Lifecourse Adiposity and Blood Pressure Between Birth and 17 Years Old

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BACKGROUND
Childhood obesity creates a predisposition to develop adult hypertension and diabetes. We have identified distinct childhood adiposity trajectories associated with increased insulin resistance in early adolescence. Our aim was to investigate the relationship between these adiposity trajectories with childhood blood pressure (BP) development.

METHODS
Adiposity trajectories were previously developed by semiparametric modeling using serial anthropometry from birth to age 14 from the West Australian Pregnancy Cohort. The association between these trajectories and the prevalence of hypertension and prehypertension in 17 year olds was assessed by logistic regression. The relationship between adiposity trajectories and lifecourse BP was then assessed using linear mixed modeling.

RESULTS
The study includes 1,023 adolescents with BP measured at age 17 years. Three of 7 childhood adiposity trajectories (with accelerating adiposity) previously related to increased insulin resistance were associated with an increased risk of 17-year-old prehypertension or hypertension, compared to a referent trajectory of “stable average adiposity” (odds ratio (OR) = 2.9, P = 0.007; OR = 3.5, P < 0.001; and OR = 1.8, P = 0.041). One decelerating adiposity trajectory from high birth size was associated with significant interactions with age terms (P values = 0.025–0.084 and 0.011–0.027), indicating an altered slope and therefore, relative decline in lifecourse BP compared to the reference adiposity trajectory.

CONCLUSIONS
Adiposity trajectories (which comprise 27% of the population) were associated with an increased risk of hypertension/prehypertension in adolescence. Higher BP was detectable as early as 3 years old. Consequently, targeting fat loss (catch-down growth) in the preschool years may prevent the development of hypertension and related metabolic disorders.

Keywords: blood pressure; catch-down growth; childhood; development; hypertension; obesity; Raine Study; trajectories.

doi:10.1093/ajh/hpu266

A number of different growth trajectories from infancy may be associated with childhood obesity.1–3 Childhood obesity can originate very early in life; large for gestational age babies often progress to obese children and adults. Alternatively, low birth weight infants experiencing rapid weight gain may develop obesity late in childhood.

With increasing maternal obesity and related gestational diabetes, there is a greater incidence of large babies.4 Therefore, not only is the overall prevalence of childhood obesity increasing, but there is an increasing percentage of children following a growth trajectory leading to obesity.5

Childhood obesity is a major predictor of childhood blood pressure (BP).6 However, antecedent growth history additionally affects childhood BP, independent of childhood obesity. Low birth weight and accelerated postnatal growth are both independently associated with childhood BP5 and adult hypertension.8 The manner in which one grows to become overweight or obese may be just as important as being overweight.9,10 Despite knowledge that antecedent growth contributes to hypertension, we do not yet have overarching lifecourse data associating different childhood growth trajectories with the development of BP in childhood and late adolescent. Understanding the way BP develops to 17 years is meaningful, with BPs as early as 10 years of age predicting hypertension and metabolic disease in adult life.11,12

In the West Australian Pregnancy Cohort (Raine Study) (www.rainestudy.org.au), we have previously identified 3 distinct adiposity trajectories associated with relative insulin resistance at age 14.2 These trajectories are characterized by either the early onset of elevated adiposity or a rising accelerated adiposity trajectory. As insulin resistance and BP are interrelated,13,14 we hypothesized that these adiposity trajectories also affect longitudinal BP in childhood. We investigated the effect of these childhood adiposity trajectories on BP from birth to age 17 years (as measured at 8 time points). Further we determined whether these childhood adiposity trajectories are also related to BP, prehypertension, and hypertension at age 17 years.

METHODS

The Raine Cohort enrolled pregnant women ≤18th week of gestation (1989–1991) (N = 2,900) through the antenatal clinic.
at King Edward Memorial Hospital and nearby private clinics in Perth, Western Australia. Detailed clinical assessments were performed at birth. Birth information (including birth weight) was obtained from midwife records. The children were followed-up at 1, 2, 3, 5, 8, 10, 14, and 17 years of age (Supplementary Table 1) by questionnaire that included sociodemographic and behavioral data and by physical assessments including weight, height, and BP. The Human Ethics Committees (King Edward Memorial Hospital and/or Princess Margaret Hospital) approved all protocols. Informed, written consent to participate in the study was obtained from the mother of each child at enrollment and at each subsequent follow-up.

**Loss to follow-up at 17-year contact**

A total of 1,754 of the original 2,868 live births participated in the 17-year follow-up (414 deferred from participating, 480 had withdrawn, 184 were lost to follow-up, and 36 were deceased, but not due to cardiometabolic disease). Preterm births <37 weeks gestation were excluded. BP measurements were taken on 1,023 of the remaining participants. The original cohort more closely reflected those participants referred to a tertiary center, overrepresenting socially disadvantaged families. Socially disadvantaged participants were less likely to remain in the study beyond 3 years. The remaining study participants had sociodemographic characteristics equivalent to the general Western Australian population by the time they were 3 years old and 14 years old.

**Comparison of participants with nonparticipants in BP follow-up at age 17 years**

Those without BP data at 17 years had lower paternal education. There were no differences in family income, parity, birth weight, gestational age at delivery, or mother’s weight at delivery (Supplementary Table 2).

**Blood pressure**

Resting BP was measured at 1, 3, 5, 8, 10, 14, and 17 years old by trained nursing staff. The participants were rested in the seated posture for 5 minutes. Systolic BP (SBP) and diastolic BP (DBP) were measured using an oscillometric sphygmomanometer (Dinamap vital signs monitor 8100, DINAMAP XL vital signs monitor, DINAMAP PROCARE 100 (DPC100X – EN)) with appropriate cuff size for arm circumference. The Dinamap was set automatically to record every 2 minutes for 3 readings to age 10 and subsequently for 6 readings. After discarding the first reading, the average of subsequent readings was calculated.

**Anthropometry**

Anthropometry was measured at birth and each subsequent follow-up. Height and weight were measured by Holtain Infantometer and Stadiometer (nearest 0.1 cm) and Wedderburn Chair Scales (nearest 100 g). Body mass index (BMI) z-scores (2–14 years) and weight-for-height z-scores (<2 years) customized by age and gender were calculated using US Centers for Disease Control and Prevention (CDC) growth chart software recommended for Australian children. As CDC does not provide BMI z-scores for children ≤24 months old, weight-for-height z-scores were used as the best adiposity measurement available for this age. BMI-for-age and weight-for-length z-scores are age-appropriate surrogate measures for adiposity and highly correlated to each other. Using the same CDC reference populations, z-score values for adiposity at different ages were calculated. Z-scores (weight-for-height and BMI) have a consistent mean and SD, creating a consistent scale for approximating adiposity from birth to 14 years old.

**Other measures**

Socioeconomic status was assessed by maternal education. Maternal weight and height was measured by a trained midwife at 18 and 34 weeks gestation. Prepregnancy weight was self-reported by questionnaire. Occurrence of self-reported gestational diabetes was recorded by midwives 2 days after delivery. Gestational age was based on the date of the last menstrual period unless there was discordance with ultrasound biometry at the dating scan.

**Childhood adiposity trajectories.** Semiparametric mixed modeling was used on CDC-derived age and sex-adjusted adiposity z-scores to identify 7 adiposity trajectory groups (reproduced with the permission of Diabetes Care (Figure 1)).

The 3 childhood adiposity growth patterns previously identified as high risk for relative insulin resistance were lifelong high adiposity from birth to adolescence (z-score ≥1 at each time point) (trajectory 1) and 2 rising adiposity trajectories (trajectories 2 and 4).

Trajectory 1 ("stable high") consists of infants of above average birth weight (3.66, 95% confidence interval (CI) = 3.53 to 3.79 kg). Trajectory 2 ("rising to high") consists of accelerated gain in adiposity from average birth weight (3.41, 95% CI = 3.33–3.49 kg). Trajectory 4 ("rising to moderate") consists of accelerated gain in adiposity from below average weight (3.22, 95% CI = 3.14–3.29 kg).

The “reference group” (trajectory 5) (35% of the cohort) included participants for which age- and sex-adjusted BMI z-scores were approximately zero throughout childhood reflecting “normal” growth.

**Definition of hypertension and prehypertension in 17 year olds**

Hypertension was defined using adult criteria as a SBP ≥140 mm Hg and/or DBP ≥90 mm Hg. Prehypertension was defined as SBP between 120 and 139 mm Hg and/or DBP between 80 and 89 mm Hg, according to the Joint National Committee on Prevention, Detection, Evaluation and Prevention of High Blood Pressure (JNC7) criteria.

