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Pediatrics 2003;111:851-859
DOI: 10.1542/peds.111.4.851

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Obesity and Psychiatric Disorder: Developmental Trajectories

Sarah Mustillo, PhD; Carol Worthman, PhD; Alaattin Erkanli, PhD; Gordon Keeler, MS; Adrian Angold, MRCPsych; and E. Jane Costello, PhD

ABSTRACT. Objectives. To identify age-related trajectories of obesity from childhood into adolescence, and to test the association of these trajectories with the development of psychiatric disorders (conduct disorder, oppositional defiant disorder, attention-deficit/hyperactivity disorder, substance abuse, depression, and anxiety).

Methods. White children (N = 991) 9 to 16 years old from the Great Smoky Mountains Study, a representative sample of rural youth, were evaluated annually over an 8-year period for height, weight, psychiatric disorder, and vulnerabilities for psychiatric disorder. Longitudinal analyses on the repeated measures data were conducted using developmental trajectory models and generalized estimating equation models.

Results. Obesity was 3 to 4 times more common than expected from national rates using Centers for Disease Control and Prevention 2000 criteria. Four developmental trajectories of obesity were found: no obesity (73%), chronic obesity (15%), childhood obesity (5%), and adolescent obesity (7%). Only chronic obesity was associated with psychiatric disorder: oppositional defiant disorder in boys and girls and depressive disorders in boys.

Conclusions. In a general population sample studied longitudinally, chronic obesity was associated with psychopathology. Pediatrics 2003;111:851–859; obesity, psychopathology, developmental trajectories, depression, conduct disorder.

Abbreviations. GSMS, Great Smoky Mountains Study; BMI, body mass index; NHANES, National Health Interview Survey; CAAPA, Child and Adolescent Psychiatric Assessment; ADHD, attention-deficit/hyperactivity disorder; SPMM, semiparametric mixture model; GEE, Generalized Estimation Equation; CDC, Centers for Disease Control and Prevention.

Obesity among children and adolescents is a serious public health issue in the United States. The percentage of children defined as overweight has more than doubled since the 1970s and is still on the rise. Many studies have documented a secular trend in childhood obesity that is associated with increased health risks, both during childhood and later in adulthood. During childhood, obesity is associated with neurologic, pulmonary, gastrointestinal, circulatory, and endocrine conditions. In adulthood, a history of childhood obesity is associated with an increased risk for cardiovascular disease, diabetes, colon cancer, and all-cause mortality.

Overweight and obesity, or perceptions thereof, may also affect self-esteem, body image, and social mobility, and it is well-known that adults who are overweight or obese are at an increased risk for psychological disorders. Less is known about whether childhood obesity is associated with concurrent psychopathology, or with specific types of psychiatric disorder. Most of the published research on obesity and psychopathology in children has used clinical samples recruited because of obesity or depression. The risk of psychiatric disorder in obese children in these referred samples may not reflect what is seen in the general population.

Among the structural, familial, and environmental vulnerabilities associated with both obesity and psychopathology in childhood are being from a poor or single-parent household, having harsh or abusive parents, and, for some psychiatric disorders, being female. However, there is little research that establishes the direction of effect, or the role of these factors as mediators or moderators of the link between obesity and psychopathology.

Weight fluctuations are common in childhood, and even more so in adolescence. Because most studies of childhood obesity and psychopathology are cross-sectional or retrospective, we do not know whether there are distinct patterns of obesity throughout childhood, nor do we know whether certain patterns are associated with particular outcomes. This study provides repeated measures of height and weight taken annually over 8 years, together with annual information about family characteristics and mental health. Longitudinal data permit identification of distinct developmental trajectories of obesity, separating the transiently obese from the chronically obese, and those whose obesity is confined to childhood or adolescence.

The goals of this study are 1) to determine the number and type of distinct obesity trajectories in a general population sample of white children 9 to 16 years old; 2) to examine whether trajectory membership is associated with family vulnerabilities such as gender, poverty, single-parent family, parenting style, parental history of mental illness or drug abuse, and life events; and 3) to test the relationship between obesity trajectory membership and psychopathology, controlling for a range of potential mediators.
METHODS

Study Design and Sample

A full description of the setting, sample, and data collection methods can be found elsewhere. The Great Smoky Mountains Study (GSMS) is a longitudinal study of the development of psychiatric disorder and need for mental health services in rural youth. A representative sample of 3 cohorts of children, 9, 11, and 13 years old at intake was recruited from 11 counties in western North Carolina. Participants were selected from the population of some 20,000 children using a household equal probability, accelerated cohort design. The accelerated cohort design means that over several years of data collection each cohort reaches a given age in a different year, which controls for cohort effects.

