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Trajectories of Childhood Blood Pressure and Adult Left Ventricular Hypertrophy: the Bogalusa Heart Study

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Abstract

This longitudinal study aims to characterize longitudinal BP trajectories from childhood and examine the impact of level-independent childhood BP trajectories on adult left ventricular hypertrophy (LVH) and remodeling patterns. The longitudinal cohort consisted of 1,154 adults (787 whites and 367 blacks) who had repeated measurements of BP 4–15 times from childhood (4–19 years) to adulthood (20–51 years) and assessment of echocardiographic LV dimensions in adulthood. Model-estimated levels and linear slopes of BP at childhood age points were calculated in one-year intervals using the growth curve parameters and their first derivatives, respectively. Linear and nonlinear curve parameters of BP showed significant race and sex differences from age 15 years onwards. Adults with LVH had higher long-term BP levels than adults with normal LVM in race-sex groups. Linear and nonlinear slope parameters of BP differed consistently and significantly between LVH and normal groups. Associations of level-independent linear slopes of systolic BP with adult LVH were significantly inverse (odds ratio=0.75~0.82, p=0.001~0.015) in pre-adolescent children of 4–9 years but significantly positive (odds ratio=1.29~1.46, p=0.001~0.008) in adolescents of 13–19 years, adjusting for covariates. These associations were consistent across race-sex groups. Of note, the association of childhood BP linear slopes with concentric LVH was significantly stronger than that with eccentric LVH during the adolescence period of 12–19 years. These observations indicate that the impact of BP trajectories on adult LVH and geometric patterns originates in childhood. Adolescence is a crucial period for the development of LVH in later life, which has implications for early prevention.

Keywords

Blood pressure; Childhood; Left ventricular hypertrophy; Longitudinal study; Growth curve

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DISCLOSURES

None.

INTRODUCTION

Left ventricular hypertrophy (LVH), an increase in left ventricular mass (LVM), is an independent predictor of cardiovascular disease (CVD) and is regarded as a subclinical surrogate cardiovascular endpoint [1–3]. LVH is a cardinal manifestation of hypertensive organ damage; concentric LVH which is considered to carry the higher risk for CVD events than eccentric LVH [2] is the predominant form in hypertensive patients [4]. Numerous clinical and epidemiologic studies have demonstrated an important role of elevated blood pressure (BP) in the development of LVH [5–8]. Extensive observations have indicated that elevated BP levels play a pivotal role in the development of concentric LVH through chronic hemodynamic overload and increased central pressure [6,9]. Our early studies found that adult concentric LVH had a stronger association than adult eccentric LVH with BP measured in both childhood and adulthood [9,10].

The concept of “childhood origins” of CVD is supported by numerous publications from large-scale population-based cohorts of CVD risk factors followed since childhood [11]. Previous studies, including those from the Bogalusa Heart Study, have established the notion that elevated BP early in life is a risk factor for adult LVH and CVD [9,12–14]. It is well-known that high levels of childhood BP are associated with increased LVM in both children and adults [15,16]. In addition to absolute levels of BP, evidence has been increasingly accumulated that longitudinal BP growth trajectories during particular periods of life also has impact on development of subclinical CVD [14,17–19].

Despite a large body of literature detailing the relationship between BP and LVH, there are significant gaps in understanding of the association between childhood BP trajectories and adult LVH. BP levels at different ages in childhood represent the BP status at a particular time point, and the instantaneous rate of increase in BP at specific childhood age windows reflects the velocity of an increase in BP. A steeper increase in the slope of BP in childhood predicts a higher BP later in life. We hypothesized that the increasing slopes of BP in childhood are predictive of adult LVH risk and abnormal LV geometry, independent of BP levels. Using data from the Bogalusa Heart Study, we aimed to characterize longitudinal BP trajectories from childhood and to examine the association of level-independent trajectories of childhood BP with adult LVH and ventricular wall geometry.

MATERIALS AND METHODS

All data and materials have been made publicly available at the NHLBI Biologic Specimen and Data Repository and can be accessed at https://biolincc.nhlbi.nih.gov/studies/bhs.

Study cohort

The Bogalusa Heart Study, a series of long-term observations in a semi-rural biracial (65% white and 35% black) community in Bogalusa, Louisiana, was founded by Dr. Gerald Berenson in 1973. The study focused on understanding the early natural history and childhood risk factors of CVD [20]. In the community of Bogalusa, Louisiana, nine cross-sectional surveys of children aged 4–19 years and eleven cross-sectional surveys of adults aged 20–51 years who were previously examined as children were conducted between 1973

and 2010. Linking these repeated cross-sectional examinations conducted every 2–3 years provides serial observations from childhood to adulthood in the same individuals. In the longitudinal cohort, 2,732 adult subjects (1,772 whites and 960 blacks; 44.9% males) had been examined 4–15 times for BP and body mass index (BMI) (7.0 times on average, at least 2 times in childhood and at least 2 times in adulthood). Among these 2,732 individuals, 1,154 adult subjects (787 Whites and 367 Blacks; 42.5% males, mean age=42.0 years) who had echocardiographic LV dimensions measured in adulthood during 2004 to 2010 formed the current longitudinal study cohort.

At each examination, written informed consent was obtained from each study participant or from a parent/guardian in those under 18 years of age. Study protocols were approved by the Institutional Review Board of the Tulane University Health Sciences Center.

General examinations

All measurements were obtained by trained staff members who followed a standard protocol [20]. BMI (weight in kilograms divided by height in meters squared) was used as a measure of adiposity. BP levels were measured between 8:00 AM and 10:00 AM on the right arm of study participants in a relaxed, sitting position by 2 trained observers (3 replicates each). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded using a mercury sphygmomanometer. The fourth Korotkoff phase was used for DBP in children and adults to avoid bias resulting from using different phases for DBP. The mean values of the 6 BP readings obtained at each visit were used for the current analysis. A SBP/DBP value of 140/90 mm Hg was assigned to hypertensive patients (n=473) who received antihypertensive drug therapy. Information on smoking and alcohol use was obtained by means of a staff-administered standardized questionnaire. Current smoking and drinking were defined as smoking at least one cigarette per day and consuming alcohol every day, respectively, during the prior 12 months.

