Birth Weight Versus Childhood Growth as Determinants of Adult Blood Pressure

Bonita Falkner, Sonia Hulman, Harvey Kushner

Abstract—In older white American adults, recent retrospective studies have demonstrated a relationship between lower birth weight and hypertension. Black Americans have a higher occurrence of both lower birth weight and hypertension than do white Americans. To test the low birth weight–high blood pressure hypothesis, data from a prospective study (Perinatal Collaborative Project) were examined. The study followed a sample of 137 black Americans, with nine examinations. Data on birth weight, growth, and blood pressure from birth through 28.0±2.7 years were obtained longitudinally. Bivariate correlations among parameters were computed with the Pearson r. Birth weight and blood pressure at age 28 years are not correlated (Pearson r = .06). However, systolic blood pressures measured at 0.3 years and thereafter are correlated with adult systolic blood pressure. Also, weight at 0.3 years and body mass index at 7 years and thereafter are correlated with adult weight. Our data did not confirm the birth weight–blood pressure hypothesis. Rather, we detected significant correlations between preadult measurements of blood pressure and weight with adult measurements. These results indicate that in black Americans, childhood growth is a stronger determinant than intrauterine growth of adult blood pressure. (Hypertension. 1998;31[part 1]:145-150.)

Key Words: blood pressure ■ birth weight ■ blacks

Recently, several reports from the United Kingdom1-2 and Sweden3,4 have suggested that low birth weight is a causative variable for EH in adulthood.1,5 These studies were retrospective analyses of the relationship between birth weight and current BP in white men during their fifth through ninth decades. Others5,6,7 propose that reduced birth weight is also a causative variable for the development of non–insulin-dependent diabetes mellitus in adulthood. Hinchliffe et al8 proposed a potential pathogenesis for the linkage between low birth weight and EH in adulthood: the undernourished, growth-retarded fetus becomes a low-birth-weight newborn whose kidneys have a reduced number of nephrons. These authors suggest that fetal pathology sets the stage for adult-onset EH.

Black Americans have an increased incidence of low birth weight when compared with white American newborns.9,10 Black Americans also have an increased incidence of adult hypertension.11 Because of these two characteristics of the black American population, if a relationship between birth weight and adult BP exists, it will probably be found in an black American population sample.

Testing the hypothesis that low birth weight is a determinant of subsequent hypertension requires a prospective outcome study of a population sample from which anthropometric and BP data have been obtained longitudinally and periodically. The purpose of this study was to test the low birth weight–high BP hypothesis in a population sample of adult black American men and women whose mothers were enrolled in the Philadelphia site of the Perinatal Collaborative Project (PCP) from 1959 to 1966. Offspring of these women make up a cohort that has been observed prospectively from birth to current age (28 years). To determine (1) if birth weight is correlated with adult BP; (2) which (if any) anthropometric measurements in early life are correlated with EH in adulthood; and (3) if BP in early life is correlated with BP in adulthood, we examined weight and length data obtained on PCP subjects at birth and their weight, height, and BP data obtained during childhood, adolescence, and early adulthood.

Methods

All subjects reported in this study are currently adult black Americans who were the offspring of pregnant women enrolled in the PCP in Philadelphia from 1959 through 1965.12,13 Offspring of these women have been followed longitudinally from birth to current age (28 years). The PCP gathered maternal and perinatal/neonatal data, which included gestational age (by maternal dates), birth weight, length, head circumference, and chest circumference.14 Subjects’ growth was followed prospectively, with examinations at 0.3, 1, 4, and 7 years of age.12 From 1977 to 1980, the Philadelphia Blood Pressure Project collected growth, BP, and heart rate data on the black American subjects as adolescents.15 Subsequently, subjects between the ages 18 to 21 years were enrolled in a study of BP sensitivity to sodium and growth.14 During this study, subjects were assessed with measurements of body size, BP, cellular sodium transport, and mental stress testing before and after oral sodium loading. In 1987, the subjects were enrolled in a study of insulin resistance and BP.16 The insulin resistance–BP study again provided anthropometric and BP data at age 22 to 25 years.

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These data, collected on PCP subjects from birth through about 25 years, were compared with measurements made on the same subjects as adults during a subsequent study on insulin resistance, weight, and BP. None of the subjects included in this analysis had diabetes or was receiving antihypertensive medication at the time of the adult measurement.

