Secular Trends in BMI and Blood Pressure Among Children and Adolescents: The Bogalusa Heart Study

**WHAT'S KNOWN ON THIS SUBJECT:** Although obesity is correlated with levels of systolic and diastolic blood pressure, there is little evidence if the increases in obesity over the last 40 years have resulted in increased blood pressure levels.

**WHAT THIS STUDY ADDS:** Despite increases in obesity in Bogalusa, Louisiana between 1974 and 1993, there was no increase in systolic or diastolic blood pressure levels. It should not be assumed that trends in high blood pressure have paralleled those for obesity.

**OBJECTIVE:** The prevalence of obesity among children and adolescents increased by almost threefold from the 1970s to 2000. We examined whether these secular changes in BMI were accompanied by increases in blood pressure levels.

**METHODS:** A total of 24,092 examinations were conducted among 11,478 children and adolescents (aged 5–17 years) from 1974 to 1993 in the Bogalusa Heart Study (Louisiana).

**RESULTS:** The prevalence of obesity increased from 6% to 17% during this period. In contrast, only small changes were observed in levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP), and neither mean nor high (based on the 90th percentile from the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents) levels increased over the 20-year period. Within each race–gender group, mean levels of SBP did not change, whereas mean levels of DBP decreased by 2 mm Hg ($P < .001$ for trend). Levels of BMI were positively associated with levels of SBP and DBP within each of the 7 examinations, and controlling for BMI (along with other covariates) indicated that only ~60% as many children as expected had high levels of blood pressure in 1993.

**CONCLUSIONS:** Our finding that levels of DBP and SBP among children in this large sample did not increase despite the increases that were seen in obesity indicates that changes in blood pressure levels in a population do not necessarily parallel changes in obesity. Additional study of the potential characteristics that have ameliorated the expected increase in high blood pressure could lead to further reductions in risk. *Pediatrics* 2012;130:e159–e166
The high prevalence of overweight and obesity, based on levels of BMI, among children and adolescents in the United States continues to be of concern. Compared with thinner children, obese children are much more likely to become obese adults1 and to have adverse levels of insulin, lipids, and blood pressure.2 Secular trends in obesity have been well documented among children in the United States and in other countries,3,4 but the increases have slowed since 1999.5

Because of the association between obesity and blood pressure,5,6 it might be expected that levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP) among children would have also increased over the last several decades. Although some authors have reported increases in mean (or high) levels of blood pressure among children,7–9 several studies have found that, in at least some subgroups, blood pressure trends have not paralleled those for BMI.3–11 An increase (from 5.5% to 8.5%) in the prevalence of obesity among children in Seychelles from 1998 to 2006, for example, was accompanied by a mean 3–mm Hg decrease in SBP.12 Although methodologic differences in blood pressure measurements over time should be considered, these concerns also apply to the small increases (≤3 mm Hg) that have been reported by some investigators.7,8

We used data from 24,092 examinations of 5- to 17-year-olds in the Bogalusa Heart Study to determine whether there were secular increases in SBP and DBP levels from 1973–1974 to 1992–1994. During this ~20-year period, 7 cross-sectional examinations of schoolchildren were conducted, and the prevalence of obesity increased substantially.12,14

METHODS

Study Population

Bogalusa is a biracial (one-third black) community in Washington Parish, Louisiana. The Bogalusa Heart Study has examined the natural history of cardiovascular disease,15 and 7 cross-sectional studies of schoolchildren were conducted between 1973–197416 and 1992–1994. On average, each study examined ~3500 children. For simplicity, we refer to each study by using the year in which most of the examinations were conducted. The first (1974) study examined 5- to 14-year-olds, and 5- to 17-year-olds were examined in subsequent studies.

Eighty-nine participants were excluded who were missing information for weight or height and another 83 because information on SBP or DBP was missing. This resulted in 24,092 examinations (from 11,478 children) available for analysis. Written, informed consent was obtained, and study protocols were approved by human subjects review committees.

Examinations and Laboratory Determinations

Height was measured to the nearest 0.1 cm and weight to the nearest 0.1 kg; BMI was calculated as kilograms divided by meters squared. BMI-for-age z (SD) scores and percentiles were calculated from the Centers for Disease Control and Prevention (CDC) growth charts.17,18 Obesity was defined as a BMI-for-age ≥95th percentile of the CDC reference population or a BMI ≥30.

As previously described,15,19 right arm, sitting SBP and DBP levels were measured 3 times each by 2 trained observers by using a mercury sphygmomanometer. The mean of the 6 measurements was used in all analyses. Attempts were made to reduce the influence of the emotional state of the child on the measurements.16 Cuff sizes (child, adult, obese) were selected according to a protocol based on the circumference and length of the upper arm, by using a bladder width as large as possible without obstructing the stethoscope.16,20 Blood pressure protocols did not change over the 20-year period.

As recommended by the 197721 and 198722 Task Force reports on blood pressure in children, our focus was on the fourth Korotkoff sound to characterize DBP. Previous studies of DBP from the Bogalusa Heart Study have been based on this definition.2,15,16,19,23 There was no association between the absence of the fourth sound and age (r = −0.01). Although the fifth Korotkoff sound was also measured, ~8% (n = 1824) of these measurements were <30 mm Hg, and 29 measurements were recorded as 0 mm Hg. Among these 1824 measurements, the mean fifth Korotkoff sound was 27 mm Hg lower than the fourth sound. The true intraarterial diastolic pressure seems to be between the fourth and fifth sounds.24

To account for differences in blood pressure levels according to gender, age, and height, blood pressures were converted into z scores by using formulas in the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents (hereafter referred to as the Fourth Report).25 Approximately 16,000 SBP measurements from the Bogalusa Heart Study were included in this report, which recommended that hypertension be defined as an SBP or DBP level ≥95th percentile for a child’s gender, age, and height on at least 3 occasions; levels between the 90th and 94th percentiles or ≥120/80 mm Hg were considered to be prehypertensive.

Because only 2.1% of the children in Bogalusa had an SBP ≥95th percentile and only 1.5% had a DBP ≥95th percentile (based on the fourth sound), we focus on levels of SBP and DBP that are ≥90th percentile of the Fourth Report.25 These blood pressures were referred to as “high.” Additional analyses that focused on the 95th percentile yielded similar results. Some analyses also examined a fifth Korotkoff sound above the 90th percentile, but only 106 (0.4%) children had a high fifth sound.
Statistical Analyses

Analyses were performed with the statistical language R. Characteristics of the sample were summarized within each of the 7 examinations, and the statistical significance of changes in levels of SBP and DBP (and the prevalence of high levels) over time was assessed in regression models that included study period as a categorical predictor. Blood pressure levels did not vary linearly over time, and study year was therefore categorized into 3 periods: 1974–1977 (first 2 examinations), 1979–1985 (3 examinations), or 1988–1993 (2 examinations).

Because levels of blood pressure among children vary by gender, race, age, and height, we controlled for these characteristics by using either: (1) $z$ scores calculated from equations in the Fourth Report; or (2) by including these characteristics in regression models. Continuous variables (age, height, and BMI) were modeled by using restricted cubic splines to allow for nonlinearity in the associations.

Because most (56%) of the children participated in 2 or more examinations, estimates of variability needed to account for the within-subject correlation. We accounted for this nonindependence by using the “bootcov” function in the rms package for the statistical language R. This method calculates SEs by treating each child as a cluster during bootstrap resampling, and we took 200 samples (with replacement) of the data. When the number of clusters is $>5$, multilevel regression and the resampling of clusters perform equally well in the estimation of SEs in clustered data. Because of the large sample size, $P < .001$ was used to indicate statistical significance in all analyses.

The number of children in each examination was also estimated who would have had high blood pressure if the 1974–1977 prevalence rates within 6 BMI categories had remained constant over time. This standardized total was obtained by multiplying the 1974–1977 prevalence rates by the number of children in each BMI in the succeeding years.

RESULTS

Table 1 shows mean levels of various characteristics in the 7 examinations among boys and girls. The proportion of black children increased from 37% to 45% over the 2 decades largely because of increases in the last 2 (1988 and 1993) studies, and the mean BMI $z$ score increased by 0.5 SD over the 20-year period. There were also small increases in height. In contrast, SBP and DBP $z$ scores, and the prevalence of high blood pressure, were no higher in 1993 than in 1974. Among boys, for example, mean SBP $z$ scores were −0.28 in both 1974 and 1993, and mean DBP $z$ scores were 0.15 in 1974 and −0.02 in 1993. The prevalence of a high fifth Korotkoff sound was slightly higher in 1993 than in 1974, but only 14 (of 3105) subjects in 1993 had a fifth sound that was ≥90th percentile.

Figure 1 shows the prevalence of obesity and high blood pressure over the 20-year period within race–gender groups. Although the increase in obesity was fairly linear over this period, trends in blood pressure were weaker and inconsistent. For example, the prevalence of high blood pressure among white and black girls reached a maximum (∼10%) in the early 1980s, and then decreased over the last 3 examinations. In all 4 groups, the prevalence of high blood pressure was slightly lower in 1993 than in 1974.

The prevalence of obesity and blood pressure levels are shown for 3 time periods in Table 2. Within each race–gender group, increases in obesity from the first (1974–1977) to the second (1979–1985) periods were accompanied by increases in SBP, but not DBP, levels. However, the larger obesity increases that occurred between the second and third periods were accompanied by 2–3-mm Hg decreases in both SBP and DBP. From 1979–1985 through 1988–1993, for example, the prevalence of obesity among black girls increased from 10% to 15%, but the prevalence of high blood pressure decreased from 11% to 6%. None of the SBP differences between the first and last time periods were statistically significant; levels of DBP were consistently lower ($P < .001$) in 1988–1993 than in 1974–1977.

We then examined whether the blood pressure differences across these 3 periods were independent of age, BMI, and height. Within each race–gender group, predicted levels of SBP (Fig 2) and DBP (Fig 3) are shown according to BMI (x-axis), with the 3 lines representing blood pressure levels in the 3 time periods. BMI, height, and age were modeled by using cubic splines to allow for nonlinearity. Within each time period, BMI was positively related to SBP and DBP levels.

Controlling for age, height, and BMI did not substantially alter the differences in levels of SBP over the 3 time periods (Fig 2). Independently of the covariates (including race and gender), mean SBP levels increased slightly (∼1.5 mm Hg) between 1974–1977 and 1979–1983, and subsequently decreased by ∼2.5 mm Hg through 1988–1993 ($P < .001$ for differences in SBP across the 3 periods). Although the differences between time periods varied somewhat by BMI ($P < .001$ for interaction), predicted SBP levels in 1988–1993 were lower than those in 1974–1977 at all levels of BMI.

Levels of DBP also varied ($P < .001$) across studies independently of BMI and other covariates (Fig 3). There were differences according to BMI level ($P < .001$ for interaction), but among boys, predicted DBP levels generally
TABLE 1  Mean Levels of Various Characteristics According to Age Group and Examination

<table>
<thead>
<tr>
<th>Year</th>
<th>n</th>
<th>Black, %</th>
<th>Age, y</th>
<th>BMI</th>
<th>BMI z Score</th>
<th>Height z Score</th>
<th>SBP, mm Hg</th>
<th>SBP z Score</th>
<th>DBP</th>
<th>DBP z Score</th>
<th>High Blood Pressure, %</th>
<th>High Fifth Sound, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boys</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1974</td>
<td>1821</td>
<td>37</td>
<td>10.3 ± 3</td>
<td>17.5 ± 3</td>
<td>−0.08</td>
<td>−0.13</td>
<td>100 ± 10</td>
<td>−0.28</td>
<td>62 ± 8</td>
<td>0.15</td>
<td>5.8</td>
<td>0</td>
</tr>
<tr>
<td>1977</td>
<td>2087</td>
<td>35</td>
<td>11.6 ± 4</td>
<td>18.7 ± 4</td>
<td>0.04</td>
<td>−0.09</td>
<td>102 ± 11</td>
<td>−0.38</td>
<td>62 ± 10</td>
<td>−0.01</td>
<td>4.7</td>
<td>0.2</td>
</tr>
<tr>
<td>1979</td>
<td>1828</td>
<td>36</td>
<td>11.3 ± 4</td>
<td>18.6 ± 4</td>
<td>0.11</td>
<td>−0.10</td>
<td>104 ± 11</td>
<td>−0.17</td>
<td>65 ± 8</td>
<td>0.16</td>
<td>7.2</td>
<td>0.3</td>
</tr>
<tr>
<td>1982</td>
<td>1684</td>
<td>36</td>
<td>11.2 ± 4</td>
<td>18.7 ± 4</td>
<td>0.17</td>
<td>−0.05</td>
<td>104 ± 11</td>
<td>−0.16</td>
<td>65 ± 9</td>
<td>0.09</td>
<td>5.4</td>
<td>0.7</td>
</tr>
<tr>
<td>1985</td>
<td>1629</td>
<td>36</td>
<td>11.3 ± 4</td>
<td>19.0 ± 4</td>
<td>0.18</td>
<td>0.02</td>
<td>104 ± 11</td>
<td>−0.23</td>
<td>60 ± 9</td>
<td>−0.09</td>
<td>5.0</td>
<td>0.2</td>
</tr>
<tr>
<td>1988</td>
<td>1638</td>
<td>40</td>
<td>11.0 ± 3</td>
<td>19.1 ± 4</td>
<td>0.32</td>
<td>0</td>
<td>101 ± 11</td>
<td>−0.41</td>
<td>59 ± 10</td>
<td>−0.23</td>
<td>3.5</td>
<td>0.2</td>
</tr>
<tr>
<td>1993</td>
<td>1521</td>
<td>44</td>
<td>11.4 ± 3</td>
<td>20.0 ± 5</td>
<td>0.47</td>
<td>0.12</td>
<td>103 ± 10</td>
<td>−0.28</td>
<td>62 ± 9</td>
<td>−0.02</td>
<td>4.1</td>
<td>0.2</td>
</tr>
</tbody>
</table>

