

A Prospective Study of the Role of Depression in the Development and Persistence of Adolescent Obesity

Elizabeth Goodman, MD*§, and Robert C. Whitaker, MD, MPH‡§

ABSTRACT. *Background.* Adolescent obesity is a strong predictor of adult obesity, and adult obesity has been associated with depression, especially in women. Studies have also suggested an association between depression in adolescence and higher body mass index (BMI) in adulthood. Whether depression leads to obesity or obesity causes depression is unclear.

Objective. To determine in longitudinal analyses whether depressed mood predicts the development and persistence of obesity in adolescents.

Methods. A prospective cohort study of 9374 adolescents in grades 7 through 12 who completed in-home interviews for the National Longitudinal Study of Adolescent Health. Assessments were made at baseline (1995) and at follow-up 1 year later. Depressed mood was assessed with the Center for Epidemiologic Studies Depression Scale. BMI (kg/m²) was calculated from self-reported height and weight. BMI percentiles and z scores were computed using the 2000 Centers for Disease Control and Prevention growth charts. Obesity was defined as BMI ≥95th percentile, overweight as BMI ≥85th percentile and <95th percentile, and normal weight as BMI <85th percentile. A parental respondent gave information on household income, parental education, and parental obesity.

Results. At baseline, 12.9% were overweight, 9.7% were obese, and 8.8% had depressed mood. Baseline depression was not significantly correlated with baseline obesity. Among the 9.7% who were obese at follow-up, 79.6% were obese at baseline, 18.6% were overweight at baseline, and 1.8% were normal weight at baseline. Having depressed mood at baseline independently predicted obesity at follow-up (odds ratio: 2.05; 95% confidence interval: 1.18, 3.56) after controlling for BMI z score at baseline, age, race, gender, parental obesity, number of parents in the home, and family socioeconomic status. This finding persisted after controlling further for the adolescents' report of smoking, self-esteem, delinquent behavior (conduct disorder), and physical activity. After controlling for all these same factors, depressed mood at baseline also predicted obesity at follow-up among those not obese at baseline (odds ratio: 2.05; 95% confidence

interval: 1.04, 4.06) and follow-up BMI z score among those obese at baseline ($\beta = 0.11$; standard error $\beta = 0.05$). In contrast, baseline obesity did not predict follow-up depression.

Conclusions. Depressed adolescents are at increased risk for the development and persistence of obesity during adolescence. Understanding the shared biological and social determinants linking depressed mood and obesity may inform the prevention and treatment of both disorders. *Pediatrics* 2002;109:497–504; *depression, obesity, adolescence.*

ABBREVIATIONS. BMI, body mass index; Add Health, National Longitudinal Study of Adolescent Health; CES-D, Centers for Epidemiologic Studies Depression Scale; OR, odds ratio; CI, confidence interval; SE, standard error.

Obesity has become a major public health problem.^{1,2} More than 300 000 deaths each year have been linked to obesity,³ and it causes multiple medical complications.⁴ Although obesity is increasing in all age groups and among all racial/ethnic groups and educational levels, young adults ages 18 to 29 are experiencing the highest rate of increase.⁵ This suggests that there may be factors operating during adolescence that predispose to obesity risk in early adulthood, making adolescence a critical period for the development of obesity.⁶ Obesity during adolescence carries with it important psychosocial sequelae, in addition to the medical complications. Obese female adolescents become adults who, on average, earn lower wages and are at increased risk of living in poverty, and obese male adolescents are less likely to marry as adults.^{7,8} Studies also suggest that obesity may lead to lower self-esteem among adolescents and young adults, especially Hispanic and non-Hispanic white females.^{9,10}

Studies on the psychological correlates and sequelae of obesity have usually characterized the relationship between depression and obesity as unidirectional.^{9–16} The social stigmatization associated with obesity is believed to engender chronic embarrassment, shame, and guilt, all of which may lead to affective disorders.¹⁷ Within the context of the family, obesity is believed to increase depressive symptoms because “the obese child is reared in a milieu in which he becomes the focus of family conflicts and the recipient of subtle hostility and rejection and is treated differently than his siblings.”¹⁸

Although these are plausible explanations of how obesity leads to depression, few longitudinal studies have assessed the obesity-depression relationship to

From the Divisions of *Adolescent Medicine and †General and Community Pediatrics, Children's Hospital Medical Center, Cincinnati, Ohio, and §University of Cincinnati College of Medicine, Cincinnati, Ohio.

This work was presented, in part, at the North American Association for the Study of Obesity Annual Meeting, Quebec City, Canada, October 10, 2001. Received for publication Dec 26, 2001; accepted Mar 27, 2002.

Address correspondence to Elizabeth Goodman, MD, Heller School for Social Policy and Management, Brandeis University, MS035, 415 South St, Waltham, MA 02454. E-mail: goodman@brandeis.edu

These data are not available from the author. Persons interested in obtaining data files from the National Longitudinal Study of Adolescent Health should contact Francesca Florey, Carolina Population Center, 123 W Franklin St, Chapel Hill, NC 27516-3997. E-mail: fflorey@unc.edu

PEDIATRICS (ISSN 0031 4005). Copyright © 2002 by the American Academy of Pediatrics.

help establish the causal direction of this association. Most studies have been cross-sectional and have relied on clinic-based populations. In a recent prospective study, Pine et al¹⁹ showed that childhood depression was associated with an increased body mass index (BMI) in adulthood. This association persisted after controlling for socioeconomic factors. Although the study was not drawn from a nationally representative sample, experienced losses to follow-up, had limited data on childhood BMI, and had no data on parental BMI, it provided the strongest evidence to date that depression may be a cause and not just a consequence of obesity.¹⁹ Additional studies are important to help clarify the timing of depression in relation to the development of obesity. Understanding that depression can lead to obesity may enlighten obesity prevention strategies and lead to more focus on the biological and social determinants shared by both disorders. The objective of this study was to determine, in a nationally representative sample, whether depression increases risk for obesity during adolescence. We hypothesized that high levels of depressive symptoms would increase the risk of obesity at 1-year follow-up among teens.