We used a 2-phase process to select the final sample for the longitudinal study. A screening questionnaire was administered to a parent (usually the mother) of the first stage random sample (N = 3896; 95% of those contacted). The questionnaire consisted mainly of the externalizing (behavioral) problems scale of the Child Behavior Checklist, and was administered by telephone or in person. All children scoring above a predetermined cut point (the top 25% of the total scores, in this case it was a score of 20) plus a 1-in-10 random sample of the remaining 75%, were recruited for detailed interviews. Eighty percent of those recruited agreed to participate. The contribution of each participant was weighted by the inverse of their selection probabilities, stratified by age and gender, to provide accurate prevalence estimates for the population of the study area. Families were reinterviewed annually until the child was 16, and every 2 to 3 years thereafter. The data presented here, based on the first 8 annual waves of the study (1993-2000), consist of 4600 interviews with 991 non-Hispanic white participants and their parents.

Fewer than 10% of the area residents and the sample were African American. Because racial/ethnic differences in adiposity and its correlates are well-documented, non-white children were excluded from these analyses as power was not adequate for comparisons.

The study protocol was approved by the Duke University School of Medicine Institutional Review Board.

Procedures

Two interviewers visited the family each year, either at home or in a location convenient for them. Before the interviews began, parent and child signed informed consent forms. They were then interviewed in separate rooms. Each parent and child was paid $10 after the interview.

Measures

Obesity

Height and weight were both assessed at 2 different points during the interview, and the 2 averaged for these analyses. Participants wore normal clothing without shoes, socks, and belts. Height was measured to the nearest 0.1 cm using a stadiometer (CMS Weighing Equipment, London, United Kingdom) and standard techniques. The measurement was repeated if the first 2 measurements differed by >0.5 cm, and the nearest 2 of the resultant 3 were averaged. Participants were weighed twice to the nearest 0.1 kg on a portable digital scale (Soehnle, Murrhardt, Germany); a repeat measurement was taken if the first 2 differed by >0.2 kg, and averaged as for height. Body mass index (BMI) was calculated using the standard wt/ht^2 formula.

For adults, obesity is generally defined as BMI >30 kg/m^2, but no equivalent standard exists for children. In this study we use age- and sex-specific 95th percentiles from the revised (2000) growth charts of the Centers for Disease Control and Prevention (CDC), which are based on data from the National Health Interview Survey (NHANES). The reference values are based on US national survey data and are intended for US children and adolescents. Because the GSMS children were weighed clothed, and the NHANES children were weighed wearing only a gown, undergarments, and foam slippers, we subtracted 1 kg from weight before calculating the BMI.

Psychopathology

The psychopathology outcomes were assessed using the Child and Adolescent Psychiatric Assessment (CAPA), a psychiatric interview for children 9 to 17 years old. The CAPA is an interviewer-based interview. The goal of interviews using this format is to combine the advantages of clinical interviews with those of highly structured and epidemiologic interview methods. While using a highly structured format of questions and probes, the interviewer-based approach trains the interviewer to ensure that the parent or child being interviewed understands the construct under review, and provides enough detail and examples for a clear rating of the clinical severity of each symptom to be made. A detailed glossary provides the operational rules for identifying a clinically significant level of severity for each symptom.

The CAPA interviews parent and child separately, using different interviewers. The presence of a symptom can be determined on the basis of information from a single respondent, or using the either/or rule common in clinical practice. For these analyses, we coded a symptom as present if reported by either parent or child or both. The time frame of the CAPA for determining the presence of most psychiatric symptoms is the past 3 months. Diagnoses were based on Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria. For these analyses we combined psychiatric diagnoses into 7 groups: conduct disorder, oppositional defiant disorder, depressive disorders (including major depression, dysthymia, and depression not otherwise specified), anxiety disorders (including separation anxiety, generalized anxiety disorder, simple phobia, social phobia, agoraphobia, and panic), bulimia, substance abuse, and attention-deficit/hyperactivity disorder (ADHD).

Background and Family Variables

Potential mediators and moderators of the relationship between obesity and psychopathology included in these analyses were gender; family income (coded $0–$25,000, $25,001–$45,000, and $45,001+); 1 or both parents with less than an 11th-grade education; single parent family; parental history of treated mental illness, drug abuse, or criminal conviction; harsh or overprotective parenting style; lax supervision; and traumatic life events.

Season

We included in the trajectory models a variable to account for the season when the child was weighed and measured. All participants were interviewed each year on a day as close as possible to their birthday. Small seasonal variations in weight gain have been noted in 2 European samples, and studies relying on self-reports of weight have found that healthy adults believe their weight increases by ~5 pounds in the winter. Although clinical research has failed to find support for seasonal weight gain, there is little literature on seasonal effects on children’s weight. Despite the lack of consensus on seasonal weight variation in the literature, we noted a substantial seasonal weight variation in our sample. On average, 29% of children measured during the winter months (January, February, and March) were obese, compared with 20% during the remaining months, with a mean BMI difference of 1. To correct for this, we included a 4-category variable for season (January to March, April to June, July to September, and October to December) in the trajectory model as a time-dependent covariate.