| Girls |     |          |        |       |             |               |            |             |     |             |                        |                    |
| 1974 | 1677 | 37       | 10.3 ± 3 | 17.8 ± 4 | −0.04 | −0.03 | 100 ± 10 | −0.25 | 65 ± 8 | 0.17 | 8.1 | 0 |
| 1977 | 1689 | 36       | 11.5 ± 4 | 18.7 ± 4 | 0.04 | −0.10 | 101 ± 11 | −0.31 | 62 ± 10 | 0.04 | 7.3 | 0.2 |
| 1979 | 1747 | 37       | 11.1 ± 4 | 18.5 ± 4 | 0.09 | −0.10 | 103 ± 11 | −0.05 | 64 ± 9 | 0.24 | 10.0 | 0.5 |
| 1982 | 1698 | 38       | 11.1 ± 4 | 18.8 ± 4 | 0.14 | −0.05 | 103 ± 11 | −0.05 | 64 ± 9 | 0.26 | 10.8 | 1.4 |
| 1985 | 1623 | 38       | 11.5 ± 4 | 19.5 ± 5 | 0.24 | 0.05 | 103 ± 11 | −0.14 | 63 ± 9 | 0.13 | 8.7 | 1.2 |
| 1988 | 1575 | 41       | 11.0 ± 3 | 19.6 ± 5 | 0.35 | −0.01 | 100 ± 10 | −0.32 | 60 ± 10 | −0.15 | 5.0 | 0.4 |
| 1993 | 1584 | 45       | 11.4 ± 3 | 20.2 ± 5 | 0.42 | 0.12 | 102 ± 10 | −0.28 | 62 ± 9 | 0.03 | 5.8 | 0.7 |

These SDs do not account for the within-person clustering of levels and therefore underestimate the variability.

* Estimates of the SDs of BMI-for-age and other z scores were close to 1.0.

FIGURE 1
Prevalence of obesity and high blood pressure over time according to race–gender group. High blood pressure was defined as an SBP or DBP level that was ≥90th percentile in the Fourth Report.25

decreased in a stepwise manner over the 3 periods. Among girls, in contrast, DBP tended to increase slightly from 1974–1977 to 1979–1983 (whites) or showed no change (blacks). However, at all levels of BMI, race, and gender, DBP levels were lower in 1988–1993 than in 1974–1977.

Table 3 shows the prevalence of high blood pressure levels within BMI categories in each examination. In 1974–1977, the prevalence of high blood pressure varied from 4% to 24% across the 5 BMI-for-age categories. Within each BMI category, however, the prevalence of high blood pressure levels decreased across examinations, and this was most evident at high BMI levels. Among children with a BMI ≤50th percentile, the prevalence of high blood pressure decreased from 4% to 3% through 1993, whereas the prevalence decreased from 24% to 13% among children who had a BMI ≥97th percentile.

