Psychiatric Symptoms in Adolescence as Predictors of Obesity in Early Adulthood: A Longitudinal Study

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ABSTRACT

Objectives. This study examined the longitudinal relationship between psychopathology and obesity in young adulthood.

Methods. More than 700 youth in a population-based sample were psychiatrically assessed in 1983 (mean age = 14 years) and 1992 (mean age = 22 years). Self-reported body mass index (BMI) in 1992 was regressed on measures of depression and conduct disorder as well as a set of covariates including indices of physical health, social class, intelligence, and cigarette and alcohol use. Associations were examined with BMI treated as a continuous variable and with a binary index of obesity derived from the BMI distribution in each gender.

Results. BMI in young adults was positively related to a number of covariates. With all covariates controlled, BMI was inversely related to adult depressive symptoms in males but not females. BMI was positively related to adolescent symptoms of conduct disorder in both sexes. Similar associations were found between psychiatric symptoms and obesity.

Conclusions. Conduct disorder symptoms in adolescence predicted BMI and obesity in early adulthood. These associations remained after controlling for factors that can affect the association between psychopathology and obesity. (*Am J Public Health.* 1997;87:1303–1310)

Introduction

Concern with medical correlates of obesity has risen with accumulating evidence of associations between obesity and physical health.[1–7] Renewed interest in psychiatric correlates of obesity may follow recent research findings. Epidemiological studies have noted risk factors that are common to psychopathology in youth and obesity in adulthood, including poverty, low IQ, and harsh parenting.[8–13] Biological studies suggest that abnormalities in brain serotonin may result in a predisposition to some forms of psychopathology and obesity.[14–23] Epidemiological research, however, provides meager evidence of cross-sectional associations between psychopathology and obesity.[24] In this report, we examine prospective relationships between psychopathology in youth and obesity in early adulthood.

There are disparities between clinic-based and epidemiological studies in this area. While clinic-based studies have found an association between obesity and psychopathology, particularly depression,[25–27] these associations appear weak or inconsistent in epidemiological studies.[24,28–35] In fact, the only replicated association in epidemiological research is an *inverse* relationship between current depressive symptoms and obesity, particularly among men.[29–32] While differences among studies may derive from referral biases, the cross-sectional nature of most epidemiological studies could also be important. For example, acute depression often produces weight loss, and this might explain inverse cross-sectional relationships between depression and obesity.[33,36,37]

Because virtually all studies in this area have been cross-sectional, prospective epidemiological studies are needed. Studies among youth may be particularly important, since two correlates of psychopathology in youth, low social class of a child's parents and school failure in childhood, consistently predict obesity in adulthood.[10–12,38,39] The association between obesity and school failure has been attributed to other correlates of psychopathology in youth, including low IQ and childhood aggression.[8–11,38,39] Inadequacies in children's social or physical rearing environments may also predict adult obesity.[10,13] For example, a recent study found that children who did not receive harmonious support from parents faced a sevenfold increased risk for obesity as adults.[13] Such environmental risk factors consistently relate to psychopathology, particularly conduct disorder, in youth.[40,41] These suggestive prospective findings have not been pursued in psychiatric epidemiological studies of youth, which remain exclusively cross-sectional.

Methodological factors may contribute to negative findings on the relationship between psychopathology and obesity in cross-sectional studies. First, smoking, alcohol use, and medical illness may attenuate associations between obesity and psychiatric morbidity, much as they attenuate associations with medical morbidity.[1,7,33,34] These variables may attenu-

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TABLE 1—Sample Characteristics: 776 Adolescents Who Were Psychiatrically Assessed in Adolescence and Early Adulthood

	Full Adolescent Sample[a] (n = 776)	Adolescent Sample[b] (n = 644)	Young Adult Sample[b] (n = 644)
Mean age, y	14.1	13.8	22.0
Age range, y	9–18	9–18	17–28
Body mass index, kg/m^2			
Males	...	...	24.6 ± 4.1
Females	...	...	23.1 ± 4.4
Male, %	50	48	48
IQ	98 ± 14.2	100.7 ± 14.8	...
Positive parental sociopathy, %	17	18	...
White, %	90	91	91