METHODS

Sample

The data for this study were drawn from the National Longitudinal Study of Adolescent Health (Add Health), a nationally representative, comprehensive, school-based study of youth in grades 7 to 12.²⁰ The current study used data from the weighted in-home sample at baseline (Wave 1 in April-December 1995) and first follow-up (Wave 2 in April-August 1996). The sample used in our analyses was defined by 5 inclusion criteria. The subjects were those who 1) were <20 years old at Wave 1; 2) completed the in-home interviews in both waves, 3) had a biological, step, foster, or adoptive parent (the "parental respondent" for purposes of this study) who completed an in-home interview at Wave 1; and 4) provided self-reported height and weight at both waves. Because of significant racial/ethnic differences in prevalence of obesity^{21,22} and depression²³ and because the subgroup sizes for some racial/ethnic groups were so small, our study sample was further restricted to individuals who were white, non-Hispanic; black, non-Hispanic; or Hispanic. The final sample size was 9374 adolescents.

Measures

Obesity

Subjects self-reported their height in feet and inches and weight in pounds in both waves. From these data, BMI percentiles and z scores were calculated using the 2000 Centers for Disease Control and Prevention growth charts.²⁴ Obesity was defined as a BMI (kg/m²) greater than or equal to the 95th percentile for age and gender, overweight as a BMI greater than or equal to the 85th percentile but less than the 95th percentile, and normal weight as BMI less than the 85th percentile. Because the 95th percentile BMI in the reference growth charts was >30.0 in late adolescence and because some subjects were over 20 years of age at follow-up, a BMI of 30.0 or higher was considered obese in accordance with standard adult criteria.²⁵

Depression

A slightly modified version of the Center for Epidemiologic Studies Depression Scale (CES-D, 18 of 20 total items) was used to assess depressive symptoms.²⁶ The CES-D is a well-validated and widely used instrument that was developed to measure symptoms of depression within the community. The scale is valid for use in both junior and senior high school student populations.²⁷ Roberts et al²⁸ determined that scores of 24 in females and 22 in males maximized the sensitivity and specificity of the CES-D for predicting major depressive disorder among adolescents. Thus, we cre-

ated a dichotomous variable indicating depressed mood based on these cutpoints.

Sociodemographic Variables

Age was determined as the date of the interview minus the date of birth. The Wave 1 in-home interviewer determined gender. Female gender was used as the reference category as evidence suggests obesity is more common among adolescent males.^{21,29} Race/ethnicity was categorized as white, non-Hispanic; black, non-Hispanic; or Hispanic based on self-identified membership in race/ethnic groups. White, non-Hispanic was used as the reference category in regression analyses. Number of "parents" in the home was determined by categorizing answers to the relationship of the teen to those individuals identified by the teen as living in the house. If a member of the household roster was identified as mother, father's wife, or father's partner, "mother" was considered to be living in the house. If a member of the household roster was identified as father, mother's husband, or mother's partner, then "father" was considered to be living in the house. The final measure was a dichotomous variable representing 2 parents in the home as opposed to 1 or none.

Two ordinal measures of socioeconomic status were drawn from information obtained during the parental interview. Parental respondents were asked to report, in thousands of dollars, how much before-tax total income the household received in 1994 by including income from all household members, dividends, welfare benefits, and other sources. As previously described,²⁹ we created an ordinal 5-level variable for income ranging from level 1, those <1.5 times the federal poverty threshold in 1994, to level 5, those in the top 5% of US household incomes in 1994. Parental respondents reported their own and their current spouse or partner's educational attainment. The higher of these 2 education levels was used to create a variable corresponding to educational level of the highest educated parent.²⁹ Categories of this 5-level ordinal measure of parental education ranged from less than a high school degree¹ to professional training beyond college.⁵ Parental respondents also reported whether the teen's biological mother and father "was obese," but did not report actual parental height and weight. The number of obese parents was determined by summing responses to these questions (range: 0-2).

Psychological and Behavioral Covariates

We defined 4 psychological and behavioral covariates based on previous research linking each to obesity and/or depression. These covariates were all measured at baseline. A description of each follows.

Self-Esteem

Self-esteem was measured by the 6-item personal self-image scale developed by Resnick et al for Add Health analyses.³⁰ The scale has excellent reliability (Cronbach's $\alpha = 0.85$). Higher scores indicate lower self-esteem. For logistic regression analyses, low self-esteem, defined as scoring in the highest quartile on this scale, was compared with a referent group of those in the other quartiles.

Smoking

A 5-level variable was created from the adolescents' responses to items assessing cigarette smoking behavior. Those who reported they had never tried cigarette smoking "not even 1 or 2 puffs," were considered to have "never smoked." Those who smoked, but who had never smoked a whole cigarette and those who had smoked a whole cigarette, but had not smoked in the past 30 days, were considered "experimenters." Current smokers were those who had smoked in the past 30 days, and these were divided into the 3 remaining categories as follows. The number of cigarettes smoked per day by current smokers was multiplied by the number of days the teen smoked in the past 30 days to categorize subjects into those who, on average, 1) smoked less than a pack per week (<80 cigarettes per 30 days), 2) smoked between a pack per week and a pack per day (between 80 and 599 cigarettes per 30 days), and 3) smoked at least a pack per day (at least 600 cigarettes per 30 days). In regression analyses, "never smoked" was used as the reference category.

