

Rapid Child Growth Raises Blood Pressure in Adolescent Boys Who Were Thin at Birth

Linda S. Adair, Tim J. Cole

Abstract—Catch-up growth in previously growth-restricted children is a suggested risk factor for chronic disease risk. We use data from 2026 Filipino adolescents to identify periods of growth that matter more for risk of high blood pressure (BP). Subjects were drawn from the Cebu Longitudinal Health and Nutrition Survey, which enrolled pregnant women and followed up their offspring through age 14 to 16 years. High BP was defined as the top 10% of residuals from gender-specific regressions of systolic and diastolic BP on age and height. After controlling for birth length, current body mass index, age, and height, the odds of high BP in males were significantly decreased with each kilogram increase in birth weight. The highest odds of elevated BP occurred among males who were relatively thin at birth but relatively heavy as adolescents. Larger weight increments from birth to 2 years decreased the odds of high BP in boys, whereas larger increments from 8 to 11 and 11 to 16 years increased the odds of high BP. Thinness at birth significantly interacted with growth rate after age 8, such that a high rate of weight gain increased risk only among boys who were in the lower two thirds of the body mass index distribution at birth. Results in girls indicated small or no effects of early growth. The synergistic effect on adolescent BP of rapid weight gain from late childhood into adolescence with thinness at birth is further evidence of fetal programming of BP in males and suggests long-term health risks associated with rapid growth, even in the absence of obesity. (*Hypertension*. 2003;41:451-456.)

Key Words: birth weight ■ blood pressure ■ child growth ■ infant nutrition ■ adolescents

Many studies that examine the relation of size at birth to blood pressure (BP) later in life find an inverse relation that is evident or stronger only after controlling for current body size.¹ Children at greatest risk of high BP and other cardiovascular disease (CVD) risk factors are typically those who are relatively small at birth but relatively large at the time when CVD risk is assessed. This suggests an important role for postnatal changes in body size.² A recent review³ identified numerous studies that associated more rapid postnatal growth with increased BP in childhood or adulthood. However, there remain important questions about whether the timing of rapid growth affects later risk of disease. A higher-than-average rate of early postnatal growth is often observed among infants who were growth restricted in utero and is generally regarded as a desirable marker of infant health. For children in developed countries, rapid growth in childhood and adolescence is associated with increased prevalence of overweight and its associated risks. For children in developing countries, many of whom were growth retarded throughout infancy, more rapid childhood growth represents an opportunity for recovery and avoidance of the health risks more typically associated with poor growth and nutritional status.⁴ Changes associated with modernization and economic development may promote higher energy intakes and reduced

physical activity in children, thus creating more opportunities for rapid growth. We know relatively little about the extent to which more rapid childhood growth is associated with chronic disease risk, particularly whether risk differs depending on when a child experiences more rapid growth. Few studies from developing countries with repeated growth measurements throughout childhood and adolescents are available to address this gap in the literature.

We used data from a longitudinal cohort study of youths from Cebu, Philippines, to examine the relation of high BP in adolescence to size at birth and patterns of growth from birth to adolescence. The Cebu sample is characterized by a high level of early child growth restriction but more recent improvements in socioeconomic status associated with rapid economic development in the Philippines.

Methods

Survey Design and Sample

Data were collected during the Cebu Longitudinal Health and Nutrition Survey (CLHNS). This community-based survey followed up a birth cohort of more than 3000 infants born between 1983 and 1984. Participants were originally inhabitants of 33 randomly selected communities of Metro Cebu, which comprises The Philippines' second largest city and its surroundings. Sample communities included densely populated urban and periurban neighborhoods, as

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TABLE 1. Characteristics of the CLHNS Sample of Adolescents

Characteristic	Females (n=967)	Males (n=1059)
Age, y	15.0±0.3	16.1±0.3
Birth weight, kg	2.98±0.41	3.03±0.42
Birth length, cm	48.9±2.0	49.3±2.0
Adolescent height, cm	149±5.6	158.4±6.8
Adolescent weight, kg	41.9±6.4	46.8±8.4
Adolescent BMI, kg/m ²	18.8±2.4	18.6±2.5
Systolic BP, mm Hg	97.2±8.9	105.9±10.8
Diastolic BP, mm Hg	66.0±7.4	69.4±8.9
High BP, %	10.0	11.8

Data are mean±SD or %, as indicated.

well as more isolated rural villages in the mountains or on nearby islands.

