Journal of the American Heart Association

ORIGINAL RESEARCH

Prevalence of and Factors Associated With High Blood Pressure at 15 Years of Age: A Birth Cohort Study

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BACKGROUND: Arterial hypertension is the greatest cause of morbidity and mortality worldwide. Our aim was to investigate the prevalence of and factors associated with high blood pressure (HBP) among adolescents.

METHODS AND RESULTS: The Pelotas 2004 Birth Cohort included 4231 newborns from hospital births in Pelotas, Brazil. A digital automatic OMRON sphygmomanometer (model HEM 742) was used to measure blood pressure on 3 occasions (at 6, 11, and 15 years of age). Those with blood pressure ≥95th percentile for age, height, and sex on each of the 3 occasions were considered as presenting HBP. Independent variables included family (income and history of arterial hypertension), maternal (schooling, age, pregestational body mass index, and smoking during pregnancy), and adolescent characteristics at birth (sex, skin color, gestational age, intrauterine growth, and systolic and diastolic genetic factors), and at 15 years (sleep, physical activity, sodium intake, screen time, work, body mass index, fat mass index, fat-free mass index, growth pattern, and puberty status). The prevalence of HBP (95% CI) was calculated. Crude and adjusted odds ratios (ORs) stratified by sex were obtained by logistic regression. A total of 1417 adolescents with complete information on blood pressure on the 3 occasions were analyzed. The prevalence of HBP was 3.2% (95% CI, 1.9%–4.5%) in female adolescents and 4.3% (95% CI, 2.8%–5.8%) in male adolescents. Female adolescents with a family history of arterial hypertension had a 3 times higher chance of HBP than their counterparts (OR, 3.1 [95% CI, 1.26–7.54]). In male adolescents, excessive maternal pregestational weight was associated with a 2.3-fold increase in the chance of HBP. In both sexes, excessive adolescent weight was associated with HBP (ORs, 3.5 and 5.0, for female and male adolescents, respectively). A higher fat mass index and fat-free mass index in female (ORs, 1.4 and 1.2, respectively) and male adolescents (ORs, 2.5 and 3.0, respectively) increased the chance of HBP. Among male adolescents, the chance of HBP was higher among those with rapid weight gain between 48 months and 6 years and between 6 and 11 years and rapid height gain between 6 and 11 years.

CONCLUSIONS: Higher fat mass in both sexes and rapid weight gain in male adolescents are risk factors for HBP in adolescents aged 15 years, potentially amenable to prevention.

Key Words: adolescent ■ blood pressure ■ cohort ■ high blood pressure

Arterial hypertension (AH) is 1 of the most critical modifiable risk factors for cardiovascular disease and 1 of the largest contributors to the global burden of disease.[1,2] The 2017 GBD (Global Burden of Disease) Study showed that high systolic blood pressure (SBP) was the leading risk factor, accounting for 10.4 (95% CI, 9.39–11.5) million deaths and 218 (95% CI, 198–237) million disability-adjusted life-years.[1] According to the analysis of 1201 population-based studies, involving 104 million participants,

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Supplemental Material is available at https://www.ahajournals.org/doi/suppl/10.1161/JAHA.123.029627

For Sources of Funding and Disclosures, see page 13.

JAHA is available at: www.ahajournals.org/journal/jaha

CLINICAL PERSPECTIVE

What Is New?

- Determination of the prevalence of high blood pressure (HBP) in a birth cohort study at 3 moments of life (6, 11, and 15 years) using an oscillometric method.
- Pioneer study in a Brazilian pediatric and adolescent population evaluating factors associated with increased blood pressure.
- No association of HBP with genetic factors.
- A higher fat mass index and fat-free mass index were associated with higher HBP prevalence in both sexes.
- There was a higher chance of HBP in male adolescents with rapid weight gain between 48 months and 6 years and between 6 and 11 years and rapid height gain between 6 and 11 years.

What Are the Clinical Implications?

- Because HBP is 1 of the clinical manifestations of cardiovascular diseases, with the highest morbidity and mortality in the world, knowledge of its risk factors in the pediatric population has great potential in the primary prevention of cardiovascular events.

Nonstandard Abbreviations and Acronyms

AH	arterial hypertension
DBP	diastolic blood pressure
FFM	fat-free mass
FFMI	fat-free mass index
FM	fat mass
FMI	fat mass index
HBP	high blood pressure
SBP	systolic blood pressure

the number of people aged 30 to 79 years with AH doubled from 1990 to 2019, from 331 (95% CI, 306–359) million women and 317 (95% CI, 292–344) million men in 1990 to 626 (95% CI, 584–668) million women and 652 (95% CI, 604–698) million men in 2019.[2] In Brazil, AH prevalence is 32.3% (95% CI, 31.1%–33.0%) in adult individuals.[3]

There is evidence that the processes leading to atherosclerotic cardiovascular disease start in childhood and progress over time,[4] thus suggesting that atherosclerosis has a lifelong course and the risk factors known to be important in adults are also likely to be important in the pediatric population. The prevalence of childhood AH is increasing even at earlier ages and has become a public health issue.[5] A systematic review and meta-analysis described a global AH prevalence of 4.3% (95% CI, 2.8%–6.6%) at 6 years and 7.8% (95% CI, 5.7%–10.7%) at 14 years of age.[6] In Brazil, a nationwide school-based cross-sectional study with 73 399 adolescents showed that the prevalence of high blood pressure (HBP) was 9.6% (95% CI, 9.0%–10.3%).[7]

Methodological differences between the studies concerning blood pressure (BP) measurements, such as the type of device used, number of measurements on a single occasion, number of occasions when measures were taken, and the time intervals between these measurements, may explain part of this variation.[8,9]

The main current international guidelines are consensual in that for the diagnosis of AH in childhood and adolescence BP should be measured on 3 different occasions, besides establishing reference values for normal BP, elevated BP, and HBP criteria.[10] Nonetheless, there is a scarcity of population-based studies that have assessed the prevalence of HBP in this age group following those guidelines, particularly in Brazil. Furthermore, the primary prevention of AH involves the identification and modification of its risk factors. Therefore, the objective of the present study was to describe the prevalence of and the factors associated with HBP at 15 years of age among the participants of the Pelotas 2004 Birth Cohort study.