Interviewers and Interviewer Training

Interviewers were residents of the area in which the study took place. All had at least bachelor’s level degrees. They received 1 month of training and constant quality control, maintained by postinterview reviews of each schedule, notes, and tape recordings by experienced interviewer supervisors and study faculty. Interviewers were trained by Department of Social Services staff in the States requirements for reporting abuse or neglect.

Data Management and Analysis

Scoring programs for the CAPA, written in SAS software (SAS Institute, Cary, NC), combine information about the date of onset, duration, and intensity of each event and symptom to create scale scores and diagnoses. Prevalence rates were calculated using Stata 7.0 (Stata Corporation, College Station, TX), taking the survey design into account through weighting and clustering. Significant differences were calculated using a design-based Pearson χ^2 with a second-order correction converted to an F statistic.
Obesity trajectories were determined by fitting a semiparametric mixture model (SPMM) to the data, using PROC TRAJ in SAS.42 SPMMs identify distinct groups of individual trajectories within the population. This approach to modeling growth curves is different from traditional latent growth curve modeling in that the latter assumes the random parameters to be bivariate normal distributed. In other words, all individuals belong to a single class of individuals who vary continuously on a latent trait. In contrast, the group-based method employed here assumes a number of discrete classes, each having a specific intercept and age slope and an estimated population prevalence.42 Because we suspected that individuals do not vary continuously on obesity, but rather that there are a distinct number of obesity trajectories, the group-based method was the most appropriate.

The logit model was used to model the presence or absence of obesity predicted by age. By specifying the sampling weights, we invoked the robust variance estimator (ie, sandwich type estimator) to adjust the standard errors of the parameter estimates to account for the 2-phase sampling design. Thus, the classes that were identified, and their estimated prevalences and correlates, relate to the entire population of white children in the 11-county area from which participants were sampled.

Children were classified into their most probable obesity trajectory class by the use of posterior probabilities. A posterior probability is the probability of each individual belonging to each group.42 Thus, individuals are assigned to the group to which they have the highest probability of belonging. Once children were assigned to a trajectory class, we conducted analyses in 3 stages.

First, we computed bivariate statistics to obtain prevalence rates of various background, familial, and psychopathological characteristics by obesity trajectory. An F test for each variable tested whether there was any difference among obesity groups. Second, we fit multinomial logit models to determine how the background and familial characteristics identified as significant in the first stage distinguished children on the different obesity trajectories from the comparison group of never-obese children. Third, we fit Generalized Estimation Equation (GEE) models to estimate the relationship between the different obesity trajectories and psychiatric disorders, controlling for the background factors identified in the second stage. The GEE models were run using binary regression with logit link function for dichotomous outcomes, such as the presence or absence of a psychiatric diagnosis, using the Stata program xtgee. The use of multiwave data with the appropriate sample weights capitalized on the availability of multiple observation points over time, while controlling for the effect on variance estimates of repeated measures on the same child, for overlapping cohorts, and for design effects.

RESULTS

Obesity and Age

Table 1 shows the association between age and mean BMI for boys and girls. Gender differences in mean BMI were small and insignificant. BMI rose steadily through late childhood and adolescence, beginning at ~18 for 9 year olds, and rising to ~20 by age 12, 22 by age 14, and 24 by age 16.

Fig 1 presents the overall prevalence of obesity, using the criterion of weight in the top 95th percentile for age and sex according to the 2000 CDC tables. Obesity rose between 9 and 10 years, dipped be-

<table>
<thead>
<tr>
<th>Age</th>
<th>Girls</th>
<th>Boys</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>18.0</td>
<td>17.8</td>
</tr>
<tr>
<td>10</td>
<td>18.7</td>
<td>19.5</td>
</tr>
<tr>
<td>11</td>
<td>19.2</td>
<td>19.1</td>
</tr>
<tr>
<td>12</td>
<td>20.1</td>
<td>19.6</td>
</tr>
<tr>
<td>13</td>
<td>21.2</td>
<td>20.9</td>
</tr>
<tr>
<td>14</td>
<td>22.0</td>
<td>21.6</td>
</tr>
<tr>
<td>15</td>
<td>22.9</td>
<td>23.2</td>
</tr>
<tr>
<td>16</td>
<td>23.6</td>
<td>24.3</td>
</tr>
</tbody>
</table>

Fig 1. Percent obese by age.
between 10 and 11 years, flattened out between 11 and 13 years, then rose again steadily to 16 years, at which point ~20% were obese. Generally, the prevalence of obesity increased with age. These results show that this rural, Southern sample was substantially heavier than the general population of children and adolescents in the United States, based on the CDC criteria, as 20% of the sample was above the 95th percentile by 16 years.