[a]Includes all subjects in time 1 (1983) sample.
[b]Sample used for body mass index analyses. Includes only subjects with complete time 1 (1983) and time 2 (1994) data.

ate associations because they relate to psychopathology and may produce changes in weight. Existing epidemiological studies rarely control for these confounders. Second, studies often rely on brief rating scales to assess psychiatric symptoms.[9,28–35] At least in youth, standardized multi-informant interviews provide more sensitive assessments.[42]

Finally, interest in associations between psychopathology and obesity may follow recent research on the serotonergic nervous system. Serotonin has been implicated in the development of both obesity[14–19] and psychiatric syndromes, such as conduct disorder, which are characterized by a pattern of recurrent impulsive aggression.[20–23] Few epidemiological studies have examined associations between obesity and such syndromes; the only relevant study in youth found relationships between teacher-rated aggression and concurrent as well as later obesity.[9] Results in this study, however, were based on brief global ratings by teachers, were not adjusted for potential confounding variables, and were somewhat inconsistent across aggression measures.

In summary, prospective epidemiological studies have found that correlates of youth psychopathology are associated with obesity, but, to our knowledge, no epidemiological study has documented direct associations between psychopathology (defined according to the *Diagnostic and Statistical Manual of Mental Disorders* [DSM]) in youth and obesity, either concurrently or prospectively. In this study, we describe a prospective relationship between psychopathology in youth and obesity in early adulthood.

Methods

Sample

Details of the sample used in this study are described elsewhere.[42–44] The study is based on a cohort of 776 adolescents who were psychiatrically assessed in 1983 (time 1), when they were 9 to 18 years old, and in 1992 (time 2), when they were 17 to 28 years old. For convenience, time 1 is referred to as adolescence and time 2 is referred to as young adulthood. Adolescents were selected by means of a multistaged random design with complete enumeration from two semirural upstate New York counties. Complete information on sampling procedures and losses to follow-up is provided elsewhere.[42]

Assessment

Assessment protocols were approved by appropriate institutional review boards. All subjects provided informed consent.

Obesity. Body mass index (BMI) is frequently used to quantify obesity in epidemiological research, partially because of the ease with which it is measured.[1–7,32,34] Despite some disadvantages as an obesity measure, BMI correlates with more accurate measures of obesity and is consistently related to morbidity from illness associated with obesity.[1–7,45]

Height and weight were assessed by self-report in young adulthood. BMI was calculated (BMI = weight [kg] divided by height [m] squared). At time 1, weight was assessed only in a subset of the sample who reported a recent loss or gain in weight. We therefore did not examine associations using time 1 BMI. Although self-reported BMI is measured with some systematic biases, validation studies suggest that the magnitude of the bias is too small to affect conclusions about associations in large-scale epidemiological studies.[46–48]

Psychopathology. Interviews with parents and adolescents, administered by trained lay interviewers, assessed psychopathology in youth at time 1. Only youth were interviewed for psychopathology at time 2. Symptoms of DSM-III or DSM-III-R diagnoses were assessed with the use of modifications of the Diagnostic Interview Schedule for Children, as described elsewhere.[42–44] Psychopathology was assessed both as disorders and by means of diagnostic criteria–based symptom scales. Symptom scales incorporated items on impairment associated with each syndrome. Evidence documenting the reliability and validity of this assessment is presented elsewhere.[42–44]

We restricted our analyses to two specific psychiatric domains. First, on the basis of extensive clinical research, we examined relationships between major depressive disorder and BMI. Second, because epidemiological studies suggest that adult obesity is predicted by correlates of conduct disorder, including youth aggression, school failure, and parental maltreatment, we examined relationships between conduct disorder and BMI. In nonreferred samples, symptoms of depression[49,50] or conduct disorder[41,51,52] lie on a continuum. Therefore, we used continuous symptom scales, rather than the diagnoses, for both major depression and conduct disorder.

