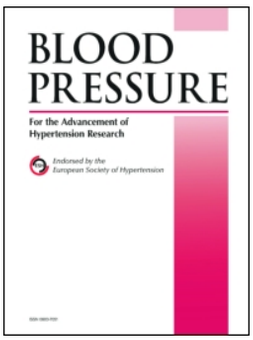

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Association of lifetime blood pressure with adulthood exercise blood pressure response: the cardiovascular risk in young Finns study

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ORIGINAL ARTICLE

Association of lifetime blood pressure with adulthood exercise blood pressure response: the cardiovascular risk in young Finns study

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ABSTRACT

Purpose: Elevated blood pressure (BP) in childhood has been associated with increased adulthood BP. However, BP and its change from childhood to adulthood and the risk of exaggerated adulthood exercise BP response are largely unknown. Therefore, we studied the association of childhood and adulthood BP with adulthood exercise BP response.

Materials and methods: This investigation consisted of 406 individuals participating in the ongoing Cardiovascular Risk in Young Finns Study (baseline in 1980, at age of 6–18 years; follow-up in adulthood in 27–29 years since baseline). In childhood BP was classified as elevated according to the tables from the International Child Blood Pressure References Establishment Consortium, while in adulthood BP was considered elevated if systolic BP was $\geq$120 mmHg or diastolic BP was $\geq$80 mmHg or if use of antihypertensive medications was self-reported. A maximal cardiopulmonary exercise test with BP measurements was performed by participants in 2008–2009, and exercise BP was considered exaggerated (EEBP) if peak systolic blood pressure exceeded 210 mmHg in men and 190 mmHg in women.

Results: Participants with consistently high BP from childhood to adulthood and individuals with normal childhood but high adulthood BP had an increased risk of EEBP response in adulthood (relative risk [95% confidence interval], 3.32 [2.05–5.40] and 3.03 [1.77–5.17], respectively) in comparison with individuals with normal BP both in childhood and adulthood. Interestingly, individuals with elevated BP in childhood but not in adulthood also had an increased risk of EEBP [relative risk [95% confidence interval], 2.17 [1.35–3.50]).

Conclusions: These findings reinforce the importance of achieving and sustaining normal blood pressure from childhood through adulthood.

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Introduction

Hypertension is an important global public health challenge, with a predicted 1.56 billion hypertensive adults in 2025 worldwide [1]. Hypertension is estimated to cause approximately 13% of deaths globally [2]. Thus, the prevention and control of hypertension is one of the most important ways to reduce deaths and disability from cardiovascular diseases [3]. In addition to elevated resting blood pressure (BP), an exaggerated response of the cardiovascular system to stress, such as exercise (i.e. exaggerated exercise blood pressure (EEBP) response), predicts adverse outcomes such as the future development of hypertension, and the risk of cardiovascular diseases and mortality in adulthood [4–7]. Therefore, early identification of young individuals at risk for elevated BP or adverse

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BP reaction to exercise would have important implications for achieving effective prevention through lifestyle modifications or drug treatment.

Elevated BP has been reported to track from childhood to adulthood [8,9]. Importantly, the influence of elevated BP in childhood on carotid atherosclerosis is reduced if these individuals become adults with normal blood pressure [10], and a favourable change in the BP profile from childhood to adulthood also appears to reduce the risk of high arterial stiffness in adulthood [11]. However, the association of BP change from childhood to adulthood on adulthood exercise blood pressure response remains largely unknown. Therefore, in this study, we sought to determine for the first time the effects of childhood and adulthood elevated BP on adulthood EEBP in the ongoing prospective Cardiovascular Risk in Young Finns Study.

Methods

Study population

The Cardiovascular Risk in Young Finns Study is an ongoing study of cardiovascular risk factors in Finland. The study design and protocol have been described in detail previously [12]. The first cross-sectional study was conducted in 1980 with 3596 participants aged 3 – 18 years. Several follow-up studies have been performed since then. The fourth large follow-up was conducted in 2007, with a total of 2204 participants. During the time period of 2008–2009, a cardiopulmonary exercise test was performed on a total of 538 participants at Tampere and Turku study centres. Participants with diabetes ($n = 3$), pregnant women ($n = 2$), participants with submaximal exercise output ($n = 37$) and participants with undefined maximal oxygen uptake ($n = 9$) were excluded. In addition, participants aged 3 years in 1980 ($n = 81$) were not included in the analyses because blood pressure measurements were performed with an ultrasound device. In total, 406 participants were included in this study. The study was conducted according to the guidelines of the Declaration of Helsinki, and the study was approved by local ethics committees. Informed written consent was obtained from all participants or their closest relatives in case of youngest participants.