Echocardiographic LV structure measurements

LV dimensions were assessed using 2-dimensional guided M-mode echocardiography with 2.25- and 3.5-MHz transducers according to American Society of Echocardiography recommendations [21]. Parasternal long- and short-axis views were used for measuring LV end-diastolic and end-systolic measurements in duplicate, and the mean was computed. LVM was calculated using a necropsy-validated formula based on thick-wall prolate ellipsoidal geometry [22]. To account for body size, LVM was indexed for body height ($m^{2.7}$) as LVM index (LVMI). LV relative wall thickness (RWT) was calculated as septal wall thickness plus posterior wall thickness divided by LV end-diastolic diameter [23]. The presence of LVH was defined by LVMI>46.7 $g/m^{2.7}$ in women and >49.2 $g/m^{2.7}$ in men; LV geometry was considered concentric when RWT was >0.42 [24]. Four patterns of LV geometry were defined: 1) normal LV geometry (normal RWT with no LVH, n=914), 2) concentric remodeling (CR, increased RWT but no LVH, n=97), 3) eccentric hypertrophy (EH, normal RWT with LVH, n=91), and 4) concentric hypertrophy (CH, increased RWT with LVH, n=52) [23–26]. Adult LVH was also defined by cut-offs of 115 (male)/95 (female) of LVM indexed to body surface area (BSA, g/m^2) [27].

Statistical methods

Nonlinear growth curve parameters of BMI and BP measured at multiple time points from childhood to adulthood were estimated using a random-effects mixed model by SAS proc MIXED, as previously reported [28,29]. The mixed model incorporates fixed and random effects and allows the intercept, linear and nonlinear parameters to vary from individual to individual. The random effect coefficients represent the difference between the fixed effect parameters and the observed values for each individual. This model allows for repeated measurements and different numbers of unequally spaced observations across individuals. The mixed linear model computes maximum likelihood estimates of curve parameters, generating 2,732 different sets of curve parameters for all the study participants. The model selection was based on the Akaike's information criterion (AIC) [30]. The most parsimonious model was selected based on p-values of the independent variable (age) at a significance level of 0.05. Age and its higher-order terms were included one by one for model building. The higher-order terms of age were not included in the model if they were not significant, or made lower-order terms not significant, or did not improve the goodness-of-fit of the model based on AIC values. Cubic curves were fitted for SBP, DBP and BMI in race-sex groups.

$$BP_i = (\beta_0 + b_{0i}) + (\beta_1 + b_{1i})\ age + (\beta_2 + b_{2i})\ age^2 + (\beta_3 + b_{3i})\ age^3 + \varepsilon$$

where $\beta = (\beta_0, \beta_1, \beta_2, \beta_3)'$ is a vector of fixed effect parameters, $b = (b_0, b_1, b_2, b_3)'$ is a vector of random effect parameters, and ε is an unknown error term. Age was centered to the mean age (20.1 years) to remove the collinearity of age with its higher-order terms. The term age^2 was divided by 10 and age^3 by 20 to improve the model fitting.

Supplemental Figure S1 shows the SBP growth curve of a white male participant as an example to describe the calculation of model-estimated linear slopes of BP in childhood. The levels and linear slopes (tangent lines as shown in solid lines) of childhood BP during the 4–19-year age period were calculated using each individual's curve parameters and their first derivatives, respectively. Levels and linear slopes of childhood BMI for each individual were calculated in the same manner. Prior to logistic regression analyses, childhood linear slope values of SBP, DBP and BMI at each age point were adjusted for their corresponding levels by regression residual analyses, then standardized with Z-transformation (mean=0, SD=1) by race-sex group to avoid collinearity of levels and linear slopes in the same model.

The significance of difference in mean levels of study variables and curve parameters between race-sex groups was tested using analysis of covariance in a generalized linear model. Based on our experience, a sufficient number of participants are needed for valid growth curve model fitting and reliable parameter estimation. Therefore, 2,732 individuals who had BP and BMI measured 4–15 times were used for growth curve model construction. Then, the derived curve parameters of BP, including levels and linear slopes, at childhood ages were used for the association analyses with LVH in 1,154 adults. A logistic regression model was used to examine the association of linear slopes of BP at childhood age points with adult LVH, adjusting for adult age, sex, race, adult BP, adult smoking and alcohol use, and childhood levels of BMI and BP at age points. Adult BMI was not included in the

regression models because it was highly correlated with childhood BMI. The adjustment for childhood BMI had substantially removed the impact of adult BMI on LVH.

RESULTS

Table 1 summarizes the study variables by age periods in race-sex groups. BMI had sex differences from adolescence to mid adulthood in Blacks (males<females) and during adolescence in Whites (males>females), and BMI had race difference from adolescence to mid adulthood in females only (Blacks>Whites). SBP showed significant sex difference (males>females) and race difference (blacks>whites) during adolescence; DBP showed the same trends in sex and race differences during adulthood. Blacks and males had significantly higher LVM and LVMI ($g/m^{2.7}$) values than Whites and females, respectively, with a borderline significance for race difference in LVM among males. RWT had significant sex difference in Whites (males>females) and race difference in females (Blacks>Whites). LVH, concentric and eccentric LVH defined by LVMI ($g/m^{2.7}$) showed significant race differences for both sexes, while only eccentric LVH had significant sex differences in both Whites and Blacks. LVH was also defined by LVMI indexed to BSA (g/m^2). The prevalence of LVH was 2.9% in white males, 5.6% in white females, 11.5% in black males and 10.5% in black females, with similar trends in race and sex differences to those in the prevalence defined by LVMI ($g/m^{2.7}$).