**Adult Anthropometric Measurements**

Height and weight were measured, and BMI was calculated. BMI, an index of adiposity, is weight in kilograms divided by the square of height in meters. Adult obesity is defined as BMI $>27.3$ kg/m$^2$ in females and $>27.8$ kg/m$^2$ in males. At birth, PI is the assessment of adiposity. PI is weight in kilograms divided by the cube of length in meters. $^3$

**BP Measurements**

BP measurements were conducted as previously described. $^14$ Measurement protocols for BP determination were similar at childhood, adolescent, and young adult assessments. In each study, BP measurements were obtained by trained technicians, using standardized conditions of position, rest, and cuff size. Casual systolic (first phase) and diastolic (fifth phase) BP measurements were obtained by auscultation with a mercury column sphygmomanometer, with subjects in the seated position after a 10-minute rest period. The average of two determinations was used as the BP. In cases in which there was a deviation of $\pm 5$ mm Hg in SBP or DBP, BP measurements were repeated until consistent measurements were obtained.

**Statistical Methods**

Bivariate correlations among parameters were computed with the Pearson $r$. The standard error of $r$ can be computed by using the formula $(1-(r^2)/n-2)^{1/2}$. The student’s $t$ test for $r$ equals $r$ divided by the standard error of $r$, and the statistic $t$ has $n-2$ degrees of freedom. Associations among categorical groupings of birth weight and adult BP were tested with the $\chi^2$ statistic. All results are considered statistically significant for $P<.05$. All data are expressed as mean±SD.

**TABLE 1. Mean Values of Anthropometric Measurements in Males at Each Examination**

<table>
<thead>
<tr>
<th>Exam</th>
<th>n</th>
<th>Age, y</th>
<th>Weight, kg</th>
<th>Height, cm</th>
<th>BMI, kg/m$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>70</td>
<td>Birth</td>
<td>3.19±0.51</td>
<td>50.0±2.4</td>
<td>24.7±3.0*</td>
</tr>
<tr>
<td>2</td>
<td>66</td>
<td>0.30±0.03</td>
<td>6.51±0.92</td>
<td>63.0±3.0</td>
<td>16.4±1.9</td>
</tr>
<tr>
<td>3</td>
<td>64</td>
<td>2.2±1.8</td>
<td>12.7±5.0</td>
<td>85.7±17.0</td>
<td>16.6±1.4</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
<td>7.0±0.4</td>
<td>25.9±5.8</td>
<td>123.3±5.9</td>
<td>16.7±3.0</td>
</tr>
<tr>
<td>5</td>
<td>60</td>
<td>13.1±1.4</td>
<td>51.1±16.8</td>
<td>159.4±13.6</td>
<td>19.7±4.1</td>
</tr>
<tr>
<td>6</td>
<td>67</td>
<td>14.3±1.4</td>
<td>56.9±18.2</td>
<td>165.3±12.1</td>
<td>20.4±4.4</td>
</tr>
<tr>
<td>7</td>
<td>64</td>
<td>20.1±1.5</td>
<td>74.3±17.9</td>
<td>178.2±8.2</td>
<td>23.4±5.2</td>
</tr>
<tr>
<td>8</td>
<td>60</td>
<td>21.1±1.4</td>
<td>75.8±18.1</td>
<td>178.2±8.2</td>
<td>23.8±5.2</td>
</tr>
<tr>
<td>9</td>
<td>70</td>
<td>27.7±2.5</td>
<td>84.6±20.1</td>
<td>177.2±8.4</td>
<td>27.2±6.7</td>
</tr>
</tbody>
</table>

*Ponderal index in kilograms/m$^3$.
greater. Of these, 40 (40/57 = 70.1%) were normotensive; 17 (17/57 = 29.8%) were borderline hypertensive. The data again indicate no trend in a relationship of birth weight with adult blood pressure. Although the numbers of individuals in each birth weight–BP group are too small to perform a meaningful statistical analysis, there is no indication that the individuals who were smaller newborns grew up to be adults with higher BP (defined as SBP >135 mm Hg, DBP >85 mm Hg).

**Anthropometric Variable–Adult SBP Correlation**

Since we found no significant correlation (positive or negative) of birth weight and birth PI with adult BP, we examined the anthropometric data obtained after the newborn period for relationships with adult BP. An analysis was conducted on height and weight data obtained in the sample population during childhood, adolescence, and young adulthood to determine if there was a correlation of these variables with adult BP. Tables 4 and 5 contain values of the Pearson $r$, which correlate measures of height and weight with adult SBP, and the corresponding probability values for each $r$ value. In males (Table 4), there are significant correlations of weight and height with adult systolic BP. At each examination, the correlation of height is more significant than weight with adult SBP. In females (Table 5), the correlation of weight with adult systolic BP is highly significant. There are no significant correlations of height with adult SBP in females.

Overall, while no relationship of birth weight with adult BP was detected, there are highly significant correlations of adolescent growth and adult BP.

**Preadult BP–Adult BP Correlation**

An analysis was conducted to examine correlations of BP measures obtained during childhood and adolescence with adult SBP. Fig 2 displays the Pearson $r$ values for the correlation of SBP at each examination with adult SBP. As early as 0.3 years, there is a significant positive correlation of childhood BP with adult SBP. The Pearson $r$ value increases with increasing age. These data demonstrate statistically significant tracking of BP as early as 0.3 years.

