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The Association of Adolescent Obesity with Risk of Severe Obesity in Adulthood

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Abstract

Context—Although the prevalence of obesity has increased in recent years, individuals who are obese early in life have not been followed 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—A US nationally representative cohort was followed from adolescence through adulthood to determine incidence of severe obesity in adulthood and which groups are at highest risk.

Design, Setting, and Participants—Subjects included 8,834 individuals enrolled in wave II (1996: 12–21 y) of the National Longitudinal Study of Adolescent Health and followed into adulthood [wave III (2001–2002: 18–27 y), and wave IV (2007–2009: 24–33 y)]. Data come from measured height and weight 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 (<20 y, 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 (≥20 y, BMI≥40.0), adjusting for race/ethnicity and age and weighted for national representation.

Results—In 1996, 1.0% (95% Confidence Interval [CI], 0.7%–1.4%; n=79) of adolescents were severely obese and 70.5% (95% CI, 57.2%–83.9%; n=60) remained severely obese in adulthood. By 2009, 7.9% (95% CI, 7.4%–8.5%; n=703) of non-severely obese adolescents became severely obese in adulthood, with highest rates for non-Hispanic black females. Obese adolescents were significantly (Hazard Ratio, 16.0; 95% CI, 12.4, 20.5) more likely to develop severe obesity in young adulthood than normal weight or overweight adolescents.

Conclusions—Obesity in adolescence was significantly associated with increased risk of incident severe obesity in adulthood, with variation across sex and race/ethnicity.

Individuals with severe obesity (BMI ≥ 40 kg/m²) encounter serious and potentially life-threatening complications, including diabetes, hypertension, hyperlipidemia, asthma, arthritis,¹ and substantial reductions in life expectancy.² Repeated cross-sectional and self-report data suggest that severe obesity prevalence has risen substantially over the past few decades,^{3–7} potentially rising at a faster rate than moderate obesity.⁶ In 2000, an estimated 2.2% of adults,³ or 4.8 million individuals were severely obese,^{8–9} with a disproportionately higher prevalence in women and race/ethnic minorities.^{3–4, 7} Yet, few national studies track individuals over time to understand the progression of obesity to severe obesity.

Diet, exercise, and/or behavioral modification are recommended as initial treatments for severe obesity, resulting in short-term weight loss, which when combined with pharmacotherapy can be associated a 5–10% reduction in weight.^{10–11} However, anti-obesity pharmacological agents have substantial adverse side effects, which when discontinued often result in weight regain.¹¹ In contrast, bariatric surgery results in weight loss ranging from 60–70% for ≥ 10 years and commonly results in complete resolution or improvement in co-morbidities after surgery.¹² As such, bariatric surgery is the only treatment that has been shown to have long-term success,¹³ yet this procedure has major potential complications including leakage, pneumonia, pulmonary embolism, band slippage, and band erosion.¹⁴ Given the lack of successful treatment options, risks associated with treatment, and the numerous health consequences of severe obesity, primary prevention is critical.

Understanding which individuals are at risk for 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 prevalence of overweight, obesity, and severe obesity has increased in recent years,^{8, 15} individuals who are obese early in life have not been followed to determine their risk of developing severe obesity in adulthood. To this end, a US nationally representative, longitudinal cohort was used 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, aged 11–20 years, mean age: 15.9 y) drawn from a sample of 80 high schools and 52 middle schools from the US was selected with unequal probability of selection. Incorporating systematic sampling methods and implicit stratification into the Add Health study design ensured this sample is representative of US schools in 1994–95 with respect to region, urbanicity, school size, school type, and ethnicity. Post-stratification 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 U.S. school population in grades 7 to 12 in 1994–95 (wave I) as they are followed into adulthood. Wave II (1996, n = 14,738, mean age: 16.5 y) included wave I adolescents still of school-age by design (including those currently in high school and high school drop outs). Wave III (2001–2002, n= 15,197, mean age: 22.3 y) and wave IV (2007–2009, n=15,701, mean age: 28.9 y) followed 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, with 80.25% consenting to participate in wave IV (excluded: 96 deceased at wave III and 687 not sampled at wave III; ineligible: 184 moved out of the country, 87 active duty military stationed out of the country, and 126 deceased at wave IV). Survey procedures have

been described elsewhere and were approved by the Institutional Review Board at the University of North Carolina at Chapel Hill.¹⁶

Measures

Weight and height were measured in waves II–IV, during in-home surveys using standardized procedures. Wave I self-reported height and weight data were excluded since the gain in one additional year of follow-up was not an acceptable trade-off for the error that would have been introduced with the use of a combination of self-report (wave I) and measured (waves II–IV) height and weight data. Body mass index (BMI [kg/m²]) and BMI percentiles from measured height and weight were derived for age and gender using the US 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 ≤ BMI < 85th percentile or a 18.5 ≤ BMI < 25 for individuals ≤ 20 years and 18.5 ≤ BMI < 25 for individuals > 20 years; 2) *overweight*: 85th ≤ BMI < 95th percentile or 25 ≤ BMI < 30 for individuals ≤ 20 years and 25 ≤ BMI < 30 in individuals > 20 years; 3) *obesity*: 95th ≤ BMI < 120% of 95th percentile or 30 ≤ BMI < 40 for individuals ≤ 20 years and 30 ≤ BMI < 40 in individuals > 20 years; and 4) *severe obesity*: 120% of BMI ≥ 95th percentile ≤ 20 years¹⁹ and BMI ≥ 40 for individuals > 20 years. Respondents who exceeded scale capacity (wave III: 330 lbs, n=12, wave IV: 440 lbs, n=2) were classified as severely obese. Incident severe obesity in adulthood was classified as non-severe 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 exam. Age at onset of severe obesity was defined as the age at the wave in which the individual was initially classified as severely obese. We observed a non-linear relationship between age at onset of severe obesity and the development of severe obesity in young adulthood; thus, we categorized age at onset of severe obesity as: <20 (referent), 20–24.9, 25–29.9, and ≥30 years.

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

Statistical Analyses

Statistical analyses were conducted using Stata (Release 10.1, Stata Corporation, College Station, TX). To account for Add Health's stratified sampling strategy, clustered sampling design, and non-response 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 7–11 in 1994 and followed 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 to individuals without severe obesity, a two-sided t-test and F-statistic were used to test statistical differences (p < 0.05). 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 two-sided F-statistic was used to compare the incidence of severe obesity by these categories and the Bonferroni correction (p = 0.0167) 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,^{24–}

25 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 were normal weight as adolescents, the three categories used to obtain absolute incidence rates (normal weight, overweight, and obese) were condensed to two categories obese vs. non-obese (i.e., collapsing normal weight and overweight into the non-obese category) for the hazard analyses. Given the particular discrete time interval based on the exam dates and obesity data, models were conditioned on time as a unit of analysis with age at exam during which severe obesity was first recorded serving as the primary time variable in all models. Age-specific hazard ratios (HRs [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 became severely obese, they remained 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 (versus non-obesity) 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 three-way interaction was used to examine effect modification using Wald Tests ($p=0.10$). Despite borderline significance ($p=0.14$), differences in the associations across race/ethnicity are clinically important given the race/ethnic disparities in the prevalence of obesity and its co-morbidities. 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 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 1) 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 ($n=45$), individuals missing: sampling weights ($n=3,699$; needed to correct for non-response bias and sample design); height and weight data at wave II ($n=46$), III or IV ($n=436$); or race/ethnicity ($n=74$); individuals who were underweight ($n=1,381$; 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), and females 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 race/ethnic minorities (than participants included in the analytic sample) and a majority ($n=60$; 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 8,834 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 more likely to be race/ethnic minorities than 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 males and 51.3% (95% CI, 44.8%–57.8%) in females. Incident severe obesity was highest among black females 52.4% (95% CI, 40.9%–63.8%). Across all sex and race/ethnic groups, less than 5% of individuals who were normal weight in adolescence became severely obese in adulthood.