Figure 1 presents longitudinal trajectories of BP from childhood to adulthood in 2,732 individuals by race-sex group. BP did not differ markedly during the age range of 5–14 years. The growth curves of SBP and DBP were separated from around 15 years and beyond, with males having higher levels and slopes of SBP and DBP than females before 45 years in Blacks and before 50 years in Whites. There was a crossover between sexes during the previously mentioned age points for both Blacks and Whites. Curve parameters (β_0+b_0, β_1+b_1, β_2+b_2 and β_3+b_3) were all different from 0 ($p<0.001$) except $\beta_2+b_2=-0.05$ ($p=0.023$) and $\beta_3+b_3=0.0001$ ($p=0.965$) for DBP in black males. β_0+b_0 is the intercept (the level of BP at age of 20.1) because age was centered to this mean value; β_1+b_1 describes the linear slopes (the tangent lines) at age point of 20.1; β_2+b_2 describes rate of increase for 20–35 years; and β_3+b_3 describes change in rate of increase for 36–51 years of age. Race and sex differences in the curve parameters were all significant except for the sex difference in β_0+b_0 for DBP in Blacks and race difference in β_0+b_0 for DBP in males as presented in Supplement Table S1.

Table 2 and Supplement Table S2 present curve parameters in those with LVH and their counterparts who had a normal LVM by race and sex. All curve parameters (β_0+b_0, β_1+b_1, β_2+b_2 and β_3+b_3) were significantly different from 0 ($p<0.001$). The curve parameters (β_0+b_0, β_1+b_1 and β_2+b_2) of BP were all significantly greater in the LVH group than in the normal group; however, the curve parameters (β_3+b_3) were significantly smaller in the LVH group than in the normal group. To evaluate the potential influence of antihypertensive medication on β_3+b_3, a sensitivity analysis was conducted by removing hypertensives under treatment. Values of β_3+b_3 were slightly changed in both SBP and DBP (data not shown).

Figure 2 presents the longitudinal trajectory patterns of SBP in those with LVH and a normal LVM by race and sex. Compared with the normal group, those with LVH had higher SBP levels from childhood to adulthood and higher slopes in young adulthood (20–35 years), but lower slopes in mid adulthood (36–51 years). The cubic growth curves of DBP had similar differences in trajectory patterns between LVH and normal groups (Supplement Figure S2). The growth curves of both SBP and DBP during pre-adolescence and adolescence age periods were consistently higher in LVH than in normal adults in the four race-sex groups.

Supplement Table S3 shows model-estimated linear slopes of SBP during childhood (4–19 years) in one-year intervals by race-sex group. The linear slopes of SBP during childhood were calculated for each individual based on the first derivatives of their curve parameters. The linear slopes of SBP were all significantly positive and decreased as childhood age increased. The SBP linear slopes during childhood showed consistently significant race (Blacks>Whites) and sex (males>females) differences. The model-estimated linear slopes of childhood DBP showed similar age-, race- and sex-related trends in differences (Supplement Table S4). Correlations between the model-estimated levels and slopes of BP at childhood age points were significantly negative ranging from −0.47 to −0.13 ($p<0.001$) for SBP and from −0.43 to −0.05 ($p<0.05$) for DBP during the age period (4–7 years) and significantly positive ranging from 0.10 to 0.54 ($p<0.001$) for SBP and from 0.16 to 0.37 ($p<0.001$) for DBP during the age period (9–19 years). The level-slope correlation patterns were consistent in race-sex groups with slight changes in the correlation coefficient size.

Figure 3 shows standardized odds ratios (OR) and 95% confidence intervals (CI) of model-estimated levels and level-adjusted linear slopes of childhood SBP for adult LVH, following adjustment for adult age, sex, race, adult SBP, adult smoking and alcohol use, and childhood BMI levels. The OR values of SBP linear slopes increased with childhood age. The ORs were significantly lower than 1.0 during the pre-adolescence period of 4–9 years (OR=0.75~0.82, p=0.001~0.015), whereas the ORs were significantly greater than 1.0 during the adolescence period of 13–19 years (OR=1.29~1.46, p=0.001~0.008). In addition, model-estimated levels of SBP were significantly associated with adult LVH during the age period (4–6 years), but the ORs were not significant during the age period (7–19 years). Supplement Figure S3 shows the ORs of levels and level-adjusted linear slopes of childhood SBP for adult LVH by race-sex group. The trend in ORs was similar in white females and black females. Supplement Figures S4 and S5 show the associations of model-estimated levels and level-adjusted linear slopes of childhood DBP with adult LVH in the total sample and race-sex groups, respectively. The trends in ORs of levels and slopes of DBP were similar to the trends in ORs of SBP. The associations of model-estimated levels and level-adjusted linear slopes of childhood BP with adult LVH were also analyzed with additional adjustment for adult BMI. The trends in ORs across childhood ages were very much the same as those in Figure 3, Supplement Figures S3, S4 and S5 with slight changes in OR values (data not shown).

Figure 4 shows ORs of model-estimated, level-adjusted linear slopes of SBP for adult eccentric and concentric LVH by childhood age, following adjustment for adult age, race, sex, adult SBP, adult smoking and alcohol use, and childhood BMI levels. The trends in ORs for eccentric and concentric LVH were substantially similar with those for overall LVH. Of

interest, the associations of SBP linear slopes with concentric LVH were significantly stronger than those with eccentric LVH during the adolescence period of 12–19 years. The ORs of model-estimated, level-adjusted linear slopes of DBP were similar for adult eccentric and concentric LVH (Supplement Figure S6).

