Body mass index and waist circumference in midchildhood and adverse cardiovascular disease risk clustering in adolescence

Sarah P Garnett, Louise A Baur, Shubha Srinivasan, Jenny W Lee, and Chris T Cowell

ABSTRACT

Background: Body mass index (BMI) may not indicate the level of central adiposity associated with the clustering of cardiovascular disease (CVD) risk factors. Hence, it has been recommended that waist circumference be used as an alternative measure.

Objective: The objective was to investigate whether waist circumference in midchildhood is more effective at predicting cardiovascular disease risk clustering in adolescence than is BMI.

Design: Anthropometric measurements were made in 342 children aged 8 y. Seven years later, anthropometric measurements were made in 290 participants, and metabolic profiles were determined in 172 participants.

Results: At 15 y, between 9.4% and 11.0% of adolescents were defined as having CVD risk clustering. Children who were overweight or obese at 8 y of age were 7 times (odds ratio: 6.9; 95% CI: 2.5, 19.0; P < 0.001) as likely to have CVD risk clustering in adolescence than were their peers who were not overweight or obese. Those with an increased waist circumference at 8 y were 4 times (3.6; 1.0, 12.9; P = 0.061) as likely to have CVD risk clustering in adolescence than were children with a smaller waist circumference. Neither BMI nor waist circumference were predictive of CVD risk clustering if adiposity status was not included as a risk factor.

Conclusions: The association between measures of adiposity in midchildhood and later adverse CVD risk is a result of the tracking of adiposity status. Our results do not support the need to measure waist circumference in children, in addition to BMI, to identify those at increased risk of CVD risk factor clustering in adolescence.


KEY WORDS Body mass index, waist circumference, metabolic syndrome, longitudinal study, children

INTRODUCTION

The long-term health outcomes of children and adolescents with different amounts of total body fat are unknown because most large-scale studies of the effects of childhood obesity have not used measures of body fat, but rather proxies such as body mass index (BMI). The use of BMI to classify children and adolescents as overweight or obese is well established (1). The 2 most widely recommended indicators of overweight and obesity are 1) age- and sex-specific BMI values presented by the International Obesity Task Force (IOTF) that correspond to BMI values (in kg/m²) of 25 and 30 at 18 y (2) and 2) BMI for age and sex percentile reference data (eg, US CDC 2000 growth reference (3), with arbitrary cutoffs, often the 85th and 95th percentiles. Both definitions are strong predictors of metabolic complications, including a poor lipid profile and increased blood pressure and insulin resistance in young adulthood and the predictive capacity of both is similar (4).

However, BMI may not indicate the level of central adiposity, which is also associated with the clustering of cardiovascular disease (CVD) risk factors, including dyslipidemia, hypertension, and insulin resistance. Clusters of risk factors are fairly stable characteristics that tend to track from adolescence to adulthood, hence the early identification of children who are likely to develop an elevated risk profile is of interest (5). Waist circumference has been recommended as a means of identifying persons at risk of morbidity associated with central adiposity. For example, European adult men and women with a waist circumference of >102 cm and >88 cm, respectively, are considered to have a higher risk of obesity-related disorders than do those with smaller measurements (6).

Only one study to date, from the United States and based on cross-sectional data from the Bogalusa Heart Study, has published age- and sex-specific waist circumference cutoffs for children and adolescents on the basis of an adverse CVD risk factor profile (7). Yet, the long-term health outcomes for children with large waist circumferences are currently unknown.

The aim of this study was to determine whether waist circumference cutoffs in midchildhood are more effective at predicting CVD risk factor clustering in adolescence than are IOTF BMI cutoffs (2). Increased central adiposity was arbitrarily defined as more than the 91st percentile. This cutoff was chosen because it was previously used in studies in young people (8, 9) and is similar to the cutoffs used to define overweight on the basis of BMI (2).