We estimated SPMMs that allowed for 1, 2, 3, 4, and 5 classes of obesity over time. Improved fits were obtained from 1 to 2, from 2 to 3 classes, and from 3 to 4 classes. However, no improvement was obtained when 5 classes were allowed. The Bayesian Information Criterion supported the 4-class model as the best fitting. Thus, the analyses presented here are based on the 4-class model (Fig 2), with individuals being assigned to their most likely obesity class using posterior probabilities.

Of the 4 trajectories of obesity, 1 consisted of children who were never or rarely obese. Children who fell into this category were not obese in any of the waves, or only in 1 wave. This group included 72.8% of the population. The second trajectory included the 7.5% of children who started off in the normal weight range during childhood but became obese over time (adolescent obesity). This curve was relatively flat until 12 years, at which point the prevalence of obesity rose sharply. The third trajectory consists of chronically obese children who were obese for most or all waves of observation. The chronically obese trajectory included 14.6% of children. The fourth trajectory, including 5.1% of the population, consisted of those who were obese during late childhood, and whose weight fell during adolescence (childhood obesity).

**Differences Among Obesity Trajectories**

Table 2 shows the prevalence of a range of child and family characteristics in each obesity group. There were no overall differences among the groups in gender, family structure, parenting style, family history of mental illness, drug abuse, crime, or traumatic events. Childhood and chronic obesity, but not adolescent obesity, were associated with having uneducated parents and lower income.

Bivariate analyses showed that childhood and chronically obese children were significantly more likely than nonobese children to have depression and oppositional defiant disorder. There was no significant difference among the trajectories in the proportion with anxiety disorders, conduct disorder, ADHD, or substance abuse disorders.

**Predictors of Obesity Trajectory Membership**

We fit multinomial regression models to determine which factors predicted group membership (Table 3). Taking the never obese as the referent group, chronically obese children were significantly more likely to be in the lower and middle income groups, and to have uneducated parents. The childhood obesity group was also more likely to have uneducated parents. There were no other significant differences between the never-obese group and children on any of the other trajectories.

Multinomial models run separately for boys and girls showed that adolescent-onset obese boys were less likely to be lower income, compared with the never obese. Chronically obese boys were more likely than never obese boys to have families with middle incomes, and to have uneducated parents. Compared with the never obese group, boys in the childhood-limited obesity group did not show any excess risk factors, nor did any of the factors examined predict chronic obesity among the girls. Uneducated parents were more common among girls with childhood-limited obesity than among the never obese. The pseudo R squared was small in all of the multinomial models, indicating that the familial and environmental variables tested did not explain a substantial portion of the variation in trajectory membership.

**Obesity Trajectories and Psychopathology**

We tested for increased risk of any of 7 psychiatric disorders in the 3 obesity groups, relative to the
nonobese group. Age, sex, and income were included in each model, as well as the other psychiatric disorders, to control for comorbidity. Oppositional disorder was more common in chronically obese boys and girls (odds ratio: 2.5; 95% CI: 1.36–4.61) and depression in chronically obese boys (odds ratio: 3.7; 95% CI: 1.27–10.2) but not girls. There were no significant associations between obesity trajectory membership and bulimia, ADHD, substance use, conduct disorder, or anxiety, controlling for comorbid psychiatric disorders. As shown in Table 3, the risk of both oppositional disorder (both sexes) and depression in boys were increased in youth with low incomes, and the risk of oppositional disorder was increased in youth with middle incomes as well. Testing for mediational effects, we found no mediational effects of income or any other family variables to explain the link between obesity trajectories and psychopathology.

**DISCUSSION**

This study explores developmental aspects of obesity in a representative, predominantly rural, population of white non-Hispanic children between 9 and 16 years. The sample used for these analyses is quite small compared with the several national surveys on which data are available (reviewed in Ref. 31), and is restricted to white non-Hispanic children. However, it has 2 advantages: 1) repeated annual measurements on the same subjects, using careful measurement of height and weight (not self-report), and 2) measurement of a wide range of risk factors and correlates, including state-of-the-art assessment for psychiatric disorder. Although 73% of the study participants were never obese across the 8 years of observation, >22% of the young people were either chronically obese (15%) or had become obese by 16 years (7%). A smaller group (5%) started obese as children and then dropped below the obesity threshold by adolescence. Defining obesity, as we did, using the CDC 2000 formula which classifies as obese children in the top 5% of the age-matched NHANES population sample, between 16% and 24% of our sample were obese at any 1 of the 8 measurement points. Our data are consistent with both the widely noted secular trend toward an increasing prevalence of early obesity,3 and an increased risk of obesity with increased age.3 There is also evidence that children in the Southern states are heavier; for example, using the 1993 CDC growth curves, Strauss and Pollack1 found that in the Southern states the proportion of children who were “overweight” (above the 95th percentile) had increased to 17.1% by 1998, from 7.6% in 1968.