DISCUSSION

In this community-based longitudinal cohort with repeated measurements of BP from childhood to midlife, we demonstrated that linear slopes of BP at different ages during childhood were significantly associated with adult LVH, especially concentric LVH, independent of childhood and adult BP levels. Despite the overwhelming evidence regarding the predictive value of childhood BP levels for LVH in later life, no previous studies have concurrently considered the importance of linear slopes and levels of BP at different age periods during childhood for the prediction of adult LVH. The observations in our study provide new insights into the early life origins of LVH and emphasize the importance of the level-independent BP trajectories during the adolescence period for later life LVH risk.

The association of BP with excessive cardiac growth occurs in children and adolescents [13,15,16,31], and elevated levels of BP in early life are significantly associated with adult LVH and LV geometric patterns [12]. The Bogalusa Heart Study has reported that childhood BP levels significantly predict midlife LVH and LV geometric patterns [9,32]. Recent studies have shown that BP trajectories are the strongest predictors, among various BP measures, of cardiovascular morbidity and mortality, and all-cause mortality [33,34]. The British birth cohort reported that rate of increase in SBP in adults aged 43–53 years was associated with greater LVM index at follow-up, independent of SBP levels [18]. The CARDIA study found that higher BP trajectories among young adults were associated with an increased risk of coronary artery calcification in middle age [17]. A recent publication from the Georgia Stress and Heart study has demonstrated the association between SBP trajectories derived from childhood and subclinical cardiovascular risk in young adulthood [19].

To date, only limited information is available to detail trajectory parameters of longitudinal BP profiles in childhood and their relationship to the development of LVH in later life. In the current study, we found that β_0+b_0 (levels at age 20.1), β_1+b_1 (overall linear slope) and β_2+b_2 (rate of increase in 20–35 years of age) were all significantly greater in the LVH than in normal groups, whereas the values of β_3+b_3 (change in rate of increase in 36–51 years of age) were significantly smaller in the LVH group. The lower values of β_3+b_3 among LVH subjects might be affected by two possible factors: anti-hypertensive medication and regression-to-the mean phenomenon. To evaluate the potential influence of antihypertensive medication on β_3+b_3, a sensitivity analysis was performed by removing hypertensives under treatment. The values of β_3 were slightly changed for both SBP and DBP and still significantly smaller in the LVH groups in all race-sex groups. Obviously, the possible explanation is that the change in rate of BP increase tended to slow down in middle-aged adults with LVH because there were more adults in this group who had high levels of BP in this age period. Taken together, the results of the current study indicate that the increase in BP during early life is important for the development of LVH.

The associations between BP slopes at different childhood ages and adult LVH varied during early childhood and adolescence. Importantly, these associations were independent of childhood BMI and BP levels at the same age points as well as adult BP levels. Limited literature is available for comparison in this regard. We found that the model-estimated linear slopes of BP during the adolescence age period (11–19 years) were significantly and positively associated with adult LVH in middle age, indicating that adolescence is a crucial period for the development of LVH in later life. The sexual maturation process during the adolescence period is characterized by a complex interplay among various gonadal and adrenal steroid hormones, growth hormones, and growth factors. These factors change dramatically during this age period [35,36]. Previous studies, including the Bogalusa Heart Study, indicated that insulin, lipids, and BP during adolescence were mediated by these factors, independent of adiposity [37–39]. The findings from this and previous studies suggest that adolescence is a critical temporal window for BP control to reduce the risk of adult LVH [34–36,39–45].

In contrast to the adolescence period, the model-estimated linear slopes of BP in pre-adolescence showed significantly inverse associations with adult LVH. This observation is an important and intriguing finding that has not been previously reported and warrants further study to confirm in other populations. The observed associations of greater rates of BP increase in pre-adolescence with a lower risk of developing adult LVH could be statistically explained by regression-to-the mean phenomenon. BP levels were found in this cohort to be significantly and negatively correlated with BP slopes at pre-adolescence age points of 4–7 years, that is, pre-adolescent children who had lower BP levels tended to have a greater rate of increase and vice versa. This phenomenon is known as regression-to-the mean [46]. It is well established in this and previous studies that higher childhood BP levels are associated with a greater risk of adult LVH. Children who had a greater rate of BP increase represented a subgroup who had lower BP levels and thus had a lower risk of developing LVH in later life. On the other hand, correlations between BP levels and slopes became significantly positive in age groups of 9–19 years in this study cohort, with the correlation patterns being consistent in race-sex groups. The complex relationship between BP levels and slopes in different childhood age periods might contribute to the divergent associations between childhood BP trajectories and adult LVH observed in the current study. This is a new research area and deserves an in-depth investigation.

Echocardiography allows identification of different forms of LV geometric remodeling, including eccentric and concentric hypertrophy with disproportionate septal thickness. Although the significance of the different forms is not yet entirely defined, concentric LVH is considered to carry the highest risk for cardiovascular events [2,47] and is the predominant form in hypertensive middle-aged and elderly patients [4]. Previous studies, including ours, have reported stronger associations between elevated BP levels and concentric LVH [6,8,9,32,48]. We noted in this study that the association of BP slopes in adolescence with concentric LVH was significantly stronger than that with eccentric LVH as shown in Figure 4. The findings of the current study point to the importance of controlling rapid increase in BP during adolescence for reducing the risk of adult concentric LVH and subsequently the prevention of CVD in later life.

This community-based longitudinal cohort study provides a unique opportunity to examine the association between childhood BP trajectories and adult LVH; it also has certain limitations. First, the forced BP values of hypertensives on pharmacologic treatment may result in bias in the growth curve parameter estimation to some extent because these individuals represented a subgroup who, without treatment, would be expected to have the highest BP levels. Second, the assessment of cut-off values of BP in adolescence for adult LVH is an important research area. However, the sample size of the current study cohort is limited to conduct such an analysis. We plan to do this analysis in collaboration with other large-scale longitudinal studies followed since childhood with a sufficient statistical power. Third, LVMIs indexed to height ($g/m^{2.7}$) and BSA (g/m^2) were used to define LVH in the current study, and the prevalence of LVH by the two indexation methods showed a substantial difference (12.4% vs 6.5%). To be consistent and comparable with our previous studies from the Bogalusa Heart Study cohort [9,10,12,16,26,32], the results of LVMI ($g/m^{2.7}$) were reported in this study.