From the Institute of Endocrinology and Diabetes, The Children’s Hospital at Westmead, Westmead, Australia (SPG, SS, JWL, and CTC), and the University of Sydney, Discipline of Paediatrics and Child Health, The Children’s Hospital at Westmead Clinical School, Westmead, Australia (SPG, LAB, SS, and CTC)

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3 Address reprint requests and correspondence to SP Garnett, Institute of Endocrinology and Diabetes, The Children’s Hospital at Westmead, Locked Bag 4001, Westmead, NSW 2145, Australia. E-mail: sarahg@chw.edu.au.

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TABLE 1
Definitions of cardiovascular disease (CVD) risk clustering

<table>
<thead>
<tr>
<th>Glucose</th>
<th>Triglycerol</th>
<th>HDL-C</th>
<th>LDL-C</th>
<th>BP</th>
<th>BMI</th>
<th>Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>mmol/L</td>
<td>mmol/L</td>
<td>mmol/L</td>
<td>mmol/L</td>
<td></td>
<td></td>
<td>pmol/L</td>
</tr>
<tr>
<td>Risk clustering 1:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥6.1</td>
<td>≥80th percentile</td>
<td>≤20th percentile</td>
<td>—</td>
<td>SBP ≥90th percentile for age, sex, and height</td>
<td>Overweight or obese</td>
<td>≥80th percentile</td>
</tr>
<tr>
<td>(male: 1.14; female: 0.90)</td>
<td>(male: 1.1; female: 1.3)</td>
<td>—</td>
<td>SPP, DBP, or both ≥90th percentile for age, sex, and height</td>
<td>(male: 112; female: 127)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Risk clustering 2:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥6.1</td>
<td>≥80th percentile</td>
<td>≤20th percentile</td>
<td>≥80th percentile</td>
<td>SPP</td>
<td>—</td>
<td>≥80th percentile</td>
</tr>
<tr>
<td>(male: 1.14; female: 0.90)</td>
<td>(male: 1.1; female: 1.3)</td>
<td>(male: 2.6; female: 2.8)</td>
<td>—</td>
<td>—</td>
<td>(male: 112; female: 127)</td>
<td></td>
</tr>
</tbody>
</table>

1 C, cholesterol; BP, blood pressure; SPP, systolic blood pressure; DBP, diastolic pressure.
2 Cutoffs were estimated from the current study population.
3 Cutoffs were based on the National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents 1996 (14).
4 Cutoffs were defined by the International Obesity Task Force (2).
5 Based on the definition used by Lambert et al (13).
6 Based on the definition used by Katzmarzyk et al (7).

SUBJECTS AND METHODS

Subjects

In 1996–1997, 436 healthy 7–8-yr-old children (n = 215 girls) and their parents were recruited for the study. The children were participants in the longitudinal Nepean Study, which was designed to investigate the effects of birth size, body size, and genes on blood pressure and bone mass. All were born at term at Nepean Hospital, in Western Sydney, between August 1989 and April 1990 and were part of a birth cohort whose details and selection criteria were previously published (10). The children were predominantly (>96%) of European descent. Between July 2004 and March 2005, the children were recontacted and 290 (67.2%) agreed to participate in the follow-up and 172 (39% of the original cohort) agreed to have a blood sample taken. The median time between baseline and follow-up was 7.3 (range: 5.9–8.5 y).

At baseline there was no significant difference in mean height (P = 0.473), weight (P = 0.929), BMI (P = 0.379), or waist circumference z scores (P = 0.539) between the young people who were followed up and those who were not. Neither was there any significant difference in the baseline in the number who were classified as overweight or obese defined by BMI (P = 0.554) or waist circumference (P = 0.636). Similarly, there were no significant differences in mean height, weight, BMI, or waist circumference z scores in children who gave blood compared with those who did not. Written informed consent was obtained from the participants’ parents, and both The Children’s Hospital at Westmead Ethics Committee and the Ethics Committee of the Wentworth Area Health Service approved the study.