**Statistical methods**

The effect of childhood adiposity trajectories on BP at 17 years old. Linear regression models with the outcome of SBP and DBP were performed. Logistic regression was
performed for the outcome of hypertension. As there were insufficient numbers with hypertension alone for this analysis, the prehypertensive and hypertensive groups were combined and are referred to as elevated BP. The outcome was therefore hypertensive status (normotensive vs. elevated BP). All linear and logistic regression models included the adiposity trajectory group and gender. Backward selection was undertaken with further covariates which were potential confounders (gestational age at delivery, maternal education, and age). The final most parsimonious model was selected. Adjustment for correlation between siblings was undertaken using the VCE option in STATA v20.0.

The effect of childhood adiposity trajectories on longitudinal BP. Fractional polynomials were used to investigate how BP varied with age by using age terms that closely modeled longitudinal BP patterns, separately for males and females. Random coefficient, linear mixed models were used to estimate the effects of gender on BP over time via the interaction of age and gender. Interactions between the adiposity trajectory, gender, and age were also explored. Birth weight, gestational age and maternal education were additionally added to the model. Adjustment for correlation between siblings was undertaken using hierarchical mixed model. SPSS v19.0 and STATA v13.0 were used. Level of significance was set at $P < 0.05$.

RESULTS

A total of 1,023 adolescents with BP measured at age 17 years were included in the study. The characteristics of the adolescents at 14 years have been reported previously and the general characteristics for the 7 trajectory groups are shown in Table 1. The 7 adiposity trajectory groups (Figure 1) were similar to one another for demographic factors including sex, age, ethnicity, maternal education, and gestation age at delivery. In Table 1, a greater proportion of males compared to females (36.4% compared to 7.9%) were either hypertensive or prehypertensive at 17 years of age.

At 17 years—the relationship between childhood adiposity trajectories and BP

Relationship of adiposity trajectories with hypertension and prehypertension at 17 years old. Compared with the reference stable adiposity trajectory (trajectory 5), 17-year-old adolescents in trajectory 1 (lifelong high adiposity) had a significantly increased risk of elevated BP (prehypertension or hypertension) (odds ratio (OR) = 2.9, 95% CI = 1.3 to 6.2) (Table 2). Individuals in the 2 rising/accelerated childhood adiposity trajectories also had an increased risk of hypertension compared to the reference trajectory. Trajectory 2 (rising from average birth weight) was associated with an increased risk of elevated BP (OR = 3.5, 95% CI = 2.1 to 5.9). Adolescents in trajectory 4 had an increased risk of elevated BP (OR = 1.8, 95% CI = 1.0 to 3.2; Table 2). The addition of the homeostatis model assessment for insulin resistance (HOMA-IR) at 17 years as an independent covariate in the final regression models resulted in minimal changes in coefficients of the adiposity trajectories and a nonsignificant effect of HOMA-IR.

![Figure 1. Seven adiposity trajectory groups.](Image)
Table 1. General characteristics and blood pressure of childhood adiposity trajectory groups at 17 years

