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ORIGINAL RESEARCH

Life-Course Blood Pressure Levels, 38-Year Tracking, and Prediction of Hypertension

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BACKGROUND: Elevated childhood blood pressure (BP) levels may persist into adulthood, and persistent exposure to elevated BP since childhood may increase the adulthood risk of hypertension. This study examined the tracking of childhood BP into adulthood over 38 years. We also studied the association between long-term cumulative BP exposure in childhood and adolescence and the risk of developing hypertension in adulthood.

METHODS: Participants were from the YFS (Cardiovascular Risk in Young Finns Study) that began in 1980 (N=3596; ages 3–18 years). The cohort was remeasured in 1983, 1986, 1989, 2001, 2007, 2011, and 2018 through 2020. In total, 2064 attended the latest study visit (45% men; ages 40–58 years; average follow-up, 38.0 years). BP was measured repeatedly using standard methods. Cumulative BP exposure was defined using area under the curve. Hypertension was defined as systolic BP ≥140 mm Hg, diastolic BP ≥90 mm Hg, self-reported use of antihypertensive medication, or diagnosis of hypertension given by a physician.

RESULTS: A weak correlation was observed between childhood (baseline ages 3–18 years) and adulthood BP (latest follow-up, ages 41–56 years): r=0.298 (P<0.0001) in females and r=0.187 (P<0.0001) in males. Long-term cumulative exposure to elevated BP (highest versus lowest systolic BP quartile) between ages 6 and 12 years was associated with higher risk of hypertension in adulthood both in females (hazard ratio [HR], 3.85 [95% CI, 2.87–5.17]) and in males (HR, 2.66 [95% CI, 2.07–3.42]).

CONCLUSIONS: These data indicate that long-term cumulative exposure to elevated BP levels in childhood/adolescence are associated with a substantially increased risk of adult hypertension.

Key Words: blood pressure ■ child ■ epidemiologic studies ■ hypertension ■ longitudinal studies

Hypertension is the leading cause of atherosclerotic cardiovascular diseases and premature death worldwide.[1] Prospective studies have shown a strong, independent association between blood pressure (BP) and the risk of cardiovascular disease, with no apparent threshold.[2,3] According to the World Health Organization, 1.28 billion adults aged 30 to 79 years have hypertension.[4] With ever-increasing prevalence and severe consequences, hypertension is a major health risk that places a considerable burden on public health, social health care systems and economy. Since BP levels have been shown to track from childhood to adulthood, detecting elevated levels as early as possible in the life course might be an important consideration for cost-effective health care.[5]

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CLINICAL PERSPECTIVE

What Is New?

- In this study, using the longitudinal follow-up data from the YFS (Cardiovascular Risk in Young Finns Study), we observed that blood pressure levels measured in childhood are associated with corresponding blood pressure levels in adulthood over a 38-year follow-up period.
- Additionally, we found that high long-term cumulative childhood blood pressure exposure, even without exceeding the reference guideline cutoffs, is associated with a higher risk of hypertension in adulthood.

What Are the Clinical Implications?

- These data highlight the importance of repeated childhood blood pressure monitoring and early prevention.

Nonstandard Abbreviations and Acronyms

YFS	Cardiovascular Risk in Young Finns Study

As BP levels have been suggested to persist over time, the effect of BP on an individual's risk profile likely begins in childhood.[5–10] The traditional approach of treating high BP in adulthood reduces the risk of cardiovascular disease,[11–13] but the accumulation of BP exposure since childhood may also have an independent role for later cardiovascular health.[14] It is important to identify those at greatest risk of developing high BP early on, as it could provide a more effective prevention strategy for high BP and hypertension, which in turn might have wide implications on cardiovascular disease risk.

BP monitoring from childhood has been widely debated.[15–19] Significant organizations, including the National High Blood Pressure Education Program and the American Heart Association, have recommended universal routine screening of BP in childhood and adolescence.[17,20] However, the practice has been criticized since the balance of benefits and harms of screening for hypertension has not been fully understood, with the US Preventive Services Task Force concluding the evidence as insufficient to support screening for high BP in children.[18] Thus, further evidence is needed to determine the long-term tracking of BP since childhood and the role of childhood/adolescence BP exposure for adulthood hypertension risk. Prior research has demonstrated that elevated BP in childhood and adolescence is associated with target-organ damage[7,21–25] and an increased risk of hypertension in adulthood.[26–29] However, the cumulative burden of BP exposure throughout childhood and adolescence and its long-term contribution to hypertension risk in adulthood remains less explored.[30] Whether the prediction of hypertension in adulthood could be improved by considering the accumulation of long-term BP exposure from childhood is still unclear.

In this study, taking advantage of the 38-year longitudinal data on the YFS (Cardiovascular Risk in Young Finns Study) cohort, we examined (1) variations in BP levels by age and time, (2) tracking of BP from childhood to adulthood in a 38-year follow-up, and (3) the association of cumulative BP exposure in childhood with risk of hypertension in adulthood.

METHODS

Data Availability

The data that support the findings of this study are available from the YFS study group upon reasonable request. Investigators can submit an expression of interest to the chairman of the YFS steering group (Professor Olli T. Raitakari, University of Turku, Turku, Finland).

Study Population

The YFS is an ongoing multicenter follow-up study conducted in 5 Finnish university cities and their rural surroundings. The original target of the study was to detect cardiovascular risk factors of Finnish children and adolescents.[31] The study began in 1980, when 4320 Finnish children and adolescents in 6 age cohorts (ages 3, 6, 9, 12, 15, and 18 years) were randomly selected from a national register and invited to participate. Of the invited, 3596 (83.2%) participated in the first cross-sectional survey. Since the baseline, complete cardiovascular risk factor assessments have been conducted in 1983, 1986, 2001, 2007, 2011, and 2018 through 2020. Participants gave written informed consent, and the study was reviewed by local ethics committees. Study amendments were approved by the Ethics Committee of Turku University Hospital. Details of the study design were presented previously.[31]

To examine the variations in BP levels by age and time, we used data on systolic and diastolic BP from all 7 follow-ups that were conducted between 1980 and 2018 through 2020. For the tracking effect analysis, we included 1667 participants whose systolic BP was measured in baseline (childhood/adolescence; age 6, 9, 12, 15, or 18 years) and whose adult systolic BP was measured in the latest follow-up study (adulthood/midlife; age 41, 44, 47, 50, 53 or 56 years). Diastolic BP was measured both in childhood/adolescence and in adulthood/midlife from 1383 participants. To define

stability of BP levels, we included N=2935 participants who had systolic BP measured in childhood/adolescence (age 6, 9, 12, 15, or 18 years) and at least once in adulthood (follow-up studies in 2001, 2007, 2011, and 2018–2020; ages 21–56 years). For the time-to-event analyses, we included 2398 participants who participated in at least 1 of the adulthood follow-up studies conducted in 2001, 2007, 2011, and 2018 through 2020.

BP Measurement

BP was measured from the right brachial artery with a standard mercury sphygmomanometer in 1980 and 1983, with a random-zero sphygmomanometer (Hawksley & Sons, Lancin, UK) in 1986 and 2001, and with an automatic oscillometric device in the follow-up studies from 2007 to 2018. In 1980, BP from 3-year-old children was measured with an ultrasound scanning device. At all study phases, BP was measured after the participant had been seated for 5 minutes. In the auscultatory BP measurements, the first Korotkoff phase was used to determine systolic BP, and the fifth phase was used to determine diastolic BP. All BP measurements were done at least 3 times on each participant, and the average of these 3 measurements was used in the analyses.

Hypertension

Hypertension was defined as systolic BP ≥140 mm Hg, diastolic BP ≥90 mm Hg, self-reported use of antihypertensive medication, or self-reported diagnosis of hypertension given by a physician. To be included in the hypertension classification, a participant had to have data on all 4 of these variables. To calculate the hazard ratios (HRs), hypertension was defined using data collected in 2001, 2007, 2011, and 2018 to 2020.