Based on the prevalence of high blood pressure levels in each BMI category in 1974–1977 and the sample sizes of the BMI categories in subsequent examinations, the number of children in each examination were calculated who would have been expected to have had high blood pressure. These calculations indicated that 261 children in 1993 were expected (compared with 157 observed) to have had high blood pressure if BMI-specific prevalence rates had not changed. Additional standardization for race, gender, age, and height altered these expected numbers only slightly.

**DISCUSSION**

Relatively few studies have examined whether secular trends in blood pressure levels among children have paralleled trends in obesity. Our results indicate that despite the large increase in obesity in Bogalusa from 1974 to 1993, neither mean nor high (≥90th percentile of the Fourth Report) levels of blood pressure increased over this period. The blood pressure changes that we observed were small, but the most consistent findings were a small (2–3 mm Hg) decrease in DBP levels and a 30% reduction in the prevalence of high blood pressure levels. The contrasting pattern of BMI increases accompanied by decreases in blood pressure levels...
was observed in the entire sample and in stratified analyses. Controlling for height, BMI, and other covariates increased the magnitudes of these decreases.

Previous analyses of secular trends in blood pressure among children have yielded conflicting results, and a 2007 review\(^1\) found little evidence that trends in blood pressure have paralleled the increases in obesity. Various studies have found that blood pressure levels have either increased slightly (\(<3\) mm Hg)\(^7\) or decreased slightly\(^10,12,30\) over the last few decades, but the lack of adjustment for height complicates the interpretation of some results. Morrison et al.\(^8\) for example, found a 2– to 3-mm Hg increase in the mean blood pressure of children over a 15-year-period but did not control for the observed 2-cm increase in height. In contrast, mean (age- and height-adjusted) levels of SBP among children in Seychelles decreased by 3 mm Hg from 1988 to 2006 despite an increase (5.5% to 8.5%) in the prevalence of obesity.\(^12\)

A previous report from the Bogalusa Heart Study of 2 cohorts,\(^31\) following up from 1973 to 1981 and the other from 1984 to 1992, found that the second cohort had lower blood pressure levels despite their higher relative weight. In contrast, levels of triglycerides were higher in the second cohort than in the first cohort.

Data from various surveys of adults\(^32,33\) have indicated that blood pressure levels among adults likely decreased by 1 to 3 mm Hg per decade from 1950 to 2000. Because these decreases have been observed throughout the entire distribution, rather than only at the upper end, it was concluded\(^34\) that changes are not due to increases in antihypertensive medication.

To account for these contrasting trends in obesity and blood pressure, it has been suggested that substantial changes have occurred in various characteristics which influence blood pressure and these changes have outweighed the adverse effects of higher BMI levels.\(^32,35\) It has been postulated that improvements in early-life diet and increases in birth weight (which is inversely associated with subsequent blood pressure levels) may, in part, be responsible for the blood pressure decreases.\(^32\) The possible roles of breastfeeding, accelerated weight gain in early childhood, and

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**TABLE 2** Mean Levels of Blood Pressure and Prevalence of Obesity and High Blood Pressure in Various Examinations