**Discussion**

This study examined longitudinal data, obtained over a 28-year period, on 137 black Americans. These data included birth weight and length, and childhood and adolescent weight, height, and blood pressure. Analysis of data from this sample did not detect a negative or positive correlation between birth weight and adult blood pressure. Also, analysis of birth PI data derived from our sample failed to demonstrate any relationship between reduced body size at birth and higher blood pressure in adulthood. However, significant blood pressure tracking beginning at 0.3 years was detected. Moreover, significant correlations of childhood growth and adult blood pressure were demonstrated.

Several epidemiological studies report an association of low birth weight with the development of essential hypertension in later life. Other studies have demonstrated only a weak or no significant support of this association. In a recent analysis of follow-up data, Hack et al reported lower blood pressure in individuals with lower birth weight.

### TABLE 2. Mean Values of Anthropometric Measurements in Females at Each Examination

<table>
<thead>
<tr>
<th>Exam</th>
<th>n</th>
<th>Age, y</th>
<th>Weight, kg</th>
<th>Height, cm</th>
<th>BMI, kg/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>67</td>
<td>Birth</td>
<td>3.02±0.38</td>
<td>49.8±2.3</td>
<td>24.5±2.9*</td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>0.3±0.03</td>
<td>5.78±0.83</td>
<td>61.0±2.3</td>
<td>15.5±1.9</td>
</tr>
<tr>
<td>3</td>
<td>62</td>
<td>2.2±2.0</td>
<td>12.0±5.3</td>
<td>83.9±18.7</td>
<td>16.3±1.9</td>
</tr>
<tr>
<td>4</td>
<td>67</td>
<td>6.9±0.2</td>
<td>24.8±5.0</td>
<td>125.5±5.0</td>
<td>16.4±2.4</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
<td>13.3±1.4</td>
<td>51.2±12.8</td>
<td>157.1±7.3</td>
<td>20.6±4.2</td>
</tr>
<tr>
<td>6</td>
<td>65</td>
<td>14.4±1.4</td>
<td>57.9±14.0</td>
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<td>22.4±4.8</td>
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<tr>
<td>7</td>
<td>60</td>
<td>20.1±1.5</td>
<td>65.5±17.3</td>
<td>162.8±5.5</td>
<td>24.6±6.2</td>
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<td>8</td>
<td>58</td>
<td>21.1±1.4</td>
<td>66.4±18.4</td>
<td>163.3±5.4</td>
<td>24.7±6.5</td>
</tr>
<tr>
<td>9</td>
<td>67</td>
<td>28.2±2.8</td>
<td>76.4±22.3</td>
<td>162.9±6.7</td>
<td>29.2±6.7</td>
</tr>
</tbody>
</table>

*Ponderal index in kilograms/m³.

### TABLE 3. Blood Pressure (mm Hg, mean±SD) of Males and Females at Each Examination

<table>
<thead>
<tr>
<th>Age, y</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>SBP</td>
</tr>
<tr>
<td>0.3±0.0*</td>
<td>62</td>
<td>85±15</td>
</tr>
<tr>
<td>6.9±0.3</td>
<td>70</td>
<td>101±9</td>
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<tr>
<td>13.2±1.4</td>
<td>41</td>
<td>114±13</td>
</tr>
<tr>
<td>14.4±1.4</td>
<td>67</td>
<td>116±13</td>
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<td>20.1±1.5</td>
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<td>21.1±1.4</td>
<td>60</td>
<td>118±12</td>
</tr>
<tr>
<td>28.0±2.7</td>
<td>70</td>
<td>125±13</td>
</tr>
</tbody>
</table>

*SBP only measured at 0.3 years.
pressures in children who were very low birth weight. The concept that impairment of intrauterine growth, including organ growth, results in alterations in functional capacity and hemodynamic stress in later adulthood is supported by some experimental studies. However, as noted above, the epidemiological and clinical studies have not produced consistent results, and the concept remains controversial.

Mean birth weights of black American newborns are less than white newborns. An analysis of more than 60,000 singleton births from 1975 to 1992 in Cleveland, Ohio, showed that at term, the mean birth weights of black infants were about 200 g less than the mean birth weights of white infants. The difference of about one-half pound means that on average, the black infants are “thinner” than the white infants, which is consistent with the description of the at-risk individuals by Barker et al. Black American adults have greater prevalence of hypertension than white adults. Hypertension in black American adults is expressed at an earlier age and results in greater morbidity and mortality than in white adults. Because both the independent variable (low birth weight) and the dependent variable (hypertension) of the low birth weight–high blood pressure hypothesis occur with greater frequency in the black American population compared with the white population, our study sample is well suited to test this hypothesis. Of the 137 subjects in our study, 80 (58.4%) had birth weights less than 3200 g, which is below the 50th percentile for the white term infants in the Cleveland study. As adults, 8-eight (27.7%) are already in a borderline range of hypertension. Although our sample size is small, it does express the two variables, low birth weight and higher blood pressure. Despite the advantage of multiple examinations from birth to a mean age of 28 years, we detected no correlations between birth weight and adult blood pressure in this black American sample.