Delinquent Behavior

Fifteen items in Add Health assessed involvement in delinquent behaviors in the past 12 months. The scale had excellent reliability (Cronbach's $\alpha = 0.84$). Examples of behaviors assessed include deliberate destruction of someone else's property, lying to parents, stealing, physical fighting, running away, and weapon carrying. These behaviors are consistent with those used to diagnose conduct disorder,³¹ which has been associated with the development of obesity in young adulthood.³² To develop a proxy measure of conduct disorder, a dichotomous variable was created to identify those scoring in the top quartile on this scale.

Low Physical Activity

Public health guidelines suggest that adolescents should engage in at least 3 bouts of moderate to vigorous physical activity per week.³³ Thus, adolescents who did not report at least 3 bouts of moderate to vigorous physical activity per week were considered to have low physical activity. Moderate to vigorous physical activity was defined as has been done in previous analyses using Add Health data.^{34,35}

Data Analyses

We weighted the data to account for the complex sampling frame of Add Health and report unweighted sample size (N) and weighted percents in the tables. To decrease the likelihood of a type 1 error because of the extremely large weighted sample size, sample weights were normalized so that the weighted sample size equaled the observed sample size.³⁶ Descriptive statistics were generated with SPSS version 10.³⁷ All statistical significance testing (2-tailed) was performed using SUDAAN V8.0 to account for design effects (Research Triangle Institute, Research Triangle Park, NC).³⁸ We used χ^2 tests for bivariate analyses and logistic and linear regression for multivariable analyses. Multivariable analysis was performed to examine the relationships between baseline depressed mood and obesity at baseline, obesity at follow-up, and BMI z score at follow-up. Multivariable analysis was also used to examine the relationship between obesity at baseline and depressed mood at follow-up. These regressions controlled for the other variables potentially related to both depressed mood and obesity.

Because the 2 variables measuring socioeconomic status (parental education and household income) were correlated to one another (Pearson's $r = 0.50$) and because current recommendations^{39,40} indicate that these factors represent different domains of social status and should be modeled separately, multivariable analyses were run separately using parental education and household income as independent variables. Results were not substantially different between these sets of analyses, and because parental education is less dynamic than household income and fewer subjects were missing education data than income data, results are presented for parental education only. In the total study sample, analyses that included both indicators of socioeconomic status yielded essentially identical results. Subgroup analyses were not performed with both indicators because of increased sample attrition from missing data. Results from multivariable analyses using household income and using both indicators of socioeconomic status are available from the first author (E.G.).

We first developed a logistic model to assess correlates of baseline obesity. The following 7 variables were entered simultaneously in the model: age, gender, race/ethnicity, number of obese parents (0,1,2), parent education, number of parents in the house (0–1 vs 2), and baseline depressed mood. We tested for 2-way interactions between depression, age, race/ethnicity, and gender. All were nonsignificant, so none are reported. After determining correlates of baseline obesity, we next turned to determining predictors of obesity at follow-up. To do this, we developed a "core" logistic model using these same 7 independent variables described above plus baseline BMI z score. We tested the core model for 2-way interactions between depression, gender, age, and race/ethnicity. Again, no significant interactions were found, so none are reported. Using the total sample, we then took this core model and developed 4 separate models in which we added 1 of the 4 psychological and behavioral covariates to determine whether addition of the covariate into the core model significantly altered the relationship between baseline depressed mood and follow-up obesity. We also tested for interactions be-

tween depressed mood and the appropriate psychological and behavioral covariate in these models. Significant interactions are reported and were retained in models using the associated covariate. A final model including the 8 independent variables in the core model and all 4 psychological and behavioral covariates and any significant interactions was also run.

We next stratified the study sample by baseline weight status into those who were obese at baseline and those who were not to determine whether baseline obesity changed the effect of baseline depressed mood on follow-up weight status. We performed the logistic regression models described above on the sample not obese at baseline. For those obese at baseline, we used the same model building strategy to develop a series of linear regression models. Follow-up BMI z score was the dependent variable in these linear regression models because such a large proportion of those obese at baseline were obese at follow-up.

We also performed parallel logistic regression modeling to determine whether baseline obesity predicted follow-up depressed mood. In these logistic models, depressed mood at follow-up was the dependent variable. Independent variables included in the core model were age, gender, race/ethnicity, parent education, the number of parents in the house, and baseline CES-D score. The same psychological and behavioral covariates were used in these logistic models.

RESULTS

A description of the study sample is found in Table 1. At baseline, 12.9% were overweight, 9.7% were obese, and 8.8% had depressed mood. Baseline depressed mood was not significantly associated with baseline obesity—9.0% of those depressed at baseline were obese compared with 9.8% of the non-depressed and 8.2% of those obese at baseline were depressed compared with 8.9% of the nonobese ($P = .60$). In multivariable analysis, the number of obese parents was the strongest correlate of baseline obesity (odds ratio [OR]: 2.80; 95% confidence interval [CI]: 2.43, 3.80), followed by non-Hispanic black race/ethnicity, and male gender (Table 2). All 4 psychological and behavioral covariates were associated with baseline depressed mood but not with obesity at follow-up (Table 3).