<table>
<thead>
<tr>
<th>Race–Gender</th>
<th>Examination Period</th>
<th>n</th>
<th>Obese, %</th>
<th>BMI z Score</th>
<th>SBP, mm Hg</th>
<th>SBP z Score</th>
<th>DBP, mm Hg</th>
<th>DBP z Score</th>
<th>High Blood Pressure, %a</th>
</tr>
</thead>
<tbody>
<tr>
<td>White boys</td>
<td>1974–1977</td>
<td>2487</td>
<td>7.5</td>
<td>0.02</td>
<td>101 ± 10</td>
<td>−0.32</td>
<td>62 ± 9</td>
<td>0.05</td>
<td>4.6</td>
</tr>
<tr>
<td>1979–1985</td>
<td>3283</td>
<td>9.1</td>
<td>0.19*</td>
<td>104 ± 11*</td>
<td>−0.16*</td>
<td>62 ± 9</td>
<td>0.06</td>
<td>5.6</td>
<td></td>
</tr>
<tr>
<td>1988–1993</td>
<td>1945</td>
<td>16.3*</td>
<td>0.42*</td>
<td>102 ± 10</td>
<td>−0.35</td>
<td>60 ± 9*</td>
<td>−0.12*</td>
<td>3.2</td>
<td></td>
</tr>
<tr>
<td>Black boys</td>
<td>1974–1977</td>
<td>1411</td>
<td>5.2</td>
<td>−0.08</td>
<td>101 ± 11</td>
<td>−0.37</td>
<td>62 ± 9</td>
<td>0.08</td>
<td>6.4</td>
</tr>
<tr>
<td>1979–1985</td>
<td>1868</td>
<td>7.7</td>
<td>0.09*</td>
<td>104 ± 12</td>
<td>−0.23*</td>
<td>62 ± 9</td>
<td>0.05</td>
<td>3.3</td>
<td></td>
</tr>
<tr>
<td>1988–1993</td>
<td>1314</td>
<td>12.1*</td>
<td>0.37*</td>
<td>102 ± 12</td>
<td>−0.34</td>
<td>60 ± 10*</td>
<td>−0.14*</td>
<td>4.6</td>
<td></td>
</tr>
<tr>
<td>White girls</td>
<td>1974–1977</td>
<td>2317</td>
<td>4.6</td>
<td>−0.04</td>
<td>100 ± 10</td>
<td>−0.27</td>
<td>62 ± 9</td>
<td>0.09</td>
<td>7.3</td>
</tr>
<tr>
<td>1979–1985</td>
<td>3161</td>
<td>7.9*</td>
<td>0.12*</td>
<td>103 ± 10</td>
<td>−0.07*</td>
<td>64 ± 9</td>
<td>0.22*</td>
<td>9.4</td>
<td></td>
</tr>
<tr>
<td>1988–1993</td>
<td>1804</td>
<td>14.4*</td>
<td>0.37*</td>
<td>101 ± 10</td>
<td>−0.28</td>
<td>61 ± 10*</td>
<td>−0.04*</td>
<td>5.2</td>
<td></td>
</tr>
<tr>
<td>Black girls</td>
<td>1974–1977</td>
<td>1329</td>
<td>7.8</td>
<td>0.07</td>
<td>101 ± 11</td>
<td>−0.29</td>
<td>63 ± 10</td>
<td>0.12</td>
<td>8.4</td>
</tr>
<tr>
<td>1979–1985</td>
<td>1907</td>
<td>9.8</td>
<td>0.22</td>
<td>103 ± 11*</td>
<td>−0.09*</td>
<td>64 ± 9</td>
<td>0.19</td>
<td>10.6</td>
<td></td>
</tr>
<tr>
<td>1988–1993</td>
<td>1356</td>
<td>14.8*</td>
<td>0.40*</td>
<td>101 ± 11</td>
<td>−0.30</td>
<td>61 ± 10*</td>
<td>−0.09*</td>
<td>5.7</td>
<td></td>
</tr>
</tbody>
</table>

*An SBP or DBP that was ≥90th percentile of the Fourth Report.\(^1\)

*P < .001 as assessed in race- and gender-specific regression models (linear or logistic) that assessed whether the mean (or prevalence) in the second and third time periods differed from the level in 1974–1977. All models accounted for the nonlinear effects of age, as well as for the intercorrelations among subjects who were examined >1 time.
various dietary factors, such as reductions in the salt content of food, have also been considered. Although the reason for the lack of increase in blood pressure levels remains uncertain, it should be realized that given a correlation of $r = 0.30$ between BMI and blood pressure, $\sim 90\%$ of the variability in blood pressure is not accounted for by BMI. It has also been suggested that blood pressure decreases may, at least in part, be the result of the BMI to blood pressure association becoming weaker. However, little evidence was found to support this possibility. Regression models (Figs 2 and 3) and correlational analyses showed no consistent difference in the relation of BMI to blood pressure levels. Although the BMI–SBP association tended to decrease in magnitude over time among both black girls and black boys ($r = 0.40$ in 1974 to 0.25 in 1993), its magnitude increased among white girls ($r = 0.35$ to 0.41). Because most children were examined multiple times in Bogalusa, we also considered if accommodation to measurement could account for the observed blood pressure decreases. We found, however, that there was a secular decrease in DBP even after restricting the analyses to the initial examination ($n = 11,477$). Although the possibly that a more accurate estimate of body fatness might yield different results was also considered, we have found that BMI is more strongly associated with blood pressure levels than are skinfold thicknesses. Our results emphasize the potential problem of basing a secular trend on only 2 time points, as has been done in most of the previous studies. We found, for example, that levels of SBP significantly increased from 1974–1977 to 1979–1985 but subsequently showed a slightly larger decrease despite substantial increases in obesity. Different conclusions would be reached based on analyses of these 2 separate time periods.