Anthropometric measures

Height, weight, and waist circumference were measured at baseline and at follow-up with the use of standard techniques (11). Waist circumference was measured with a flexible steel tape at the level of the narrowest point between the lower costal border and the iliac crest. If there was no obvious narrowing, the measurement was taken at the midpoint between the 2 landmarks (11). BMI was calculated as weight/height2 (kg/m2). z scores from age- and sex-specific reference values were calculated for height (3), weight (3), BMI (3), and waist circumference (12). The IOTF BMI criteria were used to define overweight and obesity (total adiposity) (2). In the absence of a recognized definition of increased waist circumference in young people, increased central adiposity was arbitrarily defined as more than the 91st percentile. This cutoff was chosen because it has been previously used in studies in young people (8, 9) and is similar to the cutoffs used to define overweight based on BMI (2).

To examine the utility of using both BMI and waist circumference cutoffs in identifying CVD risk clustering, children were categorized into 4 groups: acceptable BMI with an acceptable waist circumference, an acceptable BMI with an increased waist circumference, overweight or obese with an acceptable waist circumference, and overweight and obese with an increased waist circumference (4).

Biochemistry

Morning blood samples were obtained after an overnight fast by standard venipuncture technique from the 172 children at follow-up. Lipid and glucose profiles were measured on a Roche Modular (F Hoffmann-La Roche Ltd, Basel, Switzerland). Glucose concentrations were measured by using the hexokinase method. Total cholesterol, HDL-cholesterol, and triglycerol concentrations were analyzed by using standard enzymatic colorimetric procedures. HDL cholesterol was measured after polyethylene glycol precipitation of other lipoproteins. LDL cholesterol was calculated as total cholesterol — (HDL cholesterol – triglycerol/2.2). Serum samples for insulin were stored at −80 °C until assayed by radioimmunoassay with Linco’s ultrasensitive human insulin kit (Linco Research Inc, St Charles, MO). One boy did not have enough blood collected for insulin analysis.

Blood pressure

An automated blood pressure monitor (Dinamap XL 9301; Johnson and Johnson Medical Inc, Arlington, TX) was used for all blood pressure measurements at follow-up. All measurements were made on the right arm with the arm supported on a pillow. The readings were performed in an air-conditioned hospital environment, and the children were encouraged to sit quietly for 6 min before the measurements began. Three readings were taken, and the mean of the last 2 measurements was used in the analysis. Twenty participants did not have their blood pressure measured. Of these 20 participants, 7 gave blood but were not included in the CVD risk clustering analysis because of the missing data.
populations (7, 13) and detailed in Table 1. CVD risk clustering 1 included a measure of adiposity and was defined as ≥3 of the following: fasting glucose ≥6.1 mmol/L; triacylglycerols ≥80th percentile; HDL cholesterol ≤20th percentile; insulin ≥80th percentile; systolic blood pressure (SBP) ≥90th percentile for age, sex, and height (7); and overweight or obese as defined by the IOTF (13).

CVD risk clustering 2 was defined as ≥3 of the following: glucose ≥6.1 mmol/L; triacylglycerols ≥80th percentile; HDL cholesterol ≤20th percentile; LDL cholesterol ≥80th percentile; insulin ≥80th percentile, or either an SBP or diastolic blood pressure (DBP) ≥90th percentile for age, sex, and height (7). Percentages for cutoffs used to define risk factors (triacylglycerols, HDL cholesterol, LDL cholesterol, and insulin) were estimated from our study population. Previously published cutoffs were used for glucose (7, 13) and SBP and DBP (14).

Statistical analysis

Data were analyzed by using the Statistical Package for Social Sciences, version 13.0 (SPSS Inc, Chicago, IL). Differences between anthropometric characteristics assessed at ages 8 and 15 y were assessed by paired t test. A chi-square test was used as a measure of association between categorical variables, and Fisher’s exact test was used as a measure of significance. To quantify the risk associated with individual explanatory variables, odds ratios were calculated. Logistic regression models were built to explore the relation between CVD risk factor clustering, overweight or obese or increased waist circumference, sex (boys = 1, girls = 2), and time between visits (5.9–7.3 y = 0, 7.3–8.5 y = 1).