In our sample, the prevalence of obesity was higher among the boys than the girls, although the difference was not significant. Several recent studies found obesity to be more prevalent among boys than girls,43–45 with rates of obesity increasing faster in boys than in girls.1 However, gender patterns may vary by race; therefore, any gender differences may depend on the race of the sample. Gordon-Larsen et al44 examined prevalence of overweight and obesity by sex and race from the National Longitudinal Sur-

### TABLE 2. Sociodemographic, Family Characteristics, and Psychiatric Disorders in 4 Obesity Trajectories

<table>
<thead>
<tr>
<th>Obesity Trajectories</th>
<th>Never Obese</th>
<th>Adolescent Obese</th>
<th>Childhood Obese</th>
<th>F Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 991</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>47.7%</td>
<td>60.5%</td>
<td>55.3%</td>
<td>64.7%</td>
<td>1.14</td>
</tr>
<tr>
<td>Income</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤$25,000</td>
<td>29.9%</td>
<td>20.3%</td>
<td>39.4%</td>
<td>40.1%</td>
<td>2.40</td>
</tr>
<tr>
<td>$25,001–$45,000</td>
<td>32.1%</td>
<td>42.2%</td>
<td>41.6%</td>
<td>27.3%</td>
<td></td>
</tr>
<tr>
<td>≥$45,001</td>
<td>38.1%</td>
<td>37.6%</td>
<td>19.0%</td>
<td>32.6%</td>
<td></td>
</tr>
<tr>
<td>Uneducated parents</td>
<td>13.1%</td>
<td>16.4%</td>
<td>29.7%</td>
<td>32.0%</td>
<td>6.21</td>
</tr>
<tr>
<td>Single parent</td>
<td>20.4%</td>
<td>23.5%</td>
<td>22.7%</td>
<td>24.4%</td>
<td>.18</td>
</tr>
<tr>
<td>Inadequate supervision</td>
<td>6.7%</td>
<td>9.0%</td>
<td>9.2%</td>
<td>10.9%</td>
<td>1.14</td>
</tr>
<tr>
<td>Harsh discipline</td>
<td>1.9%</td>
<td>40.0%</td>
<td>3.4%</td>
<td>1.4%</td>
<td>2.98</td>
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<tr>
<td>Overprotective</td>
<td>7.0%</td>
<td>4.0%</td>
<td>1.1%</td>
<td>2.1%</td>
<td>1.48</td>
</tr>
<tr>
<td>Parent treated for MH problems</td>
<td>27.1%</td>
<td>20.5%</td>
<td>29.4%</td>
<td>25.3%</td>
<td>.39</td>
</tr>
<tr>
<td>Parent treated for drug problems</td>
<td>6.4%</td>
<td>4.2%</td>
<td>8.8%</td>
<td>8.5%</td>
<td>1.15</td>
</tr>
<tr>
<td>Parent convicted of crime</td>
<td>18.7%</td>
<td>17.5%</td>
<td>18.9%</td>
<td>31.4%</td>
<td>1.45</td>
</tr>
<tr>
<td>Violence between parents</td>
<td>1.8%</td>
<td>60.0%</td>
<td>1.5%</td>
<td>2.4%</td>
<td>.41</td>
</tr>
<tr>
<td>Loss events</td>
<td>15.8%</td>
<td>10.4%</td>
<td>15.7%</td>
<td>9.3%</td>
<td>1.72</td>
</tr>
<tr>
<td>Violent events</td>
<td>15.6%</td>
<td>15.2%</td>
<td>19.3%</td>
<td>19.7%</td>
<td>.62</td>
</tr>
<tr>
<td>Adverse ongoing events</td>
<td>13.6%</td>
<td>13.8%</td>
<td>13.2%</td>
<td>21.1%</td>
<td>.78</td>
</tr>
<tr>
<td>Network events</td>
<td>12.0%</td>
<td>1.6%</td>
<td>13.1%</td>
<td>16.0%</td>
<td>.57</td>
</tr>
<tr>
<td>BMI</td>
<td>19.6</td>
<td>23.9</td>
<td>29.4</td>
<td>25.6</td>
<td></td>
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<tr>
<td>Bulimia</td>
<td>0.0%</td>
<td>0.0%</td>
<td>4.0%</td>
<td>1.5%</td>
<td>2.56</td>
</tr>
<tr>
<td>Any depression</td>
<td>2.2%</td>
<td>3.0%</td>
<td>4.0%</td>
<td>2.0%</td>
<td>3.40</td>
</tr>
<tr>
<td>Any anxiety</td>
<td>2.4%</td>
<td>1.1%</td>
<td>2.8%</td>
<td>2.6%</td>
<td>.61</td>
</tr>
<tr>
<td>Oppositional defiant disorder</td>
<td>3.2%</td>
<td>3.7%</td>
<td>6.5%</td>
<td>7.5%</td>
<td>4.51</td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>2.1%</td>
<td>1.4%</td>
<td>3.4%</td>
<td>2.0%</td>
<td>.94</td>
</tr>
<tr>
<td>Substance use</td>
<td>18.3%</td>
<td>16.0%</td>
<td>18.7%</td>
<td>21.3%</td>
<td>.20</td>
</tr>
<tr>
<td>ADHD</td>
<td>9.7%</td>
<td>9.9%</td>
<td>1.3%</td>
<td>1.3%</td>
<td>.38</td>
</tr>
</tbody>
</table>

