ASSOCIATION OF ADOLESCENT OBESITY WITH RISK OF SEVERE OBESITY IN ADULTHOOD

Natalie S. The, PhD
Chirayath Suchindran, PhD
Kari E. North, PhD
Barry M. Popkin, PhD
Penny Gordon-Larsen, PhD

INDIVIDUALS WITH SEVERE OBESITY (body mass index [BMI] ≥40) encounter serious and potentially life-threatening complications, including diabetes, hypertension, hyperlipidemia, asthma, and arthritis, and substantial reductions in life expectancy. Repeated cross-sectional and self-reported data suggest that severe obesity prevalence has increased substantially over the past few decades, potentially increasing at a faster rate than moderate obesity. In 2000, an estimated 2.2% of adults, or 4.8 million individuals, were severely obese, with a disproportionately higher prevalence in women and racial/ethnic minorities. Yet, few national studies track individuals over time to understand the progression of obesity to severe obesity.

Diet, exercise, and behavioral modification are recommended as initial treatments for severe obesity, resulting in short-term weight loss, which, when combined with pharmacotherapy, can be associated with a 5% to 10% reduction in weight. However, antiobesity pharmacological agents have substantial adverse effects, and discontinuation often results in weight regain. In contrast, bariatric surgery results in weight loss ranging from 60% to 70% for at least 10 years and commonly results in complete resolution or improvement in comorbidities after surgery. As such, bariatric surgery is the only treatment that has been shown to have long-term success, yet this procedure has major potential complications, including leakage, pneumonia, pulmonary embolism, band slippage, and band erosion.

Author Affiliations: Carolina Population Center (Drs The, Suchindran, Popkin, and Gordon-Larsen) and Departments of Nutrition (Drs The, Popkin, and Gordon-Larsen), Biostatistics (Dr Suchindran), and Epidemiology (Dr North), Gillings School of Global Public Health, University of North Carolina, Chapel Hill.

Context Although the prevalence of obesity has increased in recent years, individuals who are obese early in life have not been studied over time to determine whether they develop severe obesity in adulthood, thus limiting effective interventions to reduce severe obesity incidence and its potentially life-threatening associated conditions.

Objective To determine incidence and risk of severe obesity in adulthood by adolescent weight status.

Design, Setting, and Participants A cohort of 8834 individuals aged 12 to 21 years enrolled in 1996 in wave II of the US National Longitudinal Study of Adolescent Health, followed up into adulthood (ages 18-27 years during wave III [2001-2002] and ages 24-33 years during wave IV [2007-2009]). Height and weight were obtained via anthropometry and surveys administered in study participants’ homes using standardized procedures.

Main Outcome Measures New cases of adult-onset severe obesity were calculated by sex, race/ethnicity, and adolescent weight status. Sex-stratified, discrete time hazard models estimated the net effect of adolescent obesity (aged <20 years; body mass index [BMI] ≥95th percentile of the sex-specific BMI-for-age growth chart or BMI ≥30.0) on risk of severe obesity incidence in adulthood (aged ≥20 years; BMI ≥40.0), adjusting for race/ethnicity and age and weighted for national representation.

Results In 1996, 79 (1.0%; 95% confidence interval [CI], 0.7%-1.4%) adolescents were severely obese; 60 (70.5%; 95% CI, 57.2%-83.9%) remained severely obese in adulthood. By 2009, 703 (7.9%; 95% CI, 7.4%-8.5%) non–severely obese adolescents had become severely obese in adulthood, with the highest rates for non-Hispanic black women. Obese adolescents were significantly more likely to develop severe obesity in young adulthood than normal-weight or overweight adolescents (hazard ratio, 16.0; 95% CI, 12.4-20.5).

Conclusion In this cohort, obesity in adolescence was significantly associated with increased risk of incident severe obesity in adulthood, with variations by sex and race/ethnicity.
Given the lack of successful treatment options, risks associated with treatment, and numerous health consequences of severe obesity, primary prevention is critical. Understanding which individuals are at risk of severe obesity is essential for determining when interventions would need to be implemented to prevent obese individuals from progressing to severe obesity. Although observational studies have reported that the prevalences of overweight, obesity, and severe obesity have increased in recent years,6,17 individuals who are obese early in life have not been studied longitudinally to determine their risk of developing severe obesity in adulthood. To this end, we used a US nationally representative, longitudinal cohort to determine the incidence and risk of severe obesity in adulthood among individuals who were obese during adolescence.

