Relation of body mass index and skinfold thicknesses to cardiovascular disease risk factors in children: the Bogalusa Heart Study1–4

David S Freedman, Peter T Katzmarzyk, William H Dietz, Sathanur R Srinivasan, and Gerald S Berenson

ABSTRACT

Background: Adverse levels of cardiovascular disease (CVD) risk factors are related to skinfold thicknesses and body mass index (BMI) among children, but the relative strengths of these associations are unknown.

Objective: The objective was to determine whether the sum of the triceps and subscapular skinfold thicknesses (SF sum) is more strongly related to levels of 6 risk factors (triglycerides, LDL and HDL cholesterol, insulin, and systolic and diastolic blood pressure) than is BMI.

Design: Cross-sectional analyses of schoolchildren examined in the Bogalusa Heart Study from 1981 to 1994 (n = 6866) were conducted. A risk factor summary index was derived by using principal components analysis.

Results: After race, sex, study period, and age were controlled for, almost all comparisons indicated that BMI was more strongly related to risk factor levels than was the SF sum. Although the differences were generally small, many were statistically significant. Associations with the risk factor summary, for example, were \( r = 0.50 \) for BMI and \( r = 0.47 \) for SF sum (\( P < 0.001 \) for difference). Furthermore, an adverse risk factor summary was observed among 62% of the children with the highest (upper 5%) BMI levels but among only 54% of children with the highest SF sum levels.

Conclusions: BMI is at least as accurate as SF sum in identifying children and adolescents who are at metabolic risk. Because of the training and errors associated with skinfold-thickness measurements, the advantages of BMI should be considered in the design and interpretation of clinical and epidemiologic studies. Am J Clin Nutr 2009;90:210–6.

INTRODUCTION

A high body mass index (BMI; in kg/m\(^2\)) in children is associated with adverse levels of various CVD risk factors, the initial stages of atherosclerosis, and obesity and mortality in adulthood (1–4). However, because BMI is based on only weight and height, both of which change greatly during growth and development, a high BMI can reflect increases in either fat mass or fat-free mass (5, 6). Although a child with a very high BMI is likely to have elevated body fatness (7), BMI can be an inaccurate indicator of body fatness in normal-weight children (8).

Skinfold thicknesses are more strongly associated with body fatness, as estimated by various reference methods, than is BMI (8–12). Because of these stronger associations with body fatness, it is frequently assumed that skinfold thicknesses would be better predictors of adverse health outcomes than BMI. Based on cross-sectional associations with levels of cardiovascular disease risk factors, various cutoffs for excess body fatness (estimated from skinfold thicknesses) in children have been proposed (13–15).

Few studies, however, have examined whether levels of cardiovascular disease risk factors are more strongly related to skinfold thicknesses than to BMI. Our objective was to determine whether the sum of subscapular and triceps skinfold thicknesses (SF sum) is more strongly related to blood pressure and concentrations of lipids and fasting insulin in children and adolescents (n = 6866) than is BMI.

SUBJECTS AND METHODS

Study population

Bogalusa is a biracial (one-third black) community in Louisiana (16). Seven cross-sectional studies of schoolchildren aged 5–17 y, with >3000 children participating in each examination, were conducted from 1973 to 1994; many children were examined in more than one of these examinations. Although BMI, blood pressure, and concentrations of lipids and lipoproteins were measured in each study, fasting insulin concentrations were not available for most children until the fourth (1981–1982) examination. Our analyses are therefore limited to 5- to 17-yr-olds who participated in studies conducted in 1981–1982, 1983–85, 1987–1988, and 1992–1994 (12,923 examinations of 7852 different children). Informed consent was obtained from all participants, and study protocols were approved by human subjects review committees.

We excluded children who were not fasting (n = 1654) and those without recorded values for weight, height, and skinfold thicknesses (n = 23). Another 611 children were excluded because they were missing information on any of the 6 cardio

1 From the Division of Nutrition, Physical Activity and Obesity, Centers for Disease Control and Prevention, Atlanta, GA (DSF and WHD); the Pennington Biomedical Research Center, Baton Rouge, LA (PTK); and the Tulane Center for Cardiovascular Health, Tulane University School of Public Health and Tropical Medicine, New Orleans, LA (SRS and GSB).

