Serial Childhood BMI and Associations With Adult Hypertension and Obesity: The Fels Longitudinal Study

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Previous studies estimated critical periods of childhood BMI growth and linked these events to adult adiposity and cardiovascular health. We expand upon both results to link childhood BMI growth patterns with adult blood pressure (BP). Data from male and female participants in the Fels Longitudinal Study (FLS) were used to estimate childhood BMI growth curves, from which we isolate ages of childhood BMI divergence based upon adult BMI and BP measurements. Repeated measure analysis of variances models were used to estimate BMI growth curves from age 2 to age 17.5 based on both adult BMI (< 25 kg/m² or ≥ 25 kg/m²) and adult BP (< 120 mm Hg or ≥ 120 mm Hg for systolic BP (SBP); < 80 mm Hg or ≥ 80 mm Hg for diastolic BP (DBP)). Participants with lower body weight throughout childhood had lower SBP and DBP in early adulthood. Any relationships between childhood adiposity and adult body weight and BP disappeared by age 60. These results were independent of adult BMI and were observed in both men and women. Increased adult BP has its genesis in part from increased childhood BMI.

The existence of “critical periods” of adiposity growth—as measured by BMI—has been known for some time, corresponding with early childhood, adolescence and post-adolescence (1). The age patterns at which these landmarks occur have been previously explored for BMI (2). While associations between ages-of-attainment of growth milestones in children and adult adiposity have been previously established (3), little is known about the relationships between the timing of achieving these growth landmarks and adult blood pressure (BP). Thus, we aim to study the associations between childhood growth patterns, as measured by BMI, on adult systolic and diastolic BPs (SBP and DBP). Using serial childhood data from the Fels Longitudinal Study (FLS), we estimated childhood BMI growth curves, based on adulthood prehypertension and overweight status, to estimate the ages of divergence in BMI for those groups of participants, where divergence is defined as the age at which the average BMI levels for any two groups become significantly different.

METHODS AND PROCEDURES

Participants
This study examined predominantly white male and female participants of the FLS who were at least 20 years of age by the end of data collection on 30 June 2009. All participants in this study provided cardiac function measurements, including SBP and DBP, and also had long-term serial adiposity data from early childhood to around age 18. FLS participants are generally enrolled at birth and are not selected in regard to factors known to be associated with disease, body composition, or other clinical conditions. Children were examined semiannually (near their birthdays and 6 months following) until 18 years of age, whereas adults were examined every 2 years following their 18th birthday. All participants signed an informed consent statement, and procedures were approved by the institutional review boards at Wright State University and Virginia Commonwealth University.

Measurements
Anthropometric measurements were taken following recommendations in the Anthropometric Standardization Reference Manual (4). Weight is measured to 0.1 kg using a SECA scale. Height is measured to 0.1 cm using a Holtain stadiometer. BMI is then calculated as the ratio of weight to height (in meters) squared (kg/m²). SBP (mm Hg) and DBP (mm Hg) were measured in adult FLS participants, with both measures recorded as the average of three readings. Each reading was taken by rapidly inflating the arm cuff to the maximum level and deflating at a rate of 2 mm Hg per second, with 30 s rest between each determination. Measurements were taken in a standardized manner with participants in a seated position using a mercury sphygmomanometer as recommended by the Second National Heart Lung and Blood Institute Task Force on BP Control.

Statistical analyses
All analyses were performed using SAS/STAT software version 9.2 (SAS Institute, Cary, NC). Each subject is assigned an adult classification value based on overweight status (Yes if BMI ≥ 25 kg/m², No if BMI < 25) and prehypertensive status (Yes if SBP ≥ 120 mm Hg or DBP ≥ 80 mm Hg; no if SBP < 120 and DBP < 80) for a given age.
Thus, there are four possible classifications: not-overweight and non-pre-hypertensive, not-overweight and prehypertensive, overweight and not-pre-hypertensive, and overweight and prehypertensive. This classification was made for each subject at every tenth year, from age 20 to age 70; little data were available beyond this point. These classifications (rather than using hypertension and obese status) ensured sufficient numbers of participants in each of the four groups. A mixed-effect repeated measures analysis of variance is used to estimate childhood BMI growth trends. Childhood age, adult BP/BMI status, and the interaction between childhood age and adult BP/BMI status are included as fixed effects, while a random subject effect is used to account for within subject correlation, which is modeled using an auto-regressive covariance pattern. Childhood age is included as a third-degree polynomial since the pattern of BMI growth is nonlinear in childhood (2). This model allows for testing of divergence of mean childhood BMI growth profiles based on adult BP/BMI status. Multiple comparisons from ages 2 to 17.5 (at 0.5-year increments) are adjusted using the step-down approach to the Bonferroni correction (5), since there are 198 comparisons to be made for each model (six comparisons between the adult BP/BMI groups at 33 age levels). Since boys and girls have different BMI growth patterns, these models are analyzed separately for each gender. To account for possibly changing associations over time, these models are analyzed separately for each adult age group used for classification (age 20, 30, 40, 50, 60, and 70); however, we only show the results for age 20.