Covariates

Age, gender, and ethnicity served as covariates. Parental social class at time 1 served as a covariate, assessed with a composite measure (described elsewhere) combining parental income and education.[42] A social class measure was not used for young adults, who often were no longer living with parents but were in transitional socioeconomic states. An index of parental sociopathy was included as a covariate, stimulated by recent research implicating this factor in the risk for obesity.[53] This measure was formed by combining dichotomous measures for each parent.[44] Eight items on physical health status, as described elsewhere,[43,44] were summed to create continuous scales

TABLE 2—Pearson Correlations among Adult Body Mass Index, Demographic Variables, Covariates, and Psychiatric Scale Scores: 644 Subjects Assessed in Adolescence and Early Adulthood

	BMI_2	Age_2	Sex	Ethn	SES_1	Alc_2	Tob_2	Tob_1	$Socio_1$	IQ_1	III_2	III_1	Dep_2	CD_2	Dep_1
Age_2	+.12**														
Sex	+.17***	+.00													
Ethn	+.13***	−.02	−.01												
SES_1	−.10**	+.03	+.06	−.27***											
Alc_2	+.00	+.20***	+.28***	−.11**	+.20***										
Tob_2	−.01	+.02	−.02	+.03	−.16***	+.16***									
Tob_1	−.02	+.34***	−.13**	−.04	−.14***	+.07	+.36***								
$Socio_1$	−.02	+.00	−.05	+.15***	−.25***	−.04	+.15***	+.09*							
IQ_1	+.00	+.03	+.13***	−.18***	+.40***	+.19***	−.11**	−.11**	−.14***						
III_2	+.12***	−.06	−.24***	−.04	−.13***	−.11**	+.08*	+.11**	−.02	−.01					
III_1	+.15***	+.03	−.06	+.05	−.15***	−.04	+.05	+.10**	+.05	−.06	+.26***				
Dep_2	−.05	−.03	−.18***	−.03	−.06	+.12**	+.11**	+.11**	+.06	+.00	+.21***	+.23***			
CD_2	−.01	−.17***	+.22***	+.08*	−.05	+.31***	+.24***	+.01	+.10**	+.03	−.01	+.04	+.26***		
Dep_1	+.09*	−.01	−.18***	+.09**	−.21***	−.07	+.07*	+.23***	+.16***	−.16***	+.17***	+.39***	+.30***	+.06	
CD_1	+.17***	+.03	+.22***	+.19***	−.22***	+.13***	+.22***	+.30***	+.20***	−.14***	+.00	+.11**	+.09*	+.29***	+.37***

Note. BMI = body mass index; sex = gender (0 = female, 1 = male); Ethn = ethnicity (0 = White, 1 = other); SES = socioeconomic status; Alc = alcohol use; Tob = tobacco use; Socio = family history of sociopathy (0 = negative, 1 = positive); IQ = measured intelligence quotient; III = physical health scale; Dep = major depressive symptoms; CD = conduct disorder symptoms. Subscript refers to adolescent (1) or adulthood (2) measure.
*$P < .05$; **$P < .01$; ***$P < .005$.

that indexed physical health in adolescence and in early adulthood. Poor physical health may predispose to obesity through inactivity but may also predispose toward thinness owing to illness-related weight loss. Since childhood IQ has been related to BMI, we also included this measure as a potential confounder, using a picture vocabulary test in adolescence. Finally, smoking and alcohol use were assessed by self-report, as described in detail elsewhere, and served as covariates.[42–44]

Data Analysis

We examined associations both with BMI as a continuous variable and with obesity (defined by a cutoff on the BMI distribution), since these two analytic approaches answer conceptually different questions. The continuous approach examines the association between psychiatric symptoms and normal variations in relative weight, as distributed throughout the population. The categorical approach, in contrast, considers the association with obesity as an abnormal condition. For categorical analyses, subjects were dichotomized at the 80th percentile of the BMI distribution for each gender (25.86 kg/m^2 in females, 27.31 kg/m^2 in males). The 80th percentile was selected because BMI values approximated commonly used cutoffs for definition of obesity among young adults.[5,7–11]

Univariate associations with BMI treated continuously were quantified with Pearson correlations. This procedure provides estimates of univariate associations that can be compared with findings from previous studies. The correlation matrix among all variables used in this study is provided to facilitate an appraisal of the degree to which multicollinearity complicates the results from multivariate analyses. Univariate associations with obesity were quantified with odds ratios. Multivariate associations were examined by regressing young adulthood BMI on a series of psychiatric scales and potential confounding variables, using both linear and logistic regression.[54] Various multivariate models were fitted to consider only cross-sectional associations, to consider associations with discrete psychiatric scales, to consider the influence of multicollinearity, and to ensure that results were statistically stable.

