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Childhood overweight and cardiovascular disease risk factors: The National Heart, Lung, and Blood Institute Growth and Health Study

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Abstract

Objective—To estimate the prevalence and incidence of overweight in black and white girls, and to examine associations between adolescent overweight and cardiovascular disease (CVD) risk factors.

Study design—In the National Heart, Lung, and Blood Institute Growth and Health Study (NGHS), annual measurements were obtained from girls followed longitudinally between the ages of 9 or 10 and 18; self-reported measures were obtained at ages 21–23. Participants were 1,166 white and 1,213 black girls. Childhood overweight as defined by the Centers for Disease Control and Prevention (CDC) was the independent variable of primary interest. Measured outcomes included blood pressure and lipid levels.

Results—Rates of overweight increased through adolescence from 7% to 10% in white girls and 17% to 24% in black girls. Incidence of overweight was greater during ages 9–12 than in later adolescence. Girls who were overweight during childhood were 11 to 30 times more likely to be obese in young adulthood. Overweight was significantly associated with increased percent body fat, sum of skinfolds and waist circumference, unhealthful systolic and diastolic blood pressure, HDL cholesterol and triglycerides.

Conclusion—The relationship between CVD risk factors and CDC-defined overweight is present at age 9.

Keywords

Obesity; Blood pressure; Cholesterol; Triglycerides; Body Mass Index

Findings from the National Health and Nutrition Examination Surveys have shown that the prevalence of childhood overweight has continued to increase with each survey, from 4–6% in 1976–80 to 16% in 1999–2002.^{1–2} Overweight during childhood is not benign. Adverse levels of cardiovascular (CVD) risk factors—increased blood pressure, serum total cholesterol (TC),

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low-density lipoprotein cholesterol (LDL), triglycerides, fasting insulin and reduced high-density lipoprotein cholesterol (HDL)—are associated with increased body mass index (BMI).³⁻⁷ The greater the increase in BMI, the greater the increase in risk factor levels.

Obesity and cardiovascular risk factors track from childhood into adulthood.⁸⁻¹¹ In addition, adults who were overweight in childhood have higher levels of lipids, blood pressure, and fasting insulin and are thus at increased risk for coronary heart disease compared with adults who were thin as children.^{8-9, 12}

BMI has been the measure of choice in national surveys for defining overweight in children and has been recommended by health experts for use in clinical practice.¹³⁻¹⁴ Precise measurements of adiposity are impractical to implement; however, the components of BMI, height and weight, can be measured easily with high accuracy and reliability and have been correlated with body fat in children and adolescents, including young African-American girls.¹⁵⁻¹⁷ The definition of overweight that is commonly used is the age- and sex-specific 95th percentile for BMI from the 2000 Growth Charts.¹⁸ The utility of the Centers for Disease Control and Prevention (CDC) cutpoints to classify overweight children would be greatly enhanced if the magnitude of increased risk associated with those cutpoints were assessed.

The National Heart, Lung, and Blood Institute Growth and Health Study (NGHS) is a longitudinal study involving a cohort of 2,379 black and white females, examined annually from ages 9-10 through ages 18-19 and later interviewed at ages 21-23.¹⁹⁻²¹ The purpose of NGHS was to investigate correlates and outcomes of overweight in black and white females to better understand why black women experience greater rates of obesity and cardiovascular disease mortality compared with their white peers.²² Data collected from the NGHS cohort provides the opportunity to examine overweight and its relationship to adiposity and cardiovascular risk factors, using CDC 2000 growth chart BMI cutpoints for childhood overweight. Also, incidence of overweight can be assessed in this cohort. Knowledge of the incidence of obesity across the adolescent years may help determine whether there are critical time periods when obesity incidence is greatest and when the odds of being obese during young adulthood are greatest. In addition, measures of blood pressure and serum lipids obtained at clinic visits during adolescence allow for an assessment of the relationship between the CDC definition of overweight at each age and prevalence of adverse levels of CVD risk factors based on clinical cutpoints used for screening and risk assessment.

METHODS

Participants and Recruitment

NGHS was designed to have a sample of adequate size to permit comparisons of indicators of obesity by race.¹⁹ Although there was some geographic diversity, the sample was not designed to be nationally representative. As previously reported,¹⁹ 2,379 black and white girls who were 9 or 10 years old at study entry were recruited at three institutions: University of California at Berkeley; University of Cincinnati / Cincinnati Children's Hospital Medical Center; and Westat, Inc. (Rockville, MD). The Maryland Medical Research Institute served as coordinating center, and the National Heart, Lung, and Blood Institute also participated. Participants were recruited from schools, health maintenance organizations and Girl Scout troops. Areas selected were based on census tract data in order to include a wide distribution of household incomes and parental education within each race. Eligibility criteria were few¹⁹: girls were age 9 or 10 years, identified themselves as "black" or "white," were non-Hispanic, and had racially concordant parents or guardians. Eligible participants were invited to enroll until the target sample size was attained; an effort was made to enroll each eligible girl to minimize the risk of sample bias. All girls who enrolled in the study had assented, and their parents (or guardian)

had provided written informed consent. The study protocol was approved by the Institutional Review Boards of all participating institutions.

During 1986-1997, girls attended 10 annual “clinic visits” (visits 1-10). Retention rates were very high at visits 2-4 (96%, 94%, 91%), declined to a low of 82% at visit 7, and increased to 89% at visit 10. Data were collected at the three clinic sites or, if the girl was unable to travel to the site, at her home, using standardized procedures. Between 1998 and 2001, a single telephone interview (NGHS-Wave II²¹) was conducted with 2,054 NGHS participants (991 White and 1,063 Black) who were then between ages 21 and 23 (mean age = 21.5 years, SD = 0.6).

Instruments and Measures

Only instruments and measures relevant to the present report are described below. Physical measures were obtained annually at all 10 visits when girls were ages 9-10 (visit 1) through 18-19 years (visit 10), unless stated otherwise.

Demographic information—Information regarding age and race/ethnicity was collected at study entry from girls and their parents (or guardians) by self-report, using United States census categories. Age was coded as age at last birthday, except that age in months was used to classify overweight by the CDC definition.

Height, weight, and waist circumference—Central training sessions were conducted annually to train and certify data collectors to follow a common measurement protocol. Data collectors obtained two measurements of height and weight and took a third if the first 2 differed by a pre-determined amount. The average of all measurements was used. In NGHS-Wave II, participants were asked during the telephone interview to self-report their current weight.²¹ BMI was calculated as weight in kilograms divided by height in meters squared. Beginning with visit 2, waist circumference (narrowest part of the torso) was also measured annually.²³

Childhood overweight and young adult obesity—The Centers for Disease Control and Prevention age-specific 95th percentile for girls was used to classify overweight at ages 9-18 years (CDC Growth Charts, available at <http://www.cdc.gov/growthcharts/>). Young adult obesity (ages 21 and up) was defined as BMI of 30 kg/m² or greater.²⁴

Body fat—Resistance and reactance measures of bioelectrical impedance were collected using bioelectrical impedance (PJL Systems model 10, Detroit, MI). Percent body fat was computed as $\beta_{0e} + \beta_{1e} * (\text{height}^2/\text{resistance}) + \beta_{2e} * \text{weight} + \beta_{3e} * \text{reactance}$, where $\beta_{0e}, \dots, \beta_{3e}$ are ethnicity-specific coefficients (e = 1 if white, 2 if black) from predictive models of fat-free mass (based on dual energy x-ray anthropometry, $R^2 = 0.99$), developed with a separate sample of 126 black and white girls, ages 6-17.^{10, 25}

Skinfolds—Skinfold measurements at the triceps, suprailiac, and subscapular sites were obtained following a standard protocol;²⁶ these measures were added to compute sum of skinfolds.

Blood pressure—Blood pressure was measured three times, with measurements taken at least 60 seconds apart using a standard mercury sphygmomanometer with cuffs that were appropriate for size of arm circumference. The second and third measurements were used to calculate the mean systolic (K_1) and diastolic (K_5) blood pressures (SBP and DBP, respectively). The elevated range was defined as at or above the sex- and age-specific 95th percentile for SBP and DBP, also taking height into account.²⁷

Lipids—Overnight fasting blood specimens were obtained in the morning at visits 1, 3, 5, 7, and 10. The total cholesterol and HDL cholesterol levels were determined using the CHOD-PAP method.²⁶ Triglycerides were analyzed enzymatically using a commercially available method, and LDL cholesterol was calculated using the Friedewald formula²⁹ as modified based on the Lipid Research Clinics data:³⁰ $LDL\ Cholesterol = Total\ Cholesterol - HDL\ Cholesterol - (Triglycerides/6.5)$. High levels of lipid measures were defined as above 200 for total cholesterol, above 130 for LDL cholesterol, and 130 or above for triglycerides;³¹ HDL cholesterol levels below 50 were considered low.³²

Incidence (new onset) of childhood overweight during NGHS was determined as follows. Girls who were overweight at the first visit were excluded from the analyses of overweight onset. Girls who were not overweight at the first visit could experience onset of overweight from the second visit onward. The percent of new onset cases was computed at each age as the number of new onset cases at that age, divided by the total number of girls with BMI data for each age between study entry (age 9 or 10) and that age. Girls were excluded from the “risk set” (denominator) at a given age if at any prior age they were overweight or their overweight status was unknown.

Statistical analysis

The association of overweight prevalence with age and race (including the interaction) was computed using mixed logistic regression to account for the repeated measurements.³³⁻³⁴ To test hypotheses about the association of incidence (onset of overweight) with age and race, the length of time to first onset of overweight was modeled (piecewise exponential accelerated failure time model)³⁵ The degree of association between overweight and three indicators of body fatness (i.e., percent body fat from bioelectric impedance measures, sum of skinfolds, and waist circumference) was estimated with linear mixed models adjusting for age, race, and their interactions with overweight. Logistic regression was used to estimate the association between overweight at each age in childhood and obesity in early adulthood, adjusting for race. Finally, mixed logistic regression was used to examine the association of overweight with the probability of developing elevated (or, for HDL cholesterol, low) levels of CVD risk factors, adjusting for age, race, their interactions with overweight. In addition to the variables mentioned above, all analyses adjusted for study site. For outcomes with small samples in some analysis cells (i.e., incidence of overweight and CVD risk factors at specific ages), age effects were modeled using two age segments, 9-12 and >12-18;³⁶ the segmentation was at age 12 because this was the approximate mean age of menarche in this cohort.²⁰ A separate effect for each age was estimated in all other models. Direct likelihood estimation or multiple imputation was used to account for missing data.³⁷⁻³⁹ SAS 9.1 (SAS Institute, Cary, NC) was used for all analyses. P-values less than 0.05 were considered statistically significant.

Results

Prevalence and Incidence of Childhood Overweight

Table I reports the number of participants with BMI data by age, and shows the prevalence and incidence (new onset) of overweight in black and white girls at each age. Overweight was more prevalent in black girls than white girls (odds ratio = 4.9, 95% CI: 1.6-8.2; $p < 0.0001$). Rates of overweight differed significantly by age ($p < 0.0001$), tending to increase as girls grew older, with the increase in age stronger in black girls than white girls, although the interaction term was not significant (age-by-race interaction, $p = 0.06$).

The percent of new onset cases (i.e., incidence) of childhood overweight ranged from 2-5% through age 12, after which the annual incidence was generally lower, approximately 1-2%. The estimated instantaneous risk (hazard) of experiencing overweight was greater during ages

9-12 than it was above age 12 (hazard ratio = 1.6, 95% CI: 1.1-2.3, $p=0.03$). For black girls, the risk of experiencing overweight onset at any given time was about 1.5 times greater than that for white girls (hazard ratio = 1.5, 95% CI: 1.2-2.0, $p=0.003$).

To examine the possibility that changes in incidence across age were an artifact of using a BMI-based definition of overweight, which includes a lean body mass component, an analogous analysis was conducted, with overweight defined as being above the age-specific 95th percentile of sum of skinfolds for the NGHS sample. The results were very similar: the incidence of overweight during ages 9-12 was 1.7 times greater (95% CI: 1.2-2.5, $p=0.008$) than it was after age 12.

Childhood Overweight and Obesity in Young Adulthood

Table II shows the percent of girls who were obese in young adulthood (NGHS Wave II, ages 21-23) by their overweight status at each age from 9 to 18, inclusive. Compared with girls who were not overweight, those who were overweight during ages 9-18 were much more likely to be obese as young adults ($p's < 0.0001$).

Childhood Overweight and Other Adiposity Indicators

Table III shows the association between childhood overweight and three indicators of body fatness by age. For all indicators, the mean values for overweight girls were greater than the mean values for non-overweight girls ($p's < 0.0001$), and the differences between the measures for overweight and non-overweight girls tended to increase as girls grew older (overweight-by-age interaction, $p's < 0.0001$). All measures increased with age ($p's < 0.0001$). For percent body fat and waist circumferences, means were greater for black girls ($p's < 0.0001$) by approximately 5.7% (95% CI: 4.4-7.1) and 2.8% (95% CI: 1.9-3.7), respectively, but there was no racial difference for sum of skinfolds ($p=0.09$). There were significant race-by-overweight interactions for percent body fat and sum of skinfolds ($p's < 0.0003$) but not waist circumference ($p = 0.81$). For percent body fat, the difference between overweight and non-overweight girls was greater among white girls (percent difference = 14.6%, 95% CI: 12.6-16.5) than among black girls (percent difference = 11.2%, 95% CI: 9.4-13.0), although the opposite was true for sum of skinfolds (percent difference = 39.5%, 95% CI: 35.8-43.2, and 48.4%, 95% CI: 43.3-53.7, for white and black girls, respectively).

Based on results of mixed models technique, which is analogous to partial R^2 in ordinary multiple regression,⁴⁰⁻⁴¹ overweight was most strongly associated with sum of skinfolds and waist circumference, and less strongly (albeit significantly) associated with percent body fat, even after adjusting for study site, age and race.

Childhood overweight and outcomes

Table IV shows the percent of girls with high levels of blood pressure and lipids (low levels for HDL), by age and overweight.

Compared with non-overweight girls, overweight girls were more likely to exhibit elevated SBP and elevated DBP ($p=0.01$). Rates of elevated SBP and DBP also changed with age, but these changes depended on overweight. Among overweight girls, rates of elevated SBP increased between ages 9-12 ($p=0.03$) and decreased thereafter ($p < 0.0001$), although rates of elevated SBP did not change with age among non-overweight girls. Among non-overweight girls only, rates of elevated DBP decreased during early adolescence (ages 9-12, $p=0.01$); among both overweight and non-overweight girls, rates of elevated DBP tended to decrease after age 12 ($p=0.005$). Neither measure of elevated blood pressure varied by race ($p's > 0.08$).

Compared with non-overweight girls, overweight girls were more likely to exhibit decreased HDL ($p < 0.0001$) and elevated triglycerides ($p = 0.002$). Overweight was not significantly associated with elevated LDL or elevated total cholesterol (p 's > 0.06).

Among both overweight and non-overweight girls, there were significant decreases in rates of unhealthy total cholesterol (TC), LDL and HDL during ages 9-12 (p 's < 0.05); further, among non-overweight girls, unhealthy triglycerides (TG) increased between ages 9-12 ($p = 0.001$). Overweight and non-overweight girls showed different age trends after age 12: among overweight girls, rates of unhealthy TC and LDL (p 's < 0.002) tended to increase, although among non-overweight girls, there was an increase after age 12 for low HDL, but a decrease for elevated TG (p 's < 0.05). Non-overweight black girls were more likely to exhibit elevated TC than were non-overweight white girls (OR for race = 1.6, 95% CI: 1.1-2.4, $p = 0.02$), whereas overweight black girls were less likely to exhibit elevated TC (OR for race = 0.6, 95% CI: 0.1-1.0, $p = 0.03$). Regardless of overweight, black girls were less likely than white girls to exhibit unhealthy HDL and TG (for HDL, OR for race = 0.6, 95% CI: 0.6-0.7; for TG, OR for race = 0.5, 95% CI 0.4-0.6, p 's < 0.0001). There were no racial differences in LDL (p 's > 0.31).

Pubertal maturation has been associated with lipids.⁴²⁻⁴³ If maturation explains some of the variation in unhealthy lipid levels, above and beyond that due to age, maturation may impact the estimated association of lipids with overweight. Therefore, as a secondary analysis, the association of overweight with unhealthy lipid levels was re-estimated in models controlling for maturation as well as age (maturation stage was represented as pre-pubertal, pubertal, post-menarchal < 2 years, or post-menarchal ≥ 2 years). Although the estimates were generally similar (Table IV), adding maturation tended to result in tighter confidence intervals. One consequence was that in the model controlling for maturation, unhealthy LDL was significantly associated with overweight ($p = 0.01$).

DISCUSSION

The aims of the present study were to examine racial differences in females in the prevalence and incidence of overweight using the CDC definition; compare overweight with several other adiposity indicators; estimate the association between childhood overweight and obesity in young adulthood; and examine relations between overweight and unhealthy high (or low) levels of CVD risk factors. NGHS offered a unique opportunity to fulfill these aims, due to high-quality measures of height, weight, adiposity, blood pressure and lipids collected longitudinally on a large and ethnically diverse sample during an important developmental period encompassing childhood and adolescence.

Our findings point to two useful areas to target obesity prevention efforts. First, in accordance with earlier studies reporting high rates of childhood overweight for African Americans,^{1-2, 44} black girls in NGHS had higher prevalence rates and also experienced overweight onset at a significantly faster rate than white girls. This finding highlights the importance of prevention efforts that consider cultural differences.⁴⁵ Second, the incidence of overweight in NGHS was significantly higher at ages 9 to 12 than at older ages; this observation was true for the BMI-based definition of overweight as well as for a definition based on sum of skinfolds. The pubertal period, a time when body fat is rapidly deposited, may be a vulnerable period during which risk for overweight becomes pronounced.^{20, 46-47} The rapid increase in overweight during ages 9-12 suggests that this pubertal period may be an especially important time for clinical and public health interventions to prevent overweight.

Our data suggest that childhood overweight may have serious consequences in both the short and long term. Our data highlight that the relationship between CVD risk factors and CDC-

defined overweight is already present at age 9 and suggest that pediatricians need to address the health correlates of overweight during childhood.⁴⁷ Girls who were overweight were 3-10 times more likely to be assessed in the “risk” range on four of six health indicators (systolic and diastolic blood pressure, HDL, and triglycerides), and had 3 times greater odds of having elevated LDL in a model controlling for maturation ($P=.01$). Freedman et al⁵ found similar results among 5-17 year-olds using seven cross-sectional studies. These findings also suggest that the CDC definition of overweight based on BMI is valid and that it provides a useful standard for classifying overweight in both African American and white adolescent girls in clinical and research settings.^{5, 48-50} Consistent with others,⁴⁸⁻⁵⁰ percent body fat, sum of skinfolds, and waist circumference were substantially higher in overweight girls of both races, compared with nonoverweight girls, illustrating that measures of height and weight that comprise BMI are strongly related to these indicators of adiposity.

Over the longer term, childhood overweight was strongly associated with young adult obesity, even measured more than 10 years apart. Consistent with others,⁹⁻¹¹ we found that childhood overweight confers a 10-fold or greater increase in risk for being overweight in early adulthood, relative to children who were not overweight at the same age. One limitation is that young adult weight was self-reported, and the study did not assess the reliability of self-reported weight. Although self-reported body weight may be subject to bias, it is noteworthy that young adult obesity was strongly associated with childhood overweight, and the strength of this association was similar in girls of both races to that observed in previous studies, providing some assurance that there was no large, systematic bias.

Interventions to address childhood obesity and unfavorable CVD risk factors levels have primarily been conducted in school settings.⁵¹ Although changes in food intake and physical activity in the desired directions were observed in some studies,⁵²⁻⁵³ success in reducing BMI has been observed in only a few studies conducted in schools.⁵⁴⁻⁵⁶ Intervention studies in community settings have begun to specifically address childhood obesity in African American girls.⁵⁷⁻⁵⁸ Several community outreach programs addressing eating behavior and physical activity for parents and their children are currently under way in more than 95 communities across the country.⁵⁹ Pediatricians and primary care practitioners need to support public health and societal efforts to prevent childhood overweight by monitoring growth and providing advice to parents on the need for their children to maintain a healthy weight during adolescence through healthy eating and regular physical activity.⁶⁰ The challenge for clinicians, community leaders, researchers and public health officials will be to develop effective innovative obesity prevention interventions that are generalizable and disseminable so that the dire prediction that deaths related to obesity will soon become the leading cause of mortality in the U.S. does not come to pass.⁶¹

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References

1. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999-2000. *JAMA* 2002;288:1728–32. [PubMed: 12365956]
2. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. *JAMA* 2004;291:2847–50. [PubMed: 15199035]
3. Clarke WR, Woolson RF, Lauer RM. Changes in ponderosity and blood pressure in childhood: the Muscatine Study. *Am J Epidemiol* 1986;124:195–206. [PubMed: 3728436]

4. Gidding SS, Bao W, Srinivasan SR, Berenson GS. Effects of secular trends in obesity on coronary risk factors in children: the Bogalusa Heart Study. *J Pediatr* 1995;127:868–74. [PubMed: 8523181]
5. Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics* 1999;103:1175–82. [PubMed: 10353925]
6. Srinivasan SR, Myers L, Berenson GS. Temporal association between obesity and hyperinsulinemia in children, adolescents, and young adults: the Bogalusa Heart Study. *Metabolism* 1999;48:928–34. [PubMed: 10421238]
7. Srinivasan SR, Myers L, Berenson GS. Distribution and correlates of non-high-density lipoprotein cholesterol in children: the Bogalusa Heart Study. *Pediatrics* 2002;110:e29. [PubMed: 12205279]
8. Obarzanek, E. Obesity in children, adolescents, and families. In: Fletcher, GF.; Grundy, SM.; Hayman, LL., editors. *Obesity: impact on Cardiovascular Disease*. Armonk, NY: Futura Publishing Co., Inc.; 1999. p. 31-53.
9. Steinberger J, Moran A, Hong CP, Jacobs DR Jr, Sinaiko AR. Adiposity in childhood predicts obesity and insulin resistance in young adulthood. *J Pediatr* 2001;138:469–73. [PubMed: 11295707]
10. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *Am J Clin Nutr* 2002;76:653–8. [PubMed: 12198014]
11. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Interrelationships among childhood BMI, childhood height, and adult obesity: the Bogalusa Heart Study. *Int J Obes* 2004;28:10–16.
12. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics* 2001;108:712–8. [PubMed: 11533341]
13. Himes J, Dietz W. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. *Am J Clin Nutr* 1994;59:307–16. [PubMed: 8310979]
14. Dietz WH, Robinson TN. Overweight children and adolescents. *N Engl J Med* 2005;352:2100–9. [PubMed: 15901863]
15. Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, HEymsfield SB. Body mass index as a measure of adiposity among children and adolescents: a validation study. *J Pediatr* 1998;132:204–10. [PubMed: 9506629]
16. Frontini MG, Bao W, Elkasabany A, Srinivasan SR, Berenson G. Comparison of weight-for-height indices as a measure of adiposity and cardiovascular risk from childhood to young adulthood: the Bogalusa Heart Study. *J Clin Epidemiol* 2001;54:817–22. [PubMed: 11470391]
17. Bedogni G, Iughetti L, Ferrari M, Malavolti M, Poli M, Bernasconi S, et al. Sensitivity and specificity of body mass index and skinfold thicknesses in detecting excess adiposity in children aged 8-12 years. *Ann Human Biol* 2003;30:132–9. [PubMed: 12637189]
18. Centers for Disease Control and Prevention [homepage on the Internet]. Hyattsville, MD :CDC ; c2000. National Center for Health Statistics. Available from: http://www.cdc.gov/nchs/about/major/nhanes/growthcharts/clinical_charts.htm
19. National Heart, Lung, and Blood Institute Growth and Health Study Research Group. Obesity and cardiovascular disease risk factors in black and white girls: the NHLBI Growth and Health Study. *Am J Public Health* 1992;82:1613–20. [PubMed: 1456335]
20. Kimm, SYS.; Barton, BA.; Obarzanek, E.; McMahon, RP.; Sabry, ZI.; Waclawiw, MA., et al. Racial divergence in adiposity during adolescence: the NHLBI Growth and Health Study. *Pediatrics*. 2001. Available at: <http://www.pediatrics.org/cgi/content/full/103/3/e34>
21. Striegel-Moore RH, Dohm FA, Kraemer HC, Taylor CB, Daniels S, Crawford PB, et al. Eating disorders in white and black women. *Am J Psychiatry* 2003;160:1326–31. [PubMed: 12832249]
22. Gillum RF, Liu KC. Coronary heart disease mortality in United States Blacks, 1940-1978: trends and unanswered questions. *Am Heart J* 1984;108:653–657. [PubMed: 6475736]
23. Callaway, CW.; Chumlea, WC.; Bouchard, C.; Himes, JH.; Lohman, TG.; Martin, AD., et al. Circumferences. In: Lohman, TG.; Roche, AF.; Martorell, R., editors. *Anthropometric standardization reference manual*. Champaign, IL: Human Kinetics; 1988. p. 41-42.

2. National Heart, Lung, and Blood Institute, National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. *Obesity Research* 1998;6:54S.
25. Morrison JA, Guo SS, Specker B, Chumlea WC, Yanovski SZ, Yanovski JA. Assessing the body composition of black and white girls in field studies, 6 to 17 Years of Age. *Am J Hum Biol* 2001;13:249–54. [PubMed: 11460870]
26. Cameron, N. *The Measurement of Human Growth*. London, England: Croom Hehn; 1984.
27. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The Fourth Report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics* 2004;114:555–76. [PubMed: 15286277]
28. Bachorik P, Walker RE, Virgil DG. High-density-lipoprotein cholesterol in heparin-MnCl₂ supernates determined with the Dow enzymic method after precipitation of Mn²⁺ with HCO₃⁻. *Clinical Chemical* 1984;30:839–42.
29. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972;18:499–502. [PubMed: 4337382]
30. DeLong DM, DeLong ER, Wood PD, Lippel K, Rifkind BM. A comparison of methods for the estimation of plasma low- and very low-density lipoprotein cholesterol. The Lipid Research Clinics Prevalence Study. *JAMA* 1986;256:2372–7. [PubMed: 3464768]
31. National Cholesterol Education Program. Report of the Expert Panel on Blood Cholesterol in Children and Adolescents. *Pediatrics* 1992;89:525–84. [PubMed: 1538956]
32. Morrison JA, Friedman LA, Harlan WR, Harlan LC, Barton BA, Schreiber GB, et al. Development of the metabolic syndrome in black and white adolescent girls: a longitudinal assessment. *Pediatrics* 2005;116:1178–82. [PubMed: 16264006]
33. Verbeke, G.; Molenberghs, G. *Linear mixed models for longitudinal data*. New York: Springer; 2000.
34. Prescott, P.; Brown, H. *Applied mixed models in medicine*. Chichester, NY: J Wiley & Sons; 1999.
35. Allison, PD. *Survival analysis using the SAS system: a practical guide*. Cary, NC: SAS Publishing; 1995.
36. Harrell, FE. *Regression modeling strategies*. New York: Springer-Verlag; 2001.
37. Allison, PD. *Missing data*. Thousand Oaks, CA: Sage Publications; 2002.
38. Beunckens C, Molenberghs G, Kenward MG. Direct likelihood analysis versus simple forms of imputation for missing data in randomized clinical trials. *Clinical Trials* 2005;2:379–86. [PubMed: 16315646]
39. Little, R.; Rubin, DB. *Statistical analysis with missing data*. New York: Wiley; 1987.
40. Bryk, AS.; Raudenbush, SW. *Hierarchical linear models: applications and data analysis methods*. Newbury Park: Sage Publications; 1992.
41. Neter, J.; Kutner, M.; Wasserman, W. *Applied linear statistical models: regression, analysis of variance, and experimental designs*. 3. Homewood, IL: Irwin; 1990.
42. Berenson GS, Srinivasan SR, Cresanta JL, Foster TA, Webber LS. Dynamic changes of serum lipoproteins in children during adolescence and sexual maturation. *Am J Epidemiol* 1981;113:157–70. [PubMed: 7468573]
43. Kwiterovich PO, Barton BA, McMahon RP, Obarzanek E, Hunsberger S, Simons-Morton D, et al. Effects of diet and sexual maturation on low-density lipoprotein cholesterol during puberty. The Dietary Intervention Study in Children (DISC). *Circulation* 1997;96:2526–33. [PubMed: 9355889]
44. Haas JS, Lee LB, Kaplan CP, Sonneborn D, Phillips KA, Liang SY. The association of race, socioeconomic status, and health insurance status with the prevalence of overweight among children and adolescents. *Am J Public Health* 2003;93:2105–10. [PubMed: 14652342]
45. Rochon J, Klesges RC, Story M. Common design elements of the Girls health Enrichment Multi-site Study (GEMS). *Ethn Dis* 2003;13:S6–S14. [PubMed: 12713207]
46. Freedman DS, Perry G. Body composition and health status among children and adolescents. *Prev Med* 2000;31:S34–S53.

47. Daniels SR, Arnett DK, Eckel RH, Gidding SS, Hayman LL, Kumanyika S, et al. Overweight in children and adolescents. Pathophysiology, consequences, prevention, and treatment. *Circulation* 2005;111:1999–2012. [PubMed: 15837955]
48. Taylor RW, Jones IE, Williams SM, Goulding A. Body fat percentages measured by dual-energy X-ray absorptiometry corresponding to recently recommended body mass index cutoffs for overweight and obesity in children and adolescents aged 3-18 y. *Am J Clin Nutr* 2002;76:1416–21. [PubMed: 12450911]
49. Zimmermann MB, Gübeli C, Püntener C, Molinari L. Detection of overweight and obesity in a national sample of 6-12-y-old Swiss children: accuracy and validity of reference values for body mass index from the US Centers for Disease Control and Prevention and the International Obesity Task Force. *Am J Clin Nutr* 2004;79:838–43. [PubMed: 15113723]
50. Katzmarzyk PT, Srinivasan SR, Chen W, Malina RM, Bouchard C, Berenson GS. Body mass index, waist circumference, and clustering of cardiovascular disease risk factors in a biracial sample of children and adolescents. *Pediatrics* 2004;114:e198–205. [PubMed: 15286257]
51. Summerbell CD, Waters E, Edmunds LD, Brown KS, Campbell KJ. Interventions for preventing obesity in children. *The Cochrane Database of Systematic Reviews*. 2005;(3) 10.1002/14651858.CD001871.pub2Art. No.:CD001871.pub2
52. Luepker RV, Perry CL, McKinlay SM, Nader PR, Parcel GS, Stone EJ, et al. Outcomes of a field trial to improve children's dietary patterns and physical activity: the Child and Adolescent Trial for Cardiovascular Health (CATCH). *JAMA* 1999;275:768–76. [PubMed: 8598593]
53. Caballero B, Clay T, Davis SM, Ethelbah B, Rock BH, Lohman T, et al. Pathways: a school-based, randomized controlled trial for the prevention of obesity in American Indian schoolchildren. *Am J Clin Nutr* 2003;78:1030–8. [PubMed: 14594792]
54. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA* 1999;282:1561–7. [PubMed: 10546696]
55. Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, et al. Reducing obesity via a school-based interdisciplinary intervention among youth. *Arch Pediatr Adolesc Med* 1999;153:409–18. [PubMed: 10201726]
56. Fizgibbon ML, Stolley MR, Schiffer L, Van Horn L, KauferChristoffel K, Dyer A. Two-year follow-up results for Hip-Hop Health Jr.: a randomized controlled trial for overweight prevention in preschool minority children. *J Pediatr* 2005;146:618–25. [PubMed: 15870664]
57. Beech BM, Klesges RC, Kumanyika K, Murray DM, Klesges L, McClanahan B, et al. Child- and parent-targeted interventions: the Memphis GEMS pilot study. *Ethn Dis* 2003;13:S40–S53. [PubMed: 12713210]
58. Robinson TN, Killen JD, Kraemer HC, Wilson DM, Matheson DM, Haskell WL, et al. Dance and reducing television viewing to prevent weight gain in African-American girls: the Stanford GEMS pilot study. *Ethn Dis* 2003;S65–S77. [PubMed: 12713212]
59. National Institutes of Health. [homepage on the Internet] Ways to Enhance Children's Activity and Nutrition (We CAN!). Available from: <http://www.nhlbi.nih.gov/health/public/heart/obesity/wecan/>
60. Institute of Medicine. Preventing Childhood Obesity. In: Koplan, JP.; Liverman, CT.; Kraak, VI., editors. *Health in the Balance*. Washington, D.C: The National Academies Press; 2005.
61. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *Jama* 2004;291:1238–45. [PubMed: 15010446]

Table 1

Prevalence and Annual Incidence of Overweight, by Age and Race *

Age (years)	White			Black		
	n	% overweight	% new onset (n / n at risk)	n	% overweight	% new onset (n / n at risk)
9	612	7.4%	n/a [†]	533	17.4%	n/a [†]
10	1098	8.7%	3.1% (16/523)	1159	18.2%	2.7% (11/407)
11	1024	9.9%	2.5% (22/888)	1092	20.0%	4.8% (41/860)
12	1010	10.3%	2.1% (17/794)	1100	21.5%	4.2% (32/760)
13	958	9.9%	1.8% (13/718)	1074	20.9%	1.8% (12/677)
14	856	9.6%	2.0% (12/607)	967	22.3%	1.0% (6/572)
15	811	8.3%	0.4% (2/509)	931	22.6%	2.2% (11/489)
16	852	8.8%	1.1% (5/460)	940	21.1%	0.7% (3/423)
17	904	10.4%	1.4% (6/422)	934	23.7%	0.8% (3/369)
18	873	10.1%	0.8% (3/373)	896	23.3%	1.7% (5/302)
9-12 [‡]			5.8% (55/947)			9.4% (84/892)
13-18 [§]			5.7% (41/718)			5.9% (40/677)

* At study entry, approximately half of the NGHS girls were age 9 at study entry, and about half were 10 years old.

[†] For new onset, data for the year of study entry (age 9 or 10) are not shown, because some girls were already overweight (by one or more definitions) at study entry. No new onset data are given for age 9 because all 9-year-old data were collected during girls' first visit, and data from the first visit were not included in incidence calculations.

[‡] This is the incidence estimate for years 9-12 combined.

[§] This is the incidence estimate for years 13-18 combined

Table 2

Percent (number) of girls who were overweight in young adulthood (age 21-23) among those who were overweight and those who not overweight in childhood, by age.

Age (years)	% (number) overweight in young adulthood, among those girls who were... *		Odds ratio (95% CI) †
	...overweight at age	...not overweight at age	
9	71.3% (87/122)	10.4% (90/864)	14.5 (9.6-22.0)
10	69.0% (187/271)	12.9% (218/1692)	11.2 (8.6-14.6)
11	68.9% (195/283)	12.3% (193/1574)	11.5 (8.9-14.8)
12	67.4% (211/313)	11.0% (172/1561)	10.7 (8.4-13.7)
13	72.3% (209/289)	10.8% (164/1518)	15.5 (11.6-20.9)
14	72.3% (196/271)	10.5% (145/1386)	14.9 (11.2-19.7)
15	76.2% (202/265)	9.7% (131/1352)	19.3 (13.8-27.0)
16	78.0% (202/259)	8.8% (124/1410)	24.9 (18.8-32.9)
17	79.7% (235/295)	8.4% (119/1423)	27.9 (21.5-36.2)
18	78.0% (220/282)	6.9% (95/1369)	30.3 (20.5-44.8)

* The descriptive statistics were computed using only those girls who had non-missing BMI at both the age in childhood and in young adulthood; thus, the sample sizes were smaller than those shown in Table 1.

† The odds ratios and 95% confidence intervals were estimated following multiple imputation of missing data. Descriptive statistics (columns 2 and 3) were based on sample data prior to multiple imputation.

Table 3

Mean (SD) of percent body fat, sum of skinfolds, and waist circumference, by age and overweight (OW = overweight girls, ~OW = girls who were not overweight, D = difference calculated as mean for OW minus mean for ~OW).

Age (years)	Adiposity indicators									
	% body fat			Sum of skinfolds (mm)			Waist circumference (cm)			
	OW	~OW	D	OW	~OW	D	OW	~OW	D	D
9	33 (5)	25 (10)	8	69 (16)	28 (12)	41	*	*	*	*
10	38 (5)	29 (10)	9	73 (17)	30 (13)	43	79 (6)	61 (6)	18	18
11	44 (6)	33 (7)	11	76 (19)	32 (14)	44	83 (7)	64 (6)	19	19
12	48 (6)	37 (6)	12	80 (21)	34 (15)	46	86 (8)	66 (6)	20	20
13	52 (7)	40 (6)	12	87 (20)	38 (15)	50	88 (9)	67 (6)	21	21
14	54 (7)	41 (6)	13	95 (21)	41 (16)	55	91 (10)	68 (6)	23	23
15	56 (8)	42 (8)	14	94 (20)	43 (16)	51	93 (10)	69 (6)	24	24
16	56 (8)	43 (6)	14	92 (19)	42 (16)	51	94 (11)	70 (6)	25	25
17	57 (8)	43 (5)	15	93 (20)	42 (16)	51	96 (11)	71 (7)	25	25
18	58 (8)	43 (6)	15	99 (24)	42 (17)	56	98 (10)	71 (7)	26	26

* There were no waist measurements for 9-year-olds.

Table IV Percent of overweight (OW) and non-overweight (~OW) girls with unhealthy high or low levels of cardiovascular disease (CVD) risk factors

Age (years)	CVD risk factors											
	Systolic BP \geq 95 th percentile		Diastolic (K5) BP \geq 95 th percentile		Total cholesterol > 200 mg/dL		LDL > 130 mg/dL		HDL < 50 mg/dL		Triglycerides \geq 130 mg/dL	
	OW	~OW	OW	~OW	OW	~OW	OW	~OW	OW	~OW	OW	~OW
9	7%	1%	4%	2%	14%	16%	21%	17%	4%	4%	9%	5%
10	6%	2%	3%	1%	14%	13%	20%	15%	4%	4%	20%	7%
11	9%	2%	3%	1%	8%	10%	14%	8%	6%	2%	17%	9%
12	9%	2%	2%	1%	8%	9%	11%	8%	6%	3%	16%	9%
13	10%	2%	4%	1%	10%	6%	12%	6%	6%	1%	18%	8%
14	7%	2%	3%	1%	8%	6%	9%	5%	3%	1%	15%	5%
15	6%	1%	4%	1%	12%	8%	16%	7%	7%	1%	13%	6%
16	5%	1%	4%	2%	8%	9%	14%	8%	3%	1%	14%	6%
17	4%	1%	5%	2%	8%	9%	14%	8%	3%	1%	14%	6%
18	5%	1%	6%	1%	21%	10%	28%	9%	5%	1%	21%	8%
OR (CI) [†]	10.0 (5.0-20.0),		3.0 (1.3-7.3), p=0.01		1.2 (0.4-3.1), p=0.77		2.4 (0.9-6.0), p=0.07		6.3 (3.4-11.8),		3.3 (1.6-7.1), p=0.002	
OR (CI) [§]					1.5 (0.6-3.6), p=0.37		3.0 (1.3-7.0), p=0.01		5.4 (2.8-10.3), p<0.001		5.0 (2.3-10.8), p<0.001	

* Data not shown for lipids at age 17, due to small sample size (\leq 73).

[†] These odds ratios are for models controlling for maturation stage in addition to age. This was done for lipids, based on past work indicating that maturation is associated with lipid levels.

[‡] Odds ratio (95% confidence interval) in models adjusting for age but not maturation stage.

[§] Odds ratio (95% confidence interval) in models adjusting for maturation stage as well as age (lipids only, based on hypothesis).