Using multivariate, discrete hazard models, obese adolescents were significantly more likely to develop severe obesity than normal or overweight adolescents (HR, 15.6 [95% CI, 12.1–20.0]), with variation across race/ethnicity and sex (Table 3). While the HR for males is higher than for females, the incidence of severe obesity in adulthood was higher among females (51.3% [95% CI, 44.8%–57.8%]) than males (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.

DISCUSSION

Taking advantage of a nationally representative, longitudinal dataset, we observed high rates of incident severe obesity in adulthood among individuals who were obese earlier in life, with a higher incidence in females (versus males) and highest risk for black females. These nationally representative estimates suggest 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 30's. 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. Further, 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 NHANES data suggesting a severe obesity prevalence of 4.2% (males) and 7.6% (females) in young adulthood (20–39 years) in 2008.¹⁵ Short-term, self-report data suggests that severe obesity might be rising 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 females.

While previous studies have shown a persistence of obesity from childhood/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 cutpoints. These cutpoints do not capture differences in incidence of obesity comorbidities that exist on the continuum of BMI values.²⁸ Likewise, cutpoints do not capture the complex process of body weight regulation²⁹ or the gradual process of weight gain.³⁰ However, cutpoints are needed for clinical guidance²⁸ and for comparative purposes.¹⁸ Finally, while we have unique longitudinal data over the period from adolescence to young adulthood, these data are nationally representative of the school-aged population in 1994–95 that are followed over time into adulthood, and thus are not nationally representative of the population aged 24–33 at follow-up.

In summary, data from a longitudinal, nationally representative and ethnically-diverse 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.^{4, 31–32} Findings highlight the critical 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.

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References

1. Mokdad AH, Ford ES, Bowman BA, et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA*. 2003 Jan 1; 289(1):76–79. [PubMed: 12503980]
2. Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. *JAMA*. 2003 Jan 8; 289(2):187–193. [PubMed: 12517229]
3. Freedman DS, Khan LK, Serdula MK, Galuska DA, Dietz WH. Trends and correlates of class 3 obesity in the United States from 1990 through 2000. *JAMA*. 2002 Oct 9; 288(14):1758–1761. [PubMed: 12365960]
4. Skelton JA, Cook SR, Auinger P, Klein JD, Barlow SE. Prevalence and trends of severe obesity among US children and adolescents. *Acad Pediatr*. 2009 Sep–Oct; 9(5):322–329. [PubMed: 19560993]

