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Body Composition and Health Status among Children and Adolescents[1]

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INTRODUCTION

In addition to dietary information, the assessment of nutritional status among youth includes various anthropometric dimensions that provide information on growth and body composition. These data are not always easy to interpret, but they are important to obtain because overweight youth are at increased risk for adverse health outcomes, including total mortality, in later life [*1–5*]. This review will focus on various measurements that can be used in field studies of children and adolescents. Although laboratory determinations can also be useful in assessing nutritional status, these measurements have been extensively reviewed [*6,7*] and will not be considered in this article.

Much of the presented data are from a series of nationally representative examinations that were conducted by the National Center for Health Statistics (NCHS) between 1960 and 1994 [*8*] (Table 1). Data collected from 5- to 17-year-olds who participated in the Bogalusa (Louisiana) Heart Study, a long-term study of the early natural history of cardiovascular disease risk factors [*9*], are also used extensively throughout this review. In that study, seven cross-sectional surveys were conducted between 1973 and 1994 [*10*], and many of the observed associations (e.g., levels of subscapular skinfold thickness by age) are summarized using locally weighted scatterplot smoothing (lowess) [*11*].

HEALTH EFFECTS OF OBESITY AND OVERWEIGHT

Although overweight and obesity are often used interchangeably, the former is simply an excess of weight for a given height, whereas obesity is a surplus of body fat. Overweight is a major health problem in the United States, increasing the risk for diabetes mellitus, ischemic heart disease (IHD), hypertension, and various cancers [*12–14*]. While it is frequently assumed that these increased risks are due to the excess body fat among overweight persons, rather than to increased fat-free mass, there are surprisingly little data to support this hypothesis. Indeed, almost all of the evidence concerning the relation of "obesity" to disease risk is based on simple combinations of weight and height [*12,13*], and there is some evidence suggesting that these simple indices may be better predictors of adverse health outcomes than is the triceps skinfold thickness [*15*], a standard surrogate measure of body fat. Furthermore, weight–height indices appear to be as strongly correlated with various metabolic abnormalities as are densitometric estimates of body fat [*16,17*].

Because many cohort studies have found a U- or J-shaped relation between weight-for-height indices and mortality [*14*], the idea that borderline-to-moderate overweight is detrimental has also been questioned [*18*]. It is likely, however, that much of the increased risk among persons who are underweight at an initial examination can be attributable to confounding by cigarette smoking, bias due to (previous) preclinical weight loss, or inappropriate statistical control of intervening variables (e.g., hypertension) [*19*]. The relatively low amount (or decease in) of fat-free mass among thin persons may also be detrimental [*12*], and weight–height indices cannot disentangle the effects of fat- and fat-free mass.

Overweight among children and adolescents is also associated with adverse levels of lipids, blood pressure, insulin, and other risk factors [*20*] and predicts adverse health outcomes in adulthood [*1–5*]. Furthermore, although there is conflicting evidence [*21*], some studies [*1*] have found that even after controlling for weight in adulthood, childhood overweight may influence subsequent disease risk. Although further study is needed [*22,23*], the intrauterine environment may also be an

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TABLE 1

Anthropometric Measurements Performed in Surveys Conducted by the National Center for Health Statistics

	Survey						
	HES I	HES II	HES III	NHANES I	NHANES II	Hispanic HANES	NHANES III
Years of survey	1960–1962	1963–1965	1966–1970	1971–1975	1976–1980	1982–1984	1988–1994
Age group	18–79 years	6–11 years	12–17 years	1–74 years	0.5–74 years	0.5–74 years	2 months
Skinfolds							
Triceps	+[a]	+	+	+	+	+	+
Subscapular	+	+	+	+	+	+	+
Medial calf			+			+	
Midaxillary (lateral chest)			+				
Suprailiac			+			+	+
Midthigh (anterior)							+
Circumferences							
Waist	+	+	+				+
Hip (buttocks)		+	+				+
Midthigh							+

Note. Modified from Ref. [*8*]. Abbreviations used: HES. Health Examination Survey; NHANES, National Health and Nutrition Examination Survey.

[a] + indicates that measurement was performed.

important factor in the development of various chronic diseases. Following an initial ecologic association between infant mortality rates and IHD, cohort studies have found low weights and head circumferences at birth to be predictive of IHD and diabetes mellitus [*24*]. It has been suggested that these phenotypes reflect a lack of nutrients or oxygen at critical developmental stages [*25*], but a measure of nutritional status would provide more persuasive evidence.

MEASUREMENT ISSUES IN ANTHROPOMETRY

Fat is the most variable component of body composition, and the collection and interpretation of anthropometric data have been discussed in several manuals [*6,26,27*]. Much attention has been given to the validity and repeatability of these data. Describing a measurement as valid implies that it is an unbiased estimate of the characteristic of interest (e.g., fat mass); in contrast, repeatability refers to the similarity between serial measurements. (Validity and accuracy are often used interchangeably in the literature, as are the terms repeatability, precision, reproducibility, stability, replicability, and reliability.) A short time interval between measurements is useful in assessing repeatability, whereas over longer time periods, physiologic variation would be expected to decrease the agreement between serial measurements. In general, repeatability can be maximized by using standardized techniques, providing extensive training, frequently calibrating instruments, and using mean levels of measurements performed in duplicate or triplicate [*6*]. Because the degree of repeatability can influence the associations between various body components and disease risk, information on the magnitude of the measurement errors should be included in all studies; a sample size of 50 persons would usually be adequate [*28*].

The assessment of repeatability focuses on test–retest differences, with measurement errors[3] typically calculated as $(\Sigma\Delta_i^2 / 2N)^{1/2}$, where $\Sigma\Delta_i^2$ is the sum of the squared differences over all N subjects; the coefficient of variation (CV) expresses this within-subject variability as a percentage of the overall mean. In contrast, the intraclass correlation coefficient (ICC) compares the within- and between-subject variability. This latter contrast, which is often called the "reliability coefficient" or "precision coefficient," may be important for biological characteristics that are highly regulated: if the between-subject variability is low (e.g., glucose), then the within-subject variability may also be low. The ICC can easily be obtained from an analysis of variance [*29*], and in contrast to the Pearson correlation, its magnitude is reduced by systematic test–retest differences.

The variability of the residuals from a regression of one test's value by the other's provides another measure of within-subject variability. This measure can be expressed as the variance of the residuals (which is the mean square error around the estimated regression line); the square root of this quantity (the standard deviation of the residuals) is the residual standard error. Because the magnitude of measurement errors is frequently associated with the level of obesity, a plot of

[3] The technical error of measurement is also known as the measurement error standard deviation or the unreliability standard deviation. It should be noted that this statistic is sensitive to outliers because differences are squared and that it may frequently be preferable to use the absolute values of the differences.

TABLE 2

Estimates of Repeatability of Anthropometric Dimensions in the 1992–1994 Examination Conducted by the Bogalusa Heart Study[a]

Dimension	Mean	Test–retest differences: Absolute difference	Test–retest differences: Measurement error standard deviation (technical error)	Test–retest differences: Technical error CV	Standard deviation of residuals[b]	Intraclass correlation coefficient
Weight (kg)	44.12[c]	0.14	0.14	0.3%	0.20	>0.99
Height (cm)	146.85	0.50	0.80	0.5%	1.13	>0.99
Rohrer index (kg/m^3)	13.39	0.15	0.21	1.5%	0.29	0.99
Waist circumference (cm)	66.04	1.56	1.93	2.9%	2.64	0.97
Triceps skinfold (mm)	15.29	1.18	1.30	8.5%	1.78	0.98
Subscapular skinfold (mm)	11.71	1.14	1.45	12.4%	2.02	0.98

[a] All estimates are based on 282 children who were examined twice (by the same observer) on the same day; because of the design of the screening examination, no information is available on interobserver errors.

[b] Based on the results of linear regression analyses in which the one value was used to predict the other.

[c] All values, with the exception of technical error coefficient of variation and intraclass correlation coefficient, are given in units specified by the anthropometric dimension.

test–retest differences vs the mean value may also be informative [30].

Estimates of intraobserver repeatability for several anthropometric dimensions in the 1992–1994 Bogalusa Heart Study are shown in Table 2. These values are based on a 10% sample (n = 282) of children who were reexamined by the same observer on each screening day. As expected, weight and height are the most repeatable measurements, but despite the relatively large differences in skinfold thickness measurements (CV = 8.5 to 12.4%), all intraclass correlation coefficients are ≥0.97. Despite the moderate within-subject measurement errors for the skinfold thicknesses, the range of observed values is much larger. Interobserver errors for the skinfold thicknesses would be substantially larger than the intraobserver errors seen in Table 2 (see Skinfold Thickness Measurements).

REFERENCE TECHNIQUES

Because the chemical analysis of fat is possible only for cadavers, several indirect methods have been developed to measure body composition; most of these are based on the assumption that the body consists of two (fat and fat-free mass) or four (fat, water, protein, and minerals) compartments. (Table 3 provides a brief summary of the strengths and limitations of these methods.) Although several fat-free body compartments can be accurately measured, the assumptions needed to convert these values into estimates of fat mass can lead to substantial errors. Furthermore, the values for several of the constants needed for these calculations, which have been obtained from a small number of cadavers, appear to be variable [6].

For example, although the water content of fat-free mass is assumed to be a constant (0.73 ml/g) in the estimation of total body water through dilution methods, one study [6] found this value to range from 67 to 77%, and it is likely that the variability in the general population is larger [6,31–33]. Furthermore, the density (1.10 g/cc) and potassium concentration (69.4 mmol/kg) of fat-free mass can also vary [6], and there are systematic differences in these presumed constants by race, sex, and obesity status [31,34]. This reliance on "constants" that, in fact, are variable may partly account for the differences in estimates of fat mass obtained in comparisons of various reference techniques [35].

There are additional difficulties in using these assumptions and constants to derive estimates of body composition among youth. The relative amounts of muscle and bone change substantially during growth, with the density of the fat-free mass increasing from approximately 1.08 to 1.10 g/cc between the ages of 7 and 20 years [31]. Although this range is relatively small, the use of 1.10 g/cc (the adult "constant") can result in an overestimate of 1.5 kg for the fat mass of children [31,36,37]. Comparable discrepancies can also result from the use of adult values for total body water or potassium concentration [31,32]. Estimates of body composition can be improved by the use of three- or four-compartment models in which one or more of the components (e.g., water or mineral) of fat-free mass are directly measured [34,38].

Several relatively new methods are available for the laboratory assessment of body composition. *In vivo* neutron activation analysis [39] allows the elemental composition of the whole body to be determined, and dual X-ray absorptiometry (DXA), which involves a very small radiation dose (0.05 to 1.5 mrem) (the radiation exposure from a transcontinental flight across the United States is ~5 mrem), can quantify both fat mass and fat distribution [40–43]. (With DXA, the fat content of soft tissue is estimated from the attenuations of two different energies, and it is assumed that the ratio of these

TABLE 3

Advantages and Disadvantages of Selected Methods for the Measurement of Body Composition

Method	Advantages	Disadvantages
Anthropometry	Inexpensive and rapid; little equipment needed	Varying assumptions needed to use as surrogates for fat mass
Weight, height	Very high reproducibility; very easy to measure; can be as highly correlated with metabolic/clinical complications as are fat estimates based on other techniques	No distinction between fat mass, fat-free mass, and edema
Skinfolds	A more direct measure of subcutaneous fat than is weight or circumferences; a contrast of thicknesses at different sites can provide information on the distribution of subcutaneous fat.	Interobserver error can be high, particularly among the obese; can be difficult to (re)locate sites; results depend upon examiner's skill; possibly low validity due to compressibility and other characteristics; no information on intraabdominal fat
Circumferences	Smaller errors than for skinfolds; measurements at multiple sites can be combined to provide information on fat patterning	Does not distinguish between fat and lean tissue; locations of sites differ
Bioelectrical impedance	Inexpensive and rapid; little interobserver error	Can be influenced by hydration and body build; requires fasting for a minimum of 2 h; sex- and age-specific prediction equations required to estimate fat mass from total body water
Reference methods	Very accurate measurement of some body compartments	Systematic differences across methods; assumptions used in calculating fat mass may lead to substantial errors, particularly among children
Densitometry		Subject cooperation necessary; not suitable for young children
Dilution methods		May require radiation exposure or blood sampling
Potassium counting		Expensive instrument; estimates may be influenced by body geometry; men and women may differ in potassium content of fat-free mass
Dual X-ray absorptiometry	Low interobserver error; can be used to assess fat patterning; little radiation required (<1.5 mrem)	Expensive instrument; estimates can be influenced by hydration, body thickness, and representativeness of the body area analyzed
Computerized tomography (CT) and nuclear magnetic resonance (MRI)	Both yield a direct observation of region of interest; can assess fat patterning; very accurate; MRI does not require irradiation	Very expensive instruments; CT requires radiation exposure; whole-body scans are very time consuming

two variables provides information on the proportion of fat and lean tissue.) Although estimates of body fat obtained from DXA are highly correlated with those based on densitometry, dilution techniques, and neutron activation, the actual estimates appear to be biased [*35,40,42*]; furthermore, the direction of these systematic differences can vary between men and women and between obese and lean persons [*34,35*]. Aloia et al. [*40*], for example, found that the DXA-estimated mean fat mass among 165 women was 1% (total body potassium estimate) to 14% (total body water) higher than were estimates made with other techniques. Although DXA measurements appear to be reproducible, with a technical error CV of 1 to 5% for percentage body fat [*43*], additional work is needed to assess its accuracy in the quantification of body fat among children and adolescents. The lack of an acceptable gold standard limits the assessment of the validity of field methods that can be used to estimate body fat.