METHODS

Sample Population

The present study uses data from the Pelotas 2004 Birth Cohort, performed in Pelotas, a southern Brazilian city with an estimated population of 342 000 inhabitants.[11] In 2004, a birth cohort was started, including all newborns from hospital births between January 1 and December 31 of mothers who lived in the urban area of Pelotas. A total of 4231 newborns (representing 99.2% of all births in the city that year, with 32 refusals) were included in the study. Standardized interviews were performed with the mothers soon after delivery during their hospital stay (perinatal study). After the cohort inception, the children were followed up at the mean (SD) ages of 3.0 (0.1), 11.9 (0.2), 23.9 (0.4), 49.5 (1.7), and 82.2 (4.0) months and at 6.7 (0.2), 10.3 (0.5), and 15.7 (0.20) years. All visits were performed by trained interviewers using a structured questionnaire. Information on socioeconomic, demographic, and behavioral characteristics was gathered. The newborns were examined within the first 24 hours after delivery by the study team under the supervision of a pediatrician.

Before the 6-year follow-up, the children were evaluated in their homes. The follow-up visits from 6 years onwards were held at a clinic installed at the headquarters

of the Federal University of Pelotas Epidemiologic Research Center. In the visits, the follow-up rates were 95.7% (N=3985), 94.3% (N=3907), 93.5% (N=3869), 92.0% (N=3799), 90.2% (N=3722), 86.6% (N=3566), and 50.4% (N=2131), respectively. The follow-up at 15 years started in November 2019 and was prematurely interrupted in March 2020 because of the COVID-19 pandemic. This study used data collected at the perinatal study and in the 6-, 11-, and 15-year follow-ups.

Details on the study methods are available in previous publications.[12,13] The questionnaires and interviewer guidelines from all follow-up visits are available in electronic format at http://www.epidemio-ufpel.org.br/site/content/coorte_2004/index.php.

Outcome

BP was measured using the automatic device model HEM 742 from Omron. A study to assess the validity of this equipment for measuring BP in adolescents showed a sensitivity of 100% and a specificity of 98.5%.[14] The same BP measurement protocol was used in the 3 follow-ups. All measurements were performed with the participants seated for at least 5 minutes, legs uncrossed with their feet flat on the floor, without voiding desire, without having practiced physical activity or ingesting food up to 30 minutes before. The left upper limb was used, without clothing, supported, and maintaining the same height as the limb and heart relative to the ground. Two measurements were performed with an interval of 2 minutes. When interference occurred, such as coughing during the measurement, that measurement was discarded, and another was performed with an interval of 2 minutes. Appropriate-sized cuffs were used for each arm circumference being determined by measuring the perimeter of the arm, at the midpoint between the acromion and the olecranon of the elbow, representing 80% to 100% of this value. The Rosner formula was used to transform mean SBP and diastolic BP (DBP) values into percentiles by sex, age (in complete years), and height (in centimeters), following international recommendations.[15,16]

HBP in childhood is defined by at least 3 measurements on different occasions (with at least 1- to 4-week intervals, depending on the BP level) with SBP, DBP, or both above the 95th centile for age, height, and sex.[10] In our study, the adolescent was considered as presenting HBP at the age of 15 years if his/her SBP, DBP, or both measure was ≥95th percentile in each of the 3 visits (in the 6-, 11-, and 15-year follow-ups).

Independent Variables

Family and Maternal Characteristics

Family income during the month preceding the child's birth was collected as a continuous variable by summing the monthly wages of all household members and divided into quintiles (the first quintile representing the poorest, and the fifth quintile representing the wealthiest families). Family history of AH in the parents and in the parents' first-degree relatives (grandparents of the child) was collected in the 6-year follow-up visit and was dichotomized into yes or no.

Maternal education level in number of full years of schooling was later categorized as 0 to 4, 5 to 8, and ≥9 years. Maternal age was categorized as ≤20, 21 to 25, 26 to 30, and >30 years. Prepregnancy body mass index (BMI) was calculated using the information on height and maternal weight at the beginning of the pregnancy and was classified as low/normal weight (<25.0 kg/m^2) or excessive weight (≥25.0 kg/m^2). Maternal smoking during pregnancy (yes or no), as reported by the mother, was defined as at least 1 daily cigarette during any trimester of the pregnancy.

Adolescent Characteristics at Birth

Information on the adolescent's sex (male and female) was collected from the medical records at birth. Gestational age was estimated using the first day of the last normal menstrual period or by obstetric ultrasound obtained before 20 gestational weeks when information about the last normal menstrual period was unreliable or not available. In the absence of both menstrual and ultrasound information, gestational age was estimated from the physical and neurologic assessment of the newborn, using the Dubowitz method.[17] Gestational age was categorized as <37 or ≥37 weeks. Skin color was chosen as a proxy for ancestral background, because miscegenation in Brazil is highly prevalent, and it is not feasible to classify participants into different ethnic groups in large-scale studies.[18] Based on maternal self-response on child skin color in the 6-year follow-up, 4 groups (Black, Brown, White, or other [Yellow, Indigenous, Mulatto]) were coded, according to the classification adopted by the Brazilian Census Bureau.[19] Because children in the Black and other skin color or other ethnic origin categories had similar sociodemographic characteristics, they were assembled into a single group and the variable was categorized into White and non-White (Black or mixed ethnic origin).

Intrauterine growth was defined according to the INTERGROWTH-21st parameters,[20] and classified as small for gestational age (birth weight lower than the 10th centile), adequate for gestational age (birth weight between the 10th and the 90th centiles), or large for gestational age (birth weight above the 90th centile) for a specific completed gestational age and sex. The hospital staff measured the child's birth weight using electronic pediatric scales checked daily for accuracy by the research team. The study team measured birth length using an ARTHAG infantometer with an accuracy of 1 mm.

Adolescent Characteristics at 15 Years

In the 15-year follow-up, information was collected on puberty status, sleep, physical activity, sodium intake, screen time, current work, anthropometry, and body composition.

Puberty status at 15 years was evaluated with an instrument containing line drawings depicting the 5 Tanner stages.[21] Adolescents selected their self-perceived stage of development by choosing the pictures closest to their current stage of sexual development. The figures of pubic hair scale were applied for both male and female adolescents and ranged from 1 to 5 (stage 1=hairless, stage 2=very few hairs, stage 3=plenty of hair, stage 4=the hair did not spread over the thighs, and stage 5=hair spread across the thighs); the breast development scale was applied to female adolescents and contained images varying from stage 1=breasts are flat, stage 2=the breasts form small mounds, stage 3=the breasts form larger mounds than in stage 2, stage 4=the nipple (breast nipple) and the surrounding portion (areola) make a mound that stands out from the breast, and stage 5=only the breast nipple stands out from the breast; and the external genitalia scale was applied to male adolescents and ranged from stage 1=the scrotum and penis are the same size as when you were younger, stage 2=the scrotum has descended a little and the penis is a little wider, stage 3=the penis is longer, and the scrotum is wider, stage 4=the penis is longer, and the scrotum is darker and larger than before, and stage 5=penis and scrotum are the size and shape of an adult. The sum could range from 2 to 10 and was later categorized into 3 groups (2–4, 5–7, and 8–10 points). For example, if a girl scored stage 3 (plenty of hair) on the pubic hair scale and stage 3 (the breasts form larger mounds than in stage 2) on the breast development scale, she would add 6 points and be classified in the category 5 to 7 of puberty status.