During childhood as well as during adulthood a direct relationship between weight and blood pressure has been demonstrated consistently. The data obtained during childhood and adolescence on the cohort in this study have been reported previously, and demonstrate a significant direct correlation of blood pressure with weight. Blood pressure measurements were not obtained at birth in this cohort, most likely because of the technical difficulty in obtaining accurate newborn blood pressure measurements at that time. The current availability of Doppler instrumentation has enabled reliable measurement of blood pressure in newborn infants, including very-low-birth-weight infants. Recent reports have demonstrated a very significant direct correlation of weight with blood pressure on the first day of life. The direct correlation of weight with blood pressure is sustained as the newborn undergoes the hemodynamic transition from intrauterine to extrauterine life. The direct correlation of weight with blood pressure is consistently observed, from very low birth weight newborns to normal full-term infants.

In addition to the consistently strong relationship of blood pressure with body size and growth throughout childhood, the observation of blood pressure tracking has also been

<table>
<thead>
<tr>
<th>Age, y</th>
<th>Weight r</th>
<th>Weight P</th>
<th>Height r</th>
<th>Height P</th>
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</thead>
<tbody>
<tr>
<td>13.3±1.4</td>
<td>.34</td>
<td>.005</td>
<td>.01</td>
<td>.92</td>
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<tr>
<td>14.4±1.4</td>
<td>.38</td>
<td>.002</td>
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<tr>
<td>20.1±1.5</td>
<td>.25</td>
<td>.51</td>
<td>.03</td>
<td>.83</td>
</tr>
<tr>
<td>21.1±1.4</td>
<td>.37</td>
<td>.005</td>
<td>.03</td>
<td>.85</td>
</tr>
</tbody>
</table>

Figure 1. The y-axis is adult systolic blood pressure; the x-axis is birth weight in grams. There is no relationship between birth weight and blood pressure in this sample of 137 black Americans. The Pearson r value is .06 for the entire group.

Figure 2. Bars depict the Pearson r values correlating preadult blood pressure with adult blood pressure. The Pearson r value increases with increasing age, reaching a highly statistically significant correlation by age 13 years.
reported. Data from these epidemiological studies demonstrate that children in the higher range of the blood pressure distribution at a given age continue to have higher blood pressures as they grow and age. It has also been documented that elevated blood pressure in childhood often correlates with hypertension in early adulthood. The significant correlation of adult blood pressure with blood pressure in childhood, adolescence, and young adulthood in this sample of black American men and women is consistent with previous reports. In addition, we detected a significant correlation of adult blood pressure with blood pressure as early as 0.3 years (4 months of age). In cross-sectional analysis, the most important determinant of blood pressure in childhood is body size. In the recent Working Group Report on Hypertension in Childhood, an analysis of all available US data on blood pressure in children and adolescence determined that height was the strongest correlate of blood pressure throughout childhood and adolescence. Weight was also a strong direct correlate of blood pressure. However, excessive weight (obesity) contributes to higher blood pressure and the development of hypertension. Analysis of our data from this young black American cohort supports the concept that higher body weight is a determinant of higher blood pressure and higher blood pressure in subsequent years. As shown in Tables 4 and 5, we found a direct and highly significant correlate of weight in adolescence (age 13 to 14 years) with the blood pressure in adulthood (age 28 years). Our sample has high rates of obesity, particularly among the females. The prevalence of obesity in this sample (approximately 40% among the women) is consistent with that of other reports on obesity in black Americans in the United States. Data were not available from the adolescent age examinations to determine if lean body mass versus fat mass were independent correlates of adolescent BP or predictive of adult BP. Reduced intrauterine growth and smaller body size at birth resulting in attenuation in organ function capacity throughout life is the conceptual basis for the low birth weight—high blood pressure hypothesis. Our data do not support this hypothesis in black Americans. Since our study is limited to black Americans, our findings may not necessarily be generalizable to white Americans. Our data do suggest an alternative hypothesis. The data clearly support the tracking of blood pressure from early childhood, which is consistent with underlying genetic regulation of blood pressure. In addition, excessive body growth during childhood determines higher blood pressure later in life. Thus an alternative hypothesis is that excessive growth, or obesity, in childhood imposes excessive demands on organ function and contributes to later hypertension and cardiovascular disease. The known relationship of obesity with hypertension and the increasing rates of obesity in childhood indicate that more attention should be focused on this area.

Acknowledgment
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References
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