SKINFOLD THICKNESS MEASUREMENTS

The measurement of skinfold thicknesses has been extensively discussed [*26,27*]. While it is often assumed that skinfold thicknesses provide a direct estimate of body fat, in fact they reflect only the amount of subcutaneous fat at a particular site [*44,45*]. Furthermore, estimates of the proportion of fat that is subcutaneous vary from 20 to 70% [*33,44,46*] and increase markedly with the degree of obesity [*47*]. Comparisons across persons can also be strongly influenced by fat patterning, and it is possible that the amount of intra-abdominal fat, which cannot be estimated through skinfold thickness measurements, may be the depot that is most important in determining the metabolic complications of obesity [*48*]; see Body Fat Patterning. Although skinfold thicknesses are strongly related to reference estimates of body density and body fat, with correlation coefficients reaching 0.85 [*44*], fairly similar associations have frequently been observed with various weight–height indices [*49*].

Sites

Various statistical criteria have been used to select the "best" skinfold or set of skinfolds. Investigators have focused on maximizing representativeness, correlations with reference estimates of fat, associations with clinical outcomes, sensitivity, or specificity; sites have also been chosen to minimize interexaminer errors [*50*]. Because these criteria may differ by sex, age, race/ethnicity, degree of obesity, outcome, and body fat pattern [*51*], it is not surprising that numerous combinations of sites have been proposed. Additional difficulties in selection of a subset of skinfolds include the strong intercorrelations among skinfolds [*52*], whether to use various transformations (e.g., logarithmic) or robust regression techniques for skewed distributions, and the use of quadratic terms or natural splines in regression models.

For adults, sites that have been recommended if only one skinfold is to be measured have included the subscapular or midaxillary, triceps, pectoral, thigh, or suprailiac [*44,45,50,51*]. (As lipid levels may be more strongly correlated with the thickness of subscapular, rather than triceps, skinfold [*51*], a classification of obesity based solely on the latter may not be optimal.) Furthermore, the measurement of 13 different skinfolds among middle-aged men in the Paris Prospective Study has suggested that the abdominal skinfold may be most predictive of IHD [*53*]. For studies that involve the measurement of two (or more) sites, the triceps is almost always included, along with the calf, thigh, or a truncal skinfold [*31,32,44,54,55*].

For children and adolescents, suggested single sites have included the triceps [*46*], subscapular [*50*], and midaxillary (girls) [*46*]; a combination of the triceps, subscapular, suprailiac, and abdominal skinfolds has been suggested [*56*]. Although not typically measured, a lower-body skinfold, such as the thigh, may be useful; disrobing is not required if this is measured 1 in. above the knee [*57*] (one of the most important determinants of site selection is the degree of modesty required). Given the numerous recommendations, it may be reasonable to select skinfolds based on their ability to describe body fat patterning. One possibility would be to select at least one site from various body regions, such as the trunk (suprailiac, subscapular, chest, abdominal), lower body (calf, thigh), and arm (triceps, forearm) regions. If desired, the sum (or mean) of these skinfolds could be used as a global estimate of adiposity.

Limitations

Although measurement issues have frequently focused on repeatability, the validity of skinfold thicknesses can be strongly influenced by the compressibility and fat content of a skinfold, characteristics that can vary by age, sex, site, and level of obesity [*45*]. Several formulas have been used to estimate percentage body fat from skinfold thickness measurements, but many of these equations are not generalizable [*45,49,55*], particularly among children and adolescents [*58*], and should probably be avoided. Care should also be taken when using skinfolds to assess weight change: the abdominal and iliac skinfolds show larger changes with weight loss than does the triceps skinfold [*59,60*]. In addition, it is uncertain whether skinfold thickness measurements can accurately quantify the large differences in body fat seen among children and adolescents: at the 85th skinfold percentile (sum of triceps and subscapular skinfolds), reported body fat percentages have ranged from 15 to 34% among boys and from 22 to 38% among girls [*61*].

As assessed by the test–retest differences, measurement error standard deviations generally range from 0.8 mm for the triceps skinfold (which is 5 to 10% of its median thickness among children and adolescents) to 2 mm for the thigh skinfold [*27*]. (Errors for the thigh skinfold may be due to a lack of site standardization by inexperienced measurers.) Although skinfold thickness measurements can be repeatable ($r > 0.95$) if measured by the same examiner [*28*] (as seen in Table 2), interobserver errors can be large due to difficulties in site location [*62*] and the tendency for caliper readings to decrease over time [*45,63*]. The degree of obesity also influences the magnitude of the skinfold measurement errors [*64*], with the reliability decreasing as the amount of subcutaneous fat increases. As some skinfolds are truly immeasurable [*65*], the potential effects of nonrandom missing data should be considered in data analyses.

In addition to thorough training in standardized techniques, several steps can be taken to minimize measurement error. If there are multiple examiners in a study, it may be best to (1) rotate them among subjects to reduce potential biases arising from systematic differences in measurement or (2) have each site measured by a single observer. (A single observer per site during the time frame of a longitudinal study could also substantially reduce within-subject measurement errors [*6*].) Errors could also likely be minimized if limits for test–retest differences are established before the study begins and repeatability data are regularly reviewed during data collection. Finally, because the distribution of forces applied to skinfolds may vary by type of caliper, only one of the available calipers (Harpendon, Lange, and Holtain) should be used within a study. Based on a small amount of data from adults, Lange calipers may give systematically higher skinfold readings (1 to 10%) than do either Harpendon [*66*] or Holtain calipers [*67*]. Some data (from female college athletes) also suggest that Lange calipers may yield higher interobserver measurement errors [*68*].

Reference Data

Although comparisons of skinfolds across studies should be interpreted cautiously [*69*], several nationally representative surveys (Table 1) provide reference data. These reports differ in the time periods covered, age groupings, use of race-specific data, and use of statistical smoothing techniques. Sex- and age-specific percentile curves for subscapular and triceps skinfolds (based on data collected from 1963 to 1975) have been published [*70*], and separate tabulations are available for the First and Second National Health and Nutrition Examination Surveys (NHANES I [*71,72*] and NHANES II [*73*], respectively). Race-specific data, based on the combined NHANES I and NHANES II samples, have also been published [*26*]. Data on skinfold thicknesses of children and adolescents in other countries have also been summarized [*6*].

Selected percentiles of triceps and subscapular skinfolds based on NCHS data are shown in Tables 4 (whites) and 5 (blacks and Mexican Americans); data for whites and blacks are from NHANES I and II [*26*], while data for Mexican Americans are from the Hispanic Health and Nutrition Examination Survey (HHANES) [*74*].[4] These percentiles differ substantially by age and sex, and there are racial/ethnic differences: Mexican American youth tend to have thicker triceps and subscapular skinfolds than do either whites or blacks, and white children generally have thicker triceps skinfolds than do blacks.

Body Fat during Growth and Development

An initial rise in adiposity after infancy is followed by a steady decline, which is more pronounced among boys than among girls, lasting into midchildhood. During adolescence, estimated body fat increases slightly among girls (to about 28%), but decreases (from ~22 to 13%) among boys [*49*]. Figure 1 compares average skinfold thicknesses for white and black children who participated in the Bogalusa Heart Study between 1973 and 1994. (Unless otherwise noted, data from the Bogalusa Heart Study are based on 5- to 17-years-olds who participated in any of seven cross-sectional surveys conducted between 1973 and 1994 [*10*].) Although the triceps skinfold is thicker among whites than among blacks, the difference in the thickness of the subscapular skinfold is much smaller. In addition, the triceps skinfold thickness decreases among boys after puberty, but continues to increase among girls; this sex difference in the triceps (and biceps) skinfold thickness has been consistently observed in other cross-sectional [*49*] and longitudinal [*75,76*] studies. Because race, sex, and

[4] NHANES I was conducted between 1971 and 1975, NHANES II between 1976 and 1980, and HHANES between 1982 and 1984. Because of the smaller sample sizes among blacks and Mexican Americans, the 95th percentile is not presented.

TABLE 4

Selected Percentiles of the Thickness of the Triceps and Subscapular Skinfolds, by Age and Sex for White Children

	Triceps skinfold percentiles						Subscapular skinfold percentiles					
	50		85		95		50		85		95	
Age (years)	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
5	8[a]	10	12	14	14	16	5	6	6	8	8	12
6	8	10	12	14	16	17	4	6	7	9	13	12
7	9	11	13	15	18	19	5	6	7	10	12	13
8	9	12	13	17	18	22	5	6	8	12	12	21
9	10	13	16	20	21	26	6	7	10	14	15	24
10	10	13	18	20	24	27	6	7	11	16	20	24
11	12	13	20	22	30	29	6	8	15	16	27	28
12	11	14	20	20	28	27	7	9	14	16	24	29
13	10	15	18	24	26	30	7	10	14	19	26	26
14	9	17	16	24	24	31	7	10	13	21	23	30
15	8	17	15	23	22	32	7	10	12	20	22	27
16	8	18	16	26	24	32	8	12	14	22	24	32
17	8	19	14	26	19	35	8	12	14	24	20	34

Note. Values are based on the combined NHANES I (1971–1974) and NHANES II (1976–1980) samples [Ref. *26*].

[a] Sample sizes among boys range from 231 (age 6) to 535 (age 5), among girls from 218 (age 6) to 504 (age 5). Values (in mm) represent sex- and age-specific percentile.

TABLE 5

Selected Percentiles of Thickness of the Triceps and Subscapular Skinfolds, by Sex and Age for Black and Hispanic Children

Age (years)	Triceps skinfold percentiles				Subscapular skinfold percentiles			
	50		85		50		85	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
5	7 (8)[a]	9 (10)	10 (13)	13 (14)	4 (4)	5 (6)	6 (7)	8 (10)
6	7 (8)	8 (10)	10 (12)	12 (16)	4 (4)	5 (6)	6 (7)	7 (10)
7	6 (9)	9 (11)	9 (15)	13 (18)	4 (5)	6 (6)	6 (9)	8 (13)
8	7 (10)	9 (12)	10 (17)	15 (19)	5 (5)	5 (7)	8 (11)	12 (16)
9	7 (10)	10 (14)	10 (20)	17 (21)	5 (6)	6 (10)	7 (14)	10 (20)
10	8 (12)	10 (15)	13 (22)	20 (25)	5 (7)	6 (10)	8 (21)	16 (23)
11	8 (14)	12 (14)	15 (22)	22 (22)	6 (9)	8 (10)	10 (20)	15 (18)
12	8 (11)	12 (16)	17 (21)	25 (24)	6 (8)	9 (12)	16 (18)	26 (20)
13	6 (10)	16 (17)	12 (20)	24 (26)	6 (8)	12 (13)	8 (14)	20 (22)
14	7 (10)	14 (20)	10 (21)	24 (25)	6 (8)	10 (14)	8 (16)	20 (24)
15	7 (9)	14 (19)	12 (16)	23 (26)	8 (8)	10 (16)	14 (14)	20 (26)
16	7 (10)	18 (18)	11 (17)	26 (28)	8 (10)	14 (14)	13 (19)	27 (28)
17	7 (9)	14 (18)	10 (16)	24 (26)	8 (10)	12 (16)	12 (17)	24 (28)

Note. Values for black children are based on the combined NHANES I (1971–1974) and NHANES II (1976–1980) samples [Ref. *26*]. Data for Mexican Americans are from HHANES [Ref. *74*].