Since gender differences are often found in the correlates of obesity,[7,12] associations with BMI or obesity were examined separately for males and females on an a priori basis. Significant gender differences in the results are indicated by interactions between sex and other independent variables. Similarly, interaction terms initially were fitted between age and each independent measure. Since no significant interactions with age were found, these terms were not included in our models.

To facilitate interpretations of the regression coefficients, a number of the scaled independent measures (smoking, alcohol use, social class, physical health, and psychiatric scales) were converted to standardized scores (mean = 0, SD = 1) before being entered into the regression. All linear regression models appeared satisfactory after an examination of residual plots, a search for outliers, and an examination of the overall fit. All tests are two-tailed with $\alpha = .05$. Trends to $P = .10$ are also noted.

Results

Sample Characteristics

Of the 776 youth first assessed in 1982, 716 had full psychiatric assessments in 1992 (92% of living subjects). While detailed discussions of losses to follow-up are found elsewhere,[43,44] it should be noted that the demographic features of the sample remained representative of the larger population from which it was drawn. Complete data were available for 644 subjects (83% of sample); most of the missing data were for IQ. We examined associations after excluding variables with missing values. Because results were similar across analyses, we present data using an n of 644. Demographic and psychiatric characteristics of the sample at each assessment have also been reported elsewhere[43,44] but are summarized in Table 1. Table 1 also presents summary data for the 644 subjects used in the current analyses.

TABLE 3—Unadjusted Odds Ratios (ORs) (95% Confidence Intervals [CIs]) for Obesity in Adulthood: Associations with Demographic Variables, Covariates, and Psychiatric Scale Scores Measured in Adolescence and Early Adulthood

	Males (n = 310), OR (95% CI)	Females (n = 334), OR (95% CI)	Total Sample (n = 644), OR (95% CI)
Demographic predictors			
Age[a]	1.35 (0.83, 2.20)	1.22 (0.75, 1.99)	1.28 (0.96, 1.72)
Non-White ethnicity[b]	2.48 (1.11, 5.55)	1.75 (0.77, 3.99)	2.10 (1.19, 3.70)
Predictors measured in early adulthood			
Covariates[c]			
Poor physical health	1.79 (1.03, 3.09)	2.32 (1.39, 3.86)	1.90 (1.33, 2.70)
Smoking	0.83 (0.72, 1.17)	1.08 (0.65, 1.80)	0.96 (0.68, 1.37)
Alcohol use	0.79 (0.47, 1.31)	0.43 (0.21, 0.72)	0.67 (0.45, 0.99)
Psychiatric scales[c]			
Depression	0.37 (0.16, 0.87)	1.65 (1.03, 2.64)	1.04 (0.70, 1.54)
Conduct disorder	0.77 (0.46, 1.28)	1.72 (0.81, 3.61)	0.96 (0.70, 1.31)
Predictors measured in adolescence			
Covariates			
Poor physical health[c]	2.01 (1.16, 3.49)	2.32 (1.39, 3.86)	2.05 (1.44, 2.92)
Smoking[c]	0.82 (0.37, 1.79)	1.11 (0.69, 1.45)	0.95 (0.65, 1.42)
Social class	0.55 (0.32, 0.95)	0.39 (0.23, 0.68)	0.47 (0.32, 0.69)
IQ[c]	1.35 (0.75, 2.43)	0.55 (0.31, 0.99)	0.74 (0.55, 1.33)
Positive parental sociopathy[b]	1.79 (0.94, 3.41)	1.38 (0.75, 2.56)	1.54 (0.98, 2.41)
Psychiatric scales[c]			
Depression	1.46 (0.78, 2.74)	3.06 (1.91, 4.91)	1.75 (1.23, 2.49)
Conduct disorder	1.75 (1.09, 2.80)	2.32 (1.29, 4.17)	1.93 (1.36, 2.75)

[a]Odds ratio considers magnitude of association with obesity for one group of subjects who are 5 years older than a second group of subjects (i.e., subjects who are 25 as opposed to 20 years old).
[b]Dichotomous measure, such that odds ratio is across the two groups.
[c]Continuous measure, such that odds ratio is for a 2-SD difference on the continuous scale (i.e., 1 SD above vs 1 SD below mean).