All pregnant women in the selected communities were invited to participate in the survey. They were enrolled during the second half of pregnancy, and they and their infants were followed up during bimonthly home visits from birth to 2 years postpartum. Follow-up surveys were conducted in 1991, 1994, and 1998 when the index children were, on average, 8.5, 11.5, and 15.5 years of age. CHLNS protocols have been reviewed and approved by Institutional Review Boards at the University of North Carolina and the University of San Carlos.

From the original sample of 3080 single live births, 2089 adolescents were located and included in the 1998 follow-up survey. Of these, 2026 had birth and current measurements, and they form the main analysis sample. When data were collected for the 1998 to 2000 CLHNS, a decision was made to completely survey all girls and then to begin the boys' survey. As a result, boys are, on average, ≈1 year older than girls. Characteristics of the analysis sample participants are presented in Table 1.

Data and Variables

Infant length was measured within 6 days of birth by trained project staff using custom-designed length boards. For infants born in hospitals, birth weight was measured by birth attendants using hospital scales. Infants born at home were measured by birth attendants who had been provided with Salter hanging-type scales and who had been trained in their use. Gestational age was estimated from the mother's report of the date of her last menstrual period. In cases where this date was unknown, when pregnancy complications occurred, or when the infant weighed <2.5 kg at birth, gestational age was determined by nurses using the Ballard method.⁵

From birth to 2 years, weight and recumbent length were measured bimonthly. During the subsequent follow-up surveys, anthropometric assessments included weight; height; arm, waist, and hip circumferences; and triceps and subscapular skinfolds. Relative weight is represented by body mass index (BMI; kg/m²). At birth and in adolescence, 3 groups were defined by gender-specific BMI tertiles as cutpoints. Nine groups were defined by a cross tabulation of these groups (low-low, low-middle, low-high, etc). Weight and length or height increments were calculated for the measurement intervals from birth to 2 years, 2 to 8 years, 8 to 11 years, and 11 to 15 years in girls and 11 to 16 years in boys.

In preliminary analysis, we assessed the effect of (1) diet, by using data obtained from two 24-hour recalls; (2) physical activity, measured with activity questionnaires and Caltrac accelerometers; (3) smoking habits; (4) maturation status, indicated by age at menarche or self-assessed pubic hair development in boys; and (5) household socioeconomic status, represented by total household income and television ownership. However, none of these variables proved to be confounders of the relation of body size or growth measures to risk of high BP, and thus, for simplicity, these variables were not included in the models presented below.

After a 10-minute seated rest, BP was measured in triplicate by using a mercury sphygmomanometer, and the average of the 3 measurements was used in analyses. During home visits, the same interviewer measured the BP of the index child's mother. There are no standard cutpoints to define high BP in children and adolescents because BP varies by age, gender, and height. Reference data for the United States are age-, gender- and height-percentile specific.⁶ There are no data to support the use of US reference values for Asian or other populations, nor are there good BP reference data for children in the Philippines. For purposes of this analysis, we defined sample specific "high" BP as follows. We regressed age and height on systolic and diastolic BP, for males and females separately. We then defined high BP as a residual from the systolic or diastolic BP regression greater than the gender-specific 90th percentile of the CLHNS sample. This method produced a similar prevalence of high BP in males and females.

Analysis Methods

We used logistic regression to estimate the odds of having high BP. All models were stratified by gender and were controlled for age, and, where appropriate, current height. Alternative models were specified according to the hypothesis being tested. Initially, the effect of gestational age was tested in all models. However, because gestational age was not significantly associated with a risk of high BP, nor did it modify the effects of any other variables, it was dropped from the final models.