[a] Among blacks, sample sizes range from 49 (8-year-old boys) to 163 (5-year-old girls). Among Mexican Americans from 69 (15-year-old boys) to 125 (9-year-old girls). Values (in mm) represent sex- and age-specific percentile for blacks; comparable percentile for Mexican-Americans is shown in parentheses.

age differences in the skinfold thicknesses can vary across skinfolds, it may be important to obtain measurements at several sites to obtain an estimate of overall adiposity among different subgroups. For example, whereas the mean thickness of the triceps skinfold among 17-year-old girls is almost twice that of boys, the subscapular skinfold thickness differs by ~30%. (Additional information on sex, age, and race differences in skinfold thicknesses is included under Body Fat Patterning).

CIRCUMFERENCES

Body circumferences, which typically show a moderate to high correlation ($r = 0.5$ to 0.8 [*65*]) with skinfold thicknesses, can be used to estimate obesity and describe fat patterning [*27,52*]. Furthermore, the simplicity of circumference measurements might be preferred to skinfolds in field studies. The high correlations between self-reported and technician-measured waist circumferences among adults ($r > 0.90$ [*77*]) also suggest that valid information may be obtained through questionnaire data. Although interobserver errors for circumferences are smaller than those for skinfolds (particularly among the obese), the locations of specific sites can be problematic. The "waist" circumference, for example, has been measured at the narrowest part of the torso or (less correctly) at the level of the umbilicus; furthermore, hip circumferences are more frequently measured near the levels of the buttocks than at the hips themselves [*27*].

Various circumferences were measured among children in the Second Health Examination Survey (HES II), which was conducted in 1963 to 1965 [*78*]. Between the ages of 6 and 11 years, the median waist circumference increases from about 53 to 62 cm among boys and from 52 to 60 cm among girls. At comparable ages, boys were found to have slightly larger waist circumferences, while girls had larger hip circumferences [*78*]. A comparable sex difference in waist circumference was also seen among ~3,000 school-aged children who were examined in 1992–1994 by the Bogalusa Heart Study [*79*] (Fig. 2). Although white boys generally have larger waist circumferences than other race–sex groups, additional differences emerge during puberty: the average waist circumference is lowest among white girls and intermediate among blacks. The waist, buttocks, and midthigh circumferences have been measured in NHANES III (see Table 1).

BIOELECTRICAL IMPEDANCE

Of the newer methods available for assessing body composition in field studies [*33,39*], bioelectrical impedance has received much attention. When an alternating current is passed into the body, it is partitioned according to the resistivity and volume of each tissue; because fat is a poor conductor, most of the current flows through water and its dissolved electrolytes in muscle tissue. Under various assumptions (see below), it is possible to estimate total body water and, subsequently, fat-free and fat mass from the observed resistance [*80*]. Although bioelectrical impedance has not

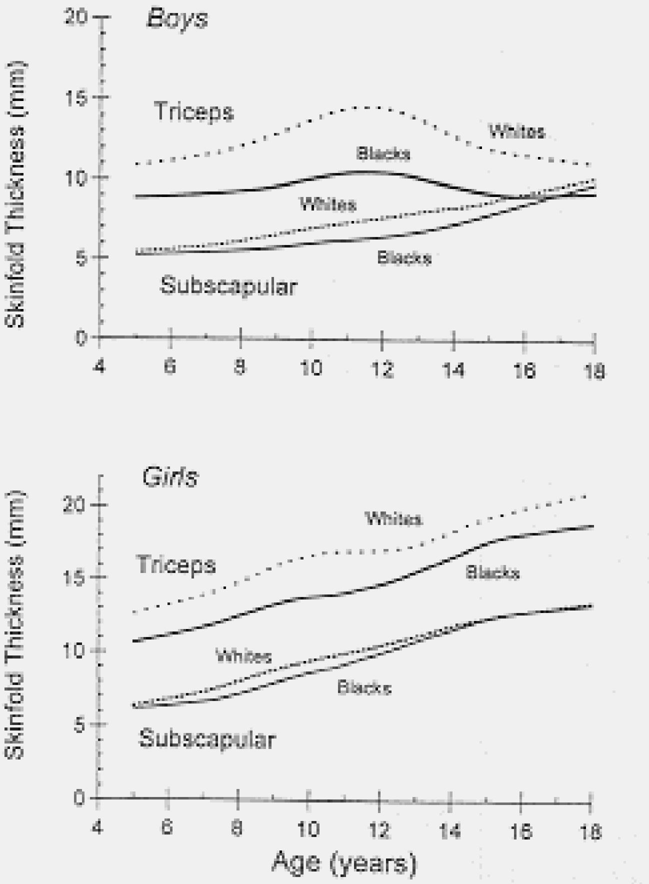

FIG. 1. Cross-sectional levels of triceps and subscapular skinfold thickness by race, sex, and age. Data are from all youths who participated in any cross-sectional examination of the Bogalusa Heart Study from 1973 to 1994 (sample is comparable to that in Ref. [10]). Data include ~24,000 triceps measurements and ~16,000 subscapular measurements; approximately equal numbers of boys and girls were examined; overall, about 40% were black. Data were smoothed using lowess curves.

consistently been found to provide more accurate estimates of adiposity than has anthropometry [81,82], this method may prove to be very useful because of its low interobserver error, moderate cost, and simplicity. Bioelectrical impedance has been used in the Framingham Heart Study and in NHANES III [82] and was the focus of an NIH Technology Assessment Conference [83]. Although the within-day technical error CV is quite low (1 to 2% [84]), bioelectrical impedance (in contrast to skinfolds and circumferences) does not provide information on fat patterning

Despite the interest in this technique, there are many unanswered questions. Whereas the trunk makes a very large contribution (>50%) to estimates of fat-free mass and body weight, whole-body impedance measurements appear to be primarily determined by the composition of the limbs near the electrodes [80]. Furthermore, because of the assumptions used in estimating fat-free mass, the accuracy of bioelectrical impedance is influenced by characteristics related to hydration (or electrical conductivity); these factors include age, sex, degree of obesity, body fat distribution, body posture, strenuous exercise, ingestion of food or drink, and timing of the menstrual cycle [85]. Thus, using appropriate sex- and age-specific equations and standardized positioning of the patient is important [37,83]. Furthermore, it is possible that equations developed among whites may underestimate the total body water of blacks [86]; additional study is needed to quantify the accuracy of this technique among various racial/ethnic groups [87].

WEIGHT AND HEIGHT

The measurement of weight and height, two of the most accurate and repeatable biologic characteristics,

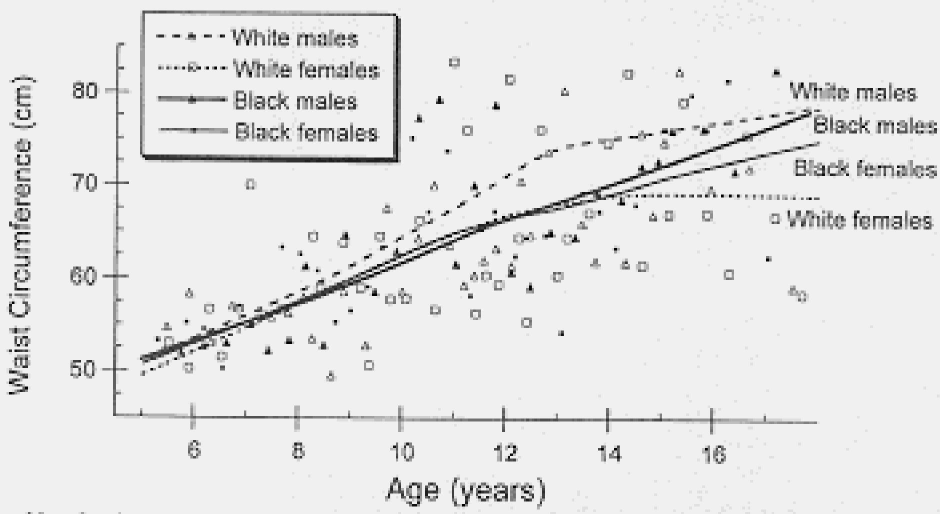

FIG. 2. Cross-sectional levels of waist circumference by race, sex, and age. Data are from 3,116 youths who participated in the 1992–1994 cross-sectional examination of the Bogalusa Heart Study [Ref. 79]. Five percent of the values is shown to illustrate the variability inherent in the data. Lines were smoothed using lowess curves.

has been extensively discussed [6,27]. Heights are generally measured with a stadiometer or portable anthropometer at maximum inspiration; minimal clothing should be worn so that posture can be observed. Weight should be measured with the bladder empty using a calibrated beam balance, but a less accurate spring balance is often used in the field. Seasonal variability due to clothing ranges up to 0.3 kg [88].

Several combinations of weight and height function fairly well as surrogate measures of obesity, accounting for about 50% of the variability in body fat [50,88–90]. Although weight–height indices may predict adiposity less accurately among children than among adults [91], this may, in part, be due to incorrect assumptions concerning the body composition of children (see Reference Techniques). It is reasonable to think that an estimate of body fatness or frame size [51] would improve the prediction of obesity-related complications beyond that achieved with weight–height indices, but there is surprisingly little evidence to support this possibility. However, it has been noted [92] that a 5-kg increase in the fat content of an adult would not necessarily be detected by skinfold-thickness measurements (due primarily to large interobserver errors), but would easily be detected by a weight determination. The widespread use of weight–height indices will almost certainly continue as these measurements are very repeatable, require relatively little technical skill to obtain, and can be compared with a large amount of reference data.

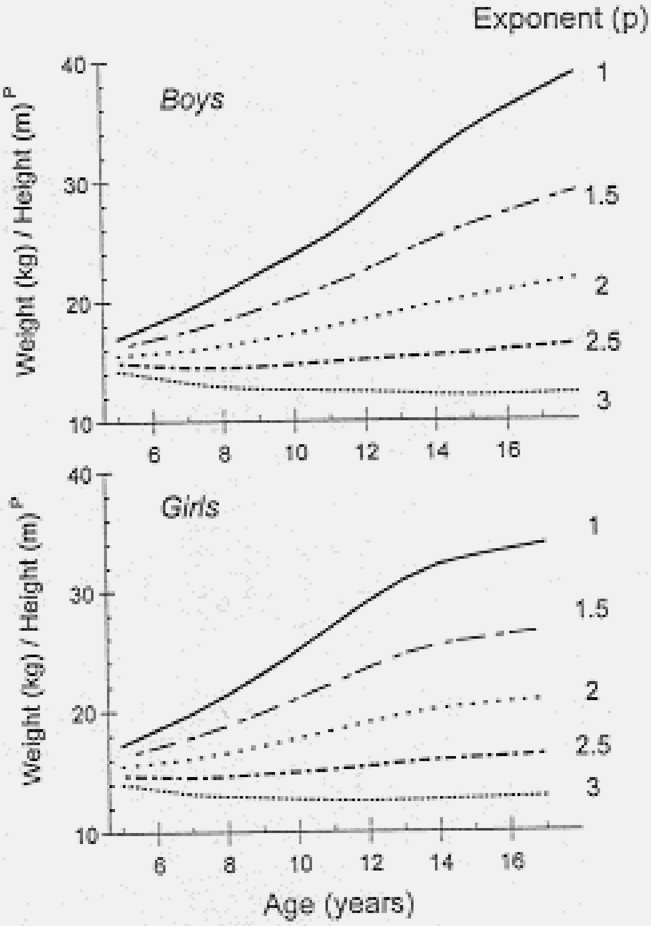

FIG. 3. Estimated levels of power indices (weight/height^p) for different exponents among boys (top) and girls (bottom). Unpublished data are from the Bogalusa Heart Study includes that all white youths (~15,000) who participated in any cross-sectional examination from 1973 to 1994 (sample is comparable to that in Ref. [10]); similar results were observed among black children (data not shown). Data were smoothed using lowess curves.