Cumulative BP Burden

The area under the curve (AUC) for both systolic and diastolic BP was defined to indicate a long-term BP exposure. The AUC variables were calculated separately for childhood (ages 6–12 years), adolescence (ages 12–18 years) and the entire period from childhood to adolescence (ages 6–18 years). For interpretability, the AUC variables were standardized, resulting in variables with a mean of 0 and an SD of 1. To classify the participants according to their cumulative BP exposure in childhood and adolescence, the continuous systolic and diastolic BP AUC variables were divided into quartiles using the cut points of 25th, 50th, and 75th percentiles, resulting in the groups 0=very low, 1=low, 2=intermediate, and 3=high cumulative systolic/diastolic BP exposure during the life stage under consideration. All AUC quartiles were defined separately for males and females. To define the mean BP levels of each group used in calculating the risks, yearly age- and sex-specific childhood mean BP levels were calculated using the childhood BP AUC variables. A detailed description of the calculation of the AUC variables is described in Data S1 and has been published previously.[32]

Statistical Analysis

The variation of BP levels according to age and time was studied by calculating the sex-specific means±SDs of systolic and diastolic BP in relation to age at all follow-up studies. The 38-year tracking of systolic and diastolic BP were first estimated by calculating Pearson's correlation coefficients stratified by sex and age. For the correlation analysis, normality of BP distributions was visually inspected and tested using the Kolmogorov–Smirnov test. The BP data in participants aged 3 years at baseline were excluded due to the methodological differences in BP measurement between the baseline and the follow-up studies. Participants using antihypertension medication (N=377) at the latest follow-up were excluded from the tracking analyses. Differences in correlation coefficients between male and female participants were formally tested using normal probability test for difference between *Z*-transformed correlation coefficients. After the correlation analyses, generalized estimating equations[33] were used to define stability of BP levels. Using the generalized estimating equation models, we estimated the tracking of BP from childhood to adulthood stratified by age when BP was first measured and the length of follow-up. From the generalized estimating equation models, regression coefficients were derived to indicate stability ("stability coefficients").[34] Cox proportional hazards regression was used to estimate the risk of hypertension in adulthood on the basis of the childhood/adolescence cumulative BP burden. The time origin was defined as the follow-up year 2001, when hypertension status was first assessed in adulthood. Follow-up time was calculated as age at the event or censoring, and the outcome was the first occurrence of hypertension defined in the follow-ups in 2001, 2007, 2011, or 2018. Participants without hypertension were censored at the last follow-up with available hypertension data (if lost to follow-up) or at the 2018 follow-up (if normotensive throughout all follow-ups). To evaluate the predictive utility of different BP classification methods in childhood, we compared 2 exposure models for systolic BP during ages 6 through 12 years: (1) cumulative BP using a quartile-based classification of the AUC variable (quartiles 0–3), and (2) the number of elevated systolic and/or diastolic BP values based on the American Academy of Pediatrics guideline thresholds[35] at age 6, 9, or 12 years (0, 1, 2, or 3 threshold

Table 1. Characteristics of the Study Population in Baseline and the Latest Follow-Up Study

	All (N=3596)		Females (N=1832)		Males (N=1764)	
Baseline (1980)	No.	Mean±SD	No.	Mean±SD	No.	Mean±SD
Age, y	3596	10.4±5.0	1832	10.5±5.0	1764	10.4±5.0
SBP, mm Hg	3549	112.5±12.2	1817	111.7±11.2	1732	113.6±12.9
DBP, mm Hg	3000	68.8±9.6	1543	68.7±9.4	1457	68.8±9.8
Height, cm	3573	141.3±26.0	1823	139.9±24.3	1750	142.6±27.5
Weight, kg	3573	38.3±18.3	1823	37.4±16.7	1750	39.3±19.8
BMI, kg/m^2	3567	17.8±3.1	1821	17.9±3.1	1746	17.8±3.1
	All (N=2064)		Females (N=1132)		Males (N=932)	
38-Year follow-up (2018)	N	Mean±SD	N	Mean±SD	N	Mean±SD
Age, y	2064	48.7±5.0	1132	48.8±4.9	932	48.6±5.1
SBP, mm Hg	2057	129.6±15.9	1127	126.6±16.4	930	133.2±14.5
DBP, mm Hg	2057	82.7±9.9	1127	80.7±9.6	930	85.2±9.7
BMI, kg/m^2	2064	27.8±5.4	1132	27.7±5.9	932	28.0±4.7
Antihypertensive medication use, %	377	18.3	182	16.1	195	20.9

BMI indicates body mass index; DBP, diastolic blood pressure; and SBP, systolic blood pressure.

exceedings). All AUC quartiles were defined separately for males and females. Guideline-based classification was based on age-, sex-, and height-specific 90th percentile ("elevated BP") cutoffs. The ability of both classification approaches to predict adulthood hypertension was assessed using area under the receiver operating characteristic curves, adjusting for age and sex. C-statistics were compared using receiver operating characteristic contrast analysis and were estimated using the full sample representing in-sample estimates. All statistical analyses were performed using SAS 9.4 software (SAS Institute, Cary, NC), and $P<0.05$ was used to indicate statistical significance.

RESULTS

Characteristics of the study population in the baseline and in the latest follow-up study are shown in Table 1. To study if the representativeness of the original study population was maintained in the present cohort, we compared the baseline (1980) characteristics between those who participated in the 38-year follow-up study and those who did not (N=1539). Those who participated in the 38-year follow-up study were older (baseline age, 10.7 versus 10.1 years; $P<0.001$) and more often females (55% versus 45%; $P<0.0001$) compared with nonparticipants. However, there were no significant differences in systolic or diastolic BP between the groups.

Variations in systolic and diastolic BP according to age and time are presented in Figure 1. We observed an increase in both systolic and diastolic BP levels with age. This upward trend relative to increasing age was noticeable in all follow-up years, with systolic BP levels tending to be slightly higher in males than in females. When BP levels assessed using different measuring methods were compared, the random-zero sphygmomanometer was found to systematically give lower values for both systolic and diastolic BP.

We found a weak tracking of BP in both male and female participants in the 38-year follow-up (Table 2). The tracking coefficients varied only slightly between the age cohorts, while a stronger tracking of BP was observed in females than in males (Z score, 2.39; $P=0.017$). When childhood BP measurement age and the length to follow-up were considered for both systolic BP (coefficients ranging from 0.22 to 0.66) and diastolic BP (coefficients ranging from 0.07 to 0.49), the degree of tracking tended to decrease with increasing length of follow-up in all age groups (Figure 2, Tables S1 and S2).

The associations between long-term systolic BP burden in childhood and adolescence and the risk of hypertension in adulthood are presented in Table 3. In these analyses, the risk of hypertension was found to increase with increasing cumulative childhood systolic BP burden. Participants with low (HR, 2.00 [95% CI, 1.47–2.72] in females; HR, 1.73 [95% CI, 1.33–2.24] in males) and intermediate (HR, 1.97 [95% CI, 1.44–2.71] in females; HR, 2.15 [95% CI, 1.66–2.77] in males) childhood cumulative systolic BP burden had approximately twice the risk of hypertension in adulthood compared with those with very low childhood systolic BP burden. For the female participants with high cumulative systolic BP burden, the risk of hypertension was nearly 4 times greater compared with the "very low" group (HR, 3.85 [95% CI, 2.87–5.17]). For males,