Sleep duration was obtained through questions extracted from the Munich Chronotype Questionnaire, referring to the 30 days before the interview: "What time are you ready to sleep?" "How many minutes do you need to fall asleep?" and "What time do you wake up?"[22] Questions were asked separately for weekdays and weekends. Sleep duration was calculated as the difference between going to bed and waking up, minus latency time ("How many minutes do you need to fall asleep?"). As sleep duration had a normal distribution, for analysis purposes, the weighted average between weekdays and weekends was used, obtained by the following formula: 24-hour weighted average sleep duration=[(5×weekday sleep duration)+(2×weekend sleep duration)]/7. The mean sleep time duration was classified as <8 or ≥8 hours. Means of <3 hours (N=8) or >15 hours (N=1) were considered aberrant, and the participants were excluded from the analysis involving this variable.

Physical activity was measured by a waterproof wrist accelerometer (ActiGraph, model wGT3X-BT and wActiSleep-BT), with 24-hour reading, with 5 consecutive reading days, 2 of which were Saturday and Sunday. The device was placed on the nondominant wrist of each child/adolescent. The results are provided in milligrams (gravitational equivalent, 1000 mg=1 g=9.81 m/s^2). The physical activity variable was described as the average number of minutes per day.

The mean sodium intake was estimated with a validated semiquantitative food frequency questionnaire, which contained 96 items evaluating food habits in the 12 months before the interview, administered to the adolescent.[23] For each food item, the adolescent was asked how many times it was consumed in a day, week, month, or year, and if the intake was usually lower than, equal to, or higher than the average portion. The average portion sizes, based on domestic measures according to the Table for Assessment of Food Intake in Household Measures, were presented to the participant verbally and with the help of images.[24] We reconfigured the 12-month food consumption to daily consumption with all portions standardized at 100 g. The sodium content of each food was estimated on the basis of the Brazilian Food Composition Table.[24] Daily sodium intake in milligrams was then calculated, which was later categorized into terciles (the first tercile representing the lowest sodium intake, and the third tercile representing the highest).

Screen time was calculated according to the adolescent's report of the number of hours in a typical week (separately from Monday to Saturday and on Sundays) he/she spent watching TV, playing on cell phones and tablets, playing videogames, and using the computer. Standardized mean screen time was obtained through the following formula: [(mean daily screen time from Monday to Saturday×6)+(daily screen time on Sundays)]/7. Screen time was then categorized into <3 or ≥3 h/d. The adolescents who were working at the time of their interview were considered as yes for current work. If they had never worked or were not working at the time of their interview, they were considered as no for current work.

Height was measured twice by trained anthropometrists using a Harpenden metal stadiometer, with 1-mm precision (Holtain, Crymych, UK), and weight was assessed using a high-precision scale (0.01 kg), part of the BodPod used for body composition assessments.[13] BMI *z* scores specific for sex and age were calculated according to the growth curves published by the World Health Organization in 2007[25] using ANTHRO PLUS software.[26] BMI *z* score was categorized as normal weight (<1 *z* score) or excessive weight (≥1 *z* score).

Body composition was evaluated by air-displacement plethysmography using a BodPod handled by specifically trained technicians. Standard equations were used to define body fat mass (FM) and fat-free mass (FFM) at 15 years of age.[27] The FM index (FMI) and FFM index (FFMI) were calculated by dividing FM (in kg) and FFM (in kg), respectively, by height (in meters squared). The FMI and FFMI were categorized in terciles.[13] The third tercile comprised those with the highest amount of FM or FFM per square meter.

Growth Pattern From Birth to 15 Years

The growth pattern was studied at several points: from birth to 3 months, from 3 to 12 months, from 12 to 24 months, from 24 to 48 months, from 48 months to 6 years, from 6 to 11 years, and from 11 to 15 years. For each age interval, the associations of weight gain and linear growth with BP were analyzed using conditional relative weight gain and conditional length/height gain, as proposed by Adair et al.[28] Conditional relative weight gain considers current height and previous weights and lengths/heights, whereas conditional length/height gain considers previous weight and length/height measurements, but not current weight. To enable the calculation of conditional measures, first, specific *z* scores were calculated by sex for each weight and length from prior follow-ups. The *z*-score measures (weight or length/height) for a given age were regressed on the *z* scores of all previous measurements using linear regressions. The conditional measure is represented by the standardized residuals of the regression and indicates the extent to which a participant's measurements deviate from the expected value, based on his/her prior growth and the cohort's mean growth. It can be interpreted as a measure of how much more quickly or more slowly weight or length/height changes over the course of a period. For example, an adolescent with a positive conditional relative weight gain value from 11 to 15 years of age gained more weight relative to his/her own previous weights and lengths/heights and in relation to all the other members of the cohort. Finally, the variables of conditional weight and length/height gains were dichotomized into rapid weight gain (conditional relative weight gain values >0) (yes or no) and rapid length/height gain (conditional length/height gain >0) (yes or no).

Polygenic Risk Score for SBP and DBP

The polygenic risk score (PRS) for SBP and DBP was obtained from saliva samples collected at age 6 years. The saliva samples for the DNA analyses were collected using the DNA Oragene Genotek–250.[13] Genomic DNA was extracted following the manufacturer's instructions. DNA was quantified and qualified by spectrophotometry using a NanoDrop. DNA samples were genotyped for ≈600000 single-nucleotide polymorphisms using an Infinium Global Screening Array 2. Imputation of remaining nongenotyped variants and quality control filtering were done as described elsewhere.[29] The PRSs for SBP and DBP were calculated as the weighted sum of risk alleles for SBP and DBP, according to the largest genome-wide association study to date.[30] The scoring files deposited in the PRS catalog were used to construct the PRS (PRS Catalog Publication ID PGP000283).[31] The calculation was performed using the PRSice software version 2.2.[32] Polygenic risk scores were transformed into *z* scores for the statistical analyses. The adjustment for genomic ancestry was also performed using the first 10 principal components. The principal component analysis was based on the whole genomic data set.