Univariate Associations

Continuous variables. As shown in Table 2, a higher BMI in early adulthood was correlated with eight independent measures: increasing age, male gender, non-White ethnicity, lower social class during adolescence, increasing degrees of physical illness in both adolescence and adulthood, increasing depressive symptoms in adolescence, and increasing conduct disorder symptoms in adolescence. While statistically significant, none of these correlations were large in magnitude.

We examined the correlation matrix for each gender and tested for interactions (data not shown). Similar associations were found across gender with four exceptions: BMI was inversely related to depression in adulthood among males ($r = -.19$) but not females, and BMI was inversely related to adult alcohol use ($r = -.16$), social class ($r = -.20$), and IQ ($r = -.14$) among females but not males. The partial correlation between BMI and IQ in females essentially disappeared ($r = -.05$) when social class was controlled for, but the partial correlation between BMI and social class remained ($r = -.16$) when IQ was controlled for. Table 2 also shows correlations among the independent measures, many of which were significant, as discussed elsewhere.[42–44] Finally, we examined potential curvilinear relationships with BMI, using quadratic and cubic terms for each independent measure. No such relationships were found.

Univariate associations with obesity. As shown in Table 3, for males, for females, and for the entire sample, obesity was inversely related to social class and positively related to poor adult physical health, poor adolescent physical health, and adolescent conduct disorder. Obesity was related to race/ethnicity both in males and in the entire sample but not in females alone. Obesity was related to adolescent depression both in females and in the entire sample but not in males alone. Obesity was related to IQ only in females. While obesity was associated with depression in adulthood in both genders, this association was negative in males and positive in females. The gender × adult depression ($\chi^2 = 8.8$, $P < .001$) and gender × IQ ($\chi^2 = 4.7$; $P < .05$) interactions, but no other interactions, were significant predictors of obesity. When the analyses were covaried for social class, the association between obesity and low IQ in females disappeared (data not shown). Although these associations were consistently significant on statistical grounds, point estimates for most unadjusted odds ratios were not large (usually between 1.5 and 2.5).

Multivariate Linear Regression

Because many independent variables were correlated, various regression models were fitted in an effort to evaluate the influence of multicollinearity. Results for psychiatric scales were essentially unchanged among analyses entering different combinations of covariates and psychiatric scales; point estimates were very similar, with somewhat different standard errors and comparable levels of significance. For completeness, in Table 4 we present results for separate linear regression models examining associations with adult BMI using only adult independent measures and using only adolescent independent measures. We also present results for a model including both adolescent and adult measures, so that coefficients with adult measures represent associations for change in the independent measure. Results from other analyses are available on request.

Relationships with covariates. As shown in Table 4, across analyses, eight covariates were significantly related to BMI in young adulthood: age, gender, adolescent and adult health, adolescent smoking, ethnicity, social class in females, and adult alcohol use in females. In the model entering all adult and adolescent covariates simultaneously, the regression step with covariates accounted for 13% of the variance in adult BMI ($F[12,631] = 7.7$; $P < .001$). Poor physical health at both ages, as indicated by a higher score on the physical health scales, was associated with a higher BMI in adulthood. Smoking status in adolescence predicted lower adult BMI. Increasing age, non-White ethnicity, and male gender

TABLE 4—Predictors of Body Mass Index among 644 Young Adults: Associations with Demographic Variables, Covariates, and Psychiatric Scale Scores Measured in Adolescence and Early Adulthood