<table>
<thead>
<tr>
<th>Trajectory Group</th>
<th>All trajectories</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 1,023</td>
<td>n = 51</td>
<td>n = 122</td>
<td>n = 173</td>
<td>n = 105</td>
<td>n = 358</td>
<td>n = 187</td>
<td>n = 29</td>
</tr>
<tr>
<td>General</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Males</td>
<td>50.5</td>
<td>63</td>
<td>56</td>
<td>48</td>
<td>44</td>
<td>48</td>
<td>58</td>
<td>42</td>
</tr>
<tr>
<td>Anthropometry</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>3.44 (3.41, 3.47)</td>
<td>3.66 (3.53, 3.79)</td>
<td>3.41 (3.33, 3.49)</td>
<td>3.66 (3.59, 3.74)</td>
<td>3.22 (3.14, 3.29)</td>
<td>3.47 (3.42, 3.51)</td>
<td>3.31 (3.25, 3.37)</td>
<td>3.12 (2.96, 3.28)</td>
</tr>
<tr>
<td>BMI at 17 years—boys</td>
<td>22.7 (22.3, 23.0)</td>
<td>29.9 (27.8, 32.0)</td>
<td>27.2 (26.0, 28.4)</td>
<td>22.7 (23.0, 24.3)</td>
<td>24.3 (23.2, 25.3)</td>
<td>21.3 (21.0, 21.6)</td>
<td>19.6 (19.3, 20.0)</td>
<td>17.7 (16.8, 18.5)</td>
</tr>
<tr>
<td>BMI at 17 years—girls</td>
<td>23.1 (22.7, 23.5)</td>
<td>30.7 (27.4, 34.0)</td>
<td>28.6 (27.4, 29.7)</td>
<td>24.4 (23.8, 25.0)</td>
<td>22.7 (21.9, 23.5)</td>
<td>21.7 (21.4, 22.1)</td>
<td>19.7 (19.2, 20.2)</td>
<td>17.9 (17.1, 18.8)</td>
</tr>
<tr>
<td>Blood pressure at 17 years</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>118 (117, 119)</td>
<td>123 (120, 127)</td>
<td>122 (120, 125)</td>
<td>117 (115, 119)</td>
<td>119 (116, 122)</td>
<td>116 (115, 117)</td>
<td>116 (115, 118)</td>
<td>115 (109, 120)</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>58 (58, 59)</td>
<td>58 (56, 60)</td>
<td>58 (56, 60)</td>
<td>58 (56, 59)</td>
<td>59 (57, 61)</td>
<td>58 (57, 59)</td>
<td>59 (57, 60)</td>
<td>58 (55, 62)</td>
</tr>
<tr>
<td>% Hypertensive or prehypertensive</td>
<td>36.4</td>
<td>51.9</td>
<td>56.1</td>
<td>36.9</td>
<td>43.8</td>
<td>30.7</td>
<td>29.2</td>
<td>23.1</td>
</tr>
<tr>
<td>Girls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>108 (108, 109)</td>
<td>110 (105, 115)</td>
<td>113 (110, 115)</td>
<td>107 (105, 109)</td>
<td>109 (107, 111)</td>
<td>108 (107, 109)</td>
<td>107 (105, 109)</td>
<td>108 (102, 113)</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>59 (59, 60)</td>
<td>58 (54, 61)</td>
<td>59 (58, 61)</td>
<td>58 (57, 59)</td>
<td>60 (59, 61)</td>
<td>60 (59, 61)</td>
<td>59 (58, 61)</td>
<td>62 (59, 64)</td>
</tr>
<tr>
<td>% Hypertensive or prehypertensive</td>
<td>7.9</td>
<td>18.2</td>
<td>18.8</td>
<td>3.4</td>
<td>8.9</td>
<td>5.6</td>
<td>2.5</td>
<td>6.3</td>
</tr>
</tbody>
</table>

Mean (95% confidence intervals) are shown for continuous measures. Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure.
Table 2. Linear regression models with the outcome of 17-year-old systolic blood pressure, diastolic blood pressure and logistic regression models of categorical outcomes of normotension vs. elevated BP (prehypertension or hypertension)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Trajectory</th>
<th>( b )</th>
<th>95% CI</th>
<th>( P ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>1</td>
<td>5.2</td>
<td>2.3 to 8.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>( N = 1,023^a )</td>
<td>2</td>
<td>5.6</td>
<td>3.6 to 7.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.6</td>
<td>-1.0 to 2.2</td>
<td>0.47</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>2.4</td>
<td>0.4 to 4.4</td>
<td>0.018</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>-0.4</td>
<td>-1.8 to 1.0</td>
<td>0.57</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>-0.8</td>
<td>-4.4 to 2.9</td>
<td>0.68</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>1</td>
<td>0.6</td>
<td>-1.0 to 2.2</td>
<td>0.47</td>
</tr>
<tr>
<td>( N = 1,023^b )</td>
<td>2</td>
<td>-0.04</td>
<td>-1.4 to 1.3</td>
<td>0.95</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>-0.8</td>
<td>-1.9 to 0.4</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.8</td>
<td>-0.6 to 2.1</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>0.2</td>
<td>-1.0 to 1.3</td>
<td>0.77</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>1.4</td>
<td>-0.7 to 3.4</td>
<td>0.19</td>
</tr>
<tr>
<td>Abnormal blood pressure</td>
<td>1</td>
<td>2.9</td>
<td>1.3 to 6.2</td>
<td>0.007</td>
</tr>
<tr>
<td>(Hypertension or prehypertension)</td>
<td>2</td>
<td>3.5</td>
<td>2.1 to 5.9</td>
<td>0.001</td>
</tr>
<tr>
<td>( N = 1,017^c )</td>
<td>3</td>
<td>1.2</td>
<td>0.8 to 2.1</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>1.8</td>
<td>1.0 to 3.2</td>
<td>0.041</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>0.8</td>
<td>0.5 to 1.4</td>
<td>0.45</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>0.8</td>
<td>0.3 to 2.5</td>
<td>0.68</td>
</tr>
</tbody>
</table>

The \( b \) coefficient or odds ratio (OR) are shown compared to the reference trajectory 5 after adjusting for gender. Abbreviations: BP, blood pressure; CI, confidence interval.