PERSPECTIVES

The current study characterized the black-white and sex specific BP growth curve patterns from 4 to 51 years of age in a longitudinal cohort of the Bogalusa Heart Study. The comparison and association analyses of the curve parameters demonstrated that higher levels and rates of increase of BP during adolescence and young adulthood were associated with adult LVH and its remodeling patterns. In contrast to the adolescence period, the present study found that pre-adolescent children with greater rates of BP increase had a lower risk of developing LVH in later life. These observations suggest that adolescence is a critical age window for BP control to reduce the LVH risk. These findings underscore the importance of controlling BP in early life to prevent the development of LVH and CVD later in life.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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ABBREVIATIONS

BMI	body mass index
CVD	cardiovascular diseases

BP	blood pressure
SBP	systolic blood pressure
DBP	diastolic blood pressure
LVH	left ventricular hypertrophy
LVM	left ventricular mass
LVMI	left ventricular mass index
RWT	relative wall thickness

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Novelty and Significance

What Is New?

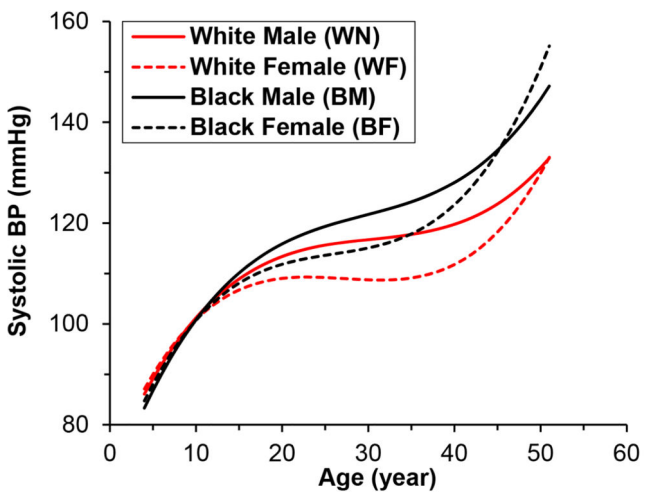
- Race- and sex-specific growth curves of BP from 4 to 51 years of age were characterized in a longitudinal cohort.
- Significant impact of level-independent childhood BP trajectories on adult LVH and remodeling patterns was found in race-sex groups.

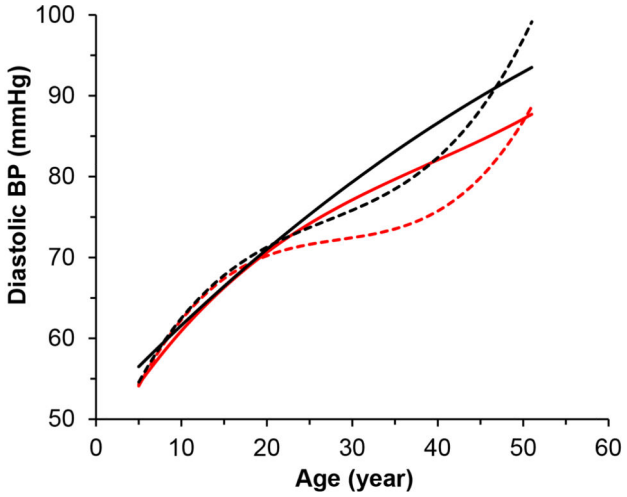
What Is Relevant?

- These observations suggest that the adolescence period is a critical age window for BP control to reduce the LVH risk. These findings underscore the importance of controlling childhood BP to prevent the development of LVH and CVD later in life.

Summary

- This longitudinal study characterized the black-white and sex specific BP growth trajectories from childhood and indicated that the impact of BP trajectories on adult LVH and geometric patterns originates in childhood. Adolescence is a crucial period for the development of LVH in later life, which has implications for early prevention.





WM: SBP = 113.5 + 0.63 age - 0.44 age^2 + 0.03 age^3
WF: SBP = 109.1 + 0.19 age - 0.41 age^2 + 0.04 age^3
BM: SBP = 116.0 + 0.87 age - 0.46 age^2 + 0.03 age^3
BF: SBP = 111.9 + 0.49 age - 0.39 age^2 + 0.04 age^3

WM: DBP = 70.7 + 0.78 age - 0.16 age^2 + 0.006 age^3
WF: DBP = 70.3 + 0.39 age - 0.28 age^2 + 0.02 age^3
BM: DBP = 71.1 + 0.88 age - 0.05 age^2 + 0.0001 age^3
BF: DBP = 71.4 + 0.56 age - 0.21 age^2 + 0.02 age^3

Figure 1. Growth curves of blood pressure by race-sex group

Curve parameters were all different from 0 ($p<0.001$) except −0.05 ($p=0.023$) and 0.0001 ($p=0.965$) for DBP in black males (see detailed information on the curve parameters in Supplemental Table S1).