Statistical Analysis

Only adolescents with measures of SBP and DBP at 6, 11, and 15 years were included in the analyses. First, adolescents followed up at 15 years with complete information on BP at 6, 11, and 15 years were compared with the remaining adolescents who were followed up at 15 years but had missing information on BP on at least 1 occasion (at 6, 11, or 15 years). The associations were assessed by Fisher exact test, χ^2 test, and *t* test. Interaction between sex and the other exposures was tested. As the formal tests for interaction lack power, $P<0.20$ was considered statistically significant. The *P* value for interaction between sex and FFMI was 0.179, so all the analyses were stratified by sex.[33]

Descriptive analyses were initially performed, calculating the prevalence of HBP with the respective 95% CI by sex. Crude and adjusted odds ratios (ORs) for HBP were obtained by logistic regression. The adjusted analysis was based on a hierarchical model composed of 4 levels. Variables that presented a $P\leq0.20$ in each level were maintained in the adjusted model. Level 1 was composed of family (family monthly income and family history of AH) and maternal characteristics (schooling, age, pregestational BMI, and smoking during pregnancy). The adolescent characteristics at birth (sex, skin color, gestational age, and intrauterine growth) were included in the second level. The third level comprised systolic and diastolic genetic factors (PRS for SBP and DBP), puberty status, sleep, physical activity, sodium intake, screen time, and current work. BMI, FMI, FFMI, and growth pattern in each age interval were analyzed separately in the fourth level. Because none of the female adolescents with HBP was in the first tercile of FMI, adjusted analyses for body composition were run with FMI and FFMI as continuous variables for both sexes. Two-tailed

$P<0.05$ was considered statistically significant in the final model. All analyses were performed with the Stata software version 16.0.

Ethical Approval

The Medical Ethics Committee of the Faculty of Medicine of the Federal University of Pelotas, affiliated with the Brazilian National Commission for Research Ethics, approved the study protocol of all follow-ups of the Pelotas 2004 Birth Cohort. At each stage of the study, all subjects' mothers or legal guardians gave written informed consent. In the 11- and 15-year follow-ups, the adolescents also gave written informed consent. In each follow-up, parents of adolescents with elevated BP levels were warned and advised to seek medical care for BP monitoring.

RESULTS

A total of 102 cohort participants had died before completing 15 years of age, and 1417 had full BP measurement information at 6, 11, and 15 years and were entered in the analyses. Table 1 shows that for both female and male adolescents, ≈20% of the families were in each of the income quintiles, a history of AH was present in about a quarter of the families, and at the time of childbirth, most mothers had completed <9 years of schooling and ≈30% were aged >30 years. Almost 40% of the mothers had excessive prepregnancy weight, and one-quarter of them smoked during their pregnancy. Current and at birth characteristics of the adolescents are also shown in Table 1.

Table 2 presents the prevalence of HBP according to the independent variables by sex. Female adolescents with a family history of AH had a higher prevalence of HBP (6.5% [95% CI, 3.5%–11.6%]) than those without a history of AH in the family (2.2% [95% CI, 1.2%–4.0%]). For male adolescents, the prevalence of HBP was higher among those whose mothers had excessive pregestational weight (7.4% [95% CI, 4.5%–11.8%]) and among those born small for gestational age (11.9% [95% CI, 5.7%–22.9%]), in comparison to their counterparts. A higher prevalence of HBP was associated with excessive weight in both sexes (6.7% [95% CI, 4.1%–10.7%] in female adolescents, and 9.3% [95% CI, 6.1%–13.8%] in male adolescents), as well as in those in the highest tercile of FMI (5.5% [95% CI, 3.4%–8.6%] in female adolescents, and 10.4% [95% CI, 6.5%–16.1%] in male adolescents) and FFMI (6.2% [95% CI, 3.0%–12.4%] in female adolescents, and 6.5% [95% CI, 4.3%–9.6%] in male adolescents). Except intrauterine growth for male adolescents, none of the remaining perinatal variables was associated with HBP in either sex. The puberty status was related to HBP only among male adolescents (16.7% [95% CI,

Table 1. Family, Maternal, and Adolescents' Characteristics at Birth and at 15 Years of Age, Stratified by Sex (N=1417)

Characteristics	Female adolescents (N=691)	Male adolescents (N=726)
Family characteristics		
Family income (quintiles) (N=2029*)		
First (poorest)	118 (17.1)	136 (18.7)
Second	148 (21.4)	143 (19.7)
Third	143 (20.7)	145 (20.0)
Fourth	148 (21.4)	161 (22.2)
Fifth (wealthiest)	134 (19.4)	141 (19.4)
Family history of hypertension (N=1753*)		
Yes	155 (25.4)	170 (26.2)
No	456 (74.6)	478 (73.8)
Maternal characteristics		
Schooling, y (N=2014*)		
≤4	103 (15.0)	100 (13.9)
5–8	282 (41.2)	298 (41.3)
≥9	300 (43.8)	323 (44.8)
Age at childbirth, y (N=2029*)		
≤20	156 (22.6)	162 (22.3)
21–25	173 (25.0)	173 (23.8)
26–30	151 (21.9)	167 (23.0)
>30	211 (30.5)	224 (30.9)
Pregestational BMI, kg/m^2 (N=1464*)		
<25.0	299 (61.8)	335 (62.2)
≥25.0	185 (38.2)	204 (37.9)
Maternal smoking during pregnancy (N=2029*)		
Yes	173 (25.0)	184 (25.3)
No	518 (75.0)	542 (74.7)
Adolescent characteristics at birth		
Gestational age, wk (N=2026*)		
<37	88 (12.8)	94 (13.0)
≥37	602 (87.3)	632 (87.1)
Intrauterine growth (N=1951*)		
SGA	54 (8.1)	59 (8.4)
AGA	506 (76.3)	519 (74.3)
LGA	103 (15.5)	121 (17.3)
Skin color† (N=1898*)		
White	467 (68.1)	485 (68.4)
Non-White	219 (31.9)	224 (31.6)
Adolescent characteristics at 15 y		
Puberty status (Tanner) (N=1813*)		
2–4	155 (23.0)	36 (5.4)
5–7	368 (54.6)	311 (46.8)
8–10	151 (22.4)	318 (47.8)
Systolic genetic factors (N=1812*)		
First	223 (34.3)	216 (31.0)
Second	226 (34.7)	232 (33.3)

(*Continued*)

Table 1. Continued

Characteristics	Female adolescents (N=691)	Male adolescents (N=726)
Third	202 (31.0)	248 (35.6)
Diastolic genetic factors (N=1812*)		
First	217 (33.3)	234 (33.6)
Second	216 (33.2)	220 (31.6)
Third	218 (33.5)	242 (34.8)
Mean sleep duration, h/d (N=1910*)		
<8	416 (62.6)	476 (67.7)
≥8	249 (37.4)	227 (32.3)
Physical activity, mean (SD), min/d (N=1484*)		
min/d	30.9 (8.4)	34.5 (12.5)
Daily sodium intake, mean (SD), g (N=1917*)	3.199 (4.189)	3.339 (3.705)
Screen time, mean (SD), h/d (N=1880*)	4.5 (3.6)	6.5 (4.6)
BMI *z* score, mean (SD) (N=1903*)	0.5 (1.3)	0.4 (1.4)
FMI, mean (SD), kg/m^2 (N=1903*)	7.3 (3.7)	4.7 (3.7)
FFMI, mean (SD), kg/m^2 (N=1903*)	15.9 (1.9)	17.7 (2.1)
Current work (N=1822*)		
Yes	72 (11.2)	85 (13.0)
No	574 (88.9)	571 (87.0)

Data are given as number (percentage) unless otherwise indicated. AGA indicates adequate for gestational age; BMI, body mass index; FFMI, fat-free mass index; FMI, fat mass index; LGA, large for gestational age; and SGA, small for gestational age.