Clinical measurements

In childhood and adulthood, height and weight were measured, and body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared. Office BP from the right brachial artery was measured in the sitting position after 5 min of rest with a standard mercury sphygmomanometer in 1980 and with a random zero sphygmomanometer (Hawksley & Sons Ltd, Lansing, United Kingdom) in 2007 as described previously in more detail [10,11]. The average of three measurements was used in the analyses.

Elevated BP was defined in childhood according to the International Child Blood Pressure References Establishment Consortium tables [13] and in adulthood according to the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood pressure [14]. BP in childhood was classified as normal if systolic and diastolic BP (SBP and DBP, respectively) were <90th percentile for age, sex, and height and elevated if SBP or DBP were ≥90th percentile. BP in adulthood was classified as normal if SBP was <120 mmHg and DBP was <80 mmHg and elevated if SBP was ≥120 mmHg or DBP was ≥80 mmHg. In addition, adulthood BP was considered elevated among those self-reporting the use of antihypertensive medications. Four groups based on childhood and adulthood BP levels were defined: (1) control group, participants who had a normal BP in childhood and normal BP as an adult; (2) resolution group, participants who had elevated BP in childhood but not as an adult; (3) incident group, participants with normal BP in childhood who had elevated BP as an adult; and (4) consistent group, participants who had elevated BP in childhood and as an adult.

Cycle ergometry and gas measurements

Cardiopulmonary exercise was performed as described in detail previously [15]. Exercise tests were performed in 2008 or 2009 on electronically braked cycle ergometers (Lode Corival 906900, Lode BV, Groningen, Netherlands) according to the American Thoracic Society guidelines and the American College of Chest Physicians Joint Statement on Cardiopulmonary Exercise Testing [16]. The participants performed an incremental test with 1-minute intervals, until exhaustion limited maximal power output [15]. Otherwise, objective test termination criteria were applied by the observers. Twelve-lead electrocardiography (ECG) was recorded during the test (Corina ECG amplifier and CardioSoft acquisition software ver. 4.2, GE Medical Systems, Freiburg, Germany). Maximal heart rate (HR) was obtained from the ECG data. Breath-by-breath measurements

Table 1. Baseline and follow-up characteristics of study participants.

Variable	EEBP −	EEBP +	*p* Value
n (males/females)	209 (80/129)	197 (109/88)	0.001
Childhood			
Age, years	11.6 (4.0)	12.4 (4.1)	0.044
Height, cm	147.7 (19.0)	151.9 (19.8)	0.817
BMI, kg/m^2	17.8 (2.9)	18.4 (3.1)	0.389
SBP, mmHg	108 (11)	113 (11)	<0.001
DBP, mmHg	68 (10)	69 (11)	0.378
BP status, *n* (%)			
Normal	144 (68.9)	99 (50.3)	
Elevated	65 (31.1)	98 (49.7)	<0.001
Adulthood			
Age, years	38.6 (4.0)	39.4 (4.1)	0.044
Height, cm	171.5 (9.3)	173.7 (8.6)	0.795
BMI, kg/m^2	24.7 (4.0)	26.8 (4.2)	<0.001
SBP, mmHg	110 (11)	124 (14)	<0.001
DBP, mmHg	68 (10)	76 (11)	<0.001
BP status, *n* (%)			
Normal	167 (79.9)	79 (40.1)	
Elevated	42 (20.1)	118 (59.9)	<0.001
Exercise test			
Peak SBP, mmHg	182 (17)	222 (18)	<0.001
Peak DBP, mmHg	87 (11)	99 (13)	<0.001
Peak HR, 1/min	175 (12)	176 (13)	0.377
Peak VO_2, mL·kg^{-1}·min^{-1}	30.6 (7.7)	29.9 (8.8)	0.010
Maximal METs, 1 MET = 3.5 mL*kg^{-1}*min^{-1}	8.7 (2.2)	8.6 (2.5)	0.010

EEBP- was defined as peak exercise systolic blood pressure ≤210 mmHg for men and ≤190 mmHg for women, and EEBP + as peak exercise systolic blood pressure >210 mmHg for men and >190 mmHg for women. Values are mean (SD) for continuous variables or n (%) for dichotomous variables unless stated otherwise. Comparisons between groups were performed using age and sex adjusted linear regression analyses for continuous variables and and χ^2 tