

# Defining Health-Related Obesity in Prepubertal Children

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## Abstract

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**Objective:** The purpose of this study was to develop percentage of fat and waist circumference cut-points in prepubertal children with the intention of defining obesity associated with cardiovascular disease (CVD) risk.

**Research Methods and Procedures:** A cross-sectional analysis of 87 prepubertal children aged 4 to 11 years was used. Percentage of body fat was determined by DXA. Waist circumference was measured to the nearest millimeter. Receiver Operating Characteristic analyses of percentage of fat and waist circumference were used to develop cut-points for individuals with adverse levels of CVD risk factors.

**Results:** The risk factors selected for analyses (i.e., fasting insulin, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides, and total cholesterol/high-density lipoprotein cholesterol) were significantly related to percentage of body fat and waist circumference. Likelihood ratios were used to identify percentage of fat and waist circumference cut-points associated with adverse cardiovascular risk profiles. Two cut-points, an upper cut-point of 33% body fat and a lower cut-point of 20% body fat, were derived. Waist circumference cut-points indicative of adverse and normal risk-factor profiles were 71 cm and 61 cm, respectively.

**Discussion:** The data indicate that children with  $\geq 33\%$  body fat and children with a waist circumference  $\geq 71$  cm were more likely to possess an adverse CVD risk-factor profile than a normal risk-factor profile. The likelihood of

children with  $<20\%$  body fat or a waist circumference  $<61$  cm possessing an adverse CVD risk-factor profile as opposed to a normal risk-factor profile was small. The cut-points describe an adequate health-related definition of childhood obesity.

**Key words:** cardiovascular disease, percentage of body fat, waist circumference, lipids, insulin, cut-points

## Introduction

The recent increase in the prevalence of childhood obesity has been well-established (1,2). In boys and girls aged 6 to 11 years, the prevalence of obesity, defined as body mass index (BMI)  $\geq 95$ th percentile, has more than doubled from the earliest National Health and Nutrition Education Surveys, 1963 through 1970, to the most recent survey undertaken in 1995 (1). Numerous studies report that adverse levels of cardiovascular disease (CVD) risk factors such as total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, fasting insulin, and systolic blood pressure are associated with adiposity in children (3–8). The development of atherosclerotic plaques in young adults was first reported by Enos et al. (9) in 1953. Since then, data from the Bogalusa Heart Study and the Pathobiological Determinants of Atherosclerosis in Youth research group have shown a relationship between elevated CVD risk factors and the early advancement of atherosclerosis (10–12). Thus, the early identification of at-risk individuals is a necessary first step in the prevention of CVD (13,14). At present little data describing a physiological definition of childhood obesity exist.

BMI is widely accepted for use in defining overweight and obesity and is a valid measure of adiposity in adults (15). Current overweight and obesity standards for adults, based on BMI, are related to the comorbidities associated with obesity (15). However, BMI standards have limitations when applied to pediatric populations. First, BMI standards are population-based measures and are not directly related to any biological disturbances in children. Second, BMI is not well correlated with fat mass in children, especially at

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younger ages (16–18). Current definitions of obesity, based on age- and gender-specific BMI, may overpredict the level of fat mass and therefore overpredict the prevalence of obesity, particularly in younger children (19).

Williams et al. (20) and Dwyer and Blizzard (21) have proposed percentage of fat standards for defining health-related obesity in children. In both studies percentage of fat was estimated from skinfold measurements and was related to elevated levels of systolic blood pressure and blood lipids. Williams et al. (20) established cut-points of 30% fat in girls and 25% fat in boys for defining obesity in children. Dwyer and Blizzard (21) established similar cut-points of 30% and 20% fat in girls and boys, respectively. Both studies estimated percentage of fat from prediction equations using skinfold measurements. The accuracy of skinfold measurements in estimating percentage of fat is questionable (22). Estimates are affected by intra- and interobserver variability and potentially confounded by differences in body composition related to ethnicity and gender (23,24). In addition, differences in the distribution of subcutaneous adipose tissue are not accounted for when skinfolds are measured at only two to four sites, leading to potential over- or underestimation of percentage of fat (16).

Therefore, the purpose of this study was to further define health-related childhood obesity using DXA, a more robust estimate of percentage of fat in children. Waist circumference cut-points associated with adverse levels of CVD risk factors were also determined. Because waist circumference reflects central adiposity, as well as general adiposity, cut-points based on waist circumference may be uniquely related to disease risk. Waist circumference cut-points also may be useful to investigators who do not have access to DXA.

## Research Methods and Procedures

### Sample

Data used in this study were derived from an ongoing, longitudinal study of childhood and adolescent obesity in Birmingham, AL. Participants were healthy African American and white boys and girls recruited through advertisements, flyers, and word-of-mouth. Children were tested annually at both the General Clinical Research Center (GCRC) and at the Department of Nutrition Sciences, University of Alabama at Birmingham. They were admitted to the GCRC in the late afternoon for an overnight visit where data on anthropometrics, sociodemographics, dietary intake, and physical activity were collected. On the following morning after an overnight fast, resting metabolic rate was determined and blood samples were obtained. Fasting insulin was calculated as the mean of three determinations taken over a 15-minute period before administration of a glucose tolerance test. A pediatrician assessed pubertal development stage with the criteria of Marshall and Tanner (25,26). Two

weeks later, the children arrived at the Department of Nutrition Sciences in the fasted state where body composition was determined by DXA.

A subset of 87 children was selected for analysis based on pubertal development stage. Because insulin and lipid profiles are affected by pubertal development (27,28), only children at Tanner stage 1 were selected for the current analysis.

### DXA

Percentage of body fat was determined in the Department of Nutrition Sciences, University of Alabama at Birmingham by DXA. A DPX-L densitometer (Lunar Radiation Corp., Madison, WI), previously validated in the pediatric body-weight range, was used (29). Subjects were analyzed in light clothing while lying in the supine position. DXA scans were performed and analyzed using pediatric software (version 1.5e; Lunar Radiation Corp.). On the day of each test, the DPX-L was calibrated according to the manufacturer's guidelines. To present a more accurate measure of percentage of body fat, fat mass determined from DXA was adjusted according to a previously established regression equation [adjusted fat mass = (fat mass measured by DXA  $\times$  0.87) + 0.19 kg] (29). Adjusted fat mass was used in the analysis.

### Anthropometry

Waist circumference was measured at the narrowest part of the torso between the lowest rib margin and the iliac crest at the end of a gentle expiration. Measurements were taken to the nearest millimeter using a flexible tape. At least two measurements were made. If the two measurements were identical, then that value was used. If the two measurements differed, then a third measurement was taken to determine which value was correct. The same observer performed all anthropometric measurements.

### Laboratory Analyses

Blood samples were collected at the GCRC after an overnight fast. For determination of lipids, 5 mL of venous blood was obtained. Serum was separated by centrifugation and stored in cryotubes at  $-85^{\circ}\text{C}$ . Blood lipid levels were analyzed using Ektachem DT slides, reagents, and a Ektachem DT60 II Analyzer from Johnson and Johnson (Rochester, NY). The Ektachem analyzer was calibrated with manufacturer's reagents following National Cholesterol Education Program guidelines.

Insulin was assayed in duplicate 200- $\mu\text{L}$  aliquots with Coat-A-Count kits (Diagnostic Products, Los Angeles, CA). In our laboratory this assay has a sensitivity of 11.4 pM (1.9  $\mu\text{IU/mL}$ ), a mean intra-assay coefficient of variation of 5%, and a mean interassay coefficient of variation of 6%.

### CVD Risk Profile

The cut-point for serum fasting insulin concentration associated with risk was set at 72 pM (12  $\mu\text{IU/mL}$ ), based on

adult data (30). In addition HDL cholesterol less than the 10th percentile, and LDL cholesterol and triglycerides greater than the 90th percentile, were selected as cut-points for adverse levels. Percentiles were age- and sex-specific, based on prevalence data from the National Cholesterol Education Program, Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents (31). Because racial variations in lipid and lipoprotein levels were found to be small, the prevalence data were not race-specific (31). Cut-points at the 10th and 90th percentiles were selected because they are considered to be representative of adverse lipid and lipoprotein levels in children (32). Based on adult observations, a total cholesterol/HDL cholesterol ratio greater than 4.5 also was considered adverse and included in the analysis (33,34). Children with adverse levels of one or more of the risk factors were considered to possess an adverse risk-factor profile.

### Statistical Analyses

Pearson correlation coefficients were used to determine the relationship between percentage of fat, waist circumference, and CVD risk factors. Five of the CVD risk factors, fasting insulin, HDL cholesterol, LDL cholesterol, triglycerides, and total cholesterol/HDL cholesterol, were correlated significantly with percentage of fat and waist circumference. Variables were log transformed to correct for nonnormal distributions. Data were analyzed using SPSS software (version 8.0; SPSS Inc., Chicago, IL).

Receiver Operating Characteristic (ROC) analysis was used to develop percentage of fat and waist circumference cut-points associated with risk for one or more of the CVD risk factors. The sample distribution of percentage of fat was divided into demarcation ranges of 1% from a lower boundary of 17% fat to an upper boundary of >47.9% fat. Similarly, the sample distribution of waist circumference was divided into demarcation ranges of 2 cm, with a lower boundary of 47 cm and an upper boundary of >99 cm. The relative frequencies of children with and without at least one elevated risk factor for each percentage of fat and waist circumference demarcation range were determined. The sensitivity and specificity at each demarcation range were calculated. Cut-points were determined using likelihood ratios (35). The likelihood ratio is defined as the ratio of individuals with and without an adverse risk profile at a specific demarcation range (36). Positive likelihood ratios [(sensitivity/1 – specificity) ( $L_{pos}$ )], and negative likelihood ratios [(1 – sensitivity/specificity) ( $L_{neg}$ )], were calculated at each percentage of fat and waist circumference demarcation range. A cut-point was determined from the highest value of  $L_{pos}$  and/or the lowest value of  $L_{neg}$  (35).

A ROC curve of sensitivity vs. (1 – specificity) was plotted for the percentage of fat demarcation range and the waist circumference demarcation range. Points on the ROC curves are representative of the sensitivity and specificity of

each percentage of fat and waist circumference demarcation range. A diagonal line from the bottom left corner to the top right corner indicates a useless test; i.e., one in which identical values of sensitivity and (1 – specificity) are present at each demarcation range (37).

## Results

Descriptive data on the subjects are presented in Table 1. Fasting insulin, HDL cholesterol, LDL cholesterol, triglycerides, and total cholesterol/HDL cholesterol levels were significantly correlated with percentage of body fat and waist circumference (Table 2). Individuals with adverse levels of one or more of these variables were considered to possess an adverse CVD risk profile. A total of 34 children had one adverse risk factor, and 21 children had more than one adverse risk factor.

### Percentage of Fat

The sensitivity, specificity, and likelihood ratios for each of the demarcation ranges are shown in Table 3. The data did not support the identification of a single cut-point, because no single cut-point simultaneously produced the highest value of  $L_{pos}$  and lowest value of  $L_{neg}$ . Therefore, two percentage of fat cut-points associated with risk were identified (35); 33% body fat corresponded to the highest value of  $L_{pos}$  (Table 3). Children with  $\geq 33\%$  fat were  $\sim 15$  times more likely to have an adverse risk profile than a normal risk profile. Body fat of 19% to 19.9% corresponded

**Table 1.** Descriptive statistics ( $n = 87$ )

Variables	Mean $\pm$ SD	Range
Age (year)	8.2 $\pm$ 1.8	4.6–11.8
Height (cm)	131.5 $\pm$ 12.4	101.5–162.5
Weight (kg)	34.8 $\pm$ 14.4	14.0–85.0
Waist circumference (cm)	65.9 $\pm$ 13.2	43.9–102.5
Percent fat*	28.2 $\pm$ 9.6	11.3–51.0
Fasting insulin (pm)	78.6 $\pm$ 46.8	24.0–240.0
Total cholesterol (mm)	3.84 $\pm$ 0.73	2.17–6.47
HDL cholesterol (mm)	1.19 $\pm$ 0.36	0.54–2.3
LDL cholesterol (mm)	2.35 $\pm$ 0.72	1.11–5.24
Triglyceride (mm)	0.68 $\pm$ 0.49	0.20–3.69
Total cholesterol/HDL cholesterol	3.5 $\pm$ 1.3	1.6–8.3
Systolic blood pressure (mm Hg)	107.3 $\pm$ 8.8	81.0–133.0
Diastolic blood pressure (mm Hg)	58.0 $\pm$ 10.2	40.0–95.0

\* (Fat mass by DXA  $\times$  0.87) + 0.19 kg/weight (kg).

**Table 2.** Correlation coefficients between risk factor variables,\* adjusted percentage of fat,\*† percentage of fat,\* and waist circumference\*

Risk factor variables	Adjusted percentage of fat		Percentage of fat		Waist circumference (cm)	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
Fasting insulin	0.74	<0.001	0.67	<0.001	0.70	<0.001
Total cholesterol	0.20	NS	0.19	NS	0.21	NS
HDL cholesterol	-0.32	<0.01	-0.29	<0.01	-0.30	<0.01
LDL cholesterol	0.22	<0.05	0.22	<0.05	0.21	<0.05
Triglyceride	0.40	<0.01	0.33	<0.01	0.45	<0.01
Total cholesterol/HDL cholesterol	0.40	<0.001	0.36	<0.001	0.39	<0.001
Systolic blood pressure	0.18	NS	0.08	NS	0.17	NS
Diastolic blood pressure	-0.14	NS	-0.19	NS	-0.13	NS
Waist circumference (cm)	0.87	<0.001	0.79	<0.001		

\* Values log transformed.

† (fat mass by DXA  $\times$  0.87) + 0.19 kg/weight (kg).

NS, Not significant.

to the lowest value of  $L_{neg}$ . Children with <20% body fat were only 0.16 times more likely to have an adverse risk profile than a normal risk profile. The ROC curve for the percentage of fat data shown in Figure 1 represents a useful test.

### Waist Circumference

The sensitivity, specificity, and likelihood ratios for each waist circumference demarcation range are shown in Table 4. Again, the data did not support the identification of a single cut-point. A waist circumference of 71 cm corresponded to the highest value of  $L_{pos}$ . Children with a waist circumference of  $\geq 71$  cm were 14 times more likely to have an adverse risk profile than a normal risk-factor profile. The lowest value of  $L_{neg}$  was present at the 59- to 60.9-cm demarcation range, indicating that children with a waist circumference <61 cm were 0.35 times more likely to have an adverse risk profile than a normal risk-factor profile. The ROC curve for waist circumference represents a useful test (Figure 2).

## Discussion

To date, few studies have defined childhood obesity based on biological risk factors for disease. This study determined two percentage of body fat cut-points associated with CVD risk factors in children. Our analysis showed that children with  $\geq 33\%$  body fat were more likely to have an adverse risk-factor profile than a normal risk-factor profile. The likelihood of children with <20% body fat possessing

an adverse risk-factor profile as opposed to a normal risk-factor profile was found to be small.

Waist circumference was strongly correlated with percentage of fat and CVD risk factors in this sample (Table 2). Waist circumference previously has been shown to be highly predictive of CVD risk factors in children (38). Waist circumference provides a measure of central adipose tissue, independent of total body fat. Therefore, waist circumference cut-points associated with CVD risk factors are presented as an easily obtained anthropometric alternative to the DXA-derived percentage of fat cut-points. Children with a waist circumference  $\geq 71$  cm were more likely to have an adverse risk profile than a normal risk profile. Children with a waist circumference <61 cm were less likely to have an adverse risk profile than a normal risk profile.

Measures of obesity are related to health status in adults (39–42). BMI is commonly used to assess overweight and obesity in adulthood. Health-related cut-points of 25 kg/m<sup>2</sup> and 30 kg/m<sup>2</sup> define overweight and obesity, respectively (43).

Current BMI standards for defining obesity in children are limited. National Health and Nutrition Education Survey standards for defining childhood obesity are based on population distribution and are not related to biological risk factors for disease. Because prevalence data were obtained from a North American population, standards have limited use internationally. Health-related definitions of childhood obesity, established by Williams et al. (20) and Dwyer and Blizzard (21), used skinfold prediction equations to estimate percentage of body fat. Error may be present in skinfold-

**Table 3.** Specificity, sensitivity, and likelihood ratios for each percentage of body fat demarcation range

Percentage of body fat range	Specificity*	Sensitivity†	L <sub>pos</sub> ‡	L <sub>neg</sub> §
>47.9	1.0	0.02	∞	0.98
47-47.9	1.0	0.02	∞	0.98
46-46.9	1.0	0.04	∞	0.96
45-45.9	1.0	0.09	∞	0.91
44-44.9	1.0	0.11	∞	0.89
43-43.9	1.0	0.13	∞	0.87
42-42.9	1.0	0.15	∞	0.85
41-41.9	1.0	0.18	∞	0.82
40-40.9	1.0	0.24	∞	0.76
39-39.9	1.0	0.27	9.00	0.75
38-38.9	1.0	0.31	10.33	0.71
37-37.9	1.0	0.33	11.00	0.69
36-36.9	0.97	0.38	12.66	0.64
35-35.9	0.97	0.44	14.66	0.58
34-34.9	0.97	0.45	15.00	0.57
33-33.9	0.97	0.47	15.66	0.55
32-32.9	0.94	0.51	8.5	0.52
31-31.9	0.94	0.51	8.5	0.52
30-30.9	0.91	0.55	6.11	0.49
29-29.9	0.91	0.56	6.22	0.48
28-28.9	0.88	0.62	5.17	0.43
27-27.9	0.72	0.64	2.29	0.50
26-26.9	0.69	0.67	2.16	0.48
25-25.9	0.66	0.71	2.09	0.44
24-24.9	0.63	0.76	1.62	0.38
23-23.9	0.59	0.78	1.90	0.37
22-22.9	0.56	0.80	1.82	0.36
21-21.9	0.41	0.83	1.41	0.41
20-20.9	0.41	0.85	1.44	0.37
19-19.9	0.32	0.95	1.40	0.16
18-18.9	0.25	0.96	1.28	0.16
17-17.9	0.25	0.98	1.31	0
<17	0.19	1.0	1.23	0

\* 1 - Not at risk cumulative frequency/total individuals not at risk.

† Cumulative frequency of at-risk individuals/total individuals at risk.

‡ Sensitivity/1 - specificity.