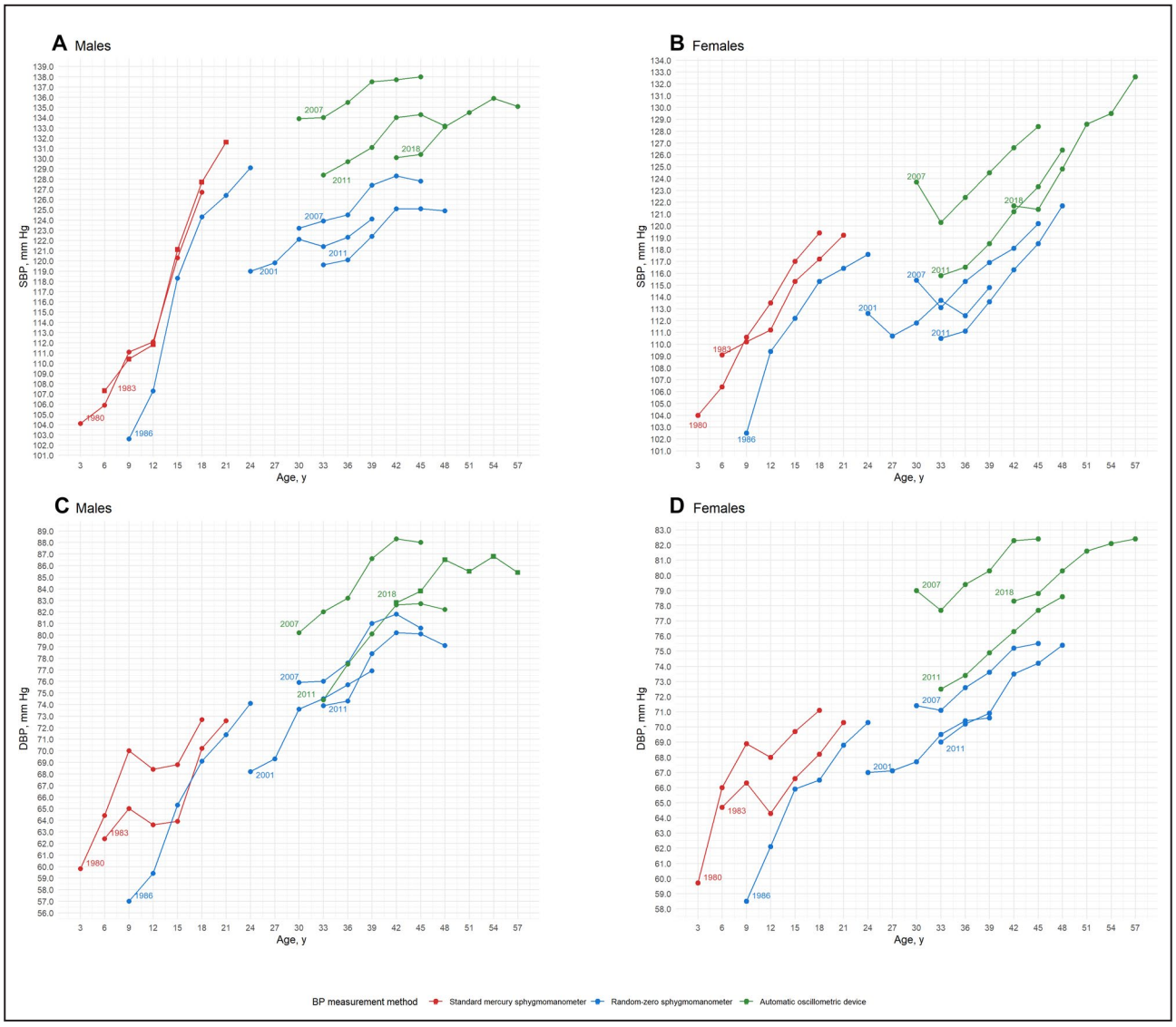

Figure 1. BP levels in relation to age and study years.
A, Males, SBP; (**B**) females, SBP; (**C**) males, DBP; (**D**) females, DBP. The colors indicate the used measurement method: red=standard mercury gravity sphygmomanometer; blue=random-zero sphygmomanometer; green=automatic sphygmomanometer. BP indicates blood pressure; DBP, diastolic blood pressure; and SBP, systolic blood pressure.

the risk was >2.5 times higher (HR, 2.66 [95% CI, 2.07–3.42]). Similarly, for diastolic BP burden (Table S3), the risk of hypertension increased with higher cumulative diastolic BP exposure. In both sexes, high cumulative diastolic BP burden associated with higher risk of hypertension (HR, 1.71 [95% CI, 1.28–2.30] in females; HR, 1.68 [95% CI, 1.30–2.18] in males). Yearly age- and sex-specific childhood mean systolic and diastolic BP levels, calculated from the AUC variables, are presented for each cumulative BP group in Table S4.

Comparisons of the prediction precision of adulthood hypertension between the cumulative systolic BP exposure (AUC variable) and the guideline-based BP classification are presented in Table S5. We found that childhood cumulative systolic BP burden, modeled using AUC quartiles (0–3), provided better predictive value for adult hypertension (C-value, 0.671 [95% CI, 0.647–0.695]) compared with the classification based on the number of childhood visits with BP above the American Academy of Pediatrics guideline threshold at age 6, 9, or 12 years (C-value, 0.578 [95% CI, 0.554–0.603]), with a statistically significant difference in discrimination (ΔC, 0.093 [95% CI, 0.067–0.118]; P<0.0001).