MH indicates mental health.
* P ≤ .05.
** P ≤ .01.
specific to white children in the United States, and this link between income and overweight may be across each of the 8 annual assessments. However, inverse relationship between BMI and income of between 0.25–0.30 may be evident of Adolescent Health and found that boys had a higher prevalence of overweight and obesity than girls among whites and Asians, but girls had a higher prevalence among Hispanics and blacks. Familial and environmental risk factors for chronic obesity were surprisingly few; of the 20 examined, only income and lack of parental education remained significant in multivariable analyses. Children with chronic or childhood obesity were more than twice as likely as other children to come from families where 1 or both parents left school before the 11th grade. Lack of parental education remained significant after controlling for having a single, unemployed, or teenage parent, and other correlated risk factors (Table 3). An inverse relationship between income and BMI has been observed in several studies.

TABLE 3. Comparison with Never Obese Trajectory: Multinomial Regression

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>Adolescent Obesity*</th>
<th>Chronic Obesity*</th>
<th>Childhood Obesity*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>1.72 (.85)</td>
<td>1.28 (.37)</td>
<td>1.95 (1.13)</td>
</tr>
<tr>
<td>Low income†</td>
<td>.67 (.30)</td>
<td>2.16 (.74)‡</td>
<td>1.05 (.73)</td>
</tr>
<tr>
<td>Middle income†</td>
<td>1.27 (.49)</td>
<td>2.31 (.72)§</td>
<td>.80 (.50)</td>
</tr>
<tr>
<td>Parental style</td>
<td>1.12 (.35)</td>
<td>1.24 (.23)</td>
<td>1.25 (.25)</td>
</tr>
<tr>
<td>Parental history</td>
<td>.74 (.13)</td>
<td>.92 (.09)</td>
<td>1.08 (.15)</td>
</tr>
<tr>
<td>Uneducated parents</td>
<td>1.61 (.78)</td>
<td>2.34 (.73)§</td>
<td>3.20 (.68)‡</td>
</tr>
<tr>
<td>Life events</td>
<td>1.37 (.28)</td>
<td>1.01 (.15)</td>
<td>.80 (.20)</td>
</tr>
<tr>
<td>Wald $\chi^2$</td>
<td>54.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudo R²</td>
<td>.04</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Girls only

| Low income†                 | 1.11 (.81)          | 2.25 (1.14)      | 1.39 (1.11)        |
| Middle income†              | .91 (.77)           | 1.79 (.86)       | 1.73 (1.02)        |
| Parental style              | 1.18 (.57)          | .80 (.23)        | 1.12 (.24)         |
| Parental history            | .89 (.18)           | .90 (.11)        | 1.16 (0.24)        |
| Uneducated parents          | 2.37 (1.65)         | 2.08 (.97)       | 7.44 (4.39)§       |
| Life events                 | 1.44 (.50)          | 1.29 (.30)       | 1.39 (.29)         |
| Wald $\chi^2$               | 88.5§               |                  |                   |
| Pseudo R²                   | .05                 |                  |                   |

Boys only

| Low income†                 | .37 (.18)‡          | 2.08 (.93)       | .93 (.83)          |
| Middle income†              | 1.53 (.60)          | 2.92 (1.10)§     | .56 (.46)          |
| Parental style              | 1.08 (.44)          | 1.75 (.40)       | 1.27 (.41)         |
| Parental history            | .62 (.16)           | .95 (.16)        | 1.08 (0.15)        |
| Uneducated parents          | 1.10 (.69)          | 2.51 (1.06)‡     | 1.70 (1.32)        |
| Life events                 | 1.25 (.33)          | .80 (.15)        | .50 (0.16)‡        |
| Wald $\chi^2$               | 47.5§               |                  |                   |
| Pseudo R²                   | .05                 |                  |                   |

* Reference group is Group 1—Never/Rarely Obese.
† Reference group is high income.
‡ $P < .05$.
§ $P < .01$.