Results

We first tested the hypothesis that the risk of high BP in adolescence is inversely related to birth weight. Among males, after controlling for birth length, current BMI, age, and height, the odds of high BP were significantly decreased (odds ratio [OR], 0.53; 95% confidence interval [CI], 0.29 to 0.94) for each kilogram increase in birth weight. All else being equal, the predicted probability of high BP was 0.068 for a 4-kg infant, 0.118 for a 3-kg (average-weight) infant, and 0.200 for a 2-kg infant. The fact that birth weight was significantly associated with high BP only after controlling for birth length suggests that it is thinness rather than weight that matters. This was further supported by a significant inverse relation of birth BMI to high BP. We used BMI rather than ponderal index because, in this sample of infants, BMI was less correlated with length. We found no significant relation of birth weight, length, BMI, or ponderal index to risk of high BP in females.

We next examined the interaction of birth size with current size by using a logistic regression model that included age, current height, and 8 dummy variables representing the BMI groups described above, with the reference category being those who were in the low-BMI group at both ages. Figure 1 shows the predicted prevalence of high BP among males in each group. The predicted prevalence of high BP was highest among males in the low-high BMI group (relatively thin at birth, relatively heavy in adolescence). The low-high BMI group was significantly different not only from the reference (low-low) group but also from the high-high BMI group, suggesting that current BMI had a stronger effect among those who were relatively thin at birth. Males in the low-high group had significantly larger subscapular skinfolds (10.5 vs 9.2 mm in the high-high group, $P<0.01$ by ANOVA). In females (not shown in the figure), the low-high BMI group had significantly greater odds of high BP compared with the low-low BMI group, but all of the adolescent high-BMI

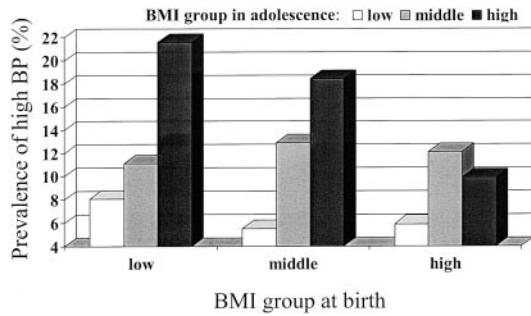


Figure 1. Age- and height-adjusted prevalence of high BP in groups of Filipino boys defined by gender-specific thirds of the BMI distributions at birth and age 16 years.

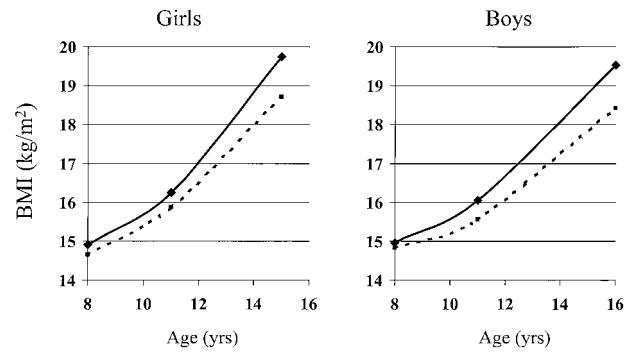


Figure 2. Mean BMI of Filipino boys and girls, with and without high BP in adolescence.

groups had a similarly elevated risk of high BP. Thus, among females, the effect of current BMI on BP was not modified by BMI at birth.

We next examined the growth patterns in youths with and without high BP. There were no consistent significant differences in attained-size measures in males or females with high vs normal BP through age 8. Figure 2 shows BMI in youths with and without high BP. Girls with high BP had marginally higher BMI at ages 8 and 11 ($P < 0.10$) and significantly higher BMI at age 15. Boys with and without high BP at age 16 did not differ in BMI at age 8 but were significantly different ($P < 0.02$) at ages 11 and 16. Compared with boys with normal BP, those with high BP were ≈ 1 cm shorter and 2 kg heavier by age 16.

The relation of growth increments at different ages to risk of high BP in adolescence was tested by using logistic regression but in this case controlling only for age, because the sum of growth increments constitutes current size (Table 2). Among boys, larger birth weight and larger weight and length increments from birth to 2 years were associated with a decreased odds of high BP. In contrast, larger weight increments from 8 to 11 years and 11 to 16 years were associated with an increased odds of high BP. Among girls, a larger weight increment from 11 to 15 years was associated with increased odds of high BP, but a larger length increment from age 2 to 8 was associated with a decreased odds of high BP. Weight increments after age 2 were strong predictors of BMI and the sum of skinfolds in the adolescent boys and girls (results not shown).