\(^a\) Additionally adjusted for maternal education and gestational age at delivery in weeks.

\(^b\) Additionally adjusted for gestational age at delivery in weeks.

\(^c\) Additionally adjusted for maternal education and gestational age at delivery in weeks.

The effect of adiposity trajectories upon longitudinal SBP throughout childhood

Figure 2a,b show the mean fitted values for the longitudinal BP models (linear mixed models) at each time point that BP was measured. These models were adjusted for age, sex, birth weight, maternal education, and gestational diabetes. Further adjustment with maternal essential hypertension or gestational hypertension did not significantly alter either the coefficients or \( P \) values (data not shown).

The effect of adiposity trajectories upon longitudinal SBP. The slope of longitudinal SBP’s modeled for individuals belonging to adiposity trajectories 1, 2, and 4 did not differ from reference trajectory 5, but all had higher intercepts (\( P = 0.008, 0.017, \) and 0.07, respectively) (Supplementary Table 3). This means that SBP was on average higher throughout childhood for individuals belonging to these 3 adiposity trajectories. (Figure 2b) To give an indication of the magnitude of elevated SBP, at 3 years, children in trajectory 1 (life-long high adiposity) had a SBP that was on average 6.4 mm Hg higher (95% CI = 3.7 to 9.1) compared to those in an optimum adiposity trajectory (trajectory 5). Children in trajectory 2 (rising from average birth weight) had a SBP on average 4.0 mm Hg higher (95% CI = 1.7 to 6.4).

The model (Supplementary Table 3) indicates that trajectories 3 (\( P \) values = 0.025–0.084) and 6 (\( P \) values = 0.034–0.047) have significant interactions with age terms (age, 1/age, 1/age\(^2\)). These interactions with age terms indicate that childhood development of SBP for trajectories 3 and 6 were different over time compared to the optimum adiposity trajectory (reference group 5) (Figure 2a). Specifically, the decelerating adiposity trajectory 3 (Figure 1) is associated with longitudinal BP that deviates down in middle childhood returning to a BP similar to the reference optimum trajectory 5 by 14–17 years old (Figure 2a).

The effect of adiposity trajectories upon longitudinal DBP. The intercept and slope of DBP for trajectories 2 and 3 were both different to the reference adiposity trajectory group 5 (Supplementary Table 4). Trajectories 2 and 3 had greater DBP in middle childhood (Figure 2b) before returning downward to a BP similar to the reference optimum trajectory 5 by 17 years old.

DISCUSSION

We previously identified 7 adiposity trajectories\(^2\) and found that 3 adiposity trajectories related to an increased risk of prehypertension and hypertension at age 17. Compared to
American Journal of Hypertension

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the optimum growth trajectory 5, a lifelong high adiposity (trajectory 1), rising trajectories from both average (trajectory 2), and low birth weight (trajectory 4) were independently associated with increased odds of hypertension and prehypertension at 17 years of age. These 3 adiposity trajectories (27% of the population) have previously been shown to be associated with higher insulin resistance at age 14 years. Therefore, there are 2 growth patterns in this study associated with subsequent elevated BP. They are accelerated infant weight gain and maintenance of high adiposity from birth. Given the known effect of adiposity on BP, these relationships are most likely to be causal. However, other lifestyle factors associated with adiposity, such as high salt intake and sedentary lifestyle may also be contributing factors.

Accelerated weight gain in infancy has previously been shown to relate to adult hypertension. We show that this relationship between accelerated weight gain in infancy and hypertension occurs irrespective of initial birth size. Elevated BP was seen in association with trajectories 2 and 4. These trajectories encompass both below average and average birth sizes. Compared to children who followed an optimal adiposity trajectory, accelerated weight gain in infancy was associated with increases in SBP 4–6 mm Hg higher by 3 years of age, magnified up to 4–8 mm Hg by age 17. Previous meta-analyses have shown that an increase in SBP of 10 mm Hg in adult populations is associated with a 33% increased elevation of stroke risk. Correspondingly, this study suggests that on a population scale, accelerated infant weight gain could substantially increase future vascular disease.