Power Indices

Various power indices, in which weight (W) is divided by height (H) raised to a power (p), are typically used to measure overweight among youth. Often the exponent is chosen to maximize the correlation with body fat and to minimize the correlation with height.[5] Because the magnitudes of these two correlations can vary by age, sex, weight, height, and other characteristics, it is not surprising that several indices have been proposed: W/H^2 (Quetelet index), W/H^3 (Rohrer index), $H/W^{1/3}$ (Ponderal index), $W/H^{1.5}$ (for women), $W^{1.2}/H^{3.3}$, and $W/H^{2.4}$ (for 5-to 12-year-olds) [93]. Although W/H^2 has sometimes been termed "body mass index," it should more properly [94] be named after Quetelet, the 19th-century investigator who noted that the weight of adults was proportional to height^2. Power indices are strongly intercorrelated among adults ($r > 0.9$), which in part reflects the greater variability in weight than in height, but it is generally accepted that the optimal exponent is near 2 among men and between 1 and 2 among women [93,95].[6] Although it has been suggested that W/H^3 may be preferred because the weight of a three-dimensional object (which is roughly equivalent to its volume) is proportional to the third power of height, this reasoning requires the (false) assumption that body dimensions remain constant with increasing height [95].

Although originally developed among adults, power indices are now also widely used as surrogates for obesity among children and adolescents. However, in contrast to the low correlations between Quetelet index and height among adults, the index is associated with height and age among youth [93]. These relations are evident in the Bogalusa Heart Study (Fig. 3), in which

[5] The general equivalence of relative weight, defined as $W - b{\cdot}H$ (where b represents the regression coefficient of weight on height), and power indices can be seen following a logarithmic transformation of W/H^p yielding $\log(W) - (p) \cdot \log(H)$. The simplest relative weight index divides a subject's weight by a reference weight, which can be obtained from the estimated regression equation $[\beta_0 + p \cdot \log(H)]$.

[6] Although several nomograms that facilitate the field calculation of Quetelet index have been published, many do not have appropriate values for school-age children or, as noted by Kahn [96], have been printed incorrectly. A useful nomogram for children, with both metric and English units, is given in Guo et al. [97].

median levels of Quetelet index increased by 30% between the ages of 5 and 17 years, yielding correlations of 0.5 to 0.6 with both age and height. Regression analyses predicting log(weight) from log(height) indicate that power indices with exponents of 2.8 (boys) and 2.9 (girls) would show virtually no correlation with height (or age) among the entire sample. Age-specific regression models, however, indicate that the optimal p increases from 2.5 (5-year-olds) to 3.5 (9-to 11-year-olds) and then decreases to 2.0 (17-year-olds) (data not shown).

Although the positive association between Quetelet index and age (height) among youth does not necessarily argue against the use of this index (obesity and height are correlated before puberty [*98*]), differences in age or height, which could confound comparisons across groups, need to be carefully assessed in analyses. A possible solution in multiple regression models that include Quetelet index as a predictor would be to also control age (or height) using quadratic terms, splines, or indicator variables; a single linear term would likely be inadequate. Another possibility would be to include both weight and height as separate predictor variables; the coefficient for weight would then reflect the relation of weight to the outcome (e.g., physical activity) while holding height constant. These latter models would be more flexible, allowing for the inclusion of nonlinear effects of either weight and height, as well as their interaction. Another advantage of considering weight and height as separate variables would be improved interpretation. Among 12-year-old boys, for example, a Quetelet index of 24.1 kg/m^2 (~90th percentile) could represent a weight of 58 kg (90th percentile) and a height of 1.55 m (70th percentile) or a weight of 42 kg (50th percentile) and a height of 1.32 m (<5th percentile). The health consequences of "overweight" among these two children could differ substantially.

Another approach would be to relate the Quetelet index of each child to a sex- and age-specific reference; this has been termed "relative Quetelet index by age" [*93*] or "BMI-for-age" [*99*]. For example, a Quetelet index of 21.0 kg/m^2 for a 12-year-old boy would represent a relative Quetelet index of 117% based on the median value in NHANES I (18.0 kg/m^2). If the Quetelet was 21.4 kg/m^2 after a 1-year follow-up, the relative Quetelet index would be 113%, as the median for 13-year-old boys is 18.9 kg/m^3. Thus, although the absolute Quetelet index increased slightly (+0.4 kg/m^2), the child became slightly thinner (117 to 113%) compared with his peers. Although the use of different reference standards could complicate comparisons across studies, this relative Quetelet index could be useful in longitudinal studies.

Limitations

It is obvious that weight–height indices cannot distinguish excess weight due to adiposity from that attributable to muscularity, skeletal tissue, or edema [*100*], and therefore, a relatively high Quetelet index may indicate either obesity or an athlete with little body fat. It is also possible that the relation of weight–height indices to adiposity varies somewhat by sex, race/ethnicity, and age [*14*], and these contrasting associations contribute to the differing relation of overweight to mortality across various subgroups [*101*]. At the same Quetelet index, for example, percentage body fat increases with age and is higher among women than among men [*90*]. It is also known that white and black boys have fairly comparable levels of Quetelet index, but that skinfolds are thicker among whites (Fig. 1)[*26*]; differences in bone density may counterbalance these adipose tissue differences. In general, comparisons of Quetelet indices across subgroups should be carefully interpreted as differences in body composition may be obscured.

The interpretation of power indices is further complicated by changes in body composition and proportions that occur during growth, and the ability of Quetelet index to identify obese adolescents has been investigated [*102,103*]. Although it has been reported [*102*] that the 85th percentile of Quetelet index had moderate sensitivity (~0.70) and high specificity (~0.95) for identifying children and adolescents above the 85th percentile for percentage body fat, other investigators [*103*] have reported that Quetelet index has a much lower sensitivity (~0.25) for identifying persons above the 90th percentile. These differences may reflect (1) the techniques used to estimate body fat (DXA vs densitometry) [*35*], (2) the cutpoints chosen to define high levels of Quetelet index and body fat, or (3) different analytical adjustments for age.

Although self-reported data on weight and height can allow a larger sample to be studied at lower costs, adults typically underreport weights by 2–3% and overreport heights by ~1% [*104*]; biases among adolescents are likely to be at least as large [*105*]. These differences could result in an underestimation of Quetelet index and among persons who are short and heavy. It should also be noted any power index in which height is raised to a power >1 will magnify reporting and measurement errors in height.

Reference Data

Reference data for Quetelet index are available from several national surveys conducted in the United States [*26,72,73,106*] and in other countries [*98,107*]. These published data differ in the (1) time periods, (2) smoothing techniques used (if any), (3) racial/ethnic and age groupings, and (4) presented percentiles. Percentiles of weight for height are available for children based on several cycles (1963–1975) of the NCHS surveys, as well as for Canadian children and adolescents [*6*]. Because the relation of weight to height changes during growth (Fig. 3), it is inappropriate to apply weight-for-height percentiles from adults, such as those in the Metropolitan Life Insurance tables, to children and adolescents.

In addition to age and sex differences, levels of Quetelet index also vary by racial/ethnic group [*13*], and data from the combined NHANES [*26*] and HHANES [*74,108*] are shown in Table 6. After age 12, levels of Quetelet index among girls tend to be higher among Mexican Americans and blacks than among whites, with the magnitude of the differences increasing at the upper range of the distribution. For example, about 10 to 15% of both Mexican American and black girls are above the 95th percentile for white girls, a cutpoint that is frequently used to define "very overweight." Similar differences can also be seen in levels of Rohrer index (W/H^3) among white and black girls who participated in the Bogalusa Heart Study (Fig. 4). Despite only a small difference in average levels (Fig. 4, top), the prevalence of a Rohrer index >90th percentile among 17-year-olds was about twofold higher among black girls than among white girls (14% vs 7%). Among boys, Mexican Americans tend to be heaviest, while blacks tend to be the thinnest (Table 6).

Although the low interobserver measurement errors that are characteristic of Quetelet index facilitate comparisons with reference standards, there are several points that should be considered in its use. As with all ratio measures, differences in Quetelet index could be due to differences in the numerator (weight), denominator (height), or both; the high prevalence of overweight among Navajo adolescents, for example, is partly attributable to the large proportion of short youth [*109*]. The time frame of the study is also important to consider. Because the prevalence of overweight among youth in the United States has increased over the past few decades (the prevalence of severe overweight approximately doubled between 1963 and 1991 [*10,110*]), the estimated prevalence of overweight in any study will

TABLE 6

Selected Percentiles of Quetelet Index among Children and Adolescents

	50th percentile			85th percentile			95th percentile		
Age (years)	Whites	Mexican Americans	Blacks	Whites	Mexican Americans	Blacks	Whites	Mexican Americans	Blacks
				Boys					
5	15.5	15.6	15.6	16.8	17.4	16.7	18.1	18.9	17.6
6	15.3	15.7	15.3	17.1	17.5	17.4	19.1	20.2	20.1
7	15.7	15.9	15.3	17.7	18.8	17.0	19.2	21.5	19.6
8	16.0	16.2	15.7	18.1	19.6	18.0	20.1	23.6	19.3
9	16.4	16.8	16.0	19.4	22.2	18.0	21.8	23.8	20.6
10	17.1	17.4	16.8	20.0	23.1	19.3	23.3	*	22.7
11	17.5	19.1	16.9	21.7	23.5	19.7	26.0	27.0	24.3
12	18.1	18.4	17.4	22.1	22.4	21.7	25.9	27.0	25.1
13	19.0	19.6	17.7	23.4	22.5	21.4	26.7	*	23.7
14	19.6	20.2	18.9	23.6	25.4	21.8	27.0	*	23.8
15	20.4	20.7	20.9	23.0	23.8	23.9	26.6	*	26.4
16	21.3	21.4	21.2	25.0	25.7	24.5	27.6	*	27.3
17	21.2	21.0	20.7	24.9	25.6	24.0	27.6	*	27.4
				Girls					
5	15.3	15.3	15.2	16.9	17.7	16.9	18.6	20.1	18.8
6	15.3	15.4	14.8	17.2	18.0	16.5	18.7	21.1	18.5
7	15.4	15.7	15.5	17.6	19.0	17.0	19.7	*	18.7
8	15.9	16.6	15.5	18.8	20.1	17.8	22.0	21.7	21.1
9	16.7	17.8	15.7	19.9	21.8	18.9	23.9	23.9	23.0
10	16.8	17.9	16.9	20.5	22.4	22.2	23.7	*	25.2
11	18.1	18.0	18.3	21.7	22.7	22.2	25.8	25.5	28.0
12	18.7	19.9	19.2	22.7	24.3	26.6	26.1	26.8	29.9
13	19.2	20.1	19.9	23.7	24.6	24.2	28.7	*	28.6
14	20.4	21.6	20.2	24.3	24.9	25.4	28.5	*	29.9
15	20.1	21.4	20.7	23.4	26.5	25.3	26.9	*	35.0
16	20.9	21.2	22.4	25.6	27.0	26.8	29.1	*	35.7
17	21.4	21.5	21.2	25.8	26.9	25.9	30.7	*	34.0

Note. Data for Mexican Americans are form HHANES [Ref. *74*]. Values for whites and blacks are based on the combined NHANES I (1971–1974) and NHANES II (1976–1980) samples [ref. *26*]. Sample sizes ranged from 54 (8-year-old black girls) to 533 (5-year-old white boys). *95th percentile was not calculated if race-, sex-, and age-specific sample size was <100 [*74*].

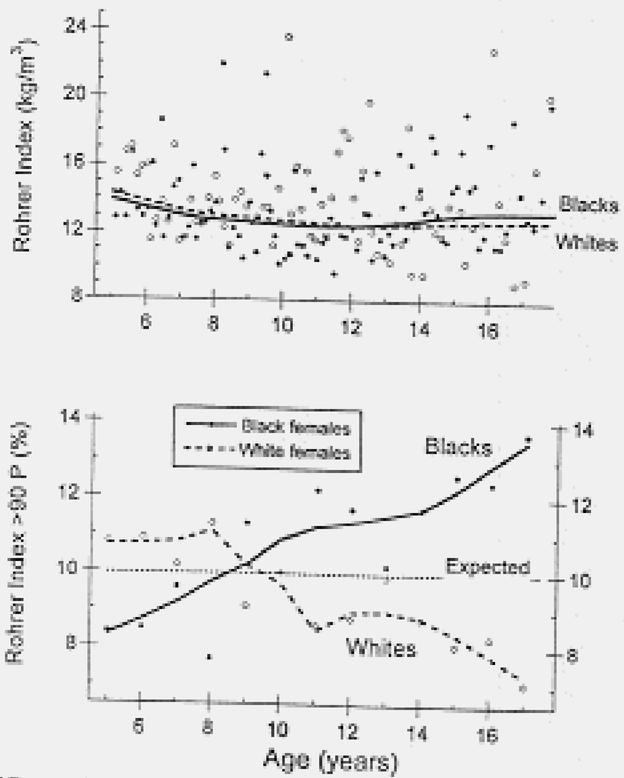

FIG. 4. Cross-sectional levels of Rohrer index (weight/height³) (top) and prevalence of overweight (bottom), by race and age among girls. Unpublished data are from girls who participated in any cross-sectional examination of the Bogalusa Heart Study from 1973 to 1994, and lines represent smoothed (lowess) associations. To indicate the variability in the data, about 1% (of ~7,300 values) for whites and 2% (of ~4,600 values) for blacks are shown in the top. Points in the bottom represent the proportions of black and white girls at each age whose Rohrer index was above the 90th percentile (combined whites and blacks) for girls of that age.

be influenced by the time period during which the reference data were collected. For example, based on data from the Bogalusa Heart Study [*10*], a 14-year-old boy with a Quetelet index of 26.8 kg/m² would be at the 95th percentile, if 1973–1974 data were used as the reference, but between the 80th and 85th percentiles using 1992–1994 data. Unless the purpose of the study is to examine secular trends, it may be best to use concurrent referent data. It should be noted that despite the growing concern with anorexia nervosa, 1988–1991 data from NHANES III do not indicate an increase in the proportion of very thin (weight–height index <5th percentile) children [*111*].