Using developmental trajectory modeling, we were able to show that chronic and childhood-only obesity were more common in the lowest income group (annual family income below $25,000) than in the middle or upper income groups. Chronic obesity was also more common than expected from children from the middle income group ($25,000–$45,000). This inverse relationship between weight and poverty, beginning in childhood and, in the case of the poorest children, persisting into adolescence, reflects earlier studies.16,50 It is particularly troubling in the light of evidence from the 1946 British birth cohort

TABLE 4. GEE Models of Association Between Obesity Trajectories and Oppositional Defiance Disorder (Both Sexes) and Depression (Boys) (Semirobust Standard Errors in Parentheses)

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>Oppositional Defiance Disorder Total Sample</th>
<th>Depression Boys Only</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>1.31 (.33)</td>
<td></td>
</tr>
<tr>
<td>Low income†</td>
<td>2.85 (.85)§</td>
<td>4.50 (2.23)§</td>
</tr>
<tr>
<td>Middle income†</td>
<td>2.06 (.60)‡</td>
<td>2.15 (1.08)</td>
</tr>
<tr>
<td>Age</td>
<td>1.08 (.06)</td>
<td>1.17 (1.11)</td>
</tr>
<tr>
<td>Adolescent obese*</td>
<td>.94 (.41)</td>
<td>.24 (.26)</td>
</tr>
<tr>
<td>Chronic obese*</td>
<td>2.57 (.79)§</td>
<td>3.72 (1.94)§</td>
</tr>
<tr>
<td>Childhood obese*</td>
<td>2.00 (.91)</td>
<td>1.68 (1.15)</td>
</tr>
<tr>
<td>Wald $\chi^2$</td>
<td>24.83§</td>
<td>26.90§</td>
</tr>
<tr>
<td>n (observations/subjects)</td>
<td>4288/983</td>
<td>2374/555</td>
</tr>
</tbody>
</table>

* Reference group is Group 1—Never/Rarely Obese.
† Reference group is high income.
‡ $P < .05$.
§ $P < .01$. 

856 OBESITY AND PSYCHIATRIC DISORDER: DEVELOPMENTAL TRAJECTORIES Downloaded from www.pediatrics.org at Duke University on September 25, 2007
that the inverse association between socioeconomic status and BMI predicted higher BMI through to middle age\textsuperscript{48} and was impervious to the child’s later academic or economic achievements.

The present study shows first and foremost that there are different types of childhood obesity. Additionally, this study shows that of the 4 basic weight trajectories—never obese, chronically obese, increasing risk, and decreasing risk—only chronic obesity was associated with a statistically significant increase in the risk of psychiatric disorder. The group with the next-highest prevalence of psychiatric disorder was in the small group who were obese in childhood but became less so as they moved through adolescence. Adolescent-onset obesity was not associated with increased risk of psychopathology.

Chronically obese children had significantly higher rates of oppositional defiant disorder, and (for boys) depression. This is, to our knowledge, the first study to show an association between chronic obesity and behavioral problems in children and adolescents. Several studies have linked child and adolescent obesity to depression, but the majority of these are based on clinic samples, referred either because of obesity\textsuperscript{11–14} or depression.\textsuperscript{15} We cannot rule out the possibility that the co-occurrence of the 2 conditions increased the likelihood of a clinical referral.\textsuperscript{51}

Among epidemiologic studies, the evidence linking obesity and depression is much less clear. The New York longitudinal study\textsuperscript{52} found an inverse relationship between adolescent depression and young adult obesity in males, and no relationship in females. One study\textsuperscript{53} argued that the association between overweight and depression may be caused by the effects of dieting, or of poor health. Another\textsuperscript{54} demonstrated that any association between depression and BMI in a sample of third graders was explained by concern about being overweight. Of course, this may be true across the whole population but not true for the smaller group of obese youth.

Some studies have found that obesity is associated with lower levels of psychopathology, as opposed to higher levels.\textsuperscript{55} Friedman et al\textsuperscript{55} note that inconsistencies in prior studies of obesity and psychopathology may be attributed to methodological and sampling limitations. Additionally, we would add that inconsistencies in prior findings may be caused, in part, by the failure to consider developmental trajectories. In our sample, being obese at 1 point in time was not associated with an increased risk in psychopathology, rather being in the chronically obese trajectory was.

One limitation of this study is that it does not address the issue of causality. Studies such as this beg the question, does obesity increase the risk of psychopathology or does psychopathology increase the risk of obesity? DiPietro et al\textsuperscript{56} rely on epidemiologic data to examine changes in weight as a function of changes in depressive symptoms and find that depression plays a role in weight change. Younger men (<55 years old) who were depressed at baseline gained more weight during the follow-up period than those not depressed, while young women who were depressed at baseline gained less weight than the nondepressed. In a small prospective case-control study, Pine et al\textsuperscript{15} found that depression during childhood was positively associated with BMI during adulthood. Conversely, Pine et al\textsuperscript{52} found an association with current but not prior depression in a large, population-based sample. These inconsistencies between prior depression and current BMI may be due to differences in type of sample, measurement of psychopathology, and the use of self-report versus measured height and weight.