*Number of individuals with information at the 15-year follow-up (independently of having information on the outcome).

†Based on classification adopted by the Brazilian Census Bureau; Black, Brown, White, Yellow, Indigenous and Mulatto.[19]

7.1%–34.3%] in those with between 2 and 4 points on the Tanner scale). In both sexes, there was no association between the PRS for SBP and DBP and the prevalence of HBP.

Table 3 shows the prevalence of HBP according to the growth pattern by sex. The prevalence of HBP was higher in female adolescents who had rapid weight gain between 12 and 24 months (4.8% [95% CI, 2.8%–7.9%]) and rapid height growth between 6 and 11 years (5.0% [95% CI, 3.0%–8.3%]). For male adolescents, a greater prevalence of HBP was observed among those with rapid weight gain between 48 months and 6 years (6.8% [95% CI, 4.4%–10.3%]), 6 and 11 years (6.1% [95% CI, 3.9%–9.4%]), and 11 and 15 years (6.6% [95% CI, 4.4%–9.8%]), and with rapid height growth between 6 and 11 years (7.2% [95% CI, 4.7%–10.7%]) and 11 and 15 years (6.3% [95% CI, 4.1%–9.4%]).

Tables 4 and 5 show the crude and adjusted ORs for HBP for the female and male adolescents, respectively, according to the variables that remained in the final analysis model. Among female adolescents, a family history of AH was associated with a 3-fold increase in the chance of HBP (adjusted OR, 3.1 [95% CI, 1.26–7.54]). Current work and screen time were kept in the model for female adolescents to control for confounding. In male adolescents, excessive maternal weight before pregnancy was associated with a 2.3-fold increase in the chance of HBP (adjusted OR, 2.3 [95% CI, 1.05–5.19]). In both sexes, adolescents with excessive weight were more likely to have HBP than those with normal weight (adjusted ORs, 4.2 and 4.4, for female and male adolescents, respectively). A higher FMI and FFMI in female (adjusted ORs, 1.2 and 1.4, respectively) and male adolescents (adjusted ORs, 1.2 and 1.4, respectively) increased the chance of presenting HBP.

No association between rapid weight gain or height growth and HBP was found among female adolescents. Male adolescents who presented rapid weight gain in the middle (48 months-6 years) of childhood had ≈3 times more chance of HBP (adjusted OR, 2.9) than their counterparts without rapid weight gain. Male adolescents who presented rapid height growth between 6 and 11 years had a 2.5 times higher chance of HBP.

Tables S1 and S2 contain median (interquartile interval) and range values for anthropometry and body composition indexes, SBP and DBP, and growth patterns between birth and 15 years, for female and male adolescents, respectively, with and without HBP. The median values for SBP and DBP at 6, 11, and 15 years among female adolescents with HBP were higher than those observed among female adolescents without HBP. The same was observed for male adolescents, except for DBP at 6 years, which was similar in adolescents with and without HBP. The interquartile intervals of the anthropometric and body composition medians for adolescents with HBP overlapped the interquartile intervals for adolescents without HBP in both sexes.

Table S3 shows the comparison between female (N=691) and male (N=726) cohort members with BP measures on 3 occasions and the ones with missing BP on at least 1 occasion (included and not included in the final analysis) (N=301 female adolescents, and N=311 male adolescents) in the analyses by sex. The losses were higher among male adolescents from mothers aged 21 to 25 years (P=0.031) and among those whose mothers had pregestational BMI <25 kg/m^2 (P=0.020).

DISCUSSION

To the best of our knowledge, this is one of the largest Brazilian cohort studies focused on describing the

Table 2. Prevalence of HBP With 95% CI Among Adolescents by Sex

	Female adolescents (N=691)		Male adolescents (N=726)	
Characteristics	Prevalence of HBP (95% CI), %	*P* value*	Prevalence of HBP (95% CI), %	*P* value*
All	3.2 (1.9–4.5)	…	4.3 (2.8–5.8)	…
Family characteristics				
Household income (quintiles)		0.554		0.481
First (poorest)	2.5 (0.8–7.6)		2.9 (1.1–7.6)	
Second	3.4 (1.4–7.9)		5.6 (2.8–10.8)	
Third	2.1 (0.7–6.3)		2.8 (1.0–7.1)	
Fourth	5.4 (2.7–10.4)		6.2 (2.2–11.2)	
Fifth (wealthiest)	2.2 (0.7–6.7)		3.6 (1.5–8.2)	
Family history of hypertension		0.017†		0.659
Yes	6.5 (3.5–11.6)		4.7 (2.4–9.1)	
No	2.2 (1.2–4.0)		4.0 (2.5–6.2)	
Maternal characteristics				
Schooling, y				
≤4	1.9 (0.5–7.4)	0.269	5.0 (2.1–11.5)	0.720
5–8	4.6 (2.7–7.8)		4.7 (2.8–7.8)	
≥9	2.3 (1.1–4.8)		3.7 (2.1–6.4)	
Age at childbirth, y				
≤20	3.2 (1.3–7.5)	0.533	1.2 (0.3–4.8)	0.109
21–25	1.7 (0.6–5.2)		5.2 (2.7–9.7)	
26–30	4.6 (2.2–9.4)		6.0 (3.2–10.8)	
>30	3.3 (1.6–6.8)		4.5 (2.4–8.1)	
Pregestational BMI, kg/m^2		0.625		0.039†
<25.0	3.3 (1.8–6.1)		3.3 (1.8–5.8)	
≥25.0	4.3 (2.1–8.4)		7.4 (4.5–11.8)	
Maternal smoking during pregnancy		0.457		0.673
Yes	4.1 (1.9–8.3)		4.9 (2.6–9.1)	
No	2.9 (1.8–4.8)		4.1 (2.7–6.1)	
Adolescent characteristics at birth				
Gestational age, wk				
<37	2.3 (0.6–8.6)	1.000	6.4 (2.9–13.5)	0.274
≥37	3.3 (2.1–5.1)		4.0 (2.7–5.8)	
Intrauterine growth				
SGA	3.7 (0.9–13.7)	0.424	11.9 (5.7–22.9)	0.019†
AGA	2.8 (1.6–4.6)		3.5 (2.2–5.4)	
LGA	4.9 (2.0–11.1)		4.1 (1.7–10.0)	
Skin color‡				
White	3.4 (2.1–5.5)	0.817	5.0 (3.3–7.3)	0.326
Non-White	2.7 (1.2–6.0)		3.1 (1.5–6.4)	
Adolescent genetic inheritance for HBP				
Systolic (*z* score) (terciles)				
First	2.7 (1.2–5.9)	0.492	5.1 (2.8–9.0)	0.365
Second	2.7 (1.2–5.8)		2.6 (1.6–5.6)	
Third	4.5 (2.3–8.3)		4.4 (2.5–7.8)	

(*Continued*)

Table 2. Continued

	Female adolescents (N=691)		Male adolescents (N=726)	
Diastolic (*z* score) (terciles)				
First	2.3 (1.0–5.4)	0.576	3.9 (2.0–7.2)	0.388
Second	3.2 (1.6–6.6)		5.5 (3.1–9.4)	
Third	4.1 (2.2–7.8)		2.9 (1.4–5.9)	
Adolescent characteristics at 15 y				
Puberty status (Tanner)				
2–4	2.8 (0.9–8.4)	0.599	16.7 (7.1–34.3)	0.033†
5–7	5.2 (3.1–8.6)		4.8 (2.7–8.4)	
8–10	3.5 (1.3–9.1)		4.2 (2.3–7.6)	
Current work				
Yes	5.6 (2.1–13.9)	0.278	2.4 (0.6–8.9)	0.568
No	3.0 (1.8–4.7)		4.7 (3.3–6.8)	
Mean sleep duration, h/d		0.107		0.687
<8	4.1 (2.6–6.5)		4.4 (2.9–6.7)	
≥8	1.6 (0.6–4.2)		3.5 (1.8–6.9)	
Physical activity, mg/d (tertiles)		0.509		0.216
First	2.6 (1.1–6.1)		5.0 (2.6–9.3)	
Second	3.2 (1.5–6.5)		6.1 (3.2–11.4)	
Third	4.9 (2.3–10.0)		2.6 (1.2–5.8)	
Daily sodium intake, g (tertiles)		0.290		1.000
First	4.6 (2.6–8.0)		4.0 (2.1–7.5)	
Second	2.4 (1.0–5.6)		4.4 (2.5–7.8)	
Third	2.3 (1.0–5.4)		4.4 (2.5–7.8)	
Screen time, h/d				
<3	2.9 (1.4–5.7)	0.827	4.6 (2.0–9.0)	0.665
≥3	3.4 (2.0–5.7)		4.0 (2.6–6.0)	
BMI *z* score				
Normal weight	1.3 (0.6–2.9)	<0.001	2.0 (1.1–3.7)	<0.001
Excessive weight	6.7 (4.1–10.7)		9.3 (6.1–13.8)	
FMI, kg/m^2 (tertiles)				
First	0.0 (0.0–0.0)	0.009†	1.8 (0.9–3.7)	<0.001†
Second	1.6 (0.7–3.9)		4.0 (1.9–8.2)	
Third	5.5 (3.4–8.6)		10.4 (6.5–16.1)	
FFMI, kg/m^2 (tertiles)				
First	1.2 (0.4–3.1)	0.005†	2.4 (0.8–7.3)	0.015†
Second	4.6 (2.6–8.2)		2.0 (0.8–4.7)	
Third	6.2 (3.0–12.4)		6.5 (4.3–9.6)	

AGA indicates adequate for gestational age; BMI, body mass index; FFMI, fat-free mass index; FMI, fat mass index; HBP, high blood pressure; LGA, large for gestational age; and SGA, small for gestational age.

*Fisher exact and χ^2 test.

†*P* values for linear trend.

‡Based on classification adopted by the Brazilian Census Bureau; Black, Brown, White, Yellow, Indigenous and Mulatto.[19]

prevalence of HBP in adolescents that used information on BP collected in 3 separate occasions (at 6, 11, and 15 years of age). The prevalence of HBP at 15 years of age was 3.2% (95% CI, 1.9%–4.5%) in female adolescents and 4.3% (95% CI, 2.8%–5.8%) in male adolescents. We did not identify other studies with the same age group or population. However, these prevalence estimates were lower than those found for female adolescents (7.3% [95% CI, 6.6%–8.2%]) and male adolescents (11.9% [95% CI, 11.1%–12.8%]) described in a nationwide school-based cross-sectional study conducted with adolescents aged 12 to 17 years

Table 3. Prevalence (95% CI) of HBP According to Adolescents' Growth by Sex

Characteristics	Female adolescents (N=691)		Male adolescents (N=726)	
	Prevalence of HBP (95% CI), %	*P* value*	Prevalence of HBP (95% CI), %	*P* value*
Rapid weight gain (*z* score)				
0–3 mo				
Yes	4.1 (2.3–7.1)	0.265	3.7 (2.2–6.3)	0.577
No	2.4 (1.2–4.5)		4.8 (3.0–7.6)	
3–12 mo				
Yes	3.9 (2.2–6.7)	0.261	5.4 (3.4–8.5)	0.356
No	2.3 (1.1–4.5)		3.7 (2.2–6.1)	
12–24 mo				
Yes	4.8 (2.8–7.9)	0.022	6.0 (3.4–10.3)	0.225
No	1.6 (0.7–3.6)		3.8 (2.4–6.0)	
24–48 mo				
Yes	4.0 (2.3–6.9)	0.275	4.7 (2.8–7.8)	0.853
No	2.5 (1.3–4.7)		4.2 (2.6–6.7)	
48 mo–6 y				
Yes	4.5 (2.6–7.5)	0.078	6.8 (4.4–10.3)	0.009
No	2.1 (1.0–4.1)		2.6 (1.5–4.7)	
6–11 y				
Yes	4.4 (2.5–7.3)	0.188	6.1 (3.9–9.4)	0.041
No	2.3 (1.2–4.5)		2.9 (1.7–5.0)	
11–15 y				
Yes	3.0 (1.6–5.5)	0.832	6.6 (4.4–9.8)	0.005
No	3.3 (1.9–5.8)		2.3 (1.2–4.4)	
Rapid length gain (*z* score)				
0–3 mo				
Yes	2.7 (1.4–5.2)	0.660	2.7 (1.4–5.1)	0.062
No	3.5 (2.0–6.1)		5.6 (3.7–8.5)	
3–12 mo				
Yes	3.0 (1.5–5.6)	1.000	3.3 (1.8–5.6)	0.199
No	3.1 (1.7–5.4)		5.5 (3.6–8.4)	
12–24 mo				
Yes	3.4 (1.9–6.1)	0.651	3.7 (2.1–6.5)	0.464
No	2.6 (1.4–5.0)		5.0 (3.2–7.8)	
24–48 mo				
Yes	4.1 (2.3–7.1)	0.267	4.2 (2.2–7.8)	1.000
No	2.4 (1.3–4.6)		4.5 (3.0–7.8)	
48 mo–6 y				
Yes	3.7 (2.1–6.5)	0.505	4.9 (3.0–8.0)	0.580
No	2.6 (1.4–4.8)		3.9 (2.4–6.3)	
6–11 y				
Yes	5.0 (3.0–8.3)	0.028	7.2 (4.7–10.7)	0.002
No	1.9 (1.0–3.8)		2.3 (1.3–4.3)	
11–15 y				
Yes	4.7 (2.8–7.8)	0.051	6.3 (4.1–9.4)	0.016
No	2.0 (1.0–4.0)		2.6 (1.4–4.7)	