At follow-up, 9.7% were obese and 8.9% were depressed. Obesity at follow-up was present in 79.7% of those obese at baseline, 18.5% of those overweight at baseline, and 1.8% of those who were of normal weight at baseline. In bivariate analyses, baseline depressed mood was associated with follow-up obesity: 12.4% of those with depressed mood at baseline were obese at follow-up compared with 9.4% of those who were not depressed at baseline ($P = .048$). This relationship remained significant in multivariable analyses with baseline depressed mood independently predicting follow-up obesity (OR: 2.05; 95% CI: 1.18, 3.56; Table 4). The magnitude of this effect was similar for the development of follow-up obesity among those not obese at baseline (OR: 2.05; 95% CI: 1.04, 4.06). Addition of adolescents' report of psychological and behavioral covariates (alone or together) to the core logistic regression model in the total sample did not alter the relationship between baseline depressed mood and follow-up obesity. Among those not obese at baseline, addition of cigarette smoking to the core model caused baseline depressed mood to lose significance (OR: 1.95; 95% CI: 0.94, 4.05). However, in the final model which adjusted for all covariates, including

TABLE 1. Description of Study Sample (*N* = 9374)

	Unweighted <i>N</i>	Percent
Male	4656	51.4
Race/ethnicity		
White, non-Hispanic	5873	74.7
Black, non-Hispanic	1881	13.6
Hispanic	1620	11.7
Educational level of the highest educated parent		
No high school degree	909	8.6
High school degree, General Educational Development certificate, or vocational training instead of high school	2166	24.5
Vocational school or some college	2695	29.5
College graduate	1563	16.3
Professional degree	1336	13.6
Missing	705	7.5
Income		
<1.5 × FPT	2261	24.0
1.5 × FPT to <2.5 × FPT	1949	20.8
2.5 × FPT* to <4 × FPT	2108	22.8
≥4 × FPT but not top 5% of US households	1433	16.4
Top 5% US households	320	3.3
Missing	1303	12.7
Depressed mood at baseline	856	8.8
Depressed mood at follow-up	855	8.9
Weight status at baseline		
Normal weight	7285	77.4
Overweight	1175	12.9
Obese	914	9.7
Obese at follow-up	892	9.7
Number of obese parents		
0	7183	77.2
1	1599	16.8
2	464	5.2
Missing	128	0.8
Two parents in the home	7442	79.7
Cigarette smoking		
Never	4091	41.9
Experimented	2800	30.3
<1 pack per wk	1301	14.3
≥1 pack per wk and <1 pack per d	780	8.7
≥1 pack per d	200	2.7
Missing	202	2.1

FPT indicates Federal poverty threshold adjusted for household size.

TABLE 2. Correlates of Baseline Obesity

	AOR	95% CI
Age	0.94	0.88, 1.01
Male gender	1.77	1.45, 2.17
Race		
White, non-Hispanic	1.00	
Black, non-Hispanic	1.94	1.51, 2.48
Hispanic	1.33	0.98, 1.80
Two parents in the home	1.06	0.81, 1.38
Number of obese parents	2.80	2.43, 3.22
Increasing parental education	0.76	0.69, 0.83
Depressed mood at baseline	0.90	0.62, 1.30

AOR indicates adjusted odds ratio from logistic regression model. These odds ratios are adjusted for all other variables in the model.

smoking, depressed mood maintained its significance (OR: 2.39; 95% CI: 1.05, 5.45).

Among those obese at baseline, linear regression analyses (Table 4) revealed that baseline depressed mood also predicted BMI *z* score at follow-up ($\beta = 0.11$; standard error [SE] $\beta = 0.05$; $P = .045$). This positive β suggests that depressed mood causes worsening obesity over the next year among obese adolescents. Controlling for psychological and behavioral covariates had some effect on this relationship. The relationship between baseline depressed

mood and follow-up BMI *z* score remained significant when adjusting for low physical activity and high delinquency. Both of these covariates were not significantly associated with BMI *z* score at follow-up. However, in models which adjusted for low self-esteem and cigarette smoking, the association of baseline depressed mood to follow-up BMI *z* score became nonsignificant. In the model adjusting for low self-esteem, neither depressed mood nor low self-esteem were associated with follow-up BMI *z* score. Although smoking at least a pack per day was associated with decreasing BMI *z* score at follow-up [$\beta_{\text{smoking at least a pack per day}} = -0.21$; SE $\beta = 0.10$; $P = .016$], a significant, positive interaction between depressed mood and smoking at least a pack per day was also present [$\beta_{\text{interaction}} = 0.50$; SE $\beta = 0.25$; $P = .048$]. This suggests that depressed obese youth who smoke heavily are at increased risk for worsening obesity.

In contrast to the findings regarding the effects of baseline depressed mood on follow-up obesity, baseline obesity did not predict follow-up depressed mood in either bivariate or multivariable analyses. Of those obese at baseline, 9.9% had depressed mood at follow-up compared with 8.7% of those who were

TABLE 3. Percentage of Subjects With Baseline Depressed Mood and Follow-up Obesity That Have the Identified Psychological and Behavioral Covariate at Baseline

	Baseline Depressed Mood			Follow-up Obesity		
	Yes (%)	No (%)	P Value	Yes (%)	No (%)	P Value
Cigarette smoking			<.0001			.72
Never	23.7	44.6		41.8	42.9	
Experimenter	27.1	31.3		33.7	30.6	
Current smoker, <1 pack per wk	23.6	13.8		13.5	14.7	
Current smoker, ≥1 pack per wk	18.9	7.9		8.8	8.9	
and <1 pack per d						
Current smoker, ≥1 pack per d	6.8	2.4		2.2	2.8	
Low self-esteem	62.7	17.0	<.0001	24.6	20.7	.16
High delinquency	46.6	19.8	<.0001	20.2	22.4	.26
Low physical activity	33.0	25.0	<.0001	28.2	25.1	.18