METHODS

National Longitudinal Study of Adolescent Health

The National Longitudinal Study of Adolescent Health (Add Health) is a cohort of adolescents (20,745 individuals aged 11-20 years at baseline; mean age, 15.9 years) drawn from a sample of 80 high schools and 52 middle schools in the United States with unequal probability of selection. Incorporating systematic sampling methods and implicit stratification into the Add Health study design ensured that this sample is representative of US schools in 1994-1995 with respect to region, urbanicity, school size, school type, and ethnicity.

Poststratification sample weights ensure that population estimates at each wave conform to population estimates from individuals eligible for each interview; thus, the respondents are representative of the US school population in grades 7 through 12 in 1994-1995 (wave I) as they are followed into adulthood. Wave II, conducted in 1996 (n=14,738; mean age, 16.5 years) included wave I adolescents still of school age by design (including those currently in high school and high school dropouts). Wave III, conducted in 2001-2002 (n=15,197; mean age, 22.3 years) and wave IV, conducted in 2007-2009 (n=15,701; mean age, 28.9 years) included all wave I respondents, regardless of wave II participation. The most recent data collection (wave IV) includes follow-up interviews from 15,701 wave I respondents drawn from 19,962 of the original 20,745 wave I respondents (exclusions: 96 deceased at wave III and 687 not sampled at wave III), with 80.25% of the eligible respondents (ineligible: 184 who moved out of the country, 87 military stationed out of the country, and 126 deceased at wave IV) consenting to participate in wave IV.

Written informed consent was obtained for all wave I participants. Survey procedures have been described elsewhere and were approved by the institutional review board at the University of North Carolina at Chapel Hill.6

Measures

Weight and height were measured in waves II through IV during in-home surveys using standardized procedures. Wave I used self-reported height and weight data, which were excluded from this analysis because the gain in 1 additional year of follow-up was not an acceptable trade-off for the error that would have been introduced with use of a combination of self-report (wave I) and measured (waves II-IV) height and weight data.

Body mass index (calculated as weight in kilograms divided by height in meters squared) and BMI percentiles from measured height and weight were derived for age and sex using the Centers for Disease Control and Prevention National Center for Health Statistics growth charts.17 Given that adolescent BMI (wave II; 1996) was not linearly associated with incident severe obesity, BMI was categorized using the recommended definitions for comparability across adolescence and adulthood.18 These categories were defined as: (1) normal weight (≥5th to <85th percentile on BMI-for-age growth chart or BMI of ≥18.5 to <25 for individuals aged <20 years; BMI of ≥18.5 to <25 for individuals aged ≥20 years); (2) overweight (≥85th to <95th percentile or BMI of ≥25 to <30 for individuals aged <20 years; BMI of ≥25 to <30 for individuals aged ≥20 years); (3) obesity (≥95th to <120% of 95th percentile or BMI of ≥30 to <40 for individuals aged <20 years; BMI of ≥30 to <40 for individuals aged ≥20 years); and (4) severe obesity (≥120% of 95th percentile for individuals aged <20 years19; BMI of ≥40 for individuals aged ≥20 years). Respondents who exceeded scale capacity (for wave III: 330 lb [148.5 kg] [n=12]; for wave IV: 440 lb [198 kg] [n=2]) were classified as severely obese. Incident severe obesity in adulthood was classified as nonsevere obesity at adolescence (wave II) and severe obesity at adulthood (wave III or IV; 2001-2009).

Age was recorded as the respondent’s age on the date of examination. Age at onset of severe obesity was defined as age at the wave in which the individual was initially classified as severely obese. We observed a nonlinear relationship between age at onset of severe obesity and development of severe obesity in young adulthood; thus, we categorized age at onset of severe obesity as younger than 20 years (reference), 20 to 24.9 years, 25 to 29.9 years, and 30 years or older.

Consistent with previous Add Health research,20,21 race/ethnicity was obtained from a combination of in-home surveys from parents and adolescents and was categorized as non-Hispanic white, non-Hispanic black, Hispanic (Cuban, Puerto Rican, Central/South American, Mexican, or other Hispanic), or Asian American (Chinese, Filipino, or other Asian).

Statistical Analyses

Statistical analyses were conducted using Stata software, version 10.1 (Stata Corp, College Station, Texas). To account for Add Health’s stratified sampling strategy, clustered sampling design, and nonresponse bias,22,23 sample weights and survey analysis techniques were used in all analyses. All results are nationally representative of adolescents who were enrolled in grades.
7 through 12 in 1994 and followed up into adulthood.

For descriptive analyses, percentages were calculated for categorical variables, while means were calculated for continuous variables. To compare individuals with incident severe obesity with individuals without severe obesity, a 2-sided t test and F statistic were used to test for statistical differences (P < .05). Incidence rates of severe obesity during the transition from adolescence to adulthood were calculated by sex, race/ethnicity, and adolescent weight status (normal, overweight, and obese). A 2-sided F statistic was used to compare the incidence of severe obesity by these categories, and the Bonferroni correction (P = .0167) was applied for multiple comparisons.

Discrete time hazard models (with a complementary log-log link), a type of a survival analysis model appropriate when the outcome is ascertained at periodic measurements, were used to determine the relationship between adolescent obesity and incidence of severe obesity in adulthood. Given the relatively low incidence of severe obesity in individuals who had normal weight as adolescents, the 3 categories used to obtain absolute incidence rates (normal weight, overweight, and obese) were condensed to 2 categories, obese vs nonobese (ie, collapsing normal weight and overweight into the nonobese category) for the hazard analyses. Given the particular discrete time interval based on the examination dates and obesity data, models were conditioned on time as a unit of analysis, with age at the examination during which severe obesity was first recorded serving as the primary time variable in all models. Age-specific hazard ratios (incidence rate ratios) were calculated for the probability of becoming severely obese during a given age range, conditioned on no severe obesity at the beginning of that interval. Discrete time hazard models assume that once individuals become severely obese, they remain severely obese and, thus, while included in models, they no longer contribute to the analysis.

The hazard models included only race and sex to provide net effects of risk rather than causal modeling of these relationships. Thus, a parsimonious model was used to describe the relationship between adolescent obesity (vs nonobesity) and risk of severe obesity in adulthood. To determine whether the relationship between adolescent obesity and severe obesity risk varied by sex and race/ethnicity, a 3-way interaction was used to examine effect modification using Wald tests (P = .10). Despite borderline significance (P = .14), differences in the associations across race/ethnicity are clinically important given the racial/ethnic disparities in the prevalence of obesity and its comorbidities. Thus, final models were sex-stratified and included interactions between adolescent obesity and race/ethnicity. Additionally, effect measure modification by age at severe obesity onset with adolescent obesity and race/ethnicity. Additionally, effect measure modification by age at severe obesity onset with adolescent obesity and age at severe obesity onset with race/ethnicity was tested, but neither showed effect modification.

**RESULTS**

Data from the initial 14,738 participants measured at wave II (FIGURE) were included in the analytic sample frame, with a total of 29,476 observations spanning 1996 (wave II) to 2009 (wave IV), excluding participants of Native American race/ethnicity (n = 45); individu-
als missing sampling weights (needed to correct for nonresponse bias and sample design) (n = 3699), height and weight data at wave II (n = 46) or wave III or 4 (n = 436), or race/ethnicity (n = 74); individuals who were underweight (because the amount of weight gain necessary to shift from underweight to severe obesity in the 13-year time frame of the study could indicate a different phenotype or surrogate for other metabolic conditions) (n = 1381); and girls/women who were pregnant at baseline (n = 144). Given interest in incident severe obesity, individuals who were already severely obese at baseline (n = 79 [1.0%; 95% confidence interval{CI}, 0.7%-1.4%]) were excluded; these 79 individuals were more likely to be racial/ethnic minorities than participants included in the analytic sample and most (n = 60 [70.5%; 95% CI, 57.2%-83.9%]) remained severely obese in adulthood (result not shown).

The final analytic sample included all available exposure, outcome, and covariate data across waves II, III, and IV, totaling 15 598 observations across 8834 individuals. The analytic sample included significantly more whites, older individuals, and individuals of higher parental education than those excluded. However, inverse probability weighting showed no evidence of selection bias by these factors in final models.

Over the 13-year period between adolescence (1996) and adulthood (2007-2009), a total of 703 incident cases of severe obesity in adulthood were observed, indicating a total incidence rate of 7.9% (95% CI, 7.4%-8.5%) (Table 1). Individuals with incident severe obesity in adulthood had a higher adolescent BMI, were older, and were more likely to be racial/ethnic minorities compared with individuals without severe obesity.

A substantial proportion of obese adolescents became severely obese by their early 30s, with significant variation by sex (Table 2). Among individuals who were obese as adolescents, incident severe obesity was 37.1% (95% CI, 30.6%-43.6%) in men and 51.3% (95% CI, 44.8%-57.8%) in women. Incident severe obesity was highest among black women at 52.4% (95% CI, 40.9%-63.8%). Across all sex and racial/ethnic groups, less than 5% of individuals who were at a normal weight in adolescence became severely obese in adulthood.

In analysis using multivariate, discrete hazard models, obese adolescents were significantly more likely to develop severe obesity than normal-weight or overweight adolescents (hazard ratio, 16.0; 95% CI, 12.4-20.5), with variation across race/ethnicity and sex (Table 3). While the hazard ratio for men was higher than for women, the incidence of severe obesity in adulthood was higher among women (51.3%; 95% CI, 44.8%-57.8%) than men (37.1%; 95% CI, 30.6%-43.6%). Thus, the male-female differences in risk must be interpreted relative to the difference in rates of incidence.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Weighted</th>
<th>Unweighted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline age, mean (SD), y</td>
<td>16.5 (1.6)</td>
<td>16.8 (1.6)</td>
</tr>
<tr>
<td>Male, % (SE)</td>
<td>92.1 (0.4)</td>
<td>92.0 (0.3)</td>
</tr>
<tr>
<td>Female, % (SE)</td>
<td>7.9 (0.4)</td>
<td>8.0 (0.3)</td>
</tr>
<tr>
<td>White</td>
<td>70.3 (3.0)</td>
<td>68.9 (5.0)</td>
</tr>
<tr>
<td>Black</td>
<td>14.5 (2.1)</td>
<td>18.3 (3.7)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>11.3 (1.8)</td>
<td>12.1 (3.1)</td>
</tr>
<tr>
<td>Asian</td>
<td>3.9 (0.8)</td>
<td>0.7 (0.6)</td>
</tr>
<tr>
<td>Baseline body measurements</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, mean (SD)</td>
<td>22.7 (3.2)</td>
<td>22.7 (3.2)</td>
</tr>
<tr>
<td>Overweight, % (SE)</td>
<td>18.0 (0.6)</td>
<td>17.6 (0.4)</td>
</tr>
<tr>
<td>Obese, % (SE)</td>
<td>6.4 (0.4)</td>
<td>6.3 (0.3)</td>
</tr>
</tbody>
</table>

Abbreviation: BMI, body mass index, calculated as weight in kilograms divided by height in meters squared.

TABLE 1. Selected Characteristics of Participants by Longitudinal Severe Obesity Status

©2010 American Medical Association. All rights reserved.

COMMENT

Taking advantage of a nationally representative longitudinal data set, we observed high rates of incident severe obesity in adulthood among individuals who were obese earlier in life, with a higher incidence in women (vs men) and with the highest risk for black women. These nationally representative estimates suggest that approximately 125 000 individuals may have been severely obese during adolescence, while another 1 million adolescents may have become severely obese by the time they reached their early 30s. Over the 13-year study period, individuals who never developed severe obesity gained an average of 5.1 BMI units, whereas individuals who developed severe obesity as adults gained an average of 14.2 BMI units. Furthermore, obese adolescents were at substantially higher risk of developing severe obesity in adulthood than normal-weight or overweight adolescents.
Rates of obesity have increased across all age groups, with cross-sectional National Health and Nutrition Examination Survey data suggesting a severe obesity prevalence of 4.2% for men and 7.6% for women in young adulthood (aged 20-39 years) in 2008.13 Short-term self-reported data suggest that severe obesity might be increasing at a faster rate than moderate obesity.6 This increase is particularly concerning given the serious and potentially life-threatening complications associated with severe obesity. Yet, little is known regarding the persistence of severe obesity, the progression of obesity to severe obesity, and how risk differs by sex and race/ethnicity. Understanding these patterns is critical for reducing the burden of obesity and for implementing interventions to prevent the progression of obesity to severe obesity. Findings from current research suggest that interventions designed to prevent adult-onset severe obesity would best be implemented among obese adolescents, particularly black girls.

Although previous studies have shown a persistence of obesity from childhood and adolescence to adulthood,26,27 there are no known studies that have examined persistence and development of severe obesity. The current findings indicate that (1) there is strong persistence of severe obesity from adolescence to young adulthood; (2) there is a relatively high incidence rate of severe obesity during the transition from adolescence to adulthood; and (3) individuals who were obese as adolescents were significantly more likely to become severely obese in adulthood, highlighting the need for primary and secondary prevention of severe obesity early in the life course. In particular, primary prevention efforts should focus on the prevention of obesity prior to adolescence, while secondary prevention efforts should focus on the identification and treatment of high-risk groups in adolescence, including overweight and obese adolescents.