	Relationship with Adulthood Predictors Only		Relationship with Adolescent Predictors Only		Relationship with Adulthood and Adolescent Predictors	
	Regression Coefficient [a]	*t* (*df* = 633)	Regression Coefficient[a]	*t* (*df* = 632)	Regression Coefficient[a]	*t* (*df* = 626)
Demographic predictors						
Age, y	+0.22 ± 0.06	3.5†	+0.27 ± 0.06	4.3†	+0.25 ± 0.06	3.8†
Male vs female[b]	+1.93 ± 0.36	5.4†	+1.23 ± 0.35	3.5†	+1.48 ± 0.37	4.0†
Non-White ethnicity[b]	+1.94 ± 0.56	3.4†	+1.22 ± 0.60	2.1*	+1.39 ± 0.59	2.4*
Predictors measured in early adulthood						
Covariates[c]						
Physical health	+0.80 ± 0.17	4.7†	...	...	+0.66 ± 0.17	3.8†
Smoking	−0.01 ± 0.16	0.1	...	...	+0.05 ± 0.18	0.3
Alcohol use (females)[d]	−1.01 ± 0.29	3.5†	...	...	−0.85 ± 0.29	3.3†
Alcohol use (males)[d]	+0.31 ± 0.22	1.4	...	...	+0.27 ± 0.22	1.3
Psychiatric scales[c]						
Depression (females)[e]	+0.40 ± 0.22	1.9	...	...	+0.25 ± 0.22	1.2
Depression (males)[e]	−0.92 ± 0.27	3.4†	...	...	−1.15 ± 0.28	4.2†
Conduct disorder	+0.01 ± 0.19	0.0	...	...	−0.06 ± 0.19	0.3
Predictors measured in adolescence						
Covariates						
Physical health[c]	...	...	+0.51 ± 0.17	3.0***	+0.44 ± 0.17	2.8**
Smoking[c]	...	...	−0.56 ± 0.19	2.9***	−0.55 ± 0.20	2.8***
Social class (females)[c,f]	...	...	−0.77 ± 0.25	3.1***	−0.53 ± 0.25	2.2*
Social class (males)[c,f]	...	...	−0.05 ± 0.25	0.2	+0.04 ± 0.25	0.2
IQ	...	...	+0.01 ± 0.01	0.6	+0.01 ± 0.01	0.9
Parental sociopathy[b]	...	...	−0.57 ± 0.42	1.3	−0.54 ± 0.42	1.3
Psychiatric scales[c]						
Depression	...	...	+0.15 ± 0.20	0.7	+0.20 ± 0.20	1.0
Conduct disorder	...	...	+0.53 ± 0.21	2.6**	+0.62 ± 0.21	3.0***

[a]Body mass index (BMI) is regressed on all predictors in column in one model. Regression coefficients refer to change in BMI (kg/m^2) associated with each unit change in predictor. Coefficients for demographic predictors pertain to entire sample; coefficients for variables with gender qualifiers pertain only to that gender.
[b]Dichotomous variable.
[c]Scale is standardized (mean = 0, standard deviation = 1).
[d]For gender × adult alcohol use interaction, $t_{626} = 2.7$, $P < .01$.
[e]For gender × adult depression interaction, $t_{628} = 4.1$, $P < .001$.
[f]For gender × adolescent social class interaction, $t_{626} = 1.8$, $P = .079$.
*$P < .05$; **$P < .01$; ***$P < .005$; †$P < .001$.

were each associated with a higher adult BMI.

In the model including all adult and adolescent covariates, the interaction between social class and gender was significant only at the trend level ($t_{626} = 1.8$; $P = .079$). An association between increased BMI and lower social class specific to women is consistently found in epidemiological studies. We therefore included gender-specific social class variables in our models despite the marginal significance of this interaction. As shown in Table 4, social class was inversely related to BMI in females but showed no relationship to BMI in males.

While the etiology of a female-specific association between social class and BMI is poorly understood, early pregnancy could play a role.[55] Because early pregnancy may be one step on the path between conduct disorder and obesity, pregnancy status was excluded from our final models. However, number of lifetime pregnancies was inversely correlated with social class in our sample, and when this variable was entered into the model, number of pregnancies ($B = 1.3 \pm 0.8$; $t_{628} = 2.6$; $P < .01$), but not social class, gender, or gender × social class interactions, predicted young adult BMI.