TABLE 4. Regression Models* Describing the Effect of Baseline Depressed Mood on Follow-up Obesity

	Total		Baseline Nonobese		Baseline Obese	
	AOR	95% CI	AOR	95% CI	β	SE
Core model	2.05†	1.18, 3.56	2.05†	1.04, 4.06	0.11†	0.05
Core model + low self esteem	2.01†	1.11, 3.64	2.23†	1.03, 4.83	0.08	0.06
Core model + cigarette smoking	2.02†	1.12, 3.65	1.95	0.94, 4.05	0.09	0.05
Core model + high delinquency	2.24†	1.27, 3.95	2.30†	1.14, 4.64	0.13†	0.06
Core model + low physical activity	2.03†	1.17, 3.52	2.00†	1.02, 3.94	0.11†	0.05
Core model + all covariates	2.17†	1.14, 4.11	2.39†	1.05, 5.45	0.08	0.06

* For total and baseline nonobese samples, logistic regression was used with obesity at follow as the dependent variable. For those obese at baseline, linear regression was used with BMI z score at follow as the dependent variable. The core model including depressed mood at baseline plus the following 7 independent variables: age, gender, race/ethnicity, parental obesity, parental education, 2 parents in the home versus other, and baseline BMI z score. The adjusted odds ratios (AOR) and adjusted regression coefficients (β) associated with depressed mood at baseline are presented for the logistic and linear regression models, respectively.

† $P < .05$.

not obese at baseline ($P = .43$) In the core model, increasing parental education was protective (OR: 0.86, 95% CI: 0.76, 0.91), whereas older age (OR: 1.11; 95% CI: 1.04, 1.19) and higher baseline depressive symptoms increased risk of depressed mood at follow-up (OR: 1.17; 95% CI: 1.16, 1.19). Baseline obesity was not significantly associated with follow-up depressed mood in the core model (OR: 1.16; 95% CI: 0.81, 1.65). Additional modeling revealed that low self-esteem, high delinquency, and smoking behaviors remained independent predictors of follow-up depressed mood in multivariable analyses, but baseline obesity remained nonsignificant in all models.

DISCUSSION

Using data from a nationally representative cohort of >9000 adolescents who were surveyed in 1995 and again 1 year later, we have shown that depressed mood at baseline was associated with the development of obesity in those not yet obese at baseline and with an increase in age-adjusted BMI in those already obese at baseline. Among those adolescents not yet obese at baseline, the odds of becoming obese in the next year were doubled if they had a depressed mood at baseline. This risk persisted after controlling for several factors related to both depressed mood and obesity, including low self-esteem, low levels of physical activity, parental obesity, and lower levels of parent education.

The association between obesity and various psychological states has been reported in the scientific literature over the last several decades.¹⁷ Many of these studies have examined the relationship be-

tween obesity and depression, but nearly all have been cross-sectional in design, and very few have involved children or adolescents. A prospective study design is required to help distinguish whether depressed mood could be a cause, and not just an effect, of obesity.

Strauss¹⁰ prospectively examined changes in self-esteem among obese and nonobese 9- and 10-year-old children. Obese children had worsening self-esteem over a 4-year period, and this decline in self-esteem was associated with increased feelings of sadness and loneliness in early adolescence. However, this study did not assess whether either worsening self-esteem or increased sadness was associated with the development or worsening of obesity over time. Depression and self-esteem are highly interrelated, both conceptually and clinically. In this study, we found that baseline low self-esteem was not associated with obesity at follow-up among adolescents who were not obese at baseline. However, among those obese at baseline, we were unable to separate the effects of depressed mood from low self-esteem, perhaps because of low discriminant validity of the measures used in Add Health.

To our knowledge, Pine et al^{19,32} have published the only 2 prospective studies examining the association between depression in childhood and adolescence and later obesity. These studies suggested that depression and conduct disorder were associated with the development and persistence of obesity in women, but not in men. In contrast, our study did not demonstrate a moderating effect of gender. In addition, we found that delinquent behaviors, used

by us as a proxy for conduct disorder, were not associated with obesity at follow-up and did not alter the depressed mood-obesity relationship. The differences between the design of Pine's study and our own were numerous and might explain the different results. Most important among these differences is the fact that their studies used a standard psychiatric interview to establish the diagnoses of depression and conduct disorder. However, these investigators were unable to control for the BMI of the child or adolescent at baseline or for parental obesity, as we did in our study.

Although we were able to control for parental obesity in this study, parental BMI was not available in Add Health. In fact, Add Health posed a number of limitations for studying the relationship between obesity and depression. For example, measured heights and weights were available only at follow-up, although self-reported height and weight was present in both waves. Our analyses suggest that there are some systematic differences in how individuals report their heights and weights. We did assess whether depressed mood at follow-up influenced reporting of height and weight and found no difference in reporting among those with depressed mood compared with those without. In addition, previous work suggests these differences do not lead to misclassification with regard to obesity status.⁴¹ Because we focused on the dichotomous outcome of obesity, this reporting bias has little effect, except in the analyses of worsening BMI among those obese at baseline. Those obese at baseline may underreport their BMI at follow-up, and they may do so more if they are more obese at baseline. Another limitation relates to the psychological and behavioral measures in Add Health. Although these were drawn from validated instruments, the survey does not include complete scales. Thus, there is little data on the validity and reliability of these measures. The CES-D is perhaps one of the strongest in the survey, as 16 of the 20 items are included verbatim, but it is not a diagnostic tool for major depressive disorder. Last, Add Health is a study of in-school youth. Youth with chronic disease, such as depression and obesity, may be less likely to stay in school. Despite these limitations, Add Health is one of the largest, most recent epidemiologic studies of American youth and provides valuable data with which to understand the complexities between depression, obesity, and adolescent development.