Maintenance of high adiposity from birth (trajectory 1) is also associated with an elevated risk of hypertension. Our

Figure 2. The fitted lines based on the optimum linear mixed model for blood pressure from year 1 to 17 years old for each of the adiposity trajectory groups is shown. (a) Shows the fitted line for systolic blood pressure. (b) Shows the fitted line for diastolic blood pressure.
data suggest that this increased risk may be avoidable. In babies of above average birth weight, but decreasing fat mass in infancy (trajectory 3), there was a relative decline in SBP (Figure 2a) and DBP (Figure 2b) in middle childhood, compared to those who followed the referent adiposity trajectory. This averted adolescent hypertension. Interventions which protect against obesity, such as breastfeeding and limiting bottle feeding are currently advocated. These may be insufficient. More aggressive preschool obesity management may be necessary for those who already have increased adiposity at birth. Establishing safety parameters is of paramount importance, so that such organ and brain development are not compromised by weight management in infancy. Subject to that, intervention studies to evaluate the effect of reducing adiposity in preschoolers are necessary to establish if adiposity trajectories are capable of modification. As well, they will determine if the associations between falling infant adiposity and reduction in hypertension risk are causative. Inherent with this is the necessity to address parental underestimation of preschool obesity.

In biology, there is growing use of latent growth trajectory analyses. Ventura et al. showed that an upward BMI percentile group akin to the trajectory 2 (rising from average birth weight) had greater metabolic risk. Using spline modeling Ben-Shlomo et al. showed that accelerated postnatal growth in 2 time periods (0–5 months and 1 year and 9 months to 5 years) is associated with SBP. These findings concur with our data showing that adolescents in trajectories 1, 2, and 4 with accelerated postnatal growth had higher BP at 17 years. In Figure 1, these 3 trajectories have in common a rising slope in the preschool years. This is a point of difference from the other 4 trajectories. An advantage of these methods is that adiposity growth patterns are identified independently without needing to assign a-priori the relative contributions of multiple lifestyle, genetic, and epigenetic factors. Therefore, the formation of these adiposity trajectories inherently takes into account that variation in adiposity trajectories will be due to multiple factors.

In this study, 44%–56% of males with accelerated adiposity trajectories in childhood were at risk of prehypertension or hypertension. This propensity for males to be at risk of elevated BP in response to accelerated/rising childhood adiposity is consistent with sexual dimorphism repeatedly observed in BP control and cardiovascular fetal programming. Human studies are starting to confirm that males, similar to animal models, may be at greater risk of fetal programming related to low birth weight. A limitation is that this is an observational study. Accordingly, no inference can be made that the association of adiposity trajectories with BP is causal. This study is unable to give any indication as to whether a pattern of “catch-down growth” can be induced by environmental changes. Further, it does not indicate whether inducing such a pattern can change future BP development. Canalization, whereby child growth trajectories and phenotypes are robust to small perturbations, may occur following in utero exposure to maternal blood glucose levels. The evidence for this is not clear. Some studies showing that babies born to mothers with higher glucose concentrations within the normal range show greater neonatal fatness with significant “catch-down” growth in infancy. However, other studies show that higher maternal glucose levels are associated with higher birth size but not postnatal size at 2 years old. Another limitation is attrition. Only 1,023 participants with BP measurements at age 17 years were included. However, we show that selective attrition resulted in a sample more representative of the general Western Australian population than the original high-risk population. The prospective design is a strength avoiding recall bias. The standardized data collection over a 17-year period of both anthropometry and BP allows us to compare trajectories.

In conclusion, this study shows that growth patterns of adiposity incorporating birth size and the first years of life may be superior predictors of future SBP and the risk of prehypertension and hypertension in adolescents, compared to cross-sectional BMI measures. Diminishing adiposity in the early years of life was associated with a downward deviation of the longitudinal childhood BP curve. By 17 years of age, no excess risk for hypertension/prehypertension was present in this category. This study provides strong evidence that early intervention through alteration of childhood adiposity trajectories may benefit the public health management of hypertension.

SUPPLEMENTARY MATERIAL

Supplementary materials are available at American Journal of Hypertension (http://ajh.oxfordjournals.org).

ACKNOWLEDGMENTS

The authors thank all the families that took part in this study and the Raine Study team, which includes data collectors, cohort managers, clerical staff, researchers, and volunteers. This work was supported by the Raine Medical Research Foundation; Healthway, Western Australia; The Telethon Kids Institute, University of Western Australia (UWA); Faculty of Medicine, Dentistry and Health Sciences (UWA); Women and Infants Research Foundation (UWA); Curtin University; and The Australian National Health and Medical Research Council (NHMRC). R.-C.H. is supported by a NHMRC Fellowship (grant number 1053384).

DISCLOSURE

The authors declared no conflict of interest.

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