DISCUSSION

In this study, taking advantage of the longitudinal follow-up data of the YFS, we found that BP levels measured

Table 2. Age- and Sex-Specific 38-Year Tracking of Systolic and Diastolic Blood Pressure

		Systolic blood pressure, mm Hg			Diastolic blood pressure, mm Hg		
	Age in 1980, y	n	*r*	*P* value	n	*r*	*P* value
Female	6	167	0.227	0.003	166	0.197	0.011
	9	167	0.313	<0.0001	167	0.186	0.016
	12	177	0.286	<0.0001	177	0.228	0.002
	15	163	0.220	0.005	163	0.106	0.176
	18	128	0.203	0.021	128	0.237	0.007
	6, 9	334	0.295	<0.0001	333	0.201	0.0002
	12, 15, 18	468	0.249	<0.0001	468	0.191	<0.0001
	All	940	0.298	<0.0001	801	0.202	<0.0001
Male	6	123	0.247	0.006	123	−0.009	0.918
	9	129	0.218	0.013	127	0.147	0.098
	12	119	0.099	0.285	117	0.236	0.010
	15	117	0.253	0.006	117	0.297	0.001
	18	99	−0.008	0.940	98	0.215	0.033
	6, 9	252	0.247	<0.0001	250	0.125	0.048
	12, 15, 18	335	0.123	0.024	332	0.251	<0.0001
	All	727	0.187	<0.0001	582	0.201	<0.0001

Tracking coefficients were calculated using Pearson correlation.

in childhood correlate with corresponding adulthood levels in the 38-year follow-up. We also found that the risk of developing hypertension increases together with increasing cumulative BP burden from childhood to young adulthood. In relation to cumulative systolic BP burden in childhood between ages 6 and 12 years, we showed that female participants who were in the highest BP burden quartile had nearly 4 times higher instantaneous risk of adulthood hypertension compared with those in the lowest quartile. For male participants in the highest quartile, the corresponding risk was >2.5 times higher. Our findings emphasize the importance of long-term cumulative BP burden in childhood in the development of hypertension in adulthood.

Our results are in line with earlier reports from other cohorts examining tracking of BP with a shorter follow-up time.[6,27,36–38] Weakening of correlations with increasing follow-up time have also been observed in previous studies.[5,39] Although our follow-up time was longer than in similar studies, tracking of BP was observed. This reinforces the view that childhood BP levels may predict adulthood levels of BP.

In most current guidelines for healthy children, pediatric hypertension is defined as repeated BP readings in the age-, sex-, and height-specific ≥95th percentile of the distribution. However, we found significantly increased risk of developing hypertension already at lower BP levels during childhood when the cumulative

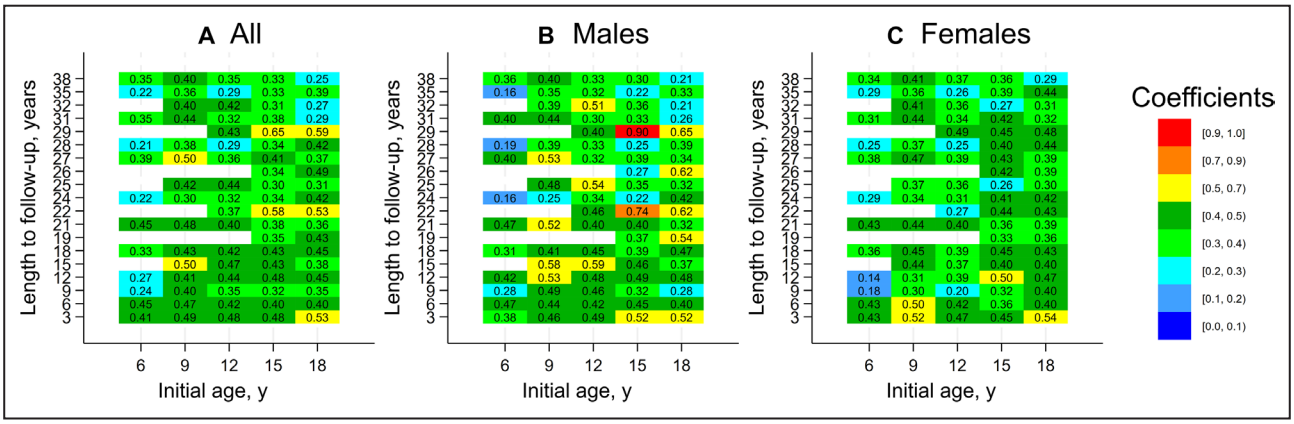

Figure 2. Stability coefficients of systolic blood pressure levels from childhood to mid-adulthood, stratified by length to follow-up.