HBP indicates high blood pressure.
*Determined using χ^2 test.

Table 4. Crude and Adjusted ORs for HBP Among Female Adolescents

Characteristics	Crude OR	95% CI	*P* value	Adjusted OR	95% CI	*P* value
Family characteristics						
Family history of hypertension						
Yes	3.1	1.26–7.54	0.014*	3.1	1.26–7.54	0.014*
Adolescent characteristics at 15 y						
Current work						
Yes	1.9	0.63–5.89	0.250	2.7	0.83–8.52	0.100
Screen time, h/d						
≥3.0	1.2	0.49–2.87	0.705	2.0	0.73–5.49	0.181
Adolescent current nutritional status†						
BMI (excessive weight)						
≥1.0	5.3	2.06–13.82	0.001*	4.2	1.53–11.30	0.005*
FMI, kg/m^2	1.2	1.11–1.32	<0.001*	1.2	1.07–1.30	0.001†
FFMI, kg/m^2	1.4	1.15–1.69	0.001*	1.4	1.11–1.68	0.003†

BMI indicates body mass index; FFMI, fat-free mass index; FMI, fat mass index; HBP, high blood pressure; and OR, odds ratio.
*Determined using χ^2 test.
†Adjusted for family history of hypertension, current work, and screen time; *P* values for linear trend.

old in Brazil (the Estudo dos Riscos Cardiovasculares em Adolescentes [ERICA] study).[7]

Methodological issues may explain the observed difference between the results. First, the ERICA study referenced in the National High Blood Pressure Education Program Working Group values, whose cutoff values for the definition of HBP differ in adolescents aged >12 years, favoring an increase in its prevalence.[34] Second, the ERICA study enrolled adolescents aged 12 to 17 years, and as the age group increases, children and adolescents are more likely to be diagnosed with HBP.[35] Third, and most important, the ERICA study had a cross-sectional design with BP measured on a single occasion, whereas our study was a cohort one, with BP measured on 3 separate occasions. With repeated BP measurements, the prevalence of confirmed HBP tends to decrease in part because of inherent BP variability and because of an adjustment to the experience of having BP measured (also known as the accommodation effect), as well as

Table 5. Crude and Adjusted ORs for HBP Among Male Adolescents

Characteristics	Crude OR	95% CI	*P* value	Adjusted OR	95% CI	*P* value
Maternal characteristics						
Pregestational BMI, kg/m^2 (excessive weight)						
≥25.0	2.4	1.05–5.19	0.037*	2.3	1.05–5.19	0.037*
Adolescent characteristics at 15 y†						
Puberty status (Tanner)						
5–7	0.2	0.07–0.70	0.045*,‡	0.2	0.05–0.59	0.113‡
8–9	0.2	0.06–0.63		0.2	0.07–0.72	
Adolescent current nutritional status§						
BMI (excessive weight; *z* score)						
≥1.0	5.0	2.32–10.85	<0.001*	4.4	1.63–11.90	0.003*
FMI, kg/m^2	1.2	1.11–1.29	<0.001*	1.2	1.07–1.29	<0.001*,‡
FFMI, kg/m^2	1.3	1.15–1.59	<0.001*	1.4	1.11–1.68	0.004*,‡
Adolescent growth patterns§						
Rapid weight gain (48 mo–6 y) (*z* score)	2.7	1.28–5.76	0.009*	2.9	1.24–6.85	0.014*
Rapid height gain (6–11 y) (*z* score)	3.3	1.51–7.01	0.003*	2.6	1.13–5.96	0.025*

BMI indicates body mass index; FFMI, fat-free mass index; FMI, fat mass index; HBP, high blood pressure; and OR, odds ratio.
*Test of heterogeneity.
†Adjusted for maternal pregestational BMI.
‡*P* values for linear trend.
§Adjusted for maternal pregestational BMI and puberty status at 15 years.

to the phenomena of regression to the mean (ie, the tendency of individuals at the extremes to have values nearer to the mean on repeated measurement).[15,36]

Several modifiable risk factors for HBP already established at more advanced ages have also become evident in adolescence.[5] In our study, in female adolescents, a family history of AH increased the chance of HBP by 3 times compared with those without a family history of AH. In male adolescents, excessive maternal pregestational weight increased the chance of HBP by 2.3 times, a finding that had already been associated with HBP in childhood.[5]

A higher BMI, FMI, and FFMI were associated with higher HBP prevalence in both sexes. The relationship of higher BMI values with HBP in adolescents has already been evidenced in other studies.[6,12,13,37] On the other hand, the observed relationship with body composition is a new contribution of our study, as the relationship between HBP and FMI and FFMI is lacking in other studies, particularly with Brazilian adolescents.[38] In 4 population-based cohort studies from Germany, consisting of individuals aged ≥20 years not taking antihypertensive medication at the baseline and followed up with follow-up times between 4 and 7 years, there were positive associations of annual changes in FM and FFM with annual changes in SBP and DBP.[39] Likewise, FM and FFM gains were associated with an increased risk of the incidence of AH, whereas decreases in FM and FFM were positively associated with BP incident normalization.[39]