2 The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

3 Supported by National Institute on Aging grant AG-16592.

4 Address reprint requests and correspondence to DS Freedman, CDC K-26, 4770 Buford Highway, Atlanta, GA 30341–3724. E-mail: dxf1@cdc.gov.

Received January 22, 2009. Accepted for publication April 8, 2009. First published online May 6, 2009; doi: 10.3945/ajcn.2009.27525.

210

vascular disease risk factors: triglycerides, LDL cholesterol, HDL cholesterol, fasting insulin, systolic blood pressure (SBP), and diastolic blood pressure (DBP). We also excluded 11 girls who reported being pregnant and 35 children who reported that they were taking insulin or had diabetes mellitus.

These exclusions resulted in 10,589 examinations, with ~60% of the children having participated in ≥2 studies. Because repeated measurements from the same child are not independent, the current analyses are based on the final examination for each child. This resulted in a sample size of 6866 children in most analyses. Analyses that are stratified by study, however, are based on all 10,589 examinations; a child was examined only once in each cross-sectional study.

Examinations and risk factors

Height was measured to the nearest 0.1 cm, weight was measured to the nearest 0.1 kg, and BMI was calculated as a measure of relative weight. The triceps and subscapular skinfold thicknesses were measured 3 times to the nearest 1.0 mm with Lange Skinfold Calipers (Cambridge Scientific Industries, Cambridge, MD), and the mean values were used in the analyses. We used the sum of the triceps and subscapular skinfold thicknesses (SF sum) as an overall measure of subcutaneous fat.

We previously reported (17) that of 323 rescreened children in the 1981–1982 examination, the intraclass correlation coefficient between replicate skinfold-thickness measurements was \( r = 0.98 \) for both the triceps and subscapular skinfold thicknesses. This estimate of reproducibility is lower than estimates for weight and height \( (r > 0.999) \) but higher than estimates for blood pressure and laboratory determinations \( (r = 0.85 \text{–} 0.95) \).

Sitting levels of SBP and DBP were measured on the right arm 3 times by 2 observers with a mercury sphygmomanometer (Baumanometer; WA Baum Co, Inc, Copiague, NY) (16, 18), and the mean of the 6 values was used in the analyses. Serum triglyceride concentrations were measured by using enzymatic procedures, and concentrations of LDL and HDL cholesterol were measured by using heparin-calcium precipitation and agar-agarose gel electrophoresis (19). Plasma insulin was measured by using a radioimmunoassay procedure (Phadebas Insulin Kit; Pharmacia Diagnostics AB, Uppsala, Sweden).

Statistical analyses

Analyses were performed by using R (20). Because the distribution of several characteristics was skewed, we used the 15th, 50th (median), and 85th percentiles to summarize levels by sex. (For a normally distributed variable, the 15th and 85th percentiles to summarize levels by sex. (For a normally distributed variable, the 15th and 85th percentiles would be \( \pm 1 \) SD from the mean.)

Levels of BMI, skinfold thicknesses, and the 6 risk factors were adjusted for age, race, and study in sex-specific regression models; the residuals from these models represent a child’s level relative to other children of the same sex, age, race, and study period. We used these models rather than the Centers for Disease Control and Prevention (CDC) growth charts of BMI-for-age (21), so that similar adjustments could be made to BMI and the skinfold thicknesses. Principal components analysis (22) was then used to derive a smaller number of uncorrelated variables from the adjusted risk factor levels (residuals). The first principal component, which accounted for 32% of the variability in levels of the 6 risk factors, was used as an overall summary of risk.

Correlations between this risk factor summary (first principal component) and levels of each risk factor were 0.72 (triglycerides), 0.49 (LDL cholesterol), −0.52 (HDL cholesterol), 0.57 (insulin), 0.57 (SBP), and 0.51 (DBP).

Correlation coefficients were used to summarize the relation of the various risk factors to levels of BMI and the skinfold thicknesses. The statistical significance of the differences between these associations with BMI or the skinfold thicknesses was assessed (23) after the association between these adiposity indexes was accounted for. For example, given the strong correlation between BMI and the SF sum \( (r = 0.86) \), we determined whether the correlation between fasting insulin and BMI \( (r = 0.46) \) differed significantly from the correlation between fasting insulin and the SF sum \( (r = 0.43) \). These associations were also examined within categories of sex, race, age, study period, and CDC BMI-for-age (21) to determine whether metabolic risk was more strongly associated with skinfold thickness than with BMI in any subgroup. Bootstrapping (24) was used to confirm the statistical significance of observed differences in the relation of various risk factors to BMI and the skinfold thickness.