Overweight and Desirable Weight

Although it would be ideal to base cutpoints for overweight and desirable weight on estimates of disease risk, even among adults there are numerous difficulties with this approach. Several cohort studies, for example, have found that after smokers and persons with preclinical disease are excluded, there is no obvious cutpoint at which the shape of the positive relation of Quetelet index to disease risk changes [*12,112*]. Furthermore, associations with disease can be influenced by age, race, sex, concurrent risk factors (e.g., total cholesterol), length of follow-up, and distribution of diseases in the sample; differences in these covariates could lead to differences in cutpoints across studies.

It is therefore not surprising that numerous definitions of overweight among adults have been suggested, with cutpoints generally ranging from 25 to 30 kg/m² [*13,92,97,113–115*]. (A value of 25 kg/m² is approximately the 50th percentile (men) or 60th percentile (women) among adults in NHANES II, while 30 kg/m² is about the 85th percentile [*73*].) Another approach has been based on levels of Quetelet index among 20- to 29-year-olds in NHANES II [*115*]: the 85th percentiles (overweight) are 27.3 kg/m² (women) and 27.8 kg/m² (men), while the 95th percentiles (severe overweight) are 32.3 kg/m² (women) and 31.1 kg/m² (men). Desirable weights have also been defined as encompassing a Quetelet index of 20–25 kg/m², the range in which the minimum morbidity in cohort studies is frequently seen [*116*]. Several other ranges of desirable weights have been recommended [*12*], and it is possible that these cutpoints should increase with age to reflect the changing associations with mortality [*117*].

The limited amount of longitudinal data greatly complicates the classification of overweight and desirable weight among youth [*5*]. Although several studies have found that levels of Quetelet index among children and adolescents are predictive of subsequent morbidity and mortality [*1–4*], a relatively small number of endpoints have been examined (often in highly selected populations), with one of the larger studies consisting of 447 deaths among 13,146 persons after a 50-year follow-up [*2*]. In that sample, mortality showed a linear trend with prepubertal (but not postpubertal) weights among boys, while among girls, only those in the upper relative weight guintile appeared to be at increased risk. Other investigators have also reported differences between boys and girls in the relation of childhood overweight to adult disease [*1*], as well as a nonlinear relation to IHD mortality [*4*]. Given the relatively small numbers, it is difficult to tell whether these sex and developmental-stage differences are real or due to sampling variability. The possible inverse association between Quetelet index in early adulthood and subsequent osteoporosis [*118*], and the smaller amount of data relating overweight among girls to subsequent disease [*5*], also complicates the classification of desirable weights and overweight among youth.

Because of the long time periods needed to relate childhood overweight to adult disease, cross-sectional associations with lipid and blood pressure levels have also been used to define desirable weight among youth. Based on association with risk factor levels, for example, cutpoints for body fat percentages of 20 to 30% have been suggested [*61,119*]. (Published equations were used in both studies to estimate percentage body fat from age and skinfold thicknesses.) However, basing a cutpoint on cross-sectional associations is less desirable than analyzing the longitudinal relation to adult mortality, and it is possible that associations may differ across risk factors. For example, the relation of Quetelet index to levels of LDL cholesterol progressively increases between the ages of 5 and 17 years, while the magnitude of its association with systolic blood pressure decreases with age [*120*].

Summary statistics can also obscure the large amount of variability in the data: the 95% confidence intervals around several of the proposed cutpoints for body fat range from <15% to >35% [*119*]. This variability is also apparent in Fig. 5, which shows the relation of systolic blood pressure to Quetelet index and skinfolds (subscapular and triceps) among 648 8-year-old white girls examined in the Bogalusa Heart Study. (Of the strata defined by race, sex, and age, 8-year-old white girls showed the strongest associations between a specific risk factor (systolic blood pressure) and the overweight/obesity indices.) Despite the fairly strong associations (r = 0.5 to 0.6), there are no obvious inflection points at which levels of systolic blood pressure begin to increase. Furthermore, the predicted increase in systolic blood pressure for a given increase in obesity tends to become *smaller* (P <0.001 for nonlinear trend) at higher levels of both skinfolds. Additional consideration should possibly be given to logarithmic transformations in which a specified percentage increase in skinfold thickness is related to the outcome.

Because of the difficulties in using biological endpoints to define overweight/obesity, recent recommendations [*54*] have used sex-and age-specific 85th and 95th percentiles[7] of Quetelet index from NHANES I (Table 7). (A simple approximation to the 95th percentile of Quetelet index among 10- to 17-year-olds can be obtained by adding 13 (for boys) or 14 (for girls) to years of age [*54*].) According to these recommendations, children above the 95th percentile would be considered overweight and referred for further assessment; those between the 85th and the 95th percentiles would be considered "at risk for overweight" and examined for various IHD risk factors. It should be noted, however,

TABLE 7

Recommended Cutpoints for Overweight (Quetelet Index) among Children and Adolescents

	Boys percentile		Girls percentile	
Age (years)	85th	95th	85th	95th
6	16.6	18.0	16.2	17.5
7	17.4	19.2	17.2	18.9
8	18.1	20.3	18.2	20.4
9	18.8	21.5	19.2	21.8
10	19.6	22.6	20.2	23.2
11	20.4	23.7	21.2	24.6
12	21.1	24.9	22.2	26.0
13	21.9	25.9	23.1	27.1
14	22.8	26.9	23.9	28.0
15	23.6	27.8	24.3	28.5
16	24.4	28.5	24.7	29.1
17	25.3	29.3	25.2	29.7

Note. Recommendations are based on smoothed race- and sex-specific percentiles from NHANES I [Ref. *72*].

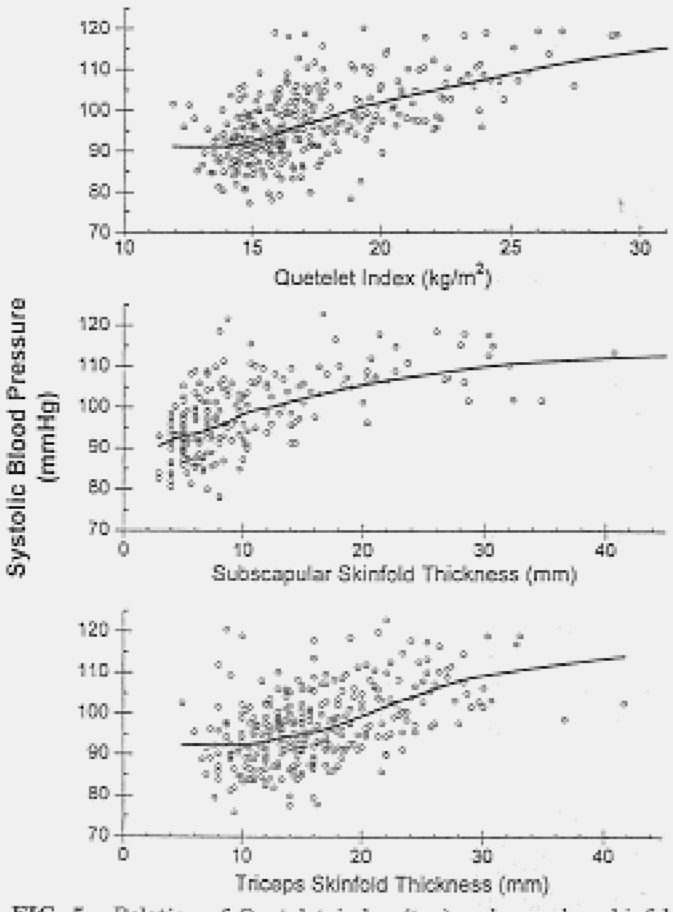

FIG. 5. Relation of Quetelet index (top), subscapular skinfold thickness (middle), and triceps skinfold thickness (bottom) to levels of systolic blood pressure among 8-year-old white girls. Unpublished data are from 8-year-old girls who participated in The Bogalusa Heart Study between 1973 and 1994; sample sizes ranged from 429 (subscapular skinfold) to 648 (Quetelet index); 50% of the data is shown. As assessed by multiple linear regression, associations with subscapular and triceps skinfolds were not linear (P < 0.001). Data were smoothed using lowess curves.

[7] Because of the secular trends in overweight that have been observed [*110*], about 10% of children and adolescents will have a Quetelet index that is greater than the 95th percentile from NHANES I. Although published percentiles of Quetelet index are available for adolescents from more recent NCHS surveys, the Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services concluded that the percentiles published by Must et al. [*72*] from NHANES I were preferable because they incorporated sampling weights and statistical smoothing and included both the 85th and the 95th percentiles.

that the relatively small sex- and age-specific sample sizes (~125) in NHANES I, along with the skewness of Quetelet index and its association with age, make it difficult to accurately estimate upper percentiles. Based on bootstrap resampling [*121*], the standard deviation of the sex- and age- specific 95th percentile is approximately 1 kg/m^2.

TRACKING OF OVERWEIGHT AND OBESITY

Tracking is usually defined as the persistence of the ranking for a characteristic over time, and there is little doubt that childhood levels of obesity and overweight are predictive of subsequent levels. However, a wide range (r= 0 to 0.84) of correlation coefficients has been reported between serial measurements during follow-up periods of <5 to 45 years [*5,122,123*]. Estimates of the proportion of overweight children who remain overweight as adults are also quite variable (26 to 77%), as are the increased risks (1.5 to 7) for overweight in later life [*124*]. In general, the magnitude of these estimates is (1) positively associated with the age at which the initial measurements are made, (2) stronger for weight–height indices than for skinfolds, and (3) inversely associated with the length of follow-up.

A recent analysis from the Fels Longitudinal Study [*125*] illustrates many of these trends. Although Quetelet index among 35-year-olds could be predicted fairly well from levels at age 18 (r ~ 0.60–0.75), correlations with childhood levels (age 7) were 0.3 (boys) and 0.5 (girls). Furthermore, the probability of overweight in adulthood increased with the age at which the initial measurement was performed. For example, a 17-year-old girl with a Quetelet index at the 85th percentile had a 40 to 80% chance of being overweight at age 35, but a severely overweight (95th percentile) 8-year-old girl had less than a 30% chance of being overweight at follow-up. Although these results emphasize the difficulties involved in weight management programs for children under 9 years of age, other results suggest an increased persistence of overweight among young children if one (or both) parents are overweight [*126*].

Other characteristics may influence the magnitude of tracking of overweight and obesity, and it is possible that there are a few critical periods [*127*]. For example, several investigators have found that the age at which the postinfancy nadir in Quetelet index is reached (typically between the ages of 5 and 7 years [*107, 125*]) is inversely related to the risk for overweight in adulthood; this phenomenon has been termed "adiposity rebound." Insulin resistance may also play an important role; among overweight adults, weight gain is inversely associated with fasting levels of insulin [*128*], and it is possible that overweight children with relatively low insulin levels may be at the greatest risk of remaining overweight. It would also be of interest to know if the development and persistence of obesity among girls is related to age of menarche.

BODY FAT PATTERNING[8]

Systematic methods to categorize human body shape were first introduced in the 1930s, and the association of body fat distribution with various chronic diseases was recognized by the 1950s [*129*]. Cohort studies have since shown that fat patterning, typically measured by various ratios (or differences) of skinfolds and circumferences, is predictive of diabetes mellitus, IHD, stroke, and total mortality [*53,130,131*]. In addition, a relative excess of adipose tissue in the abdominal, upper body, or truncal regions is associated with adverse levels of lipoproteins, blood pressure, glucose, insulin, and other metabolic risk factors [*48,132,133*]. Although these associations appear to be independent of the level of overweight and obesity, it should be emphasized that the correlation (r ~ 0.2 to 0.5) [*53,132*] between Quetelet index and various indices of fat distribution needs to be considered in data analyses and interpretation of the results. Among children and adolescents, the relative proportion of truncal fat also appears to increase with Quetelet index [*134*].