Similarly, there is a scarcity of studies that evaluate the relationship of the speed of weight gain and growth with HBP.[40] In male subjects, this relationship was present with rapid weight gain between the ages of 4 and 6 years and with the highest speed of height gain between the ages of 6 and 11 years. Previous studies have shown that faster infant and early childhood weight gain relates more strongly to adult FFM than to adiposity, whereas weight gain in later childhood and adolescence contributes more to adult adiposity, which, in turn, is related to chronic disease risk.[41–44] On the other hand, an analysis of the Pelotas 1993 Birth Cohort at 14 to 15 years of age to explore the independent effects of weight and length/height gains during the first 4 years of life on SBP found that rapid weight gain between 0 and 6 months increased adolescent BP, whereas early length/height gains were not associated with higher BP.[41]

We found no association between the PRS for SBP and DBP and HBP in the adolescents, even when we ran the analyses after excluding the family history of AH variable from the adjusted model. The risk of AH in individuals with a family history is much higher compared with nonrelatives in the general population,[45] which is consistent with our finding of an association between a family history of AH and HBP among female adolescents. Genome-wide association studies have identified many BP loci, although each individually accounts for slight BP differences (<1 mm Hg).[46] It is not clear whether the lack of association of the PRS for SBP and DBP with HBP is attributable to insufficient sample size, whether the PRS missed important genes, whether the family history in our study is more about the shared environment than shared genes, or whether this finding is attributable to unmeasured variables leading to residual negative confounding.

The Black population has a higher risk of hypertension, organ damage at younger ages, frequency of resistant hypertension, kidney disease, stroke, heart failure, and mortality than other ethnic groups.[46,47] BP level is also higher among Black, Hispanic, and Asian children. In our study, given the ethnic diversity of the Brazilian population, we used self-reported skin color instead of race categorization to explore the association between ethnic origin and HBP, and we found no association in either sex. Nonetheless, there is a lack of consensus and controversy about the use of skin color as an independent variable for health outcomes. As a social and cultural construct, ethnoracial self-identifications can vary depending on the social status and social contexts, and can change over the life course.[48] In addition, psychosocial stressors, such as racism, may explain BP variability better than skin tone or genetic ancestry, pointing to the primacy of sociocultural processes involved in the occurrence of diseases.[49,50] In agreement with this, a systematic review planned to assess BP tracking from childhood to adulthood found that BP tracking did not vary markedly across race/population groups.[51]

This study has strengths and limitations. The first limitation is that BP was evaluated only by the oscillometric method. Although we have repeated BP measurements over time, if AH is suspected on the basis of oscillometric readings, confirmatory measurements should be obtained by auscultation.[15] Nonetheless, oscillometric BP devices are an alternative for initial screening, and they have become commonplace in health care settings. The most perceived benefits of oscillometrics described are ease of use, lack of digit preference, and automation. Another limitation is the potential for white coat hypertension bias. Although the cohort participants are familiar with the research clinic environment where the follow-ups are performed, it is possibly a source of BP elevation. Recent studies indicate that up to half of children referred for evaluation of elevated office BP have white coat hypertension.[52] The study was performed in only 1 region of the country, which national studies suggest is the region with the highest prevalence of AH in adults.[9] Thus, although the associations between the independent variables and HBP are not affected by the prevalence of HBP, it is possible that the observed

prevalence of adolescents with HBP is higher in our study than in other regions of the country. Also, because maternal pregestational BMI in our sample was associated with HBP and missing measures of BP, it is possible that our prevalence of HBP among male adolescents is overestimated.

Another limitation of our study is related to the long-time interval (years) between the BP measurements that, despite strengthening the diagnosis of HBP by remaining high in the 3 moments, lacks information on the variation of BP in shorter intervals (weeks or months) as generally used at clinical settings. In addition, the family history of AH was collected only at the 6-year follow-up. Then, we have no information on family members who have been diagnosed with AH after that time. Also, residual confounding by unknown/unmeasured variables may have affected the detection of association between other variables, like socioeconomic status (that had been shown to be associated with HBP by others), and BP.[53]

Among the strengths, first, it was a longitudinal, population-based study involving a large number of adolescents. Second, the standardized BP measurements were performed in 3 follow-ups (at 6, 11, and 15 years of age). Third, our study evaluated several potential risk factors related to HBP. Early identification and prevention of these factors can significantly reduce the prevalence of this and other cardiometabolic diseases. Fourth, the inclusion of the PRS for SBP and DBP, defined according to a clear-cut and standardized protocol, is another strength of the study. Finally, although obesity is a well-known major risk factor for pediatric hypertension,[46] the novelty in our study derives from the assessment of body composition with air-displacement plethysmography, a more precise, indirect method (instead of doubly indirect methods, such as BMI and skin folds). Furthermore, studies exploring growth velocity's role on adolescents' BP are scarce. The use of body composition parameters assessed through air-displacement plethysmography and conditional growth variables is a unique contribution of our study.

Because of the heterogeneity between studies on the definition and prevalence of AH and the lack of randomized trials indicating short- and long-term benefits of managing and treating HBP in childhood and adolescence, there is a lack of evidence to recommend routine BP measurement at these stages of life.[15,54] Nonetheless, many risk factors for the development of cardiovascular diseases can start in a subclinical way and may be modifiable or even be avoided.[42] AH is 1 of these factors.[5,55,56] Our study showed that higher FM in both sexes and velocity of weight gain in male adolescents are risk factors for HBP in adolescents aged 15 years potentially amenable to prevention. Although we cannot predict with certainty the clinical course of adolescents with HBP, our findings support the importance of preventing excessive body weight and body fat deposition from childhood to adolescence, aiming to have an impact on AH, a disease that remains the greatest cause of morbidity and mortality worldwide.[56]

ARTICLE INFORMATION

Received April 12, 2023; accepted July 13, 2023.

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Sources of Funding

This article is based on data from the study Pelotas Birth Cohort, 2004 conducted by Postgraduate Program in Epidemiology at Universidade Federal de Pelotas, with the collaboration of the Brazilian Public Health Association. From 2009 to 2013, the Wellcome Trust supported the 2004 birth cohort study. The World Health Organization, National Support Program for Centers of Excellence (PRONEX), Brazilian National Research Council (CNPq), Brazilian Ministry of Health, and Children's Pastorate supported previous phases of the study. The 11- and 15-year follow-ups were supported by PRONEX, CNPq, the Research Support Foundation of the State of Rio Grande do Sul, and Research Support Foundation of the State of São Paulo. Alicia Matjasevich, Aluisio Jardim Dornellas de Barros, Ina da Silva dos Santos, and Luciana Tovo-Rodrigues are supported by CNPq. This study was financed in part by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior - Brasil - Finance Code 001, Research Support Foundation of Rio Grande do Sul, CNPq.

Disclosures

None.

Supplemental Material

Tables S1–S3

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