Based on the distribution of adjusted levels of BMI and the SF sum, we constructed 5 categories ( <25th, 25th to 49th, 50th to 84th, 85th to 94th, and ≥95th percentiles) of each characteristic to examine the similarity of BMI and SF sum levels. Agreement between the 5 categories of BMI and the SF sum was assessed by the \( \kappa \) statistic with linear weighting (25). We also examined the prevalence of adverse risk factor levels across the 5 categories of BMI and the SF sum; adverse risk factor levels were considered to be an adjusted value ≥85th percentile (<15th percentile for HDL cholesterol).

Although levels of many of the examined characteristics were not normally distributed, similar results were obtained in additional analyses that were based on various normalizing transformations or the use of robust regression methods to minimize the effects of extreme values.

RESULTS

Levels of various characteristics by sex are shown in Table 1. The examined children were relatively heavy, with a median BMI-for-age \( z \) score of ~0.3, ~13% of the children had a BMI that was greater than the 95th percentile of the CDC reference population. Girls had substantially thicker skinfold thicknesses than did boys, with the relative difference for the SF sum reaching 46% (median: 19 mm among boys and 27 mm among girls). Sex differences in levels of the various risk factors were smaller, with the largest difference (22%) seen for fasting insulin (median: 8.0 and 9.8 mU/L).

As shown in Table 2, correlations between BMI and the various risk factors ranged in magnitude from \( r = 0.19 \) (LDL cholesterol and DBP) to \( r = 0.50 \) (risk factor summary); given the large \( (n = 6866) \) sample size, all associations were statistically significant \( (H_0: r = 0) \) at the 0.001 level. As seen in the second column, none of the risk factors were more strongly related to the SF sum than to BMI, and several of the associations with the SF sum were significantly weaker. Correlations with fasting insulin, for example, were \( r = 0.46 \) for BMI and \( r = 0.43 \) for SF sum \( (P < 0.001 \text{ for difference}) \). Levels of HDL cholesterol, SBP, and the risk factor summary were more
factor summary than was BMI within any subgroup (Table 3) Within almost all categories of sex, race, age, and BMI-for-age category, the risk factor summary was more strongly ($P < 0.05$) related to BMI than to the SF sum. For example, correlations were $r = 0.51$ for BMI and $r = 0.47$ for SF sum in boys, $r = 0.53$ and $r = 0.51$ in white children, and $r = 0.54$ and $r = 0.51$ in 5- to 8-y-olds. Furthermore, BMI was more strongly related to the risk factor summary than was the SF sum among children who were examined in 3 of the cross-sectional studies; in the one exception (children examined in 1983–1985), the risk factor summary was similarly related ($r = 0.44$) to both measures. Although the magnitudes of the associations were reduced within the 2 categories of BMI-for-age (bottom 2 rows), associations with BMI were stronger ($P < 0.05$) than those with the SF sum among both normal-weight and overweight children. Associations with the subscapular skinfold thickness tended to be stronger than with the triceps skinfold thickness within all strata (last 2 columns), but neither skinfold thickness was more strongly related to the risk factor summary than was BMI.

A cross-classification of categories of BMI and the SF sum (Table 4) indicated that most children were categorized