Although indices of fat patterning based on either skinfolds or circumferences show fairly similar associations with risk factors [*57,132*], circumference measurements (possibly at the waist, thigh, and hips) may be preferred in epidemiologic studies because of their low measurement errors [*65*]. Despite the simplicity of circumference measurement, however, it is important to use standardized sites which are clearly specified [*135*]. Jakicic et al. [*134*], for example, found that levels of the waist to hip circumference ratio (WHR) among men were more strongly associated with total cholesterol levels if the hip circumference was measured at the buttocks rather than at the iliac crest (r = 0.22 vs 0.07). Small changes in the location of the waist measurement, which has frequently been obtained at either the smallest torso circumference or at the level of the umbilicus [*135*], can also influence associations with risk factors [*136*].

There are several mechanisms by which fat patterning could potentially influence metabolic and clinical complications, but attention has focused on adipocytes in the intra-abdominal region because of their proximity to the portal vein and their sensitivity to lipolytic stimuli [*48*]. The size of this fat depot, which

[8] Although "fat patterning" and "fat distribution" are often used interchangeably, fat distribution is sometimes used to describe what is seen or measured, whereas fat patterning often refers to the relative excess (or deficit) of fat at each site that would exist after adjustment for the overall level of obesity. When examining associations with metabolic and clinical outcomes, it would typically be of interest to focus on fat patterning.

can be directly measured using computerized tomography or magnetic resonance imaging [137] can be predicted fairly well ($R^2 = 0.5$ to 0.9) using age, Quetelet index, circumferences, and skinfolds [138]. Although the amount of intra-abdominal fat is moderately correlated with WHR, this fat depot may actually be more highly correlated with waist circumference (alone) [139], the waist/thigh circumference ratio [133], or the abdominal sagittal diameter [140]. There are also limitations in the use of ratios to describe fat patterning [141,142], including (1) uncertainty as to whether the numerator or denominator is responsible for any observed association, (2) an assumption that a regression of the characteristic has an intercept of 0, and (3) a loss of reliability due to the division of one imprecise number by another.

It is also likely that other fat depots are important, and adverse health outcomes have been related to circumferences measured at the neck [140] and bust [143] and to several contrasts (ratios or differences) between truncal skinfolds (subscapular, suprailiac, subumbilical) and those in the peripheral or lower-body (thigh, triceps, calf) regions [53,57]. Furthermore, several of these patterns are only weakly correlated with each other (WHR and the subscapular/triceps skinfold ratio showed correlations of 0.18 (men) and 0.22 (women) in a study of 738 Mexican American adults [132]), and it is possible that a single index (particularly one based on only two measurements) cannot convey all relevant information [132,140]. To overcome the difficulties involved in analyzing highly correlated anthropometric dimensions, principal component analysis has frequently been used to derive a smaller number of uncorrelated variables (components) [53,144,145].

Body Fat Patterning among Youth

Few studies have directly measured intra-abdominal fat among youth, and importance of fat patterning in early life is unclear. Because of the large age-related increases in hip circumference, mean levels of WHR decrease from ~1.1 to ~0.75 between the ages of 1 and 17 years ($r = -0.55$ with height) [146]. The amount of intra-abdominal fat among children is only weakly related to WHR [147–149], but stronger associations have been observed with waist circumference (alone) [149,150], the waist/thigh circumference ratio [148], and several truncal skinfolds [147,148]. In addition, the waist/thigh ratio is more strongly correlated with centralized obesity (as assessed by skinfolds) than is WHR [151]. Whereas some studies have found body fat patterning to be associated with various metabolic complications, equivocal or negative results have also been reported [150,152]. Furthermore, it is possible that the amount of fat in the subcutaneous region [86] may be as important as the small amount of intra-abdominal fat that is present before adulthood [147,149].

Despite these difficulties, a recent analysis of ~3,000 children and adolescents from the Bogalusa Heart Study [79] found that adverse lipid and insulin levels were associated with a central/abdominal distribution of body fat; furthermore, these associations were independent of weight, height, age, and other covariates. The magnitudes of the associations were similar whether fat patterning was characterized using (1) the waist circumference alone, (2) WHR, or (3) a principal component contrasting the waist circumference and the hip circumference and triceps skinfold thickness. These results suggest that the waist circumference (alone) may be useful in quantifying fat patterning in youth and that it can be used to assess the risk of adverse risk factor levels.

Age, Sex, and Racial/Ethnic Differences in Fat Patterning

Much of the information on age, sex, and racial/ethnic differences in fat patterning is based on skinfolds, and it is known that during adolescence, boys experience a marked truncal redistribution of subcutaneous adipose tissue [75,76]. This fat redistribution among boys is concurrent with a decrease in percentage body fat [49]; in contrast, there is little change in overall body fatness and little or no change in fat patterning among adolescent girls [153,154]. Because sex hormones are known to influence regional fat distribution, it is likely that the hormonal environment contributes to this fat redistribution.

Although it is difficult to assess the independent effects of chronological age and maturation on fat patterning [153,154], many of the observed associations likely reflect, at least in part, sexual maturation. For example, after controlling for chronological age, early maturation (defined by bone age or sexual maturity) among boys is associated with a relative excess of subcutaneous fat in the truncal regions [154,155]. Although postmenarcheal girls have also been found to have thicker truncal skinfolds than do premenarcheal girls, this may partly reflect small differences in chronological age [75] or the association between skinfold thickness ratios and Quetelet index [155].

Racial/ethnic differences in fat patterning have also been observed. Compared with white children, Mexican American school-aged children appear to have a relative excess of upper-body fat, and black children tend to have a more centralized fat pattern with less fat in the arms and legs [156,157]. During adolescence, Mexican American boys experience a particularly large decrease in leg fat, with the median calf skinfold thickness decreasing by ~40% (5 mm) [158]. In addition, prepubertal white children have more intra-abdominal fat than do black children [*159*].

Although relatively few studies have assessed the tracking of fat patterning, serial correlations may be weaker than those seen for the general level of overweight/obesity [*75,160,161*]. However, analogous to the trends observed for obesity and overweight, the magnitude of these correlations appears to increase with the age at which the initial measurement is obtained. For example, analyses from the Amsterdam Growth and Health Study indicate that the subscapular/triceps ratio measured at age 13 is moderately correlated ($r \sim 0.5$) with its level during the subsequent 16 years [*75*]. Furthermore, it is possible that the relative excess of truncal fat associated with early maturation may persist into adulthood [*154,162*]. Many of these associations, however, are substantially influenced by the errors inherent in longitudinal skinfold measurements, and additional studies are needed to evaluate the tracking of circumferences

SUMMARY AND RECOMMENDATIONS

Despite the advances that have been made in measuring adiposity, there is little evidence that estimates of total body fat, whether based on skinfolds, bioelectrical impedance, DXA, or densitometry, predict various metabolic and clinical complications better than do simple combinations of weight and height. This conclusion may reflect errors in the methods currently used to estimate body fat, and it is possible that more accurate methods may reveal that excess adiposity is the body component of most concern. However, the relative importance of weight–height indices compared with other methods also raises the possibility that fat-free mass may play a role in the development of diabetes mellitus, IHD, and other complications.

In contrast to a global index of adiposity, the patterning of body fat is an important risk factor for several chronic diseases. Although the best measure of body fat distribution is uncertain, it is possible that contrasts between various circumferences and skinfolds convey important, albeit somewhat different, information. Among youth, the relatively small amount of intra-abdominal fat and the poor performance of various anthropometric indices as a surrogate for intra-abdominal fat complicate the assessment of fat patterning. Because some evidence suggests that, in addition to the amount to intra-abdominal fat, an excess of subcutaneous fat in various regions [*132,140,163*] is associated with adverse health outcomes, it is probably best to obtain skinfolds or circumferences from various body regions. Fat patterning among youth may be best described by two sets of measurements: (1) at least two skinfolds, with one from the upper body (trunk) and another from the lower body, and (2) the waist (and possibly thigh) circumference or sagittal diameter. Further research, however, is needed to determine the importance of fat patterning among children and adolescents, its tracking over time, and the characteristics that influence the development and persistence of an adverse fat pattern.

It is also important to obtain additional information on the relation of overweight/obesity and body fat patterning among youth to various chronic diseases in adulthood. It would also be helpful to establish whether any associations between childhood obesity (or fat patterning) and adult disease are independent of obesity status in adulthood. These data, along with information on characteristics that influence these associations, may allow more effective interventions to be developed and more precisely targeted.

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REFERENCES

1. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med 1992;327:1350–5.

2. Nieto FJ, Szklo M, Comstock GW. Childhood weight and growth rate as predictors of adult mortality. Am J Epidemiol 1992; 136:201–13.

3. Abraham S, Collins G, Nordsieck M. Relationship of childhood weight status to morbidity in adults. HSMHA Health Rep 1971;86:273–84.

4. Hoffmans MDA, Kromhout D, de Lezenne Coulander C. The impact of body mass index of 78,612 18-year old Dutch men on 32-year mortality from all causes. J Clin Epidemiol 1988;41:749–56.

5. Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. Int J Obes 1997;21:507–26.

6. Gibson RS. Principles of nutritional assessment. New York: Oxford Univ. Press, 1990.

7. Bates CJ, Thurnham DJ. Biochemical markers of nutrient intake. In: Margetts BM, Nelson M, editors. Design concepts in nutritional epidemiology. New York: Oxford Univ. Press, 1991:192–265.

8. Kuczmarski R J, Johnson C. National nutritional surveys assessing anthropometric status. In: Himes JH, editor. Anthropometric assessment of nutritional status. New York: Wiley-Liss, 1991:319–35.

9. Berenson GS, editor. Causation of cardiovascular risk factors in children. Perspectives on cardiovascular risk in early life. New York: Raven Press, 1986.

10. Freedman DS, Srinivasan SR, Valdez RA, Williamson DF, Berenson GS. Secular trends in relative weight and adiposity obesity among children over two decades: the Bogalusa Heart Study. Pediatrics 1997;99:420–5.

11. Cleveland WS. The elements of graphing data. Monterey (CA): Wadsworth, 1985: 167–78.

12. Kushner RF. Body weight and mortality. Nutr Rev 1993;51: 127–36.

13. Hodge AM, Simmet PZ. The epidemiology of obesity. Baillieres Clin Endocrinol Metab 1994;8:577–99.
14. Baumgartner RN, Heymsfield SB, Roche AF. Human body composition and the epidemiology of chronic disease. Obes Res 1995;3:73–95.
15. Spataro JA, Dyer AR, Stamler J, Shekelle RB, Greenlund K, Garside D. Measures of adiposity and coronary heart disease mortality in the Chicago Western Electric Company Study. J Clin Epidemiol 1996;49:849–57.
16. Spiegelman D, Israel RG, Bouchard C, Willett WC. Absolute fat mass, percent body fat, and body-fat distribution: which is the real determinant of blood pressure and serum glucose? Am J Clin Nutr 1992;55:1033–44.
17. Richelsen B, Pedersen SB, Møller-Pedersen T, Schmitz O, Møller N, Børglum JD. Lipoprotein lipase activity in muscle tissue influenced by fatness, fat distribution and insulin in obese females. Eur J Clin Invest 1993;23:226–33.
18. Mann GV. The influence of obesity on health (PII). N Engl J Med 1974;291:226–32.
19. Manson JE, Stampfer MJ, Hennekens CH, Willett W. Body weight and longevity: a reassessment. JAMA 1987;257:353–8.
20. Berenson GS, Srinivasan SR, Wattigney WA, Harsha DW. Obesity and cardiovascular risk in children. Ann N Y Acad Sci 1993;699:93–103.
21. Willett WC, Manson JE, Stampfer MJ, Colditz GA, Rosner B, Speizer FE, et al. Weight, weight change, and coronary heart disease in women. Risk within the 'normal' weight range. JAMA 1995;273:461–5.
22. Paneth N, Susser M. Early origin of coronary heart disease (the "Barker Hypothesis"). Br Med J 1995;310:411–2.
23. Joseph KS, Kramer MS. Review of the evidence on fetal and early childhood antecedents of adult chronic disease. Epidemiol Rev 1996; 18:158–74.
24. Barker DJP. Mothers, babies, and disease in later life. London: BMJ Publ., 1994.
25. Lucas A. Programming by early nutrition in man. In: Bock GR, Whelan J, editors. Ciba Foundation Symposium: the childhood environment and adult disease. Chichester (UK): Wiley, 1991;156:38–55.
26. Frisancho AR. Anthropometric standards for the assessment of growth and nutritional status. Ann Arbor (MI): Univ. of Michigan Press, 1990.
27. Lohman TG, Roche AF, Martorell R, editors. Anthropometric standardization reference manual. Champaign (IL): Human Kinetics, 1988.
28. Mueller WH, Martorell R. Reliability and accuracy of measurement. In: Lohman TG, Roche AF, Martorell R, editors. Anthropometric standardization reference manual. Champaign (IL): Human Kinetics Books, 1988:83–6.
29. Snedecor GW, Cochran WG. Statistical methods. 7th ed. Ames (IA): Iowa State Univ. Press, 1980:243–4.
30. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986;1:307–10.
31. Lohman TG. Applicability of body composition techniques and constants for children and youths. Exerc Sport Sci Rev 1986; 14:325–57.
32. Roche AF. Methodological considerations in the assessment of childhood obesity. Ann N Y Acad Sci 1993;699:6–17.
33. Lukaski HC. Methods for the assessment of human body composition: traditional and new. Am J Clin Nutr 1987;46:537–6.
34. Hendel HW, Gotfredsen A, Andersen T, Højgaard L, Hilsted J. Body composition during weight loss in obese patients estimated by dual energy X-ray absorptiometry and by body potassium. Int J Obes 1996;20:1111–9.
35. Fogelholm M, van Marken Lichtenbelt W. Comparison of body composition methods: a literature review. Eur J Clin Nutr 1997; 51:495–503.
36. Martin AD, Drinkwater DT. Variability in the measures of body fat. Assumptions or technique? Sports Med 1991;11:277–88.
37. Guo SM, Roche AF, Houtkooper L. Fat-free mass in children and young adults predicted from bioelectric impedance and anthropometric variables. Am J Clin Nutr 1989;50:435–43.
38. Heymsfield SB, Wang ZM, Withers RT. Multicomponent molecular level models of body composition analysis. In: Roche AF, Heymsfield SB, Lohman TG, editors. Human body composition. Champaign (IL): Human Kinetics, 1996:129–48.
39. Jebb SA, Elia M. Techniques for the measurement of body composition: a practical guide. Int J Obes 1993;17:611–21.
40. Aloia JF, Vaswani A, Ma R, Flaster E. Comparative study of body composition by dual-energy x-ray absorptiometry. J Nucl Med 1995;36:1392–7.
41. Gutin B, Litaker M, Islam S, Manos T, Smith C, Treiber F. Body-composition measurement in 9–11-y-old children by dual-energy X-ray absorptiometry, skinfold-thickness measurements, and bioimpedance analysis. Am J Clin Nutr 1996;63:287–92.
42. Roubenoff R, Kehayias JJ, Dawson-Hughes B, Heymsfield SB. Uses of dual-energy x-ray absorptiometry in body-composition studies: not yet a "gold standard." Am J Clin Nutr 1993;58: 589–91.
43. Lohman TG. Dual energy x-ray absorptiometry. In: Roche AF, Heymsfield SB, Lohman TG, editors. Human body composition. Champaign (IL): Human Kinetics, 1996:63–78.
44. Lohman TG. Skinfolds and body density and their relation to body fatness: a review. Hum Biol 1981;53:181–225.
45. Martin AD, Ross WD, Drinkwater DT, Clarys JP. Prediction of body fat by skinfold caliper: assumptions and cadaver evidence. Int J Obes 1985; 9 Suppl 1:31–9.
46. Siervogel RM, Roche AF, Himes JH, Chumlea WC, McCammon R. Subcutaneous fat distribution in males and females from 1 to 39 years of age. Am J Clin Nutr 1982;36:162–71.
47. Allen TH, Peng MT, Chen KP, Huang TF, Chang C, Fang HS. Prediction of total adiposity from skinfolds and the curvilinear relationship between external and internal adiposity. Metabolism 1956;5:346–52.
48. Björntorp P. "Portal" adipose tissue as a generator of risk factors for cardiovascular disease and diabetes. Arteriosclerosis 1990; 10:493–6.
49. Johnston FE. Relationships between body composition and anthropometry. Hum Biol 1982;54:221–45.
50. Roche AF, Siervogel RM, Chumlea WC, Webb P. Grading body fatness from limited anthropometric data. Am J Clin Nutr 1981;34:2831–8.
51. Roche AF. Anthropometric methods: new and old, what they tell us. Int J Obes 1984;8:509–23.
52. Lean MEJ, Han TS, Deurenberg P. Predicting body composition by densitometry from simple anthropometric measurements. Am J Clin Nutr 1996;63:4–14.
53. Ducimetiere P, Richard J, Cambien F. The pattern of subcutaneous fat distribution in middle-aged men and the risk of coronary heart disease: the Paris Prospective Study. Int J Obes 1986; 10:229–40.
54. Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. Am J Clin Nutr 1994;59:307–16.

55. Norgan NG, Ferro-Luzzi A. The estimation of body density in men: are general equations general? Ann Hum Biol 1985;12: 1–15.

56. Seefeldt VD, Harrison GG. Infants, children, and youth. In: Lohman TG, Roche AF, Martorell R, editors. Anthropometric standardization reference manual. Champaign (IL): Human Kinetics, 1988:111–4.

57. Mueller WH, Wear ML, Hanis CL, Emerson JB, Barton SA, Hewett-Emmett D, et al. Which measure of body fat distribution is best for epidemiologic research? Am J Epidemiol 1991;133: 858–69.

58. Durnin JVGA, Wormersley J. Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 12–72 years. Br J Nutr 1974;32:77–97.

59. Garn SM, Sullivan TV, Hawthorne VM. Differential rates of fat change relative to weight change at different body site. Int J Obes 1987;11:519–25.

60. Bray GA, Greenway FL, Molitch ME, Dahmes WT, Atkinson RL, Hamilton K. Use of anthropometric measures to assess weight loss. Am J Clin Nutr 1978;31:769–73.

61. Williams DP, Going SB, Lohman TG, Harsha DW, Srinivasan SR, Webber LS, 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.

62. Ruiz L, Colley JR, Hamilton PJ. Measurement of triceps skinfold thickness: an investigation of sources of variations. Br J Prev Soc Med 1971;25:165–7.

63. Brans YW, Summers JE, Dweck HS, Cassady G. A non-invasive approach to body composition in the neonate: dynamic skin fold measurements. Pediatr Res 1974;8:215–22.

64. Marks GC, Habicht JP, Mueller WH. Reliability, dependability, and precision of anthropometric measurements: the second National Health and Nutrition Examination Survey 1976–1980. Am J Epidemiol 1989;130:578–87.

65. Mueller WH, Wear ML, Hanis CL, Barton SA, Schull WJ. Body circumferences as alternatives to skinfold measurements of body fat distribution in Mexican-Americans. Int J Obes 1987;11: 309–18.

66. Gruber JJ, Pollock ML, Graves JE, Colvin AB, Braith RW. Comparison of Harpendon and Lange calipers in predicting body composition. Res Q Exerc Sport 1990;61:184–90.

67. Zillikens MC, Conway JM. Anthropometry in blacks: applicability of generalized skinfold equations and differences in fat patterning between blacks and whites. Am J Clin Nutr 1990;52: 45–51.

68. Lohman TG, Pollock ML, Slaughter M, Brandon LJ, Boileau. Methodological factors and the prediction of body fat in female athletes. Med Sci Sports Med 1984;16:92–6.

69. Flegal KM, Harlan WR, Landis JR. Secular trends in body mass in the United States, 1960–80 [letter]. Am J Epidemiol 1990; 132:196–7.

70. Owen GM. Measurement, recording, and assessment of skinfold thickness in childhood and adolescence: report of a small meeting. Am J Clin Nutr 1982;35:629–38.

71. National Center for Health Statistics. Basic data on anthropometric measurements and angular measurement of the hip and knee joints for selected age groups 1–74 years of age: United States, 1971–75. Hyattsville (MD): Vital and Health Statistics, Series 11, No. 219, NCHS, 1981. [DHHS Publication No. (PHS) 81-1669]

72. Must A, Dallal GE, Dietz WH. Reference data for obesity: 85th and 95th percentiles of body mass index (wt/ht^2)—a correction. Am J Clin Nutr 1991;54:773.

73. Najjar MF, Rowland M. Anthropometric reference data and prevalence of overweight, United States, 1976–80. Washington: Public Health Service, Vital and Health Statistics, Series 11, No. 238, 1987. [USDHHS Publication No. (PHS) 87-1688]

74. National Center for Health Statistics. Anthropometric data and prevalence of overweight for Hispanics: 1982–84. Hyattsville (MD): Vital and Health Statistics, Series 11, No. 239, NCHS, 1989. [DHHS Publication No. (PHS) 89-1689]

75. Van Lenthe FJ, Kemper HCG, Van Mechelen W, Twisk JWR. Development and tracking of central patterns of subcutaneous fat in adolescence and adulthood: the Amsterdam Growth and Health Study. Int J Epidemiol 1996;25:1162–71.

76. Malina RM, Bouchard C. Subcutaneous fat distribution during growth. In: Bouchard C, Johnston FE, editors. Fat distribution during growth and later health outcomes. New York: A. R. Liss, 1988:63–84.

77. Rimm EB, Stampfer MJ, Colditz GA, Chute CG, Litin LB, Willett WC. Validity of self-reported waist and hip circumferences in men and women. Epidemiology 1990;1:466–73.

78. Malina RM, Hamill PVV, Lemeshow S. Selected body measurements of children 6–11 years, United States. Rockville (MD): Vital and Health Statistics, Series 11, No. 123. Department of Health, Education, and Welfare, 1973. [DHEW Publication No (HSM) 73-1605]

79. Freedman DS, Serdula MK, Srinivasan SR, Berenson GS. The relation of circumferences and skinfolds to levels of lipids and insulin among children and adolescents: the Bogalusa Heart Study. Am J Clin Nutr 1999;69:308–17.

80. Foster KR, Lukaski HC. Whole-body impedance—what does it measure? Am J Clin Nutr 1996;64 Suppl:388s–96s.

81. Houtkooper LB, Lohman TG, Going SB, Howell WH. Why bioelectrical impedance analysis should be used for estimating adiposity. Am J Clin Nutr 1996;64 Suppl:436s–48s.

82. Roubenoff R. Application of bioelectrical impedance analysis for body composition to epidemiologic studies. Am J Clin Nutr 1996;64 Suppl:459s–62s.

83. Bioelectrical impedance analysis in body composition measurements. NIH Technology Assessment Conference Statement. Am J Clin Nutr 1996;64 Suppl:524s–32s.

84. Kushner RF, Gudivaka R, Schoeller DA. Clinical characteristics influencing bioelectrical impedance analysis measurements. Am J Clin Nutr 1996;64 Suppl:423s–7s.

85. Heitmann BL. Impedance: a valid method in assessment of body composition? Eur J Clin Nutr 1994;48:228–40.

86. Yanovski JA, Yanovski SZ, Filmer KM, Hubbard VS, Avila N, Lewis B, et al. Differences in body composition of black and white girls. Am J Clin Nutr 1996;64:833–9.

87. Stolarczyk LM, Heyward VH, Hicks VL, Baumgartner RN. Predictive accuracy of bioelectrical impedance in estimating body composition of Native American women. Am J Clin Nutr 1994;59:964–70.

88. Frisancho AR, Flegel PN. Relative merits of old and new indices of body mass with reference to skinfold thickness. Am J Clin Nutr 1982;36:697–9.

89. Smalley KJ, Knerr AN, Kendrick ZV, Colliver JA, Owen OE. Reassessment of body mass indices. Am J Clin Nutr 1990; 52:405–8.

90. Gallagher D, Visser M, Sepúlveda D, Pierson RN, Harris T, Heymsfield SB. How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? Am J Epidemiol 1996;143:228–39.

91. Duerenberg P, Weststrate JA, Seidell JC. Body mass index as a measure of body fatness: age and sex-specific prediction formulas. Br J Nutr 1991;65:105–14.

92. Garrow JS. Treating obesity seriously: a clinical manual. Edinburgh: Churchill, 1981.

93. Cole TJ. Weight–stature indices to measure underweight, overweight, and obesity. In: Himes JH, editor. Anthropometric assessment of nutritional status. New York: Wiley-Liss, 1991: 83–111.

94. Garrow JS, Webster J. Quetelet's index (W/H^2) as a measure of fatness. Int J Obes 1985;9:147–53.

95. Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. Indices of relative weight and obesity. J Chron Dis 1972;25:329–43.

96. Kahn HS. A major error in nomograms for estimating body mass index. Am J Clin Nutr 1991;54:435–7.

97. Guo SS, Roche AF, Chumlea WC, Gardner JC, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35. Am J Clin Nutr 1994;59:810–9.

98. Lazarus R, Baur L, Webb K, Blyth F. Adiposity and body mass indices in children: Benn's index and other weight for height indices as measures of relative adiposity. Int J Obes 1996; 20:406–12.

99. WHO Expert Committee on Physical Status: the use and interpretation of anthropometry. Geneva: WHO, 1995. [Technical Report Series 854]

100. Garn SM. Fractionating healthy weight. Am J Clin Nutr 1996;63 Suppl: 412s–4s.

101. Wienpahl J, Ragland DR, Sidney S. Body mass index and 15-year mortality in a cohort of black men and women. J Clin Epidemiol 1990;43:949–60.

102. Lazarus R, Baur L, Webb K, Blyth F. Body mass index in screening for adiposity in children and adolescents: systematic evaluation using receiver operating characteristic curves. Am J Clin Nutr 1996;63:500–6.

103. Himes JH, Bouchard C. Validity of anthropometry in classifying youths as obese. Int J Obes 1989;13:183–93.

104. Rowland ML. Self-reported weight and height. Am J Clin Nutr 1990;52:1125–33.

105. Crawley HF, Portides G. Self-reported versus measured height, weight and body mass index amongst 16–17 years old British teenagers. Int J Obes 1995;19:579–84.

106. Hammer LD, Kraemer HC, Wilson DM, Ritter PL, Dornbusch SM. Standardized percentile curves of body-mass index for children and adolescents. Am J Dis Child 1991;145:259–63.

107. Rolland-Cachera MF, Cole TJ, Sempé M, Tichet J, Rossignol C, Charraud A. Body mass index variations: centiles from birth to 87 years. Eur J Clin Nutr 1991;45:13–21.

108. Roche AF, Guo S, Baumgartner RN, Chumlea WC, Ryan AS, Kuczmarski RJ. Reference data for weight, stature, and weight/stature^2 in Mexican Americans from the Hispanic Health and Nutrition Examination survey (HHANES 1982–1984). Am J Clin Nutr 1990;51:917s–24s.

109. Freedman DS, Serdula MK, Percy CA, Ballew C, White L. Obesity, levels of lipids and glucose, and smoking among Navajo adolescents. J Nutr 1997;127 Suppl:2120s–7s.

110. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents: the National Health and Nutrition Examination Surveys, 1963 to 1991. Arch Pediatr Adolesc Med 1995;149:1085–91.

111. Federation of American Societies for Experimental Biology. Third report on nutrition monitoring in the United States: Vol. I. Washington: U.S. Govt. Printing Office, 1995:171–82.

112. Lindsted K, Tonstad S, Kuzma JW. Body mass index and patterns of mortality among Seventh-day Adventist men. Int J Obes 1991;15:397–406.

113. Committee on Diet and Health, Food Nutrition Board, Commission on Life Sciences. Obesity and eating disorders. In: Diet and health. Washington: National Academy Press, 1989:563–92.

114. Abernathy RP, Black DR. Healthy body weight: an alternative perspective. Am J Clin Nutr 1996;63 Suppl:448s–51s.

115. Kuczmarski RJ. Prevalence of overweight and weight gain in the United States. Am J Clin Nutr 1992;55:495s–502s.

116. Meisler JG, St Jeor S. Summary and recommendations from the American Health Foundation's Expert Panel on Healthy Weight. Am J Clin Nutr 1996;63 Suppl:474s–7s.

117. Andres R, Elahi D, Tobin JD, Muller DC, Brant L. Impact of age on weight goals. Ann Intern Med 1985;103:1030–3.

118. Wardlaw GM. Putting body weight and osteoporosis into perspective. Am J Clin Nutr 1996;63 Suppl:433s–6s.

119. Dwyer T, Blizzard CL. Defining obesity in children by biological endpoint rather than population distribution. Int J Obes 1996;20:472–80.

120. Webber LS, Freedman DS. Interrelationships of coronary heart disease risk factors in children. In: Berenson GS, editor. Causation of cardiovascular risk factors in children. New York: Raven Press, 1986:65–81.

121. Efron B, Tibshirani RJ. An introduction to the bootstrap. London: Chapman & Hall, 1993.

122. 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;22:167–77.

123. Clarke WR, Lauer RM. Does obesity track into adulthood? Crit Rev Food Sci Nutr 1993;33:423–30.

124. Johnston FE. Health implications of childhood obesity. Ann Intern Med 103;6:1068–72.

125. Siervogel RM, Roche AF, Guo S, Mukhergee D, Chumlea WC. Patterns of change in weight/stature^2 from 2 to 18 years: findings from the long-term serial data for children in the Fels Longitudinal Growth Study. Int J Obes 1991;15:479–85.

126. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997;227:869–73.

127. Dietz WH. Critical periods in childhood for the development of obesity. Am J Clin Nutr 1994;59:955–9.

128. Eckel RH. Insulin resistance: an adaptation for weight maintenance. Lancet 1992;340:1452–3.

129. Vague J. The degree of masculine differentiation of obesities: a factor determining predisposition to diabetes, atherosclerosis, gout, and uric calculous disease. Am J Clin Nutr 1956;4:20–34.

130. Björntorp P. Abdominal fat distribution and disease: an overview of epidemiological data. Ann Med 1992;24:15–8.

131. Seidell JC. Regional obesity and health. Int J Obes 1992;16 Suppl 2:S31–4.

132. Haffner SM, Stern MP, Hazuda HP, Pugh J, Patterson JK. Do upper-body and centralized adiposity measure different aspects of regional body-fat distribution? Relationship to non-insulin-dependent diabetes mellitus, lipids, and lipoproteins. Diabetes 1987;36:43–51.

133. Terry RB, Stefanick ML, Haskell WL, Wood PD. Contributions of regional adipose tissue depots to plasma lipoprotein concentrations in overweight men and women: possible protective effects of thigh fat. Metabolism 1991;40:733–40.

134. Jakicic JM, Donnelly JE, Jawad AF, Jacobsen DJ, Gunderson SC, Pascale R. Association between blood lipids and different measures of body fat distribution: effects of BMI and age. Int J Obes 1993;17:131–7.

135. Callaway CW, Chumlea WC, Bouchard C, et al. Circumferences. In: Lohman TG, Roche AF, Martorell R, editors. Anthropometric

standardization reference manual. Champaign (IL): Human Kinetics, 1988:39–54.

136. Seidell JC, Cigolini M, Charzewska, J, Ellsinger BM, DiBiases G. Fat distribution in European women: a comparison of anthropometric measurements in relation to cardiovascular risk factors: Int J Epidemiol 1990;19:303–8.

137. Sjöström L. Measurement of fat distribution. In: Bouchard C, Johnston Fe, editors. Fat distribution during growth and later health outcomes. New York: A. R. Liss, 1988:43–61.

138. van der Kooy K, Seidell JC. Techniques for the measurement of visceral fat: a practical guide. Int J Obes 1993;17:187–96.

139. Pouliot MC, Després, JP, Lemieux S, Moorjani S, Bouchard C, Tremblay A, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol 1994;73:460–8.

140. Sjöström CD, Håkangård AC, Lissner L, Sjöström L. Body compartment and subcutaneous adipose tissue distribution—risk factor patterns in obese subjects. Obes Res 1995;3:9–22.

141. Kronmal RA. Spurious correlation and the fallacy of the ratio standard revisited. J R Stat Soc A 1993;156:379–92.

142. Goran MI, Allison DB, Poehlman. Issues relating to normalization of body fat content in men and women. Int J Obes 1995;19:638–43.

143. Freedman DS, Rimm AA. The relation of body fat distribution, as assessed by six girth measurements, to diabetes mellitus in women. Am J Public Health 1989;79:715–20.

144. Mueller WH, Wohlleb JC. Anatomical distribution of subcutaneous fat and its description by multivariate methods: how valid are principal components? Am J Phys Anthropol 1981;54:25–35.

145. Baumgartner RN, Roche AF, Guo S, Lohman T, Boileau RA, Slaughter MH. Adipose tissue distribution: the stability of principal components by sex, ethnicity, and maturation stage. Hum Biol 1986;58:719–35.

146. Weststrate JA, Deurenberg P, van Tinteren H. Indices of body fat distribution and adiposity in Dutch children from birth to 18 years of age. Int J Obes 1989;13:456–77.

147. Goran MI, Kaskoun M, Shuman WP. Intra-abdominal adipose tissue in young children. Int J Obes 1995;19:279–83.

148. Brambilla P, Manzoni P, Sironi S, Simone P, Del Maschio A, di Natale B, et al. Peripheral and abdominal adiposity in childhood obesity. Int J Obes 1994;18:795–800.

149. de Ridder CM, de Boer RW, Seidell JC, Nieuwenhoff CM, Jeneson JAL, Bakker CJG, et al. Body fat distribution in pubertal girls quantified by magnetic resonance imaging. Int J Obes 1992;16:443–9.

150. Sangi H, Mueller WH, Harrist RB, Rodriguez B, Grunbaum JG, Labarthe DR. Is body fat distribution associated with cardiovascular risk factors in childhood? Ann Hum Biol 1992;19:559–79.

151. Mueller WH, Marbella A, Harrist RB, Kaplowitz HJ, Gunbaum JA, Labarthe DR. Body circumferences as alternatives to skinfold measures of body fat distribution in children. Ann Hum Biol 1989;16:495–506.

152. Baumgartner RN, Siervogel RM, Chumlea WC, Roche AF. Associations between plasma lipoprotein cholesterols, adiposity and adipose tissue distribution during adolescence. Int J Obes 1989;13:31–41.

153. Flodmark CE, Sveger T, Milsson-Ehle P. Waist measurement correlates to a potentially atherogenic lipoprotein profile in obese 12–14-year-old children. Acta Paediatr 1995;83:941–5.

154. Beunen G, Malina RM, Lefevre J, et al. Size, fatness and relative fat distribution of males of contrasting maturity status during adolescence and as adults. Int J Obes 1994;18:670–8.

155. Deutsch MI, Mueller WH. Androgyny in fat patterning is associated with obesity in adolescents and young adults. Ann Hum Biol 1985;12:275–86.

156. Malina RM. Regional body composition: age, sex, and ethnic variation. In: Roche AF, Heymsfield SB, Lohman TG, editors. Human body composition. Champaign (IL): Human Kinetics, 1996:217–55.

157. Mueller WH. Ethnic differences in fat distribution during growth. In: Bouchard C, Johnston Fe, editors. Fat distribution during growth and later health outcomes. New York: A. R. Liss, 1988:127–45.

158. Ryan AS, Martinez GA, Baumgartner RN, Roche AF, Guo S, Chumlea WC. Median skinfold thickness distributions and fatwave patterns in Mexican-American children from the Hispanic Health and Nutrition Examination Survey (HHANES 1982–1985). Am J Clin Nutr 1990;51:925s–35s.

159. Goran MI, Nagy Tr, Treuth M, et al. Visceral fat in Caucasian and African-American pre-pubertal children. Am J Clin Nutr 1997;65:1703–8.

160. Rolland-Cachera MF, Bellisle F, Debeeger M, Pequignot F, Sempe M. Influence of body fat distribution during childhood on body fat distribution in adulthood: a two-decade follow-up study. Int J Obes 1990;14:473–81.

161. Roche AF, Baumgartner RN. Tracking in fat distribution during growth. In: Bouchard C, Johnston FE, editors. Fat distribution during growth and later health outcomes. New York: A. R. Liss, 1988:147–62.

162. van Lenthe FJ, Kemper HCG, van Mechelen W, Post GB, Twisk JWR, Snel J. Biological maturation and the distribution of subcutaneous fat from adolescence into adulthood: the Amsterdam growth and health study. Int J Obes 1996;20:121–9.

163. Abate N, Garg A, Peshock RM, Stray-Gundersen J, Adams-Huet B, Grundy SM. Relationship of generalized and regional adiposity to insulin sensitivity in men with NIDDM. Diabetes 1996;45:1684–93.