The relationship between depression and obesity during adolescence is important to understand because this is the developmental period in which both conditions may have their origins. Furthermore, both obesity⁴² and depression^{43–45} are increasing worldwide, and the reasons for these trends are unknown. Obesity is a biologically heterogeneous disorder.⁴⁶ It is likely that there is a subgroup of individuals for whom depressed mood is an important risk factor for development of obesity. These may be the same individuals who report increased, rather than decreased, appetite with the onset of depression or those who have binge eating disorder, a group in

whom depression is more common.⁴⁷ This subgroup may also represent a population for whom treatment of depressed mood could alter the course and/or development of obesity. Once obesity develops at any age, it is very resistant to treatment.^{48,49} In contrast, there are promising pharmacologic and non-pharmacologic approaches to treating depression in adults and children.^{50–54} For individuals for whom depressed mood increases risk of obesity, treatment of the depression may also prevent development of weight gain.

There is evidence of shared neurobiological mechanisms between obesity and depression, particularly in regard to serotonin and its metabolites,^{55,56} as well as evidence linking depressive symptoms and body weight to alterations in hypothalamic-pituitary-adrenal axis functioning, particularly the chronic excessive secretion of cortisol.^{57,58} Chronic stress is also believed to increase abdominal obesity and its related adverse metabolic consequences, such as hypertension, insulin resistance, and dyslipidemia.^{59,60} This stress, which may begin in early childhood and could be linked to a hostile social environment, may lead to changes in brain morphology and the neuroendocrine axis that can cause both obesity and depression.^{57,58,61} Based on these shared neuroendocrine pathways, a theoretical basis for treating obesity with antidepressants has already been suggested.⁶²

Despite the appreciation that the antecedents of obesity and depression can occur before adulthood and that both are conditions governed by the brain, little attention has been given to secular trends in the social factors that may underlie the increasing prevalence of both conditions. We postulate that there may be at least 3 such secular trends which may be interrelated—declining levels of physical activity,^{63,64} increasing social isolation,⁶⁵ and increasing socioeconomic inequality.^{57,66–69} In addition to increasing chronic levels of stress, lower socioeconomic status may lead to increased depression and obesity by constraining opportunities of physical activity because of unsafe neighborhoods or neighborhoods that lack resources such as parks, playgrounds, and organized team sports. Such structural effects also increase social isolation. Social policies, such as increasing availability of quality after school programs that increase opportunities for safe outdoor activity and peer interaction, may counteract these processes and help stem the rising rates of depression and obesity among youth.

CONCLUSION

We have shown that depressed mood in adolescence is associated with an increased risk for the development and persistence of obesity over 1 year. Understanding the neurobiological pathways and socio-environmental determinants that are shared by both depression and obesity may help inform the prevention and treatment of 2 chronic conditions with early life antecedents and the potential for life long-term morbidity.

ACKNOWLEDGMENTS

This research is based on data from the Add Health project, a program project designed by J. Richard Udry (PI) and Peter Bearman, and funded by grant P01-HD31921 from the National Institute of Child Health and Human Development to the Carolina Population Center, University of North Carolina at Chapel Hill. Cooperative funding was provided by the National Cancer Institute; the National Institute of Alcohol Abuse and Alcoholism; the National Institute of Deafness and Other Communication Disorders; the National Institute of Drug Abuse; the National Institute of General Medical Sciences; the National Institute of Mental Health; the National Institute of Nursing Research; the Office of AIDS Research, National Institutes of Health; the Office of Behavior and Social Science Research, National Institutes of Health; the Office of the Director, National Institutes of Health; the Office of Research on Women's Health, National Institutes of Health; the Office of Population Affairs, Department of Health and Human Services; the National Center for Health Statistics; Centers for Disease Control and Prevention, Department of Health and Human Services; the Office of Minority Health, Centers for Disease Control and Prevention, Department of Health and Human Services; the Office of Minority Health, Office of the Assistant Secretary of Health, Department of Health and Human Services; the Office of Assistant Secretary of Planning and Evaluation, Department of Health and Human Services; and the National Science Foundation.