Finally, we tested whether childhood weight rate had a different effect on the odds of high BP depending on size at

birth. Childhood weight rate (age 8 to 15 in girls, 8 to 16 in boys) was modestly correlated with BMI at birth (0.05 in girls, 0.11 in boys). Nine groups were defined to identify size at birth based on thirds of the gender-specific BMI distribution and thirds of the gender-specific distribution of weight rate from age 8 to 15 (see Table 3). Dummy variables representing the groups were entered into a logistic regression model, after controlling for current height and age, with the middle BMI at birth-middle childhood weight rate group as the referent. Compared with the middle-middle group, the odds of high BP were more than doubled among those who were in the low BMI group at birth but had the highest weight rate in late childhood. The odds of high BP were also high among boys who were in the middle BMI group at birth, but who had faster growth rates in late childhood. High weight rate in late childhood was not associated with high BP among boys who were in the high-BMI group at birth. These results are summarized in Figure 3, which presents the predicted probability of high BP for each of the 9 groups based on the logistic regression results. The results were confirmed in an analysis of the effects of growth increments stratified by thirds of BMI at birth. None of the weight increments significantly predicted high BP among boys in the top third of BMI at birth. Furthermore, more rapid early growth (birth to 2 years) reduced the odds of high BP only among those in the lowest third of BMI at birth. Among girls, the odds of high BP were primarily associated with weight rate in late childhood, independent of size at birth, and the most striking finding was that slow growth in weight protected against high BP. A similar analysis performed with infant weight rate

TABLE 2. Odds Ratios of High BP Associated With Birth Weight and Growth Increments at Different Ages

	Weight		Length or Height	
	Males	Females	Males	Females
Birth	0.59 (0.37–0.94)*	0.91 (0.53–1.57)	0.97 (0.87–1.07)	0.98 (0.87–1.10)
Birth–2 y	0.80 (0.65–0.98)*	0.92 (0.73–1.15)	0.93 (0.88–0.99)*	0.98 (0.91–1.04)
2–8 y	0.89 (0.80–0.98)*	0.99 (0.87–1.13)	0.97 (0.92–1.02)	0.94 (0.88–0.99)*
8–11 y	1.12 (1.04–1.20)†	1.05 (0.97–1.13)	1.05 (0.97–1.11)	0.94 (0.87–1.03)
11–15 y	1.10 (1.05–1.15)†	1.08 (1.02–1.13)†	1.01 (0.96–1.07)	0.92 (0.86–0.99)*

* $P < 0.05$; † $P < 0.01$.

TABLE 3. BMI at Birth, Weight Velocity From Ages 8–15 Years, and Odds of High BP in Cebu Adolescents

Group (BMI Group at Birth–Child Growth Rate)	n	BMI at Birth (Mean±SD)	Weight Velocity, 8–15 (kg/y)	OR (95% CI) of High BP
Males				
Low-slow	134	10.95±0.82	2.68±0.36	0.59 (0.26–1.34)
Low-middle	120	10.94±0.81	3.40±0.15	0.72 (0.31–1.68)
Low-fast	97	10.92±0.87	4.41±0.91	2.25 (1.03–4.89)*
Middle-slow	108	12.37±0.33	2.72±0.34	0.27 (0.10–0.76)*
Middle-middle	112	12.38±0.35	3.38±0.16	1.00 (referent)
Middle-fast	131	12.38±0.33	4.25±0.64	2.09 (1.00–4.37)*
High-slow	108	13.76±0.72	2.70±0.35	0.40 (0.16–1.01)
High-middle	117	13.80±0.68	3.35±0.15	0.78 (0.34–1.79)
High-fast	121	14.02±0.82	4.22±0.51	1.05 (0.46–2.37)
Females				
Low-slow	131	10.94±0.73	2.58±0.32	0.19 (0.07–0.52)†
Low-middle	111	11.13±0.61	3.25±0.17	0.61 (0.28–1.35)
Low-fast	82	11.00±0.68	4.04±0.49	0.92 (0.41–2.07)
Middle-slow	99	12.37±0.32	2.53±0.29	0.39 (0.16–0.94)*
Middle-middle	112	12.43±0.33	3.27±0.18	1.00 (referent)
Middle-fast	112	12.43±0.31	4.13±0.50	0.56 (0.24–1.29)
High-slow	90	13.97±0.83	2.59±0.28	0.24 (0.08–0.68)†
High-middle	97	13.86±0.82	3.24±0.18	0.64 (0.29–1.45)
High-fast	126	13.95±0.83	4.06±0.53	0.72 (0.33–1.54)

* $P < 0.05$; † $P < 0.01$.

showed no significant associations with high BP in adolescence.