§ 1 - sensitivity/specificity.

derived percentage of body fat measures due to intra- and interobserver variability, gender and ethnic differences, and differences in subcutaneous adipose tissue distribution

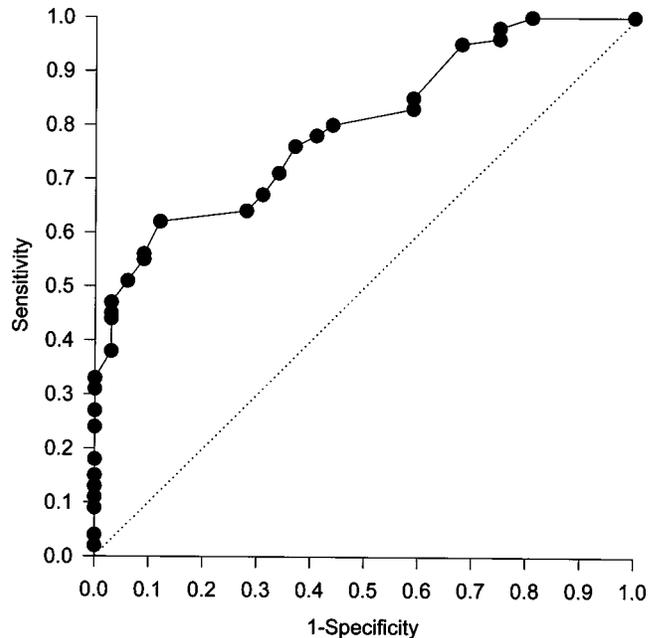


Figure 1. Percentage of fat ROC curve identifying individuals with adverse levels of one or more of the CVD risk factors in our analysis. The broken line represents a useless test.

(16,23,24). Recently Cole et al. (44) published new BMI standards for defining overweight and obesity in children. A large nationally representative sample was used. Age- and sex-specific BMI cut-points for defining overweight and obesity in children were derived from adult data by identifying percentiles in children analogous to those percentiles in adults that correspond to BMIs of 25 kg/m<sup>2</sup> and 30 kg/m<sup>2</sup>, respectively. Despite addressing two of the major limitations of previous childhood BMI standards (standards were derived from a large nationally representative sample and cut-points were predicted from adult cut-points), the new BMI standards remain unrelated to immediate biological risk factors for disease.

DXA offers a fast and noninvasive alternative to hydrostatic weighing in pediatric body-composition assessment. Several studies have cross-validated DXA body composition measures to direct carcass analysis in pig models (29,45-47). Pintauro et al. (29), using pigs in the pediatric weight range (25 ± 7.0 kg), demonstrated a small, yet significant, difference between carcass lean and fat mass and DXA measures. Specific correction factors were established to improve DXA measures of lean and fat mass. In this study, DXA percentage of fat was adjusted accordingly, thereby standardizing percentage of body fat to a known laboratory standard. Fields and Goran (48) further reinforced the use of this correction factor in a recent study comparing body composition determined by a four-compartment model with body composition determined by DXA in children. A significant difference between fat mass

**Table 4.** Specificity, sensitivity, and likelihood ratios for each waist circumference demarcation range

Waist circumference range	Specificity*	Sensitivity†	L <sub>pos</sub> ‡	L <sub>neg</sub> §
>99	1.0	0.04	∞	0.96
97–98.9	1.0	0.07	∞	0.93
95–96.9	1.0	0.09	∞	0.91
93–94.9	1.0	0.11	∞	0.89
91–92.9	1.0	0.11	∞	0.89
89–90.9	1.0	0.13	∞	0.87
87–88.9	1.0	0.16	∞	0.84
85–86.9	1.0	0.20	∞	0.80
83–84.9	1.0	0.20	∞	0.80
81–82.9	1.0	0.20	∞	0.80
79–80.9	1.0	0.27	∞	0.73
77–78.9	1.0	0.31	∞	0.69
75–76.9	0.97	0.33	11.00	0.69
73–74.9	0.97	0.36	12.00	0.65
71–72.9	0.97	0.42	14.00	0.60
69–70.9	0.94	0.45	7.5	0.59
67–68.9	0.91	0.53	5.88	0.52
65–66.9	0.88	0.56	4.66	0.50
63–64.9	0.84	0.60	4.13	0.39
61–62.9	0.78	0.65	2.95	0.45
59–60.9	0.72	0.75	2.68	0.35
57–58.9	0.28	0.84	1.17	0.57
55–56.9	0.19	0.91	1.12	0.47
53–54.9	0.09	0.91	1.0	1.00
51–52.9	0	0.95	0.95	∞
49–50.9	0	0.98	0.98	∞
47–48.9	0	0.98	0.98	∞
1<47	0	1.0	1.0	∞

\* 1 – Not at risk cumulative frequency/total individuals not at risk.

† Cumulative frequency of at-risk individuals/total individuals at risk.

‡ Sensitivity/1 – specificity.

§ 1 – Sensitivity/specificity.

determined by the four-compartment model and that determined by DXA was observed. However, after DXA fat mass was adjusted according to the correction factor of Pintauro et al. (29), there was no significant difference in fat mass measured by the two techniques.