A, All participants; (**B**) males; (**C**) females. Values indicate stability coefficients. "[" indicate more than or equal to the values; "]" indicate less than or equal to the values; ")" indicate less than the values. Model adjusted for sex and initial ages.

Table 3. HRs for Hypertension According to the Level of Cumulative Systolic Blood Pressure Burden in Childhood/Adolescence

		Childhood exposure (age 6–12 y)			Adolescence exposure (age 12–18 y)			Childhood and adolescence exposure (age 6–18 y)		
		Low	Intermediate	High	Low	Intermediate	High	Low	Intermediate	High
		HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
		P value	*P* value	*P* value	*P* value	*P* value	*P* value	*P* value	*P* value	*P* value
Female	All participants (baseline ages 3–18 y)	2.00 (1.47–2.72) <0.0001	1.97 (1.44–2.71) <0.0001	3.85 (2.87–5.17) <0.0001	2.45 (1.51–3.98) 0.0003	4.24 (2.65–6.77) <0.0001	10.79 (6.74–17.27) <0.0001	1.95 (1.39–2.73) 0.0001	2.35 (1.68–3.28) <0.0001	4.38 (3.20–6.00) <0.0001
	Younger participants (baseline ages 3, 6, 9 y)	2.68 (1.54–4.68) 0.0005	2.141 (1.19–3.86) 0.0115	5.57 (3.35–9.26) <0.0001	3.12 (1.55–6.29) 0.0015	4.90 (2.42–9.92) <0.0001	13.21 (6.61–26.42) <0.0001	2.48 (1.49–4.14) 0.0005	2.97 (1.71–5.14) 0.0001	6.73 (4.13–10.98) <0.0001
	Older participants (baseline ages 12, 15, 18 y)	1.87 (1.29–2.72) 0.0010	2.10 (1.43–3.08) 0.0001	3.59 (2.47–5.21) <0.0001	1.88 (0.92–3.82) 0.0795	2.98 (1.56–5.70) 0.0010	7.17 (3.72–13.82) <0.0001	1.81 (1.15–2.85) 0.0104	2.12 (1.38–3.26) 0.0006	3.73 (2.45–5.68) <0.0001
Male	All participants (baseline ages 3–18 y)	1.73 (1.33–2.24) <0.0001	2.15 (1.66–2.77) <0.0001	2.66 (2.07–3.42) <0.0001	2.40 (1.57–3.68) <0.0001	3.28 (2.09–5.16) <0.0001	6.15 (3.85–9.82) <0.0001	1.90 (1.45–2.50) <0.0001	2.57 (1.95–3.38) <0.0001	3.37 (2.57–4.44) <0.0001
	Younger participants (baseline ages 3, 6, 9 y)	2.18 (1.48–3.19) <0.0001	2.59 (1.74–3.84) <0.0001	3.98 (2.72–5.80) <0.0001	2.36 (1.32–4.24) 0.0039	3.33 (1.80–6.18) 0.0001	6.67 (3.46–12.84) <0.0001	2.36 (1.67–3.35) <0.0001	3.00 (2.05–4.40) <0.0001	5.17 (3.50–7.64) <0.0001
	Older participants (baseline ages 12, 15, 18 y)	1.56 (1.03–2.07) 0.0343	1.87 (1.32–2.63) 0.0004	1.99 (1.40–2.84) 0.0001	1.66 (0.72–3.81) 0.2316	2.28 (1.04–4.99) 0.0390	3.45 (1.59–7.50) 0.0018	1.42 (0.92–2.21) 0.1158	1.94 (1.29–2.92) 0.0015	2.22 (1.48–3.34) 0.0001

Analyses were conducted using Cox proportional hazard regression and AUC approach, indicating a cumulative blood pressure burden. Possible sex interaction was analyzed by adding multiplicative interaction term for each age period. These analyses indicated possible effect modification by sex (*P* values varying between 0.012 and 0.063), and thus sex-stratified analyses were conducted. The data include 1037 cases and 1361 noncases over the years 2001, 2007, 2011, and 2018. The AUC variable for systolic blood pressure was calculated separately for childhood (ages 6–12 y), adolescence (12–18 years) and the entire period from childhood to adolescence (age 6–18 y), and the participants were classified into quartiles using the cut points of 25th, 50th, and 75th percentiles resulting in groups: (1) very low, (2) low, (3) intermediate, and (4) high cumulative systolic blood pressure exposure during the life stage under consideration. The "very low" group was used as the reference group when calculating the HRs. AUC indicates area under the curve; and HR, hazard ratio.