RESULTS

Change in indicators of growth and adiposity

Between 8 y (median: 7.54; range: 7.03–8.97) and 15 y (14.91; range: 14.30–15.51), there was a significant (P ≤ 0.001) increase in the mean (± SD) z scores for weight (0.25 ± 0.67), BMI (0.22 ± 0.67), and waist circumference (0.51 ± 0.89), but no change in the mean z score for height (0.03 ± 0.57; P = 0.32) (Figure 1). Similar results were obtained when boys and girls were examined separately.

Prevalence of overweight and obesity

At both 8 and 15 y, BMI cutoffs identified a greater number of children as overweight or obese than did waist circumference cutoffs (Table 2). At 15 y, 31.7% of adolescents were defined as overweight or obese on the basis of BMI and 20.0% had increased central adiposity; 78.9% of children who were overweight or obese on the basis of BMI at 8 y were still overweight and obese at 15 y (odds ratio: 14.8; 95% CI: 7.3, 30.3), and 69.2% of children who had increased central adiposity at 8 y continued to be so at 15 y (odds ratio: 12.2; 95% CI: 5.1, 30.9). Approximately 80% of all children who had either an acceptable BMI or waist circumference at 8 y still had an acceptable BMI or waist circumference at 15 y.

CVD risk clustering

Anthropometric, blood pressure, and metabolic data are shown in Table 3. More children were identified as having at least one risk factor when the markers considered included a measure of adiposity: 40% (CVD risk clustering 1) compared with 30% (CVD risk clustering 2) (Figure 2, A and B). One boy had 5 risk factors; he had a BMI $z$ score of 2.3 at both 8 and 15 y and waist circumference $z$ scores of 2.0 and 2.8 at 8 and 15 y, respectively. The proportion of adolescents with CVD risk clustering who were overweight or obese or had an increased waist circumference at 8 y is shown in Table 4.

CVD risk clustering 1

Similar numbers of boys and girls at 15 y were identified as having CVD risk clustering: 10 (10.9%) and 9 (11.3%), respectively. As shown in Table 5, children who had CVD risk clustering at 15 y were heavier (mean difference: $z$ score = 0.69, $P =$...
TABLE 3
Blood pressure and metabolic measures at 15 y of age and the distribution of participants with values above defined cutoffs

<table>
<thead>
<tr>
<th></th>
<th>Boys</th>
<th>Girls</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of subjects</td>
<td>Median (range)</td>
<td>Subjects with values above cutoffs</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>92</td>
<td>4.8 (4.0–5.9)</td>
<td>0</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>92</td>
<td>0.8 (0.3–3.5)</td>
<td>18 (19.6)</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>92</td>
<td>1.3 (0.8–2.2)</td>
<td>27 (29.3)</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>92</td>
<td>2.3 (1.3–3.5)</td>
<td>23 (25.0)</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>130</td>
<td>111 (86–149)</td>
<td>18 (13.8)</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>130</td>
<td>55 (39–85)</td>
<td>2 (1.5)</td>
</tr>
<tr>
<td>Insulin (pmol/L)</td>
<td>91</td>
<td>87 (32–369)</td>
<td>18 (19.8)</td>
</tr>
</tbody>
</table>

The cutoffs used were as follows: glucose, ≥6.1 mmol/L; triglycerides, ≥80th percentile (boys: 1.14 mmol/L; girls: 0.9 mmol/L); HDL cholesterol (C), ≥20th percentile (boys: 1.1 mmol/L; girls: 1.3 mmol/L); LDL-C, ≥80th percentile (boys: 2.6 mmol/L; girls: 2.8 mmol/L); systolic blood pressure (SBP) or diastolic blood pressure (DBP), ≥90th percentile for age, sex, and height (7); insulin, ≥80th percentile (boys: 112 pmol/L; girls: 127 pmol/L).

0.002), had a higher waist circumference (z score = 0.82, P = 0.001), and had a higher BMI (z score = 0.63, P = 0.006) at 8 y of age than did those who were not at risk. However, there was no significant difference in height at 8 y between the 2 groups (z score = 0.55, P = 0.129). Children who had CVD risk clustering continued to be heavier (mean difference: z score = 1.15, P < 0.001), have an increased waist circumference (z score = 1.55, P < 0.001), and a higher BMI (z score = 1.08, P < 0.001) at 15 y.