REFERENCES

- Koplan JP, Dietz WH. Caloric imbalance and public health policy. *JAMA*. 1999;282:1579–1581
- US Department of Health and Human Services. *The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity*. Rockville, MD: Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001:1–35
- Allison DB, Fontaine KR, Manson JE, Stevens J, VanTallie TB. Annual deaths attributable to obesity in the United States. *JAMA*. 1999;282:1530–1538
- Must A, Spadano J, Coakley EH, Field AE, Colditz G, Heitz WH. The disease burden associated with overweight and obesity. *JAMA*. 1999;282:1523–1529
- Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP. The spread of the obesity epidemic in the United States, 1991–1998. *JAMA*. 1999;282:1519–1522
- Dietz WH. Critical periods in childhood for the development of obesity. *Am J Clin Nutr*. 1994;59:955–959
- Gortmaker SL, Must A, Perrien JM, Sobol AM, Dietz W. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med*. 1993;329:1008–1012
- Sargent JD, Blanchflower DG. Obesity and stature in adolescence and earnings in young adulthood. Analysis of a British birth cohort. [see comments]. *Arch Pediatr Adolesc Med*. 1994;148:681–687
- French SA, Story M, Perry CL. Self-esteem and obesity in children and adolescents: a literature review. *Obes Res*. 1995;3:479–490
- Strauss RS. Childhood obesity and self-esteem. *Pediatrics*. 2000;105(1). Available at: www.pediatrics.org/cgi/content/full/105/1/e15
- Sheslow D, Hassink S, Wallace W, DeLancey E. The relationship between self-esteem and depression in obese children. *Ann N Y Acad Sci*. 1993;699:289–291
- 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;90:251–257
- Wallace WJ, Sheslow D, Hassink S. Obesity in children: a risk for depression. *Ann N Y Acad Sci*. 1993;699:301–303
- Istvan J, Zavala K, Weidner G. Body weight and psychological distress in NHANES I [see comments]. *Int J Obes Relat Metab Disord*. 1992;16:999–1003
- Britz B, Siegfried W, Ziegler A, et al. Rates of psychiatric disorders in a clinical study group of adolescents with extreme obesity and in obese adolescents ascertained via a population based study. *Int J Obes Relat Metab Disord*. 2000;24:1707–1714
- Erickson SJ, Robinson TN, Haydel KF, Killen JD. Are overweight children unhappy? Body mass index, depressive symptoms, and overweight concerns in elementary school children. *Arch Pediatr Adolesc Med*. 2000;154:931–935
- Friedman MA, Brownell KD. Psychological correlates of obesity: moving to the next research generation. *Psychol Bull*. 1995;117:3–20
- Hammar SL, Campbell MM, Campbell VA, et al. An interdisciplinary study of adolescent obesity. *J Pediatr*. 1972;80:373–383
- Pine DS, Goldstein RB, Wolk S, Weissman MM. The association between childhood depression and adulthood body mass index. *Pediatrics*. 2001;107:1049–1056
- Bearman PS, Jones J, Udry JR. The National Longitudinal Study of Adolescent Health: Research Design; 1997. Available at: <http://www.cpc.unc.edu/projects/addhealth/design.html>
- Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. *Pediatrics*. 1998;101:497–505
- Rosner B, Prineas R, Loggie J, Daniels SR. Percentiles for body mass index in US children 5 to 17 years of age. *J Pediatr*. 1998;132:211–222
- Fleming JE, Offord DR. Epidemiology of childhood depressive disorders: a critical review. *J Am Acad Child Adolesc Psychiatry*. 1990;29:571–580
- National Center for Health Statistics. *CDC Growth Charts: United States*. 2001. Available at: <http://www.cdc.gov/nchs/about/major/nhanes/growthcharts/datafiles.htm>
- World Health Organization. *Physical Status: The Use and Interpretation of Anthropometry*. Geneva, Switzerland: WHO; 1995
- Radloff L. The CES-D scale: a self report depression scale for research in the general population. *Appl Psychol Meas*. 1977;1:385–401
- Radloff L. The use of the Center for Epidemiologic Studies Depression Scale in adolescent and young adults. *J Youth Adolesc*. 1991;20:149–166
- Roberts RE, Lewinsohn PM, Seeley JR. Screening for adolescent depression: a comparison of depression scales. *J Am Acad Child Adolesc Psychiatry*. 1991;30:58–66
- Goodman E. The role of socioeconomic status gradients in explaining differences in US adolescents' health. *Am J Public Health*. 1999;89:1522–1528
- Resnick MD, Bearman PS, Blum RW, et al. Protecting adolescents from harm: findings from the National Longitudinal Study of Adolescent Health. *JAMA*. 1997;278:823–832
- Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. Washington, DC: American Psychiatric Association; 1994
- Pine DS, Cohen P, Brook J, Coplan JD. Psychiatric symptoms in adolescence as predictors of obesity in early adulthood: a longitudinal study. *Am J Public Health*. 1997;87:1303–1310
- Sallis J, Patrick K. Physical activity guidelines for adolescents: consensus statement. *Pediatr Exerc Sci*. 1994;6:302–314
- Gordon-Larsen P, McMurray RG, Popkin BM. Adolescent physical activity and inactivity vary by ethnicity: the National Longitudinal Study of Adolescent Health. *J Pediatr*. 1999;135:301–306
- Gordon-Larsen P, McMurray RG, Popkin BM. Determinants of adolescent physical activity and inactivity patterns. *Pediatrics*. 2000;105(6). Available at: www.pediatrics.org/cgi/content/full/105/6/e83
- Brogan DJ. Pitfalls of using standard statistical software packages for sample survey data. In: Armitage P, Colton T, eds. *Encyclopedia of Biostatistics*. Vol. 5. New York, NY: John Wiley & Sons; 1998:4167–4174
- SPSS. *SPSS Base 10.0 User's Guide*. Chicago, IL: SPSS, Inc; 1999
- Shah BV, Barnwell BG, Bieler GS. *Sudan User's Manual Release 7.5*. Research Triangle Park, NC: Research Triangle Institute; 1997
- Libratos P, Link BG, Kelsey JL. The measurement of social class in epidemiology. *Epidemiol Rev*. 1988;10:87–121
- Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88:1973–1988
- Goodman E, Hinden BR, Kandelwal S. Accuracy of teen and parental reports of obesity and body mass index. *Pediatrics*. 2000;106:52–58
- World Health Organization. *Obesity: Preventing and Managing the Global Epidemic*. 1998. Available at: <http://www.who.int/ncd/cvd/obesityreport.pdf>
- Murray CJL, Lopez AD. *Global Burden of Disease and Injury*. Cambridge, MA: Harvard University Press; 1996
- Cross-National Collaborative Group. The changing rate of major depression. Cross-national comparisons. *JAMA*. 1992;268:3098–3105
- Klerman GL, Weissman MM. Increasing rates of depression. *JAMA*. 1989;261:2229–2235
- Perusse L, Chagnon YC, Weisnagel SJ, et al. The human obesity gene map: the 2000 update. *Obes Res*. 2001;9:135–169
- Smith DE, Marcus MD, Lewis CE, Fitzgibbon M, Schreiner P. Prevalence of binge eating disorder, obesity, and depression in a biracial cohort of young adults. *Ann Behav Med*. 1998;20:227–332
- NIH Technology Assessment Conference Panel. Methods for voluntary weight loss and control. *Ann Intern Med*. 1993;119:764–770
- National Institutes of Health. *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report*. 1998. Available at: http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.pdf. Accessed July 11, 2002
- Agency for Health Care Policy and Research. *Treatment of Depression:*