<table>
<thead>
<tr>
<th>Risk factor and category</th>
<th>BMI</th>
<th>SF sum</th>
<th>Triceps</th>
<th>Subscapular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall ($n = 6866$)</td>
<td>0.50</td>
<td>0.47</td>
<td>0.44</td>
<td>0.47</td>
</tr>
<tr>
<td>Boys ($n = 3434$)</td>
<td>0.51</td>
<td>0.47</td>
<td>0.43</td>
<td>0.48</td>
</tr>
<tr>
<td>Girls ($n = 3432$)</td>
<td>0.49</td>
<td>0.47</td>
<td>0.44</td>
<td>0.47</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whites ($n = 4358$)</td>
<td>0.53</td>
<td>0.51</td>
<td>0.47</td>
<td>0.50</td>
</tr>
<tr>
<td>Blacks ($n = 2508$)</td>
<td>0.44</td>
<td>0.41</td>
<td>0.39</td>
<td>0.41</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5–8 y ($n = 1560$)</td>
<td>0.54</td>
<td>0.51</td>
<td>0.49</td>
<td>0.49</td>
</tr>
<tr>
<td>9–11 y ($n = 1666$)</td>
<td>0.60</td>
<td>0.55</td>
<td>0.52</td>
<td>0.54</td>
</tr>
<tr>
<td>12–14 y ($n = 1920$)</td>
<td>0.49</td>
<td>0.45</td>
<td>0.40</td>
<td>0.46</td>
</tr>
<tr>
<td>15–17 y ($n = 1729$)</td>
<td>0.40</td>
<td>0.39</td>
<td>0.35</td>
<td>0.40</td>
</tr>
<tr>
<td>Examination year</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1981–1982 ($n = 2814$)</td>
<td>0.47</td>
<td>0.46</td>
<td>0.39</td>
<td>0.48</td>
</tr>
<tr>
<td>1983–1985 ($n = 2703$)</td>
<td>0.44</td>
<td>0.44</td>
<td>0.39</td>
<td>0.45</td>
</tr>
<tr>
<td>1987–1988 ($n = 2570$)</td>
<td>0.49</td>
<td>0.46</td>
<td>0.42</td>
<td>0.46</td>
</tr>
<tr>
<td>1992–1994 ($n = 2502$)</td>
<td>0.56</td>
<td>0.51</td>
<td>0.48</td>
<td>0.51</td>
</tr>
<tr>
<td>CDC BMI-for-age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;85th percentile ($n = 4987$)</td>
<td>0.24</td>
<td>0.22</td>
<td>0.17</td>
<td>0.23</td>
</tr>
<tr>
<td>$\geq$85th percentile ($n = 1879$)</td>
<td>0.38</td>
<td>0.35</td>
<td>0.29</td>
<td>0.35</td>
</tr>
</tbody>
</table>

$^1$ SF sum, sum of the triceps and subscapular skinfold thicknesses; BMI, body mass index; SF sum, sum of the triceps and subscapular skinfold thicknesses; BP, diastolic blood pressure. All correlation coefficients are significantly different from 0 ($P < 0.001$). P values were calculated by using the method proposed by Meng et al (23) for comparing correlated correlation coefficients and indicate whether the correlation between the risk factor and skinfold thickness is significantly different from the correlation between the risk factor and BMI.
similarly by the 2 measures. (Percentile cutoffs for BMI and the SF sum were based on adjusted levels among the examined children.) For example, of the 343 children who had a BMI in the upper 5% of the distribution (bottom row), 197 (57%) had an SF sum that was also in the upper 5%, and only 11 (3%) had an SF sum that was below the 85th percentile. Overall, 59% of the examined children were in identical categories of BMI and SF sum (along the diagonal), and only 4% of the children had an SF sum that differed by >1 category from the corresponding BMI category (eg, a BMI ≤ 25th percentile and an SF sum between the 50th and 84th percentiles). The χ2 statistic, with linear weighting, for the BMI and SF sum categories was 0.64, which indicated that there was moderate-to-substantial agreement between the 2 measures.

The proportion of children with adverse (>85th percentile) levels of the various risk factors varied similarly across categories of both BMI and the SF sum (Table 5). For example, only 7–8% of the 1716 children with a BMI or an SF sum in the bottom 25% of the distribution had a high triglyceride level, but the corresponding proportions were 45% (BMI) and 43% (SF sum) among children in the upper 5% of each adiposity index. A comparison of the proportions of children with high triglyceride concentrations between the 2 extreme adiposity categories yielded prevalence ratios of 6.2 for BMI (45%/7%) and 5.3 for the SF sum (43%/8%). Although the differences between the prevalence ratios for BMI and the SF sum were small, we found that BMI frequently showed a stronger (P < 0.05) association with risk factor levels than did the SF sum.

