SES×MDD	0.62	0.54	0.09	0.31	0.04	0.31
SES×Female	-0.30	0.10 *	-0.45	0.10 *	-0.50	0.21 *
MDD×Female	0.20	0.31	0.58	0.49	2.12	0.71 *
SES×MDD×Female	-0.33	0.37	0.06	0.37	0.52	0.41

^aMultivariate logistic regression models were carried out with each outcome having the referent BMI in the healthy range of 18.5–24.99 kg/m². We chose to report the β coefficients, but not OR and 95%CI because of our testing of the interaction terms.

^bModels adjusted for socio-demographic factors (age, race/ethnicity, marital status, and food insecurity)

^cSEE: Standard Error of the Estimate.

~p<0.10

* p<0.05 for null hypothesis that β or Loge(OR)=0.