RESULTS

The number of participants with available data (both SBP/DBP and BMI) gradually decreases from 491 men and 566 women at age 20 to 70 men and 81 women at age 70. Both BP measurements generally increase with age in both men and women, where mean SBP for men crosses the prehypertensive boundary (SBP ≥ 120 mm Hg) at age 40 (mean 120.5 mm Hg, s.d. = 14.82, n = 308) and mean SBP for women crosses the prehypertensive boundary at age 60 (mean 131.5 mm Hg, s.d. = 21.42, n = 165); mean DBP for men and women does not cross the conventional prehypertensive threshold (DBP ≥ 80 mm Hg) at any adult age. Mean BMI for men and women increases until age 60, after which it decreases slightly. Based on these means, mean BMI for men crosses the overweight threshold (BMI ≥ 25 kg/m²) at age 30 (mean 25.9 kg/m², s.d. = 4.55, n = 380) and mean BMI for women crosses the overweight threshold at age 40 (mean 25.7 kg/m², s.d. = 6.34, n = 312).

Figure 1 shows the BMI growth curves for male and female participants from ages 2 through 17.5 years, where the participants are divided into four groups based upon their adult BP and BMI classifications at age 20: low SBP/DBP and low

![Figure 1](image-url)
BMI, low SBP/DBP and high BMI, high SBP/DBP and low BMI, or high SBP/DBP and high BMI. In each case, we see that adult participants classified with low SBP/DBP (and either low or high BMI) have significantly lower BMI in childhood than do adult participants classified with high SBP/DBP (and either low or high BMI). For male participants, the divergence (when differences become significant) starts at age 4.5 for SBP and at age 6 for DBP, whereas for female participants the divergence starts at age 4 for SBP and at age 8 for DBP, and from that point remains significant throughout childhood. In general, the childhood BMI growth patterns for adults with high SBP/DBP and either low or high BMI are not significantly different. These trends remain for adults aged 30 and 40, but are not observed for adult classifications at 60 and 70 years (results not shown).

DISCUSSION
The results suggest that increased levels of BP in adulthood (predominantly between ages 20 and 40) have their genesis in childhood adiposity development. The childhood BMI growth curves for participants with low adult BP generally began to diverge from the growth curves for participants with high adult BP somewhere between the adiposity rebound (age 5.4 for boys and 5.3 for girls) and the attainment of peak BMI velocity (ages 14.3 for boys and 13.0 for girls) as measured in the FLS cohort (2). For SBP, these divergences began as early as 4 years in men and women, whereas for DBP the divergences occurred a little later. While the potential for the beginnings of adult hypertension being observable in childhood growth patterns can be an important tool in forecasting future health, the actual mechanism through which this relationship manifests remains unclear.

Interestingly, though perhaps not surprisingly, the participants with the largest BMI in childhood were generally those who then developed high levels of BP in adulthood. This relationship was observed in both men and women and for both SBP and DBP, and most importantly was irrespective of adult BMI. This relationship was predominantly confined to younger adults (aged 20, 30, and sometimes 40 years). This evidence shows that childhood BMI has an effect on adult BP that is independent from adult BMI.

We did not find too many discrepancies between male and female participants in their relationships between childhood BMI growth and adult BP and BMI classifications. While earlier evidence showed a stronger relationship between changes in childhood BMI and adult BMI in women than in men (2), the observed relationships were similar across genders for both SBP and DBP. Further studies are needed to examine whether these associations are independent of gender as well as adult BMI.

Since the study participants were predominantly white, we make no claims that these results apply to other races or ethnicities. Adiposity measures other than BMI could have been used to characterize overweight participants, such as percentage body fat, waist circumference, or skinfold thickness. Indeed, the FLS database contains many of these measurements which could be potentially exploited. Relationships between childhood trends for these adiposity biomarkers and adult cardiovascular health will be studied elsewhere.

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DISCLOSURE
The authors declared no conflict of interest. See the online ICMJE Conflict of Interest forms for this article.

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