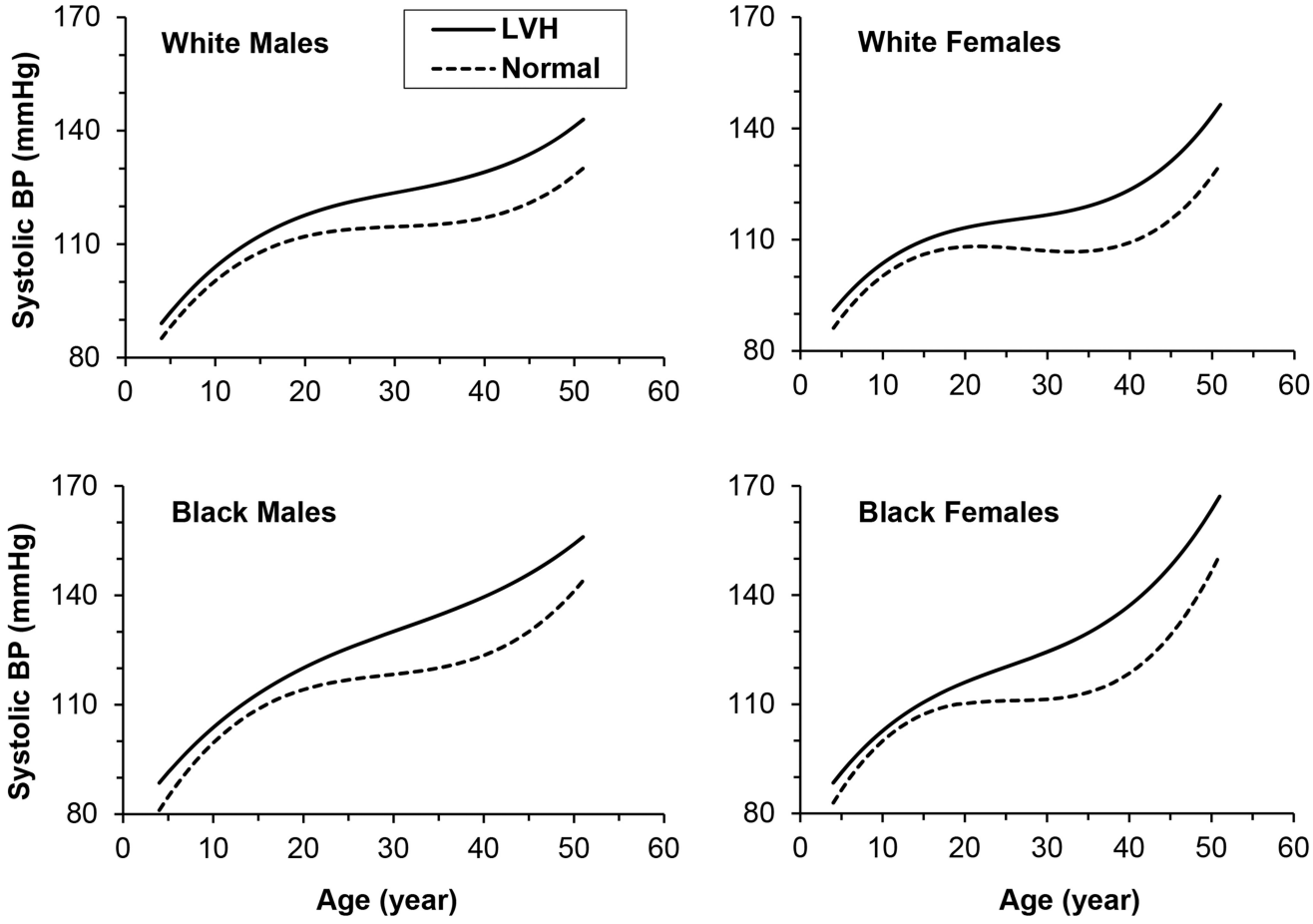


Figure 2.
Growth curves of systolic blood pressure in adults with and without LVH by race and sex