5. Sturm R. Increases in clinically severe obesity in the United States, 1986–2000. *Arch Intern Med*. 2003 Oct 13; 163(18):2146–2148. [PubMed: 14557211]
6. Sturm R. Increases in morbid obesity in the USA: 2000–2005. *Public Health*. 2007 Jul; 121(7):492–496. [PubMed: 17399752]
7. Wang YC, Gortmaker SL, Taveras EM. Trends and racial/ethnic disparities in severe obesity among US children and adolescents, 1976–2006. *Int J Pediatr Obes*. Mar 17.
8. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA*. 2002 Oct 9; 288(14):1723–1727. [PubMed: 12365955]
9. Arterburn DE, Maciejewski ML, Tsevat J. Impact of morbid obesity on medical expenditures in adults. *Int J Obes (Lond)*. 2005 Mar; 29(3):334–339. [PubMed: 15685247]
10. National Institutes of Health. *The Practical Guide: Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*. Bethesda, MD: National Institutes of Health, National Heart, Lung, and Blood Institute, and North American Association for the Study of Obesity; 2000.
11. Bray GA. Drug treatment of obesity. *Rev Endocr Metab Disord*. 2001 Oct; 2(4):403–418. [PubMed: 11725727]
12. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA*. 2004 Oct 13; 292(14):1724–1737. [PubMed: 15479938]
13. Colquitt JL, Picot J, Loveman E, Clegg AJ. Surgery for obesity. *Cochrane Database Syst Rev*. 2009; (2):CD003641. [PubMed: 19370590]
14. Tanner BD, Allen JW. Complications of bariatric surgery: implications for the covering physician. *Am Surg*. 2009 Feb; 75(2):103–112. [PubMed: 19280802]
15. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999–2008. *JAMA*. 2010 Jan; 303(3):235–241. [PubMed: 20071471]
16. Popkin BM, Udry JR. Adolescent obesity increases significantly in second and third generation U.S. immigrants: the National Longitudinal Study of Adolescent Health. *J Nutr*. 1998 Apr; 128(4):701–706. [PubMed: 9521631]
17. Ogden CL, Kuczmarski RJ, Flegal KM, et al. Centers for Disease Control and Prevention 2000 growth charts for the United States: improvements to the 1977 National Center for Health Statistics version. *Pediatrics*. 2002 Jan; 109(1):45–60. [PubMed: 11773541]
18. Must A, Anderson SE. Body mass index in children and adolescents: considerations for population-based applications. *Int J Obes (Lond)*. 2006 Apr; 30(4):590–594. [PubMed: 16570087]
19. Flegal KM, Wei R, Ogden CL, Freedman DS, Johnson CL, Curtin LR. Characterizing extreme values of body mass index-for-age by using the 2000 Centers for Disease Control and Prevention growth charts. *Am J Clin Nutr*. 2009 Nov; 90(5):1314–1320. [PubMed: 19776142]
20. The NS, Gordon-Larsen P. Entry into Romantic Partnership is Associated with Obesity. *Obesity (Silver Spring)*. 2009; 17(7):1441–1447. [PubMed: 19360012]
21. Gordon-Larsen P, Adair LS, Suchindran CM. Maternal obesity is associated with younger age at obesity onset in U.S. adolescent offspring followed into adulthood. *Obesity (Silver Spring)*. 2007 Nov; 15(11):2790–2796. [PubMed: 18070770]
22. Chantala, K.; Kalsbeek, D.; Andrace, E. Non-response in wave III of the Add Health Study. [Accessed July 7, 2010]. www.cpc.unc.edu/projects/addhealth/data/guides/W3nonres.pdf.
23. Tourangeau, R.; Shin, HC. National Longitudinal Study of Adolescent Health: Grand Sample Weights. 1999 [Accessed July 7, 2010]. <http://www.cpc.unc.edu/projects/addhealth/data/guides/weights.pdf>.
24. Prentice RL, Gloeckler LA. Regression analysis of grouped survival data with application to breast cancer data. *Biometrics*. 1978 Mar; 34(1):57–67. [PubMed: 630037]
25. Richardson DB. Discrete time hazards models for occupational and environmental cohort analyses. *Occup Environ Med*. 2010 Jan; 67(1):67–71. [PubMed: 20029026]
26. McTigue KM, Garrett JM, Popkin BM. The natural history of the development of obesity in a cohort of young U.S. adults between 1981 and 1998. *Ann Intern Med*. 2002 Jun 18; 136(12):857–864. [PubMed: 12069559]

27. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med.* 1993 Mar; 22(2):167–177. [PubMed: 8483856]
28. Stevens J. Ethnic-specific cutpoints for obesity vs country-specific guidelines for action. *Int J Obes Relat Metab Disord.* 2003 Mar; 27(3):287–288. [PubMed: 12629554]
29. Zheng H, Lenard NR, Shin AC, Berthoud HR. Appetite control and energy balance regulation in the modern world: reward-driven brain overrides repletion signals. *Int J Obes (Lond).* 2009 Jun.33 Suppl 2:S8–S13. [PubMed: 19528982]
30. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? *Science.* 2003 Feb 7; 299(5608):853–855. [PubMed: 12574618]
31. Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Study. *J Pediatr.* 2007 Jan; 150(1):12–17. e12. [PubMed: 17188605]
32. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA.* 1999 Oct 27; 282(16):1523–1529. [PubMed: 10546691]

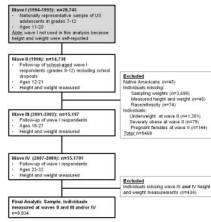


Figure 1.
Flow diagram of the National Longitudinal Study of Adolescent Health and Exclusion Criteria for Analytic Sample