BP burden during childhood and adolescence was considered. Importantly, in our cohort, we observed 2 to 3 times higher risk of developing hypertension for those with the highest cumulative BP exposure even if the BP levels were within the normal range. This observation suggests that BP exposure might be harmful at much lower levels than current cutoffs to denote elevated BP levels, particularly if the exposure accumulates from childhood. This highlights the need for follow-up measurements in individuals whose BP is slightly elevated but not markedly high. Repeated assessments in these cases could provide additional insights for risk management, particularly if BP remains consistently elevated over time.

An important issue in BP monitoring is the age at which BP measurements should be performed. Based on our results, age at the time of the measurement was not a major determinant of how well childhood BP tracked into adulthood BP. Our key finding that childhood BP burden increases the risk of hypertension in a dose–response manner emphasizes the importance of BP measurement in childhood and suggests that regular longer-term monitoring might be beneficial to estimate cumulative BP burden. Furthermore, even if the ability to predict adult BP levels from an individual childhood BP measurement might be considered weak on the basis of our results, an increase in the child's BP should raise concerns and could more often lead to preventive actions and plausibly interventions to avoid later hypertension in adulthood and possible cardiovascular outcomes later in life. Since exposure to elevated BP from childhood has also been shown to be a risk factor for atherosclerosis and cardiovascular mortality, monitoring and treating BP levels early in life may contribute not only to the risk of hypertension but also to major cardiovascular events.[24,40] Given that these actions could be started already in childhood, the focus should be on promotion of healthy lifestyle habits, such as a healthy diet and adequate physical activity, as well as preventing and controlling overweight and obesity, which all separately and in concert contribute to childhood and adolescence BP levels and could thus also reduce the risk of later hypertension.[19,41]

Our study has some limitations. One of the key challenges in this study was loss to follow-up. Nonparticipation in follow-up might have led to selection bias if loss to follow-up was differential. However, baseline characteristics, including BP levels, were comparable between those who remained in the study and those who did not. Furthermore, our cohort exhibited high participant retention compared with similar long-term studies commencing in childhood. Additionally, the statistical properties of the generalized estimating equation models include that it uses all observations. This further reduces the possibility of nonparticipation bias. Differences in BP measurement methods across the follow-up period may have affected the results. In 1980, BP was measured with a standard sphygmomanometer. Possible errors linked to this method include terminal digit preference and interobserver variability. The variation between measures is present in our multicenter study, where the same research nurse or doctor has not been able to measure BP of all individuals. Additionally, the random-zero sphygmomanometer used in some follow-up years has been shown to introduce additional variance in systolic BP measurements, potentially attenuating tracking correlations.[42] This limitation may have led to an underestimation of the strength of tracking relationships in our analyses. Furthermore, as hypertension status was assessed only at predefined follow-up visits, the exact timing of hypertension onset is unknown. Therefore, hypertension may be subject to interval censoring, which might introduce some imprecision to our results. Moreover, our cohort consists of relatively healthy and young individuals with BP levels mainly within the normal range. Thus, we divided the study population into quartiles according to their cumulative BP exposure burden and were not able to study the role of clinically abnormal BP levels for the risk of hypertension. Additionally, we did not investigate the association of a certain amount of change in BP with the risk of hypertension. An additional limitation is that we cannot completely exclude the possibility that our results are somewhat violated by the significant declines in BP levels observed in the Finnish population over recent decades.[43,44] These trends have been attributed to public health measures, including reduced salt intake, healthier lifestyle choices, and improved hypertension management. Finally, due to our racially homogeneous study cohort, the results may have limited generalizability.

In summary, these data suggest that the development of adulthood hypertension begins from early childhood. We observed that BP in childhood tracks into adulthood, over a period of up to 38 years. Our finding that high long-term cumulative childhood BP exposure, even without exceeding the reference guideline cutoffs, is associated with higher risk of hypertension in adulthood highlights the importance of repeated childhood BP monitoring and early prevention.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Data S1
Tables S1–S5

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