It is also possible that reports of secular trends in blood pressure, including the current analysis, have been influenced by differences in measurement techniques. These large effects can be seen among children who participated in national studies between 1963–1970 and 1988–1994. During this period, the prevalence of high blood pressure ($\geq 95$th percentile) decreased from 37% to 4% despite an increase in BMI. Various aspects of the examination, however, changed over this period, including the number of measurements.

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**TABLE 3 Prevalence of High Blood Pressure According to Year and BMI Category**

<table>
<thead>
<tr>
<th>Year</th>
<th>CDC BMI-for-Age Percentile</th>
<th>No. With High Blood Pressurea</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;50th</td>
<td>50–84th</td>
</tr>
<tr>
<td>1974–1977</td>
<td>4% (167/3790)</td>
<td>6% (151/2528)</td>
</tr>
<tr>
<td>1979</td>
<td>6% (101/1667)</td>
<td>8% (102/1260)</td>
</tr>
<tr>
<td>1982</td>
<td>5% (76/1544)</td>
<td>9% (116/1174)</td>
</tr>
<tr>
<td>1985</td>
<td>4% (59/1387)</td>
<td>7% (82/1115)</td>
</tr>
<tr>
<td>1988</td>
<td>2% (30/1227)</td>
<td>4% (53/1119)</td>
</tr>
<tr>
<td>1985</td>
<td>3% (33/1076)</td>
<td>4% (47/1040)</td>
</tr>
</tbody>
</table>

*a An SBP or DBP level that was $\geq 90$th percentile of the Fourth Report. Excerpted numbers represent the number of children who would have had a high blood pressure each year if the prevalence of high blood pressure had stayed the same as in 1974–1977. For example, the number of children expected to have had high blood pressure in 1982 would have been obtained by multiplying the 1974–1977 rates by the 1982 sample sizes: 0.044 x 1076 + 0.060 x 1040 + 0.099 x 69 + 0.127 x 180 + 0.341 x 340 = 261. Additional standardization for race, gender, and age altered the value only slightly ($n = 274$).c Values represent the percentage of children in specified cell who had high blood pressure.
and whether the participant was supine or sitting. It has also been estimated that the blood pressure of more than one-third of children in 1988–1994 were measured by using bladders that were too large, possibly biasing blood pressure levels downward. This over-cuffing may possibly account for the secular increases observed from 1988–1994 to the early 2000s. Attempts were made in the Bogalusa Heart Study to adhere to the same blood pressure protocol over the entire period (1974–1993). For example, the bladder cuff size in each examination was based on the circumference and length of the upper arm, and the bladder width was as large as possible. Measurements were obtained in a relaxed environment, and the mean of 6 measurements (3 readings, 2 observers) was used, resulting in levels that are lower than those in other studies. These 6 measurements, however, were from a single examination, whereas recommendations emphasize that elevated levels be confirmed on repeated visits. In addition, we cannot eliminate the possibility of methodologic differences over time as an explanation for our findings.

It should also be appreciated that the most recent examination of Bogalusa schoolchildren occurred ~20 years ago, and that the prevalence of obesity among white children in Bogalusa (Table 2) is higher (~5 percentage points [D.S.F., unpublished observation]) than that seen in NHANES III (1988–1994). Furthermore, we used the fourth Korotkoff sound to characterize DBP. The fourth sound was recommended for use among children in the 1977 and 1987 reports on blood pressure in children, and the true intra-arterial diastolic pressure seems to be between the fourth and fifth sounds. We have also found that the fourth sound is a better predictor of adult blood pressure levels than the fifth sound.

Conclusions

Despite the cross-sectional association between obesity and blood pressure levels among children in Bogalusa, we found that neither mean nor high blood pressure levels increased during a period in which the prevalence of obesity increased almost threefold. Although measurement differences over the study time period cannot be completely ruled out, these findings and those of other studies of children and adults suggest that there have been large changes in factors that have counterbalanced the expected increase in the prevalence of hypertension. Additional study of these potentially modifiable characteristics could lead to further reductions in the prevalence of high blood pressure.

Acknowledgment

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