Table 1

Selected Characteristics of Participants from the National Longitudinal Study of Adolescent Health, by Longitudinal Severe Obesity Status (n = 9,091 Individuals; measured at adolescence, Wave II [1996; ages 12–21 y], and in adulthood: Wave III [2001–2002; ages 18–27 y], and Wave IV [2007–2009; ages 24–33])^a

	Weighted		Unweighted	
	No Severe Obesity in adulthood	Incident Severe Obesity in adulthood	No Severe Obesity in adulthood	Incident Severe Obesity in adulthood
n	8,131	703	8,131	703
% (SE)	92.1 (0.4)	7.9 (0.4)	92.0 (0.3)	8.0 (0.3)
Baseline Age (y) [mean (SD)]	16.5 (1.6) ^f	16.7 (1.7)	16.8 (1.6) ^f	16.9 (1.6)
Males [% (SE)]	n = 3,991	n = 260	n = 3,991	n = 260
White	70.3 (3.0)	68.9 (5.0)	57.6 (0.8) ^f	50.0 (3.1)
Black	14.5 (2.1)	18.3 (3.7)	19.7 (0.6)	23.8 (2.6)
Hispanic	11.3 (1.8)	12.1 (3.1)	15.9 (0.6)	19.2 (2.4)
Asian	3.9 (0.8) ^f	0.7 (0.6)	6.7 (0.4)	6.9 (1.6)
Females [% (SE)]	n = 4,140	n = 443	n = 4,140	n = 443
White	70.8 (3.0) ^f	61.2 (4.9)	56.8 (0.8) ^f	46.5 (2.4)
Black	15.2 (2.1) ^f	26.2 (4.2)	22.2 (0.6) ^f	32.1 (2.2)
Hispanic	11.3 (1.7)	10.5 (2.3)	15.9 (0.6)	17.4 (1.8)
Asian	2.7 (0.7)	2.2 (1.6)	5.0 (0.3)	4.1 (0.9)
Baseline Body Weight (age 13–21) BMI [mean (SD)]	22.7 (3.2) ^f	28.8 (3.8)	22.7 (3.2) ^f	29.1 (3.8)
Overweight [% (SE)] ^d	18.0 (0.6) ^f	25.6 (2.4)	17.6 (0.4) ^f	26.6 (1.7)
Obese [% (SE)] ^e	6.4 (0.4) ^f	57.6 (2.8)	6.3 (0.3) ^f	57.9 (1.9)

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

^a Results were weighted for national representation, and the standard errors were corrected for multiple stages of cluster sample design and unequal probability of selection.

^b Adolescent (<20 y) severe obesity defined as defined as 120% of the 2000 NCHS/CDC growth chart age- and sex-specific BMIs \geq 95th percentile cut point. Adult (\geq 20 y) severe obesity defined as BMI \geq 40.

^c Adolescent (<20 y) overweight defined as defined using the 2000 NCHS/CDC growth chart age- and sex- specific 85th \geq BMIs < 95th percentile cut point or 25 \geq BMI < 30. Adult (\geq 20 y) overweight defined as 25 \geq BMI < 30.

^d Adolescent (<20 y) obesity defined as defined using the 2000 NCHS/CDC growth chart age- and sex- specific 95th \geq BMIs < 120% of 95th percentile cut point or 30 \geq BMI < 40. Adult (\geq 20 y) obesity defined as 30 \geq BMI < 40.