There was also an interaction between time 2 alcohol use and gender ($t_{626} = 2.7$; $P < .01$). As shown in Table 4, alcohol use in young adulthood was associated with a lower BMI in women but not in men. Finally, while there was a significant interaction between IQ and gender when only these variables were fitted in the model, this interaction was not included in the final model because it was redundant with the gender × social class interaction.

Relationships with psychiatric scale scores. As shown in Table 4, results for the psychiatric scale scores were consistent, whether adolescent and adult scale scores were entered alone in separate models or together in one model. Although associations were statistically significant, they were not large in magnitude. For a model entering adult psychiatric measures after all adult and adolescent covariates, adult psychiatric scales accounted for 3% of the variance in adult BMI ($F[3,628] = 6.6$; $P < .001$). As shown in Table 4, significant associations included the interaction between current depressive symptoms and gender

($t_{628} = 4.1$; $P < .001$). There was a negative association between young adult depression and BMI in males but a positive, nonsignificant, association in females.

As shown in Table 4, adolescent psychiatric scale scores related to adult BMI, whether entered only with adolescent covariates or with all covariates and adult psychiatric scale scores. In the model entering adolescent depression and conduct disorder scale scores after all covariates and adult psychiatric scale scores, the step accounted for an additional 2% of the variance in young adult BMI ($F[2,626] = 6.7$; $P = .001$). Adult BMI was significantly related to adolescent conduct disorder but not depressive symptoms. The adolescent depression and conduct scores were correlated in the sample ($r = .37$; $P < .001$). When the depression score was entered alone in the final regression step, it also showed a positive association with BMI ($B = .38 \pm 0.19$; $t_{627} = 1.8$; $P < .05$).

Multivariate Logistic Regression

Various logistic models were fitted to consider the influence of multicollinearity and the statistical independence of associations between obesity and correlated predictor variables. Gender-specific terms for alcohol use and social class were not included in multivariate models, since the gender interactions were nonsignificant in logistic models. Across analyses, obesity was predicted by adolescent conduct disorder, the gender × adult depression interaction, and by both adolescent and adult physical health.

A logistic model was fitted to include covariates related to obesity in either gender. Adjusted odds ratios with obesity were estimated for the differences in independent measures shown in Table 3 (i.e., groups differing in age by 5 years; 2-SD differences on psychiatric scale scores). Odds ratios, with 95% confidence intervals, excluded 1.0 for the following variables: age (1.65 [1.11, 2.44]), adolescent tobacco use (0.59 [0.46, 0.77]), poor physical health in both adolescence (1.65 [1.06, 1.99]) and adulthood (2.10 [1.35, 3.23]), major depression in adulthood among men (0.27 [0.16, 0.76]), and conduct disorder in adolescence (2.14 [1.34, 3.42]). There was also a trend for a relationship with social class (0.64 [0.38, 1.07]). When the model was refitted with only adolescent major depression but not adolescent conduct disorder, adolescent depression predicted obesity at the trend level (1.49 [0.97, 2.30]).

Finally, we also considered associations between obesity and number of pregnancies, though we excluded this variable from other models as its role in the causal pathway between conduct disorder and female obesity remains unclear. Number of pregnancies predicted obesity in a univariate model, with an odds ratio of 3.60 (2.00, 6.48) for women with two as opposed to no pregnancies. This odds ratio was minimally changed (3.46 [1.71, 7.00]) when social class, ethnicity, and IQ were added to the model, none of which predicted obesity with pregnancy status in the model.

Discussion

We found an association between conduct disorder symptoms in adolescence and obesity in young adulthood after controlling factors that may affect this association. These factors include physical health, smoking, and alcohol use. Few other studies examining relationships between obesity and psychopathology have controlled for these variables, all of which related to both BMI and psychopathology in our study.