Adult CVD risk factors were used as criteria for defining at-risk individuals in this study. Individuals with fasting insulin levels  $\geq 72$  pM (12  $\mu$ IU/ml) were considered at risk

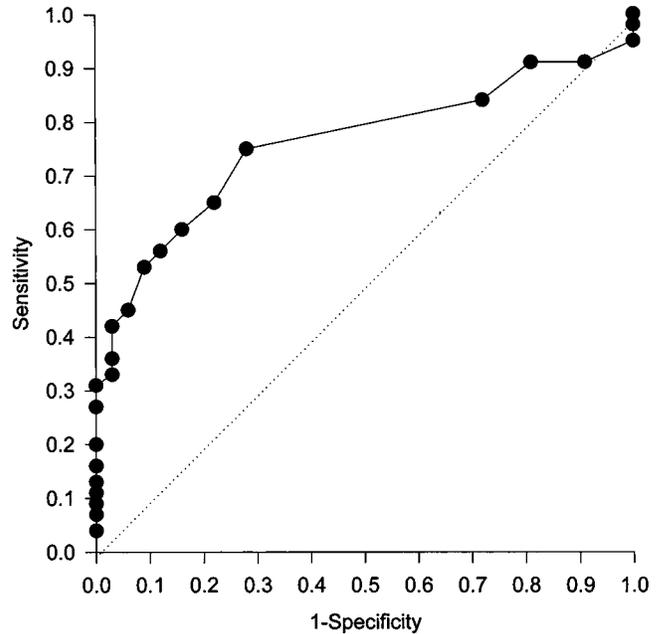


Figure 2. Waist circumference ROC curve identifying individuals with adverse levels of one or more of the CVD risk factors in our analysis. The broken line represents a useless test.

based on odds ratios for risk of ischemic heart disease in men. Triglyceride and LDL cholesterol levels greater than the age- and sex-specific 90th percentile, HDL cholesterol levels less than the age- and sex-specific 10th percentile, and a total cholesterol/HDL cholesterol ratio  $>4.5$  were also considered adverse levels of risk factors. This approach for risk assessment is likely to be valid because adverse levels of these risk factors are strongly associated with metabolic disease in adults (30,34,49–51). Furthermore, elevated CVD risk factors are related to early onset atherosclerosis and have been shown to track from childhood to adulthood (11–14,52,53).

Two previous studies established health-related definitions of childhood obesity using percentage of body fat predicted from skinfold equations. Williams et al. (20) established percentage of body fat cut-points of 25% and 30% in boys and girls, respectively. Dwyer and Blizzard (21) derived similar percentage of fat cut-points of 20% in boys and 30% in girls. Both sets of cut-points are comparable with the percentage of body fat cut-points derived using DXA in our study; an upper cut-point of 33% and a lower cut-point of 20%. Slight differences may be caused by the use of different methods of determining percentage of body fat. In addition, the different risk-factor criteria used for the assessment of CVD risk in this study vs. earlier studies may also contribute to inconsistencies. Individuals with CVD risk variables in the uppermost quintile for age-, sex-, and race-specific North American population distributions were arbitrarily considered at risk by Williams et al.

(20). Lipid and lipoprotein levels in the upper decile, and HDL cholesterol levels in the lower decile, in a sample of North American children, were considered adverse in this study. A robust percentile risk classification for plasma lipid concentration has not been established; however, the use of the 90th percentile to define risk has been suggested, because values above the 90th percentile display a high order of tracking through childhood (32).

A limitation of our study is the use of a pooled sample of boys and girls and African American and white children. However, preliminary analysis of the data used for this study showed no gender difference in the relationship between percentage of fat, waist circumference, and risk factors across the common range of percentage of fat and waist circumference values (data not shown). Furthermore, because all children in the study were prepubertal, we would not expect a strong gender influence on serum lipid, lipoprotein levels, and fasting insulin (27,54,55). Racial differences in fasting insulin levels are small before puberty (56). Racial differences in lipid and lipoprotein levels in children were also reported to be small by the Lipid Research Clinics of North America (31).

This study derived upper and lower cut-points, rather than a single cut-point, to define health-related obesity. The use of a single cut-point to define CVD risk may be impractical; a dichotomous relationship between percentage of body fat and CVD is unlikely. Rather, it is likely that CVD risk increases with an increased percentage of body fat. Therefore, a range of values may be more physiologically appropriate in identifying at-risk individuals than a single cut-point.

In conclusion, this study provides an alternative to the recent definitions of childhood obesity based on BMI percentiles that may be useful in pediatric health screenings and surveys. The cut-points established display good test sensitivity and specificity and are highly indicative of adverse and normal CVD risk-factor levels in children.

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### References

1. **Toriano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL.** Overweight prevalence and trends for children and adolescents. *Arch Pediatr Adol Med.* 1995;149:1085–91.