Discussion

Although there is substantial literature that demonstrates modest inverse associations of size at birth to levels of BP in children and adolescents,^{1,3} most studies have not specifically addressed the risk of high BP. High BP is more clinically significant⁷ and has been shown to track into adulthood,⁸ when it contributes to CVD mortality. Furthermore, because BP in children and adolescents is so highly determined by height,⁶ we selected an outcome that was height independent.

Our observation that birth size was inversely related to adolescent BP only when current size was taken into account strongly supports a role for postnatal growth.² We directly

demonstrated the role of postnatal growth by explicitly modeling growth increments across several ages. Our results address 3 important issues: the synergism of fetal and postnatal growth, the timing of rapid growth associated with disease risk, and the importance of accelerated growth in weight versus height. A relatively high BMI and larger weight increments from age 8 to 15 increased the risk of high BP only among boys who were relatively thin at birth. The interaction of small size at birth with rapid child growth is particularly noteworthy as evidence of fetal programming.

The magnitude of the effect of this combination was substantial. Among boys in the highest third of childhood weight increments, those who were thin at birth had a predicted probability of high BP nearly twice that of those who were relatively heavy at birth (24.5% vs 13.2%). Thus, higher childhood weight gain in the absence of fetal growth restriction was not a risk factor in this population. It has been postulated that fetal undernutrition results in a reduced number of nephrons.^{9,10} Such deficits may not increase disease risk in individuals who remain small, but excess growth may challenge the ability of the kidneys to effectively regulate BP. Other researchers have reported interactions of size at birth with rapid growth in childhood. Walker et al¹¹ reported larger effects of increasing weight on systolic BP in 11- to 12-year-old Jamaican children who were stunted between 9 to 24 months. Similarly, Eriksson et al¹² found that coronary heart disease (CHD) risk was only associated with more rapid growth after age 1 year among men who were thin at birth. In contrast, Williams and Poulton¹³ found no signif-

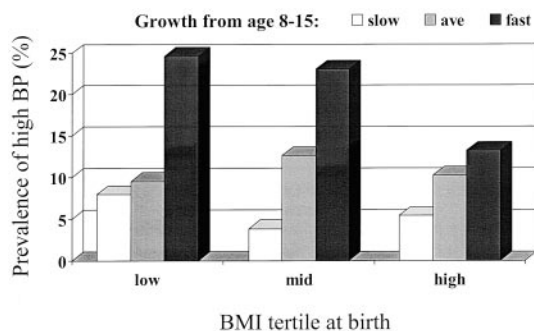


Figure 3. Predicted prevalence of high BP in Filipino boys defined by thirds of the BMI distribution at birth and thirds of the weight rate distribution from age 8 to 16 years.

icant interaction of size at birth with later obesity as determinants of BP in a cohort of young adults in New Zealand.

A second important contribution of this work is its identification of when more rapid growth confers the risk of high BP. Higher weight gain after age 8 increased the odds of high BP in boys, whereas larger infant weight and length gains reduced the odds of high BP. Eriksson et al¹² reported that low infant weight gain increased the risk of CHD in older Finnish adults, but to date, there is no other direct evidence that higher infant growth rates protect against hypertension. Eriksson et al¹⁴ found that Finnish adults who developed hypertension were shorter and lighter at birth but comparable in height and weight at age 7. This led them to the conclusion that the risk of hypertension associated with fetal growth retardation is amplified by rapid growth between birth and 7 years. However, this report did not directly compare infant versus childhood growth.