A *concurrent* association between symptoms of conduct disorder and obesity was noted in studies that predated modern DSM nosology, though most of these studies relied on subjective indices of obesity.[41,56] While little effort has been made to replicate these findings using current anthropometric and psychiatric measures, Crisp et al.[9] noted an association between teacher ratings of aggression and an objective index of obesity. However, this study used outdated measures of aggression, and the results were not as clear when other potential indices of conduct problems were employed. Using current DSM-oriented measures of conduct problems in a cross-sectional high-risk study, we noted a concurrent association between obesity and parent-rated symptoms of conduct disorder.[57] These findings suggest that a prospective relationship between symptoms of conduct disorder in adolescence and obesity in adulthood may be preceded by a concurrent association between childhood conduct disorder and obesity.

Because we did not have suitable data on adolescent BMI, it is unclear whether the longitudinal association in this study is also observable cross-sectionally in adolescence, as suggested by these other findings.[9,57] Researchers attempting a replication might consider whether a relationship between adolescent conduct disorder and adolescent BMI precedes the longitudinal association described in the present paper. Such researchers might also consider the important issue of whether childhood conduct disorder symptoms predict an increase in BMI between childhood and adulthood.

There are limitations to our findings beyond the lack of adolescent BMI data. First, we had no data on family history of obesity. Because both psychopathology and obesity are familial conditions, family history of obesity could play a role in associations between childhood psychopathology and adult obesity. Research on familial relationships between obesity and psychiatric problems may therefore enhance an understanding of the associations found in the current report. Second, the overall amount of explained variance in early adult BMI that was due to psychopathology was small in our models, and most odds ratios with obesity were not large. As a result, our findings carry unclear clinical implications. Third, there was some attrition in the sample with complete data. However, analyses were comparable after we eliminated confounding variables with missing data, and the sample studied at each assessment remained demographically representative of the population from which it was drawn. As discussed elsewhere,[42–44] findings from this study should be interpreted against these losses to follow-up. Fourth, the wide age range in our sample is a limitation, as the relationship between obesity and psychopathology may vary with age. On the other hand, the wide age range may carry some advantages. Namely, since we found no evidence of age × psychopathology interactions, the data provide some evidence that the relationship between adolescent psychopathology and adult obesity does not vary between the ages of 9 and 18. Finally, as discussed elsewhere, the use of self-reported BMI and lay interviews to assess psychopathology are other potential limitations.[42–48] It might be possible to conduct more accurate assessments of psychopathology by using experienced clinicians as interviewers.

Despite these limitations, there are reasons to believe that our findings may be replicable. Both the direction and the magnitude of associations between BMI and covariates in our study parallel results in other samples. These include the differential associations of BMI with alcohol use and social class across genders as well as the association between BMI and smoking.[7] Moreover, our results

are consistent with recent neurobiological research suggesting that abnormalities in serotonin may relate to the pathophysiology of both obesity and psychiatric syndromes that are characterized by a high degree of impulsive aggression, such as conduct disorder.[14–23]

In conclusion, in this study we found relationships between BMI and a group of factors associated with adolescent psychopathology, including low social class, poor physical health, smoking, and alcohol use. After controlling these factors, as well as concurrent depressive symptoms, we also documented a prospective relationship between symptoms of conduct disorder in adolescence and obesity in early adulthood. Efforts to replicate these findings might be encouraged in light of the strengths of this study, including its prospective epidemiological design and comprehensive psychiatric assessment. □

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NIH Consensus Panel Issues Report on Cervical Cancer

A National Institutes of Health (NIH) consensus development statement on cervical cancer may be obtained from the NIH Office of Medical Applications of Research (OMAR).

The report was prepared by a panel of experts who considered scientific evidence presented at a Consensus Development Conference on Cervical Cancer at NIH. It contains recommendations and conclusions concerning cervical cancer.

NIH consensus conferences bring together researchers, practicing physicians, representatives of public interest groups, consumers, and others to carry out scientific assessments of drugs, devices, and procedures in an effort to evaluate their safety and effectiveness.

Free, single copies of the consensus statement on cervical cancer may be obtained from the NIH Consensus Program Information Service, PO Box 2577, Kensington, MD 20891; tel: 1-888-NIH-CONS (644-2667).