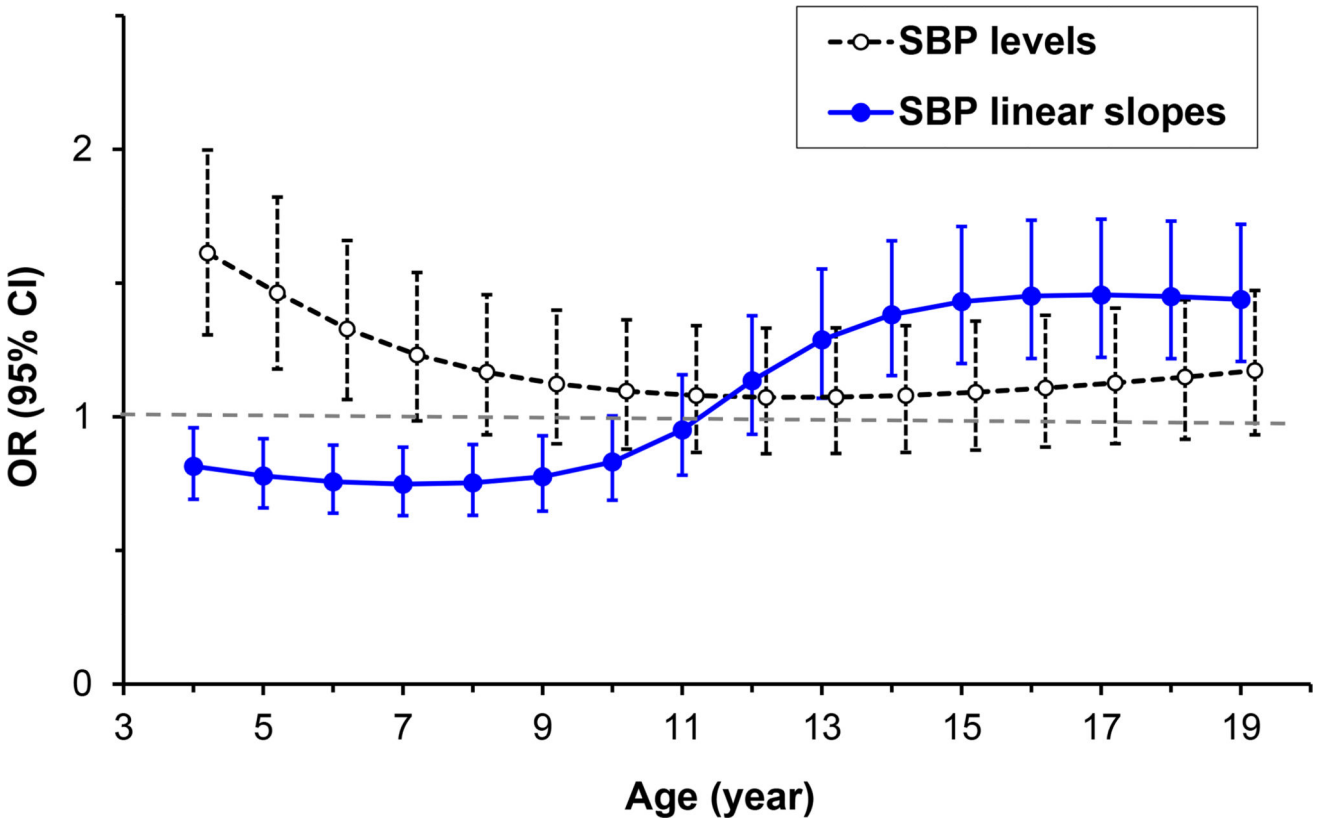


Figure 3.

Odds ratio (OR) and 95% confidence interval (CI) of model-estimated levels and level-adjusted linear slopes of SBP for adult LVH by childhood age, adjusting for adult age, race, sex, adult SBP, adult smoking and alcohol use, and childhood BMI levels

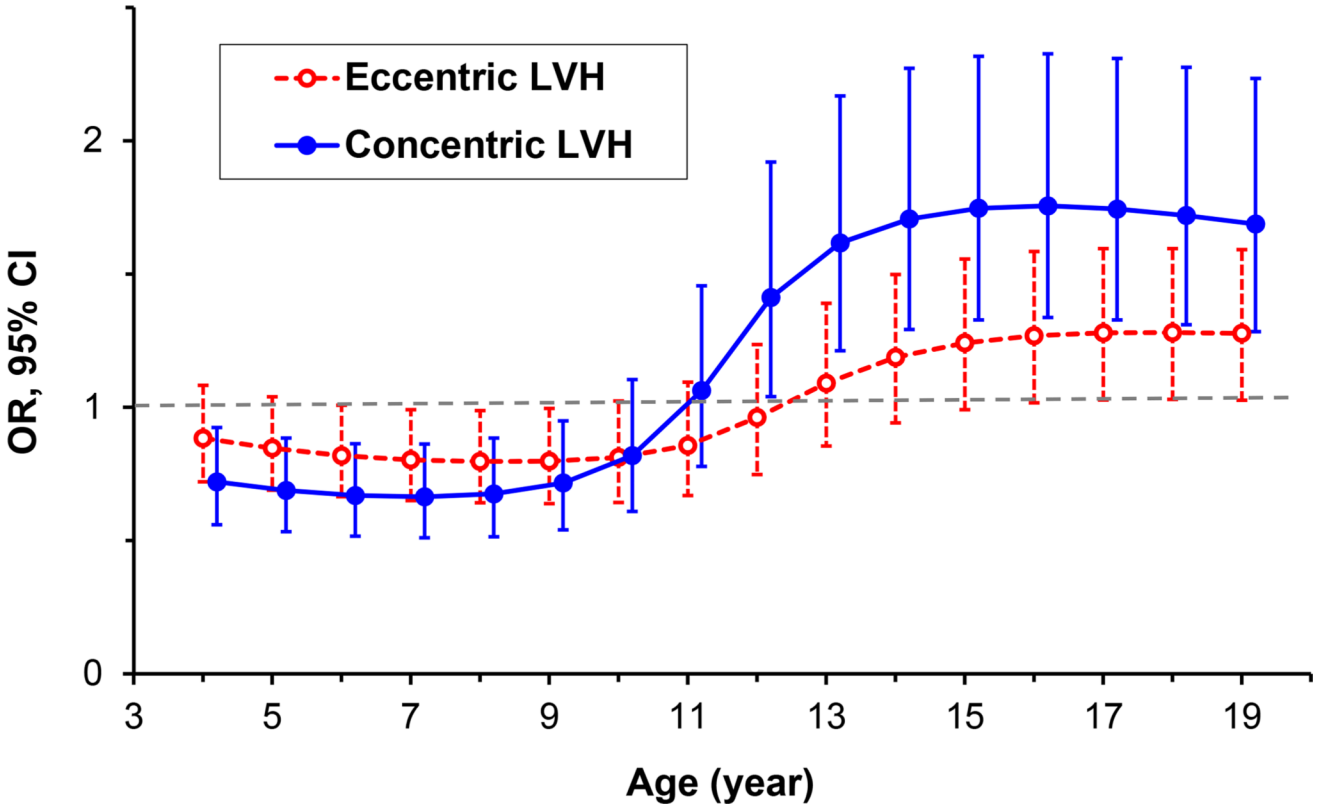


Figure 4.

Odds ratio (OR) and 95% confidence interval (CI) of model-estimated, level-adjusted linear slopes of SBP for adult eccentric and concentric LVH by childhood age, adjusting for adult age, race, sex, adult SBP, adult smoking and alcohol use, and childhood BMI levels