2. **Gortmaker SL, Dietz WH, Sobol AM, Wehler CA.** Increasing pediatric obesity in the United States. *Am J Dis Child.* 1987;141:535–40.
3. **Ronnemaa T, Knip M, Lautala P, et al.** Serum insulin and other cardiovascular risk indicators in children, adolescents and young adults. *Ann Med.* 1991;23:67–72.
4. **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.
5. **Aristimuno GG, Foster TA, Voors AW, Srinivasan SR, Berenson GS.** Influence of persistent obesity in children on cardiovascular risk factors: the Bogalusa Heart Study. *Circulation.* 1984;69:895–904.
6. **Wattigney WA, Harsha DW, Srinivasan SR, Webber LS, Berenson GS.** Increasing impact of obesity on serum lipids and lipoproteins in young adults: the Bogalusa Heart Study. *Arch Intern Med.* 1991;151:2017–22.
7. **Smoak CG, Burke GL, Webber LS, Harsha DW, Srinivasan SR, Berenson GS.** Relation of obesity to clustering of cardiovascular disease risk factors in children and young adults: the Bogalusa Heart Study. *Am J Epidemiol.* 1987;125:364–72.
8. **Laskarzewski P, Morrison JA, Mellies MJ, et al.** Relationships of measurements of body mass to plasma lipoproteins in school children and adults. *Am J Epidemiol.* 1980;111:395–406.
9. **Enos WF, Holmes RH, Beyer J.** Coronary disease among United States soldiers killed in action in Korea. *JAMA.* 1953;152:1090–3.
10. **Newman WP, Freedman DS, Voors AW, et al.** Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis: the Bogalusa Heart Study. *N Engl J Med.* 1986;314:138–44.
11. **Berenson GS, Wattigney WA, Tracy RE, et al.** Atherosclerosis of the aorta and coronary arteries and cardiovascular risk factors in persons aged 6 to 30 years and studied at necropsy: the Bogalusa Heart Study. *Am J Cardiol.* 1992;70:851–8.
12. **Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group.** Relationship of atherosclerosis in young men to serum lipoprotein cholesterol concentrations and smoking. *JAMA.* 1990;264:3018–24.
13. **Lenfant C, Savage PJ.** The early natural history of atherosclerosis and hypertension in the young: National Institutes of Health Perspectives. *Am J Med Sci.* 1995;310:S3–S7.
14. **Myers L, Coughlin SS, Webber LS, Srinivasan SR, Berenson GS.** Prediction of adult cardiovascular multifactorial risk status from childhood risk factor levels. *Am J Epidemiol.* 1995;142:918–24.
15. **Garrow JS, Webber J.** Quetelet's index ( $W/H^2$ ) as a measure of fatness. *Int J Obes Relat Metab Disord.* 1985;9:147–53.
16. **Willett W.** *Monographs of Epidemiology and Biostatistics.* New York: Oxford University Press; 1990, pp. 231–44.
17. **Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiu-mello G, Heymsfield SB.** Body mass index as a measure of overweight in children and adolescents. *J Pediatr.* 1998;132:204–10.