Others have studied growth and levels of BP, with inconsistent results. Whincup et al¹⁵ found no effect of growth rates to age 3 on BP in 3-year-old British children. In contrast, Cheung et al¹⁶ found that after controlling for birth length and ponderal index, the change in ponderal index from 6 to 18 months was inversely associated with systolic BP in young Hong Kong adults. Law et al¹⁷ found no association of growth in infancy, but more rapid weight gain from 1 to 5 years was associated with higher systolic BP in young British adults. Although more rapid infant growth has not been identified as a risk factor for elevated BP later in life, it has been identified as a risk factor for later obesity.^{18,19} The degree to which rapid infant growth represents risk may depend on whether it occurs in the context of recovery from fetal growth restriction and results in normalization of body weight (as occurred in the Cebu sample) versus excess growth that results in infant obesity.^{18,19}

The period of rapid growth that best predicts high BP in Cebu adolescents is also the period that best predicts high BMI or high triceps and subscapular skinfold thicknesses, which are associated with risk of high BP. Infant weight increments are only weakly related to adolescent BMI, and once subsequent increments are taken into account, growth in infancy is unrelated to adolescent BMI. So, in the case of Cebu, rapid growth matters during the time period of largest accretion of tissue mass typically associated with disease risk. BMI does not distinguish lean and fat mass, but whereas boys in the low-high BMI group did not differ from those in the high-high group in mean BMI, they had significantly larger subscapular skinfolds. Law et al¹⁷ interpreted the relation of early childhood weight gain to adult systolic BP as evidence that the effect of early childhood weight gain is mediated partly through its prediction of adult fatness. We found no association of childhood height increments with high BP. Some studies do not directly compare effects of growth in height versus weight. For example, Miura et al²⁰ reported an inverse relation of rate of height gain from age 3 to 20 years on systolic BP in Japanese young adults. Law et al¹⁷ reported effects of weight gain but did not show results for height. Eriksson et al,¹⁴ in their study of hypertension, found effects of both weight and height. Lundgren et al²¹ determined that linear catch-up growth did not increase the risk of elevated

BP in Swedish young adults who had been light for gestational age at birth. Rather, they reported that short adult stature was a risk factor, suggesting the importance of the absence of catch-up in height among those who were light at birth.

The Cebu study has several limitations. Because BP was measured only on 1 day, there is a substantial chance that individuals were misclassified as having high BP. Ongoing data collection in the Cebu cohort will allow replication of our analysis in young adults. Intervals between growth measurements were too long and unevenly spaced to allow for a more precise estimate of when rapid growth matters for later BP. Furthermore, we were unable to account for the large male-female differences in the relation of size at birth and postnatal growth to BP. Results from this study should only be generalized to other populations, particularly those in industrialized countries, with caution. In the Cebu sample, $\approx 20\%$ of infants were born small for gestational age, and larger weight increments in infancy largely represent compensatory growth in response to low relative weight at birth rather than excessive weight gain leading to overweight. Similarly, we define "large" weight increments relative to the Cebu sample, and $< 3\%$ of Cebu adolescents are overweight based on comparisons of their BMI to the International Obesity Task Force reference.²² In populations with excess weight gain leading to obesity, the relative importance of size at birth may be diminished.

Perspectives

Given the functional consequences of small body size among adults, compensatory growth has typically been viewed as desirable for children with poor nutritional histories in developing countries. However, a growing body of research, including the present study of Filipino youths, suggests that there may be health risks associated with more rapid child growth. We showed that Filipino adolescent boys who were relatively thin at birth and who, during late childhood, gained relatively more weight than their peers were at > 2 times higher risk of having high BP compared with boys with similar growth rates who were not poorly nourished in utero. With a mean BMI of 19.5, the Cebu boys with high BP were not overweight. Because high BP tracks into adulthood and 44.3% of Filipino adults aged 60 and older have hypertension,²³ early prevention is essential. This study suggests that children with impaired fetal growth are more sensitive to environmental factors that promote faster child growth and contribute to chronic disease risk. For disease prevention, we should therefore focus on optimization of fetal growth through good maternal nutrition and health care during pregnancy and optimization of infant growth through promotion of breastfeeding and good weaning practices. In addition, because larger weight but not height increments confer risk in those with poor fetal nutrition, promotion of healthy, proportionate child growth without excess body fat accumulation is still highly desirable. Future research should seek to identify specific modifiable factors that influence fetal growth patterns and disease development in the postnatal period.

Acknowledgments

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