BMI does not distinguish between fat mass and fat-free mass, and its limitations as an indicator of adiposity are widely known (30). Although a high BMI can identify children who have excess body fatness (7), the BMI of normal-weight children can reflect levels of either fat mass or fat-free mass (5, 6). Furthermore, several investigators (8, 31, 32) have reported that the correlation between BMI and body fatness is only moderate (r < 0.7). It has been concluded that BMI is a very poor indicator of the body fatness of normal-weight children (8).

Although skinfold thicknesses are better predictors of total body fatness than is BMI, some of the differences between the associations with body fatness have been relatively small (5, 8, 10, 33, 34). Among 8- to 19-y-old girls, for example, the triceps skinfold thickness showed only a slightly stronger association with body fatness determined by underwater weighing than did BMI (r = 0.72 and 0.67, respectively) (33). Similarly, dual-energy X-ray absorptiometry estimates of percentage body fat among 130 adolescents were almost as strongly associated with levels of BMI (r = 0.87–0.89) as with skinfold thicknesses (r = 0.92–0.93) (10). The accuracy of skinfold thicknesses in predicting body fatness may also vary according to the selected sites and prediction equation. One analysis (8), for example, found that most skinfold-thickness equations were better predictors of body fatness (determined from a 4-compartment model) than was BMI (R² = 0.85 and 0.67, respectively), but that one skinfold-thickness equation was very inaccurate (R² = 0.51). The stronger associations that we observed with the subscapular skinfold thickness than with the triceps–skinfold thickness may reflect the importance of fat distribution.

Despite the limitations of BMI as an index of body fatness, risk factor levels have been found to be related similarly to BMI and to body fatness estimates based on skinfold thicknesses (10, 35), bioelectrical impedance (36–39), air-displacement plethysmography (28), dual-energy X-ray absorptiometry (10, 38–40), and underwater weighing (29). Among adults, for example, BMI and body fatness calculated by air-displacement plethysmography showed similar associations with levels of HDL cholesterol, triglycerides, SBP, insulin resistance, and C-reactive protein (28). Although more accurate estimates of body fatness may provide additional information on risk factor levels in certain subgroups, the results have been inconsistent. A study of normal-weight adults (BMI < 25) (41), for example, found that levels of body fatness (obtained from underwater weighing) were associated with metabolic risk factors among men but not among women.

### TABLE 4

Cross-classification of categories of BMI-for-age and the sum of the triceps and subscapular skinfold thicknesses (SF sum)

<table>
<thead>
<tr>
<th>BMI category</th>
<th>&lt;25th percentile</th>
<th>25th to 49th percentile</th>
<th>50th to 84th percentile</th>
<th>85th to 94th percentile</th>
<th>≥95th percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;25th percentile (n = 1716)</td>
<td>1169 (68)</td>
<td>438 (26)</td>
<td>109 (6)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>25th to 49th percentile (n = 1717)</td>
<td>454 (26)</td>
<td>771 (45)</td>
<td>488 (26)</td>
<td>4 (0)</td>
<td>—</td>
</tr>
<tr>
<td>50th to 84th percentile (n = 2403)</td>
<td>93 (4)</td>
<td>503 (21)</td>
<td>1587 (66)</td>
<td>194 (8)</td>
<td>26 (1)</td>
</tr>
<tr>
<td>85th to 94th percentile (n = 687)</td>
<td>—</td>
<td>5 (1)</td>
<td>208 (30)</td>
<td>354 (52)</td>
<td>120 (17)</td>
</tr>
<tr>
<td>≥95th percentile (n = 343)</td>
<td>—</td>
<td>—</td>
<td>11 (3)</td>
<td>135 (39)</td>
<td>197 (57)</td>
</tr>
</tbody>
</table>

Values are n; row percentages in parentheses.

1 Cutoffs for both BMI and SF sum were based on levels (adjusted for race, sex, age, and study period) of these 2 characteristics in the examined children.

### DISCUSSION

Adverse levels of cardiovascular disease risk factors are associated with estimates of body fatness obtained from skinfold thicknesses (10, 13, 14), bioelectrical impedance (26), dual-energy X-ray absorptiometry (10, 27), air-displacement plethysmography (28), and underwater weighing (29). Although BMI provides a less accurate estimate of body fatness than do these other methods (8), our results indicate that levels of various risk factors do not show weaker associations with BMI than with skinfold thicknesses. Although the observed differences were generally small, we found that BMI frequently showed a stronger association with risk factor levels than did the SF sum.

BMI does not distinguish between fat mass and fat-free mass, and its limitations as an indicator of adiposity are widely known (30). Although a high BMI can identify children who have excess body fatness (7), the BMI of normal-weight children can reflect levels of either fat mass or fat-free mass (5, 6). Furthermore, several investigators (8, 31, 32) have reported that the correlation between BMI and body fatness is only moderate (r < 0.7). It has been concluded that BMI is a very poor indicator of the body fatness of normal-weight children (8).

Although skinfold thicknesses are better predictors of total body fatness than is BMI, some of the differences between the associations with body fatness have been relatively small (5, 8, 10, 33, 34). Among 8- to 19-y-old girls, for example, the triceps skinfold thickness showed only a slightly stronger association with body fatness determined by underwater weighing than did BMI (r = 0.72 and 0.67, respectively) (33). Similarly, dual-energy X-ray absorptiometry estimates of percentage body fat among 130 adolescents were almost as strongly associated with levels of BMI (r = 0.87–0.89) as with skinfold thicknesses (r = 0.92–0.93) (10). The accuracy of skinfold thicknesses in predicting body fatness may also vary according to the selected sites and prediction equation. One analysis (8), for example, found that most skinfold-thickness equations were better predictors of body fatness (determined from a 4-compartment model) than was BMI (R² = 0.85 and 0.67, respectively), but that one skinfold-thickness equation was very inaccurate (R² = 0.51). The stronger associations that we observed with the subscapular skinfold thickness than with the triceps–skinfold thickness may reflect the importance of fat distribution.

Despite the limitations of BMI as an index of body fatness, risk factor levels have been found to be related similarly to BMI and to body fatness estimates based on skinfold thicknesses (10, 35), bioelectrical impedance (36–39), air-displacement plethysmography (28), dual-energy X-ray absorptiometry (10, 38–40), and underwater weighing (29). Among adults, for example, BMI and body fatness calculated by air-displacement plethysmography showed similar associations with levels of HDL cholesterol, triglycerides, SBP, insulin resistance, and C-reactive protein (28). Although more accurate estimates of body fatness may provide additional information on risk factor levels in certain subgroups, the results have been inconsistent. A study of normal-weight adults (BMI < 25) (41), for example, found that levels of body fatness (obtained from underwater weighing) were associated with metabolic risk factors among men but not among women.
TABLE 5
Prevalence of adverse risk factors by category of BMI and the sum of the triceps and subscapular skinfold thicknesses (SF sum)\(^1\)

<table>
<thead>
<tr>
<th>Risk factor and measure(^2)</th>
<th>Adiposity category(^4)</th>
<th>Prevalence ratio ((\geq 95th vs &lt; 25th percentile)(^6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;25th percentile ((n = 1716))</td>
<td>25th to 49th percentile ((n = 1717))</td>
</tr>
<tr>
<td>High triglycerides BMI</td>
<td>7(^7) 10 15 33 45</td>
<td>6.2 (5.0, 7.6)</td>
</tr>
<tr>
<td>SF sum</td>
<td>8 10 15 33 43</td>
<td>5.3 (4.3, 6.5)</td>
</tr>
<tr>
<td>High LDL cholesterol BMI</td>
<td>9 12 17 22 31</td>
<td>3.3 (2.6, 4.0)</td>
</tr>
<tr>
<td>SF sum</td>
<td>10 12 17 24 28</td>
<td>2.9 (2.3, 3.6)</td>
</tr>
<tr>
<td>Low HDL cholesterol BMI</td>
<td>9 13 16 25 30</td>
<td>3.3 (2.7, 4.2)</td>
</tr>
<tr>
<td>SF sum</td>
<td>9 13 17 23 26</td>
<td>2.9 (2.3, 3.6)</td>
</tr>
<tr>
<td>High fasting insulin BMI</td>
<td>5 6 13 41 69</td>
<td>15 (12, 19)</td>
</tr>
<tr>
<td>SF sum</td>
<td>4 7 14 44 60</td>
<td>13 (11, 17)</td>
</tr>
<tr>
<td>High SBP</td>
<td>9 11 17 26 33</td>
<td>3.7 (3.0, 4.6)</td>
</tr>
<tr>
<td>BMI</td>
<td>9 12 16 27 29</td>
<td>3.1 (2.5, 3.9)</td>
</tr>
<tr>
<td>SF sum</td>
<td>10 11 17 22 29</td>
<td>2.9 (2.3, 3.6)</td>
</tr>
<tr>
<td>High DBP</td>
<td>10 13 15 23 29</td>
<td>2.8 (2.3, 3.5)</td>
</tr>
<tr>
<td>BMI</td>
<td>4 7 15 36 62</td>
<td>14 (11, 18)</td>
</tr>
<tr>
<td>SF sum</td>
<td>5 8 15 39 54</td>
<td>11 (8, 13)</td>
</tr>
<tr>
<td>High risk factor summary BMI</td>
<td>4 7 15 36 62</td>
<td>14 (11, 18)</td>
</tr>
<tr>
<td>SF sum</td>
<td>5 8 15 39 54</td>
<td>11 (8, 13)</td>
</tr>
</tbody>
</table>