Newer Pharmacotherapies: Evidence Report/Technology Assessment Number 7. Rockville, MD: Agency for Health Care Policy and Research; 1999

51. Practice parameters for the assessment and treatment of children and adolescents with depressive disorders. AACAP. *J Am Acad Child Adolesc Psychiatry*. 1998;37:63S–83S
52. Schulberg HC, Katon W, Simon GE, Rush AJ. Treating major depression in primary care practice: an update of the Agency for Health Care Policy and Research Practice Guidelines. *Arch Gen Psychiatry*. 1998;55:1121–1127
53. Birmaher B, Ryan ND, Williamson DE, et al. Childhood and adolescent depression: a review of the past 10 years. Part I. *J Am Acad Child Adolesc Psychiatry*. 1996;35:1427–1439
54. Birmaher B, Ryan ND, Williamson DE, Brent DA, Kaufman J. Childhood and adolescent depression: a review of the past 10 years. Part II. *J Am Acad Child Adolesc Psychiatry*. 1996;35:1575–1583
55. Wurtman JJ. Depression and weight gain: the serotonin connection. *J Affect Disord*. 1993;29:183–192
56. Hoebel BG, Rada PV, Mark GP, Pothos EN. Neural systems for reinforcement and inhibition of behavior: relevance to eating, addiction, and depression. In: Kahneman D, Diener E, Schwarz N, eds. *Well-Being: The Foundations of Hedonic Psychology*. New York, NY: Russell Sage Foundation; 1999:558–572
57. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998;338:171–179
58. McEwen BS. Allostasis and allostatic load: implications for neuropsychopharmacology [see comments]. *Neuropsychopharmacology*. 2000;22:108–124
59. Chrousos GP. The role of stress and the hypothalamic-pituitary-adrenal axis in the pathogenesis of the metabolic syndrome: neuro-endocrine and target tissue-related causes. *Int J Obes Relat Metab Disord*. 2000;24:S50–S55
60. Bjorntorp P, Rosmond R. Neuroendocrine abnormalities in visceral obesity. *Int J Obes Relat Metab Disord*. 2000;24:S80–S85
61. Gohil BC, Rosenblum LA, Coplan JD, Kral JG. Hypothalamic-pituitary-adrenal axis function and the metabolic syndrome X of obesity. *CNS Spectrums*. 2001;6:1–8
62. Rosmond R, Bjorntorp P. The role of antidepressants in the treatment of abdominal obesity. *Med Hypotheses*. 2000;54:990–994
63. Hu PS, Young JR. *Summary of Travel Trends. 1995 Nationwide Personal Transportation Survey*. Washington, DC: US Department of Transportation, Federal Highway Administration; 1999:1–54
64. Centers for Disease Control and Prevention. Fact sheet: youth risk behavior trends. 2001. Available at: <http://www.cdc.gov/nccdphp/dash/yrbts/00binaries/99yrbstrends.pdf>
65. Putnam RD. *Bowling Alone: The Collapse and Revival of American Community*. New York, NY: Simon and Schuster; 2000
66. Rosmond R, Bjorntorp P. The hypothalamic-pituitary-adrenal axis activity as a predictor of cardiovascular disease, type 2 diabetes and stroke. *J Intern Med*. 2000;247:188–197
67. Brunner E. Stress and the biology of inequality. *BMJ*. 1997;314:1472–1476
68. Herman JP, Cullinan WE. Neurocircuitry of stress: central control of the hypothalamo-pituitary-adrenocortical axis. *Trends Neurosci*. 1997;20:78–84
69. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Ann New York Acad Sci*. 1999;896:30–47

WHAT SEPARATES MAN AND APE THESE DAYS IS THE THUMB ACTION

“Kids and the Gadget-Crazed Use Theirs for E-mail; Finessing the ‘Splat’”

“In Tokyo, so many kids are pounding at new electronic gadgets with their thumbs they’re known as ‘oyayubi sedai’—the ‘thumb generation.’ Nokia Corp. sponsored a contest for the fastest Finnish thumbs, where 2700 players competed to thumb tap the highest score in the “Snake” game included on Nokia phones. AT&T Wireless Services Inc. is running ads featuring powerful thumbs that poke through mittens, boxing gloves and golf gloves, ready for action on a mobile phone.

Being ‘all thumbs’ used to mean you were clumsy. But phones, wireless e-mail devices, and all the other hand-held gadgets featuring ‘thumb boards’ are turning thumbs into universal index fingers for a generation of teenagers, young adults and high-tech businesspeople.”

Fowler GA. *Wall Street Journal*. April 17, 2002

Noted by JFL, MD