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Table 1

Characteristics in means (SD) of study variables by race and sex

Variable	White		Black		P for Race Difference	
	Male	Female	Male	Female	Male	Female
Pre-adolescence (4–11 yr)						
N	232	291	102	163		
Age (yr)	8.1 (2.0)	7.9 (2.0)	8.4 (2.1)	8.2 (2.0)	0.249	0.202
BMI (kg/m^2)	16.4 (2.1)	16.6 (2.8)	16.7 (2.6)	16.5 (2.8)	0.289	0.881
Systolic BP (mmHg)	97.0 (8.6)	96.2 (8.2)	96.4 (9.0)	95.6 (9.0)	0.534	0.474
Diastolic BP (mmHg)	59.0 (7.4)	58.9 (7.0)	60.7 (7.4)*	58.5 (7.9)	0.059	0.608
Adolescence (12–19 yr)						
N	332	422	147	213		
Age (yr)	16.8 (1.9)	16.8 (2.0)	17.1 (1.9)	17.1 (1.7)	0.114	0.075
BMI (kg/m^2)	22.1 (4.2)*	21.4 (4.1)	22.2 (4.8)*	23.3 (4.8)	0.859	<0.001
Systolic BP (mmHg)	112.2 (9.6)†	108.8 (8.5)	114.9 (10.7)†	110.9 (8.9)	0.007	0.003
Diastolic BP (mmHg)	68.9 (8.1)	69.7 (7.6)	68.8 (9.7)	70.5 (8.6)	0.925	0.194
Young Adulthood (20–35 yr)						
N	333	440	140	217		
Age (yr)	31.9 (3.4)†	32.6 (2.9)	31.3 (4.0)*	32.3 (3.2)	0.101	0.196
BMI (kg/m^2)	27.5 (5.0)	26.9 (7.0)	27.0 (6.6)†	30.1 (8.0)	0.402	<0.001
Systolic BP (mmHg)	116.5 (10.8)†	108.9 (12.0)	121.5 (15.8)*	117.4 (16.3)	<0.001	<0.001
Diastolic BP (mmHg)	78.1 (8.4)†	73.3 (8.7)	79.6 (11.7)	78.3 (11.7)	0.129	<0.001
Mid Adulthood (36–51 yr)						
N	296	376	128	174		
Age (yr)	43.7 (3.8)	43.5 (3.7)	43.8 (3.6)	43.8 (3.5)	0.851	0.457
BMI (kg/m^2)	29.8 (5.6)	29.0 (6.8)	29.8 (7.3)†	32.6 (8.4)	0.982	<0.001
Systolic BP (mmHg)	122.4 (13.9)†	115.7 (14.7)	134.0 (19.1)	129.6 (20.5)	<0.001	<0.001
Diastolic BP (mmHg)	83.9 (8.7)†	79.1 (8.5)	89.7 (12.3)*	86.7 (11.7)	<0.001	<0.001

Variable	White		Black		P for Race Difference	
	Male	Female	Male	Female	Male	Female
Adulthood (20–51 yr)						
N	343	444	148	219		
Age (yr)	42.3 (5.1)	41.9 (5.3)	42.2 (5.4)	41.5 (5.7)	0.866	0.340
Smokers, n (%)	90 (26.2)	129 (29.1)	58 (39.2) *	63 (28.8)	0.006	1.000
Alcohol users, n (%)	113 (32.9) †	83 (18.7)	75 (50.7) †	48 (21.9)	<0.001	0.381
Hypertension, n (%)	91 (26.5) †	84 (18.9)	43 (29.1)	65 (29.7)	0.138	<0.001
LVM (g)	173.3 (46.0) †	124.8 (40.4)	181.4 (56.2) †	137.6 (49.1)	0.095	<0.001
LVMI (g/m$^{2.7}$)	36.6 (9.3) †	33.1 (11.0)	39.1 (12.2) *	36.4 (12.5)	0.015	0.001
RWT (cm)	0.33 (0.08) *	0.32 (0.08)	0.34 (0.08)	0.34 (0.09)	0.185	0.015
LVH, n (%)	32 (9.3)	36 (8.1)	36 (24.3)	39 (17.8)	<0.001	<0.001
Concentric LVH, n (%)	5 (1.5)	19 (4.3)	9 (6.1)	19 (8.7)	0.009	0.017
Eccentric LVH, n (%)	27 (7.9) †	17 (3.8)	27 (18.2) *	20 (9.1)	0.002	0.004

BMI=body mass index; BP=blood pressure; LVM=left ventricular mass; LVMI=left ventricular mass index; RWT=left ventricular relative wall thickness; LVH=left ventricular hypertrophy

* Sex difference within racial groups: p<0.05;

† p<0.01

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Table 2

Difference in curve parameters of systolic blood pressure in means (SD) between LVH and normal groups by race and sex

Curve Parameter	White		Black		P for Race Difference	
	Male	Female	Male	Female	Male	Female
N (LVH/Normal)	32/311	36/408	36/112	39/180		
β_0+b_0 (mmHg)						
LVH	118.3 (8.0) *	113.9 (6.4)	118.4 (6.9)	116.0 (7.3)	0.961	0.171
Normal	112.8 (6.4) †	108.7 (5.7)	115.7 (7.6) †	111.4 (6.1)	<0.001	<0.001
p ≠	<0.001	<0.001	0.058	<0.001		
β_1+b_1 (mmHg/yr)						
LVH	0.799 (0.379) *	0.550 (0.479)	1.109 (0.565)	0.934 (0.717)	0.009	0.007
Normal	0.597 (0.273) †	0.163 (0.340)	0.845 (0.496) †	0.467 (0.485)	<0.001	<0.001
p ≠	<0.001	<0.001	0.006	<0.001		
β_2+b_2 (mmHg/yr^2)						
LVH	−0.443 (0.151) †	−0.304 (0.155)	−0.318 (0.268)	−0.245 (0.210)	0.020	0.165
Normal	−0.442 (0.124) *	−0.422 (0.130)	−0.485 (0.234) †	−0.405 (0.161)	0.015	0.179
p ≠	0.959	<0.001	<0.001	<0.001		
β_3+b_3 (mmHg/yr^3)						
LVH	0.025 (0.012)	0.028 (0.019)	0.024 (0.029)	0.025 (0.034)	0.841	0.667
Normal	0.029 (0.009) †	0.040 (0.013)	0.034 (0.023) †	0.045 (0.022)	0.002	0.001
p ≠	0.018	<0.001	0.031	<0.001		

LVH=left ventricular hypertrophy

* Sex difference within racial groups: $p<0.05$;

† $p<0.01$

Curve parameters were all significantly different from 0 ($p<0.001$).

≠ P values for difference in βs between LVH and normal groups were adjusted for average age, with additional adjustment for β_0+b_0 for other curve parameters.