18. **Dietz WH, Robinson TN.** Use of the body mass index as a measure of overweight in children and adolescents. *J Pediatr.* 1998;132:191–3.
19. **Obarzanek E.** Methodological issues in estimating the prevalence of obesity in childhood. *Ann N Y Acad Sci.* 1993;699:278–9.
20. **Williams DP, Going SB, Lohman TG, et al.** Body fatness and risk for elevated blood pressure, total cholesterol and serum lipoprotein ratios in children and adolescents. *Am J Public Health.* 1992;82:358–63.
21. **Dwyer T, Blizzard CL.** Defining obesity in children by biological endpoint rather than population distribution. *Int J Obes Relat Metab Disord.* 1996;20:472–80.
22. **Dezenberg CV, Nagy TR, Gower BA, Johnson R, Goran MI.** Predicting body composition from anthropometry in pre-adolescent children. *Int J Obes Relat Metab Disord.* 1999;23:253–9.
23. **Goran MI.** Measurement issues related to studies of childhood obesity: assessment of body composition, body fat distribution, physical activity, and food intake. *Pediatrics.* 1998;104:505–18.
24. **Roche AF.** Methodological considerations in the assessment of childhood obesity. *Ann N Y Acad Sci.* 1993;699:6–16.
25. **Marshall WA, Tanner JM.** Variations in the pattern of pubertal changes in girls. *Arch Dis Child.* 1969;44:291–303.
26. **Marshall WA, Tanner JM.** Variations in the pattern of pubertal changes in boys. *Arch Dis Child.* 1970;45:13–23.
27. **Amiel SA, Caprio S, Sherwin RS, Plewe G, Haymond MW, Tamborlane WV.** Insulin resistance of puberty: a defect restricted to peripheral glucose metabolism. *J Clin Endocrinol Metab.* 1991;72:277–82.
28. **Frerichs RR, Webber LS, Srinivasan SR, Berenson GS.** Relation of serum lipids and lipoproteins to obesity and sexual maturity in white and black children. *Am J Epidemiol.* 1978;108:486–96.
29. **Pintauro S, Nagy TR, Duthie C, Moran MI.** Cross validation of fat and lean measurement by dual energy X-ray absorptiometry to pig carcass assessments in the pediatric body weight range. *Am J Clin Nutr.* 1996;63:293–8.
30. **Deprés JP, Lamarch B, Mauriege P, et al.** Hyperinsulinemia as an independent risk factor for ischemic heart disease. *N Engl J Med.* 1996;334:952–7.
31. **National Cholesterol Education Program.** Report of the expert panel on blood cholesterol levels in children and adolescents. *Pediatrics.* 1992;89:519–76.
32. **Berenson GS, Gail FC, Hunter SM, Srinivasan SR, Voors AW, Webber LS.** Cardiovascular risk factors in children: should they concern the pediatrician? *Am J Dis Child.* 1982;136:855–62.
33. **Luria M, Evel J, Sopochnikov D, Gotsman S.** Cardiovascular risk factor clustering and ratio of total cholesterol to high density lipoprotein cholesterol in angiographically documented coronary artery disease. *Am J Cardiol.* 1991;67:31–6.
34. **Manninen V, Tenkunen L, Koskinen P, et al.** Joint effects of serum triglyceride and LDL cholesterol and HDL cholesterol concentrations on coronary artery disease risk in Helsinki Heart Study. *Circulation.* 1992;85:37–45.
35. **Feinstein A.** *Clinical Epidemiology: The Architecture of Clinical Research.* Philadelphia: WB Saunders; 1987, pp. 601–13.
36. **Radack KL, Rouan G, Hedges J.** The likelihood ratio. *Arch Pathol Lab Med.* 1986;110:689–93.
37. **Zweig MH, Campbell G.** Receiver-operating characteristic (ROC) plots: a fundamental evaluation tool in clinical medicine. *Clin Chem.* 1993;39:561–77.
38. **Flodmark CE, Sveger T, Nilsson-Ehle P.** Waist measurement correlates to a potentially atherogenic lipoprotein profile in obese 12- to 14-year-old children. *Acta Paediatr.* 1994;83:941–5.
39. **Van Itallie TB.** Health implications of overweight and obesity in the United States. *Ann Intern Med.* 1985;103:983–8.
40. **Harrison GG.** Height-weight tables. *Ann Intern Med.* 1985;103:989–94.
41. **Manson JE, Willet WG, Stampfer MJ, et al.** Body weight and mortality among women. *N Engl J Med.* 1995;333:677–85.
42. **Kissebah AH, Freedman SE, Peiris AN.** Health risks of obesity. *Med Clin North Am.* 1989;73:111–39.
43. **World Health Organization.** *Obesity: preventing and managing the global epidemic, report of a WHO consultation.* Geneva, Switzerland: World Health Organization; June 3–5, 1997.
44. **Cole TJ, Bellizzi MC, Flegal KM, Dietz WH.** Establishing a standard definition of childhood obesity worldwide: international survey. *BMJ.* 2000;320:1–6.
45. **Brunton JA, Bayley HS, Atkinson SA.** Validation and application of dual energy x-ray absorptiometry to measure bone mass and body composition in small infants. *Am J Clin Nutr.* 1993;58:839–45.
46. **Ellis KJ, Shypailo RJ, Pratt JA, Pond WG.** Accuracy of dual energy x-ray absorptiometry for body composition measurements in children. *Am J Clin Nutr.* 1994;60:660–5.
47. **Svendson OL, Haarbo J, Hassager C, Christiansen C.** Accuracy of measurements of body composition by dual energy x-ray absorptiometry in vivo. *Am J Clin Nutr.* 1993;57:605–8.
48. **Fields DA, Goran MI.** Body composition techniques and the four-compartment model in children. *J Appl Physiol.* 2000;89:613–20.
49. **Phillips NR, Waters D, Havel RJ.** Plasma lipoproteins and progression of coronary artery disease evaluated by angiography and clinical events. *Circulation.* 1993;88:2762–70.
50. **Reaven G.** Role of insulin resistance in human disease. *Diabetes.* 1988;37:1595–1607.
51. **Castelli WP, Garrison RJ, Wilson PWF, Abbott RD, Kalousdian S, Kannel WB.** Incidence of coronary heart disease and lipoprotein cholesterol levels: the Framingham Study. *JAMA.* 1986;256:2835–8.
52. **Clarke WR, Schrott HG, Leaverton PE, Connor WE, Lauer RM.** Tracking of blood lipids and blood pressure in school age children: the Muscatine Study. *Circulation.* 1978;58:626–34.
53. **Webber LS, Srinivasan SR, Wattigney WA, Berenson GS.** Tracking of serum lipids and lipoproteins from childhood to adulthood. *Am J Epidemiol.* 1991;133:884–99.
54. **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.
55. **Tell GS, Mittelmark MB, Vellar OD.** Cholesterol, high density lipoprotein cholesterol and triglycerides during puberty: the Oslo Youth Study. *Am J Epidemiol.* 1985;122:750–61.
56. **Gower BA, Nagy TR, Goran MI.** Visceral fat, insulin sensitivity, and lipids in prepubertal children. *Diabetes.* 1999;48:1515–21.