\(^1\) SBP, systolic blood pressure; DBP, diastolic blood pressure.

\(^2\) Adverse risk factor levels were defined as an adjusted (race, sex, age, and study) level ≥85th percentile or <15th percentile for HDL cholesterol.

\(^3\) Adiposity categories were based on adjusted levels of BMI or SF sum.

\(^4\) The proportion of children in the highest category of each adiposity index who had an adverse risk factor level divided by the comparable proportion among children in the lowest category of each adiposity index. The 95% CIs for the estimated prevalence ratios are shown in parentheses.

\(^5\) Values represent the proportion of children in the specified adiposity category who had an adverse risk factor level (positive predictive value).

Fewer longitudinal studies have compared the predictive abilities of BMI and estimates of body fatness, but some results suggest that there may be little difference in these measures. For example, BMI has been found to predict the development of type 2 diabetes as strongly as bioelectrical impedance estimates of body fatness (42). Several studies have also found that BMI is as predictive of cardiovascular disease as are most skinfold thicknesses (43–46). Although it is possible that body fatness may be a better predictor of disease than is BMI among some subgroups, results have been inconclusive. Among older (≥60 y) women, for example, both BMI and body fatness (estimated from bioelectrical impedance) were found to be inversely associated with total mortality (47).

Although BMI may convey most of the relevant information on fatness-related metabolic risk among children, the limitations of skinfold thicknesses should also be considered when interpreting our results. There is substantial variability in the measurement of skinfold thicknesses (48–50), with measurement errors increasing with the degree of adiposity (51). In addition, we focused on the sum of 2 skinfold thicknesses rather than combining skinfold thicknesses with body density to estimate body fatness (13, 15). Additional analyses, however, indicated that risk factor associations with estimates of body fatness based on skinfold-thickness equations (13) were almost identical to those for the SF sum. It should be realized, however, that another explanation for our results concerning BMI and SF sum is that lean body mass may be associated with adverse levels of some metabolic risk factors (52). For example, a higher prevalence of the metabolic syndrome has been found among adults with a high fat-free mass index (fat-free mass/height\(^2\)) (53), and increases in the fat-free mass index are associated positively with triglyceride concentrations and negatively with HDL-cholesterol concentrations (54). Despite the more accurate prediction of body fatness by skinfold thicknesses, our results indicate that levels of BMI are as strongly related to levels of lipids, fasting insulin, and blood pressure among children as are subscapular and triceps skinfold thicknesses. Because skinfold-thickness measurements require careful training of observers, our findings should be considered in the design and interpretation of studies that use anthropometric data. Although the limitations of our cross-sectional design should be considered, we found that skinfold thicknesses do not provide a more accurate assessment of metabolic risk than does BMI.

The authors’ responsibilities were as follows—DSF: data analyses, interpretation of the results, and writing of the manuscript; PTK, WHD, SRS, and GSB: interpretation of the results and editing of the manuscript; SRS and GSB: data collection; and GSB: principal investigator. None of the authors had a personal or financial conflict of interest.
REFERENCES


46. Kim J, Meade T, Haines A. Skinfold thickness, body mass index, and fatal coronary heart disease: 30 year follow up of the Northwick Park Heart Study. J Epidemiol Community Health 2006;60:275–9.