There are a few limitations to this analysis. The main objective of this study was to determine the incidence of severe obesity during the transition from adolescence to adulthood and to determine which groups are at highest risk. As such, the analytic strategy was designed to test the net effects of race/ethnicity and sex on severe obesity rather than undertake causal modeling of these relationships. Clearly, several other biological, sociocultural, and environmental factors associated with race/ethnicity and sex are likely to affect severe obesity incidence. Future research should address the specific factors associated with onset of severe obesity. A second limitation to this research is the use of conventional albeit somewhat arbitrary BMI cut points. These cut points do not capture differences in incidence of obesity comorbidities that exist on the continuum of BMI values.28 Likewise, cut points do not capture the complex process of body weight regulation29 or the gradual process of weight gain.30 However, cut points are needed for clinical guidance and for comparative purposes. Finally, while unique longitudinal data were available for the period from adolescence to young adulthood, these data are nationally representative of the school-aged population in 1994-1995 that were followed up over time into adulthood and, thus, are not nationally representative of the population aged 24 to 33 years at follow-up.

### Table 2. Incidence of Severe Obesity by Adolescent Weight Status, Stratified by Sex and Race/Ethnicity, National Longitudinal Study of Adolescent Health

<table>
<thead>
<tr>
<th>Incidence, % (95% Confidence Interval)</th>
<th>Adolescent Weight Status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Overall</td>
</tr>
<tr>
<td></td>
<td>Normal Weight</td>
</tr>
<tr>
<td></td>
<td>Overweight</td>
</tr>
<tr>
<td></td>
<td>Obese</td>
</tr>
<tr>
<td>Male, %</td>
<td>6.3 (6.2 to 7.4)</td>
</tr>
<tr>
<td>White</td>
<td>6.2 (4.9 to 7.5)</td>
</tr>
<tr>
<td>Black</td>
<td>7.8 (5.5 to 10.1)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>6.7 (3.8 to 9.6)</td>
</tr>
<tr>
<td>Asian†</td>
<td>1.3 (−0.2 to 2.8)</td>
</tr>
<tr>
<td>Female, %</td>
<td>9.5 (8.3 to 10.7)</td>
</tr>
<tr>
<td>White</td>
<td>8.3 (6.9 to 9.7)</td>
</tr>
<tr>
<td>Black</td>
<td>15.3 (12.2 to 18.3)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>8.8 (6.1 to 11.6)</td>
</tr>
<tr>
<td>Asian†</td>
<td>7.8 (−0.5 to 16.1)</td>
</tr>
</tbody>
</table>

| Overall (n = 8834 individuates with measurements in adolescence [wave II [1996; 13-21 years] and in adulthood [wave III [2001; 18-26 years] and wave IV [2007-2009; 24-33 years]). Incident severe obesity defined as individuals who became severely obese in young adulthood (waves II-V). Results were weighted for national representation. Standard errors were corrected for multiple stages of cluster sample design and unequal probability of selection. 9
tellowight status group, male-female differences are statistically significant (P < .05 by F statistic). 
9 Data for Asians should be interpreted with caution because of small sample size. 
9 Within sex and adolescent weight status groups, white-Asian differences are statistically significant (P < .0167 by F statistic). 
A Within sex and adolescent weight status groups, white-black differences are statistically significant (P < .0167 by F statistic with Bonferroni correction).
In summary, data from a nationally representative, ethnically diverse longitudinal sample suggest a high incidence of severe obesity during the transition from adolescence to adulthood. The clinical implications of these observed trends are concerning given the comorbidities and chronic disease associated with severe obesity. Findings highlight the need for interventions prior to adulthood to prevent the progression of obesity to severe obesity, which may reduce severe obesity incidence and its potentially life-threatening consequences.

Author Contributions: Dr Gordon-Larsen had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.


Financial Disclosures: None reported.

Funding/Support: This work was supported by the National Institutes of Health grant R01-HD005791. Role of the Sponsor: The National Institutes of Health had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; or preparation, review, or approval of the manuscript.

Additional Information: This research uses data from Add Health, a program project directed by Kathleen Mullan Harris, PhD, and designed by J. Richard Udry, PhD, Peter S. Bearman, PhD, and Kathleen Mullan Harris, PhD at the University of North Carolina, Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss, PhD, and Barbara Entwistle, PhD, both from the University of North Carolina, Chapel Hill, for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health Web site (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis. None of the acknowledged individuals received compensation for their assistance.

Additional Contributions: We thank Linda S. Adair, PhD, and Elizabeth J. Mayer-Davis, PhD, University of North Carolina, Chapel Hill, for their advice and Frances Dancy, BS, University of North Carolina Carolina Population Center for her helpful administrative assistance. No compensation was received.

REFERENCES