^e No observations

^f Significant differences between individuals with severe obesity and individuals without severe obesity (p < 0.05)

Table 2

Incidence of Severe Obesity^a by Adolescent Weight Status, stratified by sex and race/ethnicity, National Longitudinal Study of Adolescent Health, (n = 8,834 Individuals; measured at adolescence, Wave II [1996 (13–21 y)], and in adulthood: Wave III [2001–2002 (18–26y)], and Wave IV [2007–2009 (24–33y)])^b

	Overall	Adolescent Weight Status		
		Normal Weight	Overweight	Obese
Males (%)	6.3 (5.2, 7.4) ^d	1.2 (0.6, 1.9) ^d	6.4 (4.1, 8.6) ^d	37.1 (30.6, 43.6) ^d
White	6.2 (4.8, 7.5)	1.0 (0.2, 1.8)	6.2 (3.6, 8.8)	35.6 (27.5, 43.6)
Black	7.8 (5.5, 10.1)	2.0 (0.6, 3.5)	10.8 (2.1, 19.6)	44.7 (33.4, 56.0)
Hispanic	6.7 (3.8, 9.6)	1.7 (−0.4, 3.9)	4.0 (0.8, 7.1)	44.4 (29.3, 59.4)
Asian ^c	1.3 (−0.2, 2.8) ^e	0.1 (0.0, 0.3)	0.4 (−0.3, 1.2) ^e	13.7 (−1.6, 29.0) ^e
Females (%)	9.5 (8.3, 10.7)	2.4 (1.7, 3.1)	15.5 (11.9, 19.2)	51.3 (44.8, 57.8)
White	8.3 (6.9, 9.7)	2.2 (1.4, 3.0)	14.2 (9.8, 18.6)	51.8 (42.8, 60.1)
Black	15.3 (12.2, 18.3) ^f	3.9 (1.7, 6.0)	20.5 (12.1, 28.9)	52.4 (40.9, 63.8)
Hispanic	8.8 (6.1, 11.6)	2.9 (3.6, 5.4)	10.1 (3.6, 16.6)	47.7 (34.7, 60.8)
Asian ^c	7.8 (−0.5, 16.1)	0.0 (0.0, 0.1)	39.5 (−0.1, 79.6) ^e	37.8 (21.0, 54.5)

^a Incident severe obesity defined as individuals who became severely obese in young adulthood (waves III or IV)

^b Results were weighted for national representation, and the standard errors were corrected for multiple stages of cluster sample design and unequal probability of selection.

^c Proportions should be interpreted with caution due to small sample size

^d Within adolescent weight status group, male-female differences (F-statistics, p<0.05)

^e Within-sex and adolescent weight status group, white-Asian differences (F statistic, with Bonferoni correction, p<0.0167)

^f Within-sex and adolescent weight status group, white-Black differences (F statistic, with Bonferoni correction, p<0.0167)

Table 3

Association between Adolescent Obesity (wave II [1996; ages 12–21y]) and Incident Severe Obesity in Adulthood (Wave III [2001–2002; ages 18–27y] and Wave IV [2007–2009; ages 24–33y]).^a The National Longitudinal Study of Adolescent Health, n = 15,598 observations across 8,834 Individuals

	HR (95% CI)
Total (n = 15,598)	16.0 (12.4, 20.5)
Males (n = 7,638)	
White (n = 4,374)	21.1 (12.9, 34.7)
Black (n = 1,498)	29.2 (17.4, 48.8)
Hispanic (n = 1,232)	28.0 (15.2, 51.8)
Asian (n = 534)	6.6 (1.8, 23.7) ^b
Females (n = 7,960)	
White (n = 4,482)	16.7 (11.2, 24.9)
Black (n = 1,812)	18.1 (12.1, 27.0)
Hispanic (n = 1,261)	16.1 (9.6, 27.1)
Asian (n = 405)	12.5 (6.7, 23.4) ^b

Abbreviations: HR, hazard ratio; CI, confidence interval

^aHazard ratios (incident rate ratios) and 95% confidence intervals were obtained from sex-stratified, multivariate discrete-time hazard regression models predicting incident severe obesity by adolescent obesity (obese vs. non-obese [referent]) adjusted for age (categorized: <20, 20–24.9, 25–29.9, ≥30 years), race (White, Black, Hispanic, Asian), and the interaction terms for race X adolescent obesity.

^bEstimates unstable due to small sample size (<10 non-obese individuals in referent category (normal or overweight) became severely obese in adulthood)