In this study, we examine the association between obesity and psychopathology; however, we draw no conclusions about causal ordering. If psychopathology increases the risk of obesity, we would expect an association in the chronic and adolescent-onset groups. If obesity increases the risk of psychopathology, we would expect an association in the chronic and childhood-limited groups. Our findings show that psychopathology is most common in the chronically obese group first, and the childhood-limited group second. Therefore, these results could be suggestive of obesity increasing the risk of psychopathology; however, that conclusion is purely speculative. Future research investigating the sequence of events is necessary to draw any conclusions about causation. It is also possible that rather than 1 increasing the risk of the other, they are both associated with the same physiologic mechanisms.

Evidence, largely from adults, links obesity and depression through underlying neuro-endocrine substrates. Specifically, activity of the hypothalamo-pituitary adrenal axis appears to be altered in both depressed\textsuperscript{57–59} and obese\textsuperscript{60,61} adults, although evidence from depressed obese adults is contradictory,\textsuperscript{62} and little is known about children. The trajectories of obesity identified here may manifest in part sequelae of suboptimal conditions for early development. For example, a growing body of research documents the role of maternal-fetal stress in later mental and physical health risk. Prenatal stress followed by postnatal overnutrition leads to childhood overweight\textsuperscript{63} and adult obesity\textsuperscript{64} as well as adult depression\textsuperscript{65} and increased hypothalamo-pituitary adrenal activity.\textsuperscript{66,67} Overall, relevant findings are more suggestive than definitive, leaving etiologic interrelationships between obesity and mood or behavior disorders and their possible common neuroendocrine mediators a subject for further investigation.

Although chronic obesity and bulimia were linked in bivariate analyses, there were very few cases of bulimia in this sample, and all but 2 were comorbid for oppositional disorder and/or depression. Interestingly, bulimia was only associated with obesity when it was comorbid with a psychiatric disorder. Bulimia is clearly an important clinical problem, and the fact that in this population of 9 to 16 year olds it was seen almost exclusively in children with other psychiatric disorders argues for vigilance on the part of clinicians to identify these comorbid children early.

There is debate about the accuracy of BMI as a measure of the prevalence of obesity.\textsuperscript{68,69} For example, Frankenstein et al\textsuperscript{69} compared BMI to percent body fat as measures of obesity, and found that BMI
underidentified obesity. Defining obesity as a BMI ≥30 or at least 25% body fat for men and 30% for women, 30% of men and 40% of women who had BMI <30 had obesity-level body fat. In adolescents, however, Malina found BMI to be a highly sensitive and specific marker of obesity, if not of overweight.

A technical limitation of this study is that the children were weighed and measured clothed except for their shoes, socks, and belts. A constant 1 kg was subtracted from their weight before BMIs were calculated. The seasonal difference in weight observed in this study may have been caused in part by extra clothing worn in the winter, although American homes tend to be kept to a fairly even temperature throughout the year. Another limitation is that although the GEE models used repeated measures, they did not deal with the relative timing of obesity and psychiatric disorder. Thus, we could determine if an association existed between the obesity trajectories and Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition diagnoses, but not whether an association was more likely to occur at a particular age or if the association existed over time.

CONCLUSIONS

Chronic obesity across 8 years from 9 to 16 years of age was observed in almost 15% of a general population sample of rural white youth, 3 times the rate predicted using the most recent CDC criteria. A further 7% became obese in adolescence, while 5% who had been obese in childhood moved out of obesity by adolescence. Chronic obesity was more common in children from poor and less-educated families, and it had implications for mental health. New methods of data analysis that permit the modeling of developmental trajectories offer the opportunity to deepen our understanding of pathways to health and illness across childhood and adolescence.

ACKNOWLEDGMENTS

Work contributing to these analyses was supported by MH01167, MH57731, and MH48085 from the National Institute of Mental Health, and DA11301 from the National Institute on Drug Abuse.

We are most grateful to the people of western North Carolina for their collaboration in the GSMS, to Dan Nagin and Mike Ezell for their methodological assistance, and to the reviewers for their helpful comments.

REFERENCES

38. Wing JK, Cooper JE, Sartorius N. Present State Examination (PSE). In: *Wing JK, Cooper JE, Sartorius N, eds. The Measurement and Classification of


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“People will keep claiming to have created cloned babies and eventually someone will succeed, but at what cost? A lot of damaged children and disappointed parents.”


—Thomas Murray, Bioethicist