Children who were overweight or obese at 8 y were more likely (odds ratio: 6.9; 95% CI: 2.5, 19.04; P < 0.001) to have CVD risk clustering 1 at 15 y than were those who had an acceptable BMI. Those who had an increased waist circumference were also more likely to have increased CVD risk clustering 1; however, the odds ratio was lower than that for BMI (3.6; 1.0, 12.9; P = 0.061). Logistic regression models were built to adjust for sex and time between baseline and follow-up. Neither variable was significant in the equations (data not shown).

When children were categorized into groups on the basis of BMI and waist circumference cutoffs at 8 y, there was a significant difference (P < 0.001) in BMI z score between groups at 8 y of age. The mean (±SD) BMI z scores for children with an acceptable BMI and waist circumference, overweight or obese with an acceptable waist circumference, or overweight or obese with an increased waist circumference were −0.13 ± 0.79, 1.36 ± 0.20, and 2.00 ± 0.38, respectively. No children had an acceptable BMI and an increased waist circumference. However, there was no difference (P = 1.0) in the percentage of children identified as having CVD risk factor clustering 1 between those who were overweight or obese and had an acceptable waist circumference (33.3%) and those who were overweight or obese and had an increased waist circumference (28.6%).

**CVD risk clustering 2**

Ten (10.9%) boys and 6 (7.5%) girls at 15 y were identified as having CVD risk clustering on the basis of this definition. There were no significant differences in weight (P = 0.412), waist circumference (P = 0.221, BMI (P = 0.431), or height (P = 0.467) z scores at 8 y between those with CVD risk clustering 2 and those without CVD risk clustering. However, at 15 y, those...
Distribution of adolescents with cardiovascular disease (CVD) risk clustering 1 and 2 who were overweight or obese or had an increased waist circumference (total n = 164)

<table>
<thead>
<tr>
<th>CVD risk clustering in nonexposed group</th>
<th>CVD risk clustering in exposed group</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>CVD risk clustering 1 at 15 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight or obese at 8 y</td>
<td>8 (6.2)</td>
<td>11 (31.4)</td>
</tr>
<tr>
<td>Increased waist circumference at 8 y</td>
<td>16 (10.0)</td>
<td>4 (28.6)</td>
</tr>
<tr>
<td>CVD risk clustering 2 at 15 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight or obese at 8 y</td>
<td>11 (8.5)</td>
<td>5 (14.3)</td>
</tr>
<tr>
<td>Increased waist circumference at 8 y</td>
<td>15 (10.0)</td>
<td>1 (7.1)</td>
</tr>
</tbody>
</table>

* Fisher’s exact test.

1 Defined as ≥3 of the following: fasting glucose, ≥6.1 mmol/L; triacylglycerols, ≥80th percentile (boys: 1.14 mmol/L; girls: 0.9 mmol/L); HDL cholesterol (C), ≤20th percentile (boys: 1.1 mmol/L; girls: 1.3 mmol/L); insulin, ≥80th percentile (boys: 112 pmol/L; girls: 127 pmol/L); systolic blood pressure (SBP), ≥90th percentile for age, sex, and height (7) and overweight or obese as defined by the International Obesity Task Force (13).

2 Defined as ≥3 of the following: fasting glucose, ≥6.1 mmol/L; triacylglycerols, ≥80th percentile (boys: 1.14 mmol/L; girls: 0.9 mmol/L); HDL-C, ≤20th percentile (boys: 1.1 mmol/L; girls: 1.3 mmol/L); LDL-C, ≥80th percentile (boys: 2.6 mmol/L; girls: 2.8 mmol/L); insulin, ≥80th percentile (boys: 112 pmol/L; girls: 127 pmol/L); SBP or diastolic blood pressure, ≥90th percentile for age, sex, and height (7).

Identified as having CVD risk clustering 2 were heavier (mean difference: z score = 0.87, P < 0.001), had an increased waist circumference (z score = 1.17, P < 0.001), and had a higher BMI (z score = 0.84, P = 0.001) than did those who did not (Table 5). There was no significant difference in height (z score = 0.28, P = 0.343). Neither BMI (odds ratio: 1.8; 95% CI: 0.6, 5.5; P = 0.338) nor waist circumference (odds ratio: 0.7; 95% CI: 0.1, 5.7; P = 1.0) at 8 y of age was predictive of CVD risk clustering 2 at 15 y. The results were unchanged after adjustment for sex and time (data not shown).

Similar to CVD risk clustering 1, when children were categorized into groups on the basis of BMI and waist circumference cutoffs, there was no difference (P = 0.622) in the percentage of children identified as having CVD risk clustering 2 between those who were overweight or obese and had an acceptable waist circumference and those who were overweight and obese and had increased waist circumference.

**DISCUSSION**

The results of this study do not support the use of waist circumference measurements in midchildhood, in addition to BMI, to identify children at increased risk of later CVD risk factor clustering. BMI is a well-established measure of relative fatness in childhood and adolescence and requires measurements of height and weight, both of which are quick routine measures. In contrast, waist circumference measurements involve the location of bony landmarks (lower costal boarder and iliac crest), removal of clothing, and careful placement of the tape measure to avoid fat rolls that can be uncomfortable, awkward, or embarrassing for...
the overweight or obese child. The results of our study are consistent with a small number of pediatric cross-sectional studies (4, 15, 16). We are not aware of any other longitudinal studies that have examined the effects of an increased waist circumference in childhood on CVD risk clustering in adolescence.

We surmise that the stronger association between BMI in midchildhood than between waist circumference and later adverse CVD risk clustering was a result of the tracking of BMI status from childhood to adolescence; 78.9% of children who were overweight or obese (BMI) at 8 y were still overweight and obese at 15 y. Increased waist circumference was less persistent than was BMI; 69.2% of the children who had an increased waist circumference at 8 y continued to have such at 15 y. In addition, the association between anthropometric measures in midchildhood and CVD risk clustering in adolescence was not significant when the definition did not include adiposity as a risk factor. Tracking of BMI is well documented, and it is recognized as one of the most significant long-term consequences of childhood obesity (17–19). The association between childhood obesity and traditional atherogenic profiles in adulthood is also well established (20–23).

The results presented are in contrast with the evidence from adult and some pediatric studies, which indicate that fat distribution, as measured by waist circumference, has a stronger relation with CVD risk factors than does total adiposity (24–26). Waist circumference is considered to be a good predictor of intraabdominal fat, explaining up to 64% of its variance in children aged 7–16 y (27). However, whether the accumulation of intraabdominal fat confers an excess metabolic risk, including insulin resistance, in children remains controversial (28, 29).

Several studies have found that abdominal subcutaneous adipose tissue, and not intraabdominal adipose tissue, is independently associated with insulin resistance or that they are both equally correlated (30–32). In nonobese children, total fat or subcutaneous adipose tissue may be a primary determinant of metabolic complications (30, 33). Intraabdominal adipose tissue may only be positively associated with insulin action and lipid risk factors among obese children and adolescents (34–36).

We also examined the value of using both BMI and waist circumference cutoffs in identifying CVD risk clustering in children. Recently published results from the Bogalusa Heart Study indicate that when waist circumference was considered with BMI (as categorical variables), children and adolescents with large waist circumferences were more likely to have elevated CVD risk factors than were those with a smaller waist circumference, within a given BMI category (4). Contrary to these findings, we found no benefit of using BMI and waist circumference cutoffs together in identifying CVD risk clustering in adolescents. It is not clear why the results differ, but it may be a consequence of the smaller number of children in our study, the different ethnicities of the children, or the use of different waist circumference cutoffs.

Interpretation of the data presented is dependent on the definition of CVD risk clustering and anthropometric cutoffs. In the absence of a recognized definition to define increased waist circumference in young people, increased central adiposity was arbitrarily defined as >91st percentile of Australian age- and sex-specific reference data. This cutoff was chosen because it was previously used in young people and is similar to the cutoffs used to define overweight based on BMI (8, 9). Nevertheless, BMI identified more people as overweight or obese (19.7%) than did waist circumference (9.0%). This disparity in identification of those at risk may have led to a reduced power of waist circumference, and, in part, explain the stronger relation between BMI and metabolic risk.

However, when the analysis was repeated using age- and sex-specific waist circumference cutoffs based on data from the Bogalusa Heart Study (7), which were approximately equivalent to the 60th percentile of Australian age and sex reference data, 36.2% of young people were defined as having increased central adiposity, and our findings were similar. Those with an increased waist circumference at 8 y were still less likely to have CVD risk clustering 1 (odds ratio: 4.4; 95% CI: 1.6, 12.2) than were young people who were overweight or obese (odds ratio: 6.9; 95% CI: 2.5, 19.0). Neither increased BMI nor increased waist circumference, irrespective of definition, at 8 y was predictive of CVD risk clustering 2 at 15 y.

Currently, there is no accepted definition of CVD risk clustering or the metabolic syndrome in children and adolescents, and agreement between classifying children using existing published definitions is poor (37, 38). For many reasons, including the effects of hormone changes in puberty on insulin sensitivity and lipid profile and the complexity of diagnosing insulin resistance, defining cutoffs, and hence CVD risk clustering and the metabolic syndrome, will be difficult in this age group (34).

In the current study we used 2 definitions, similar to those previously used in pediatric populations (7, 13). The major difference between the definitions used was that one (CVD risk clustering 1) included a measure of adiposity. Nevertheless, both definitions predicted a similar number of children at risk: 11.0% compared with 9.4%. Although it is difficult to compare prevalence between populations, it is interesting to note that the prevalence of CVD risk clustering in our study is comparable with that reported in 16-y-olds participating in the Quebec Child and Adolescent Health and Social Survey: 15.2% and 12.4% in boys and girls, respectively, with the use of a similar definition (13).

Neither BMI nor waist circumference was able to identify all children at risk: 6.2% of children who had an acceptable BMI at 8 y and 10.0% of children who had an acceptable waist circumference at 8 y were classified as having CVD risk factor clustering 1 at 15 y. It is interesting to note that these children had a mean increase in BMI z score between 8 and 15 y of 1.19 ± 0.7 (weight gain: 47 ± 17 kg) and a mean increase in waist circumference z score of 1.42 ± 0.28 (27 ± 10 cm). In comparison, children who had an acceptable BMI at 8 y and no CVD risk clustering had mean increases in BMI and waist circumference z scores of 0.29 ± 0.3 (33.3 ± 8.0 kg) and 0.49 ± 0.77 (16 ± 5 cm), respectively.

A limitation of the present study was the number of adolescents who were followed up. There were no significant differences in anthropometric measurements between those who were followed up and those who were not followed up or between those who gave blood compared with those who did not give blood; hence, we believe that these factors were unlikely to have influenced the findings of the study. Nevertheless, wide 95% CIs for the odds ratios for both overweight and obesity, and for increased waist circumference, in predicting CVD risk clustering 1 were noted; this may be a result of the sample size.

In conclusion, the association between measures of adiposity in midchildhood and later adverse CVD risk is a result of the tracking of adiposity status. Our results do not support the need to measure waist circumference in midchildhood, in addition to
BMI, to identify children at increased risk of CVD risk factor clustering in adolescence.

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The authors’ responsibilities were as follows—SPG: participated in all aspects of this study and primarily responsible for drafting the manuscript; LAB: participated in the study design and supervised the study implementation, data interpretation, and preparation of the manuscript; SS: participated in data interpretation and preparation of the manuscript; JWL: responsible for the management and biochemical analysis of blood samples; CTC: participated in the study design and supervised the study implementation, data interpretation, and preparation of the manuscript. None of the authors had any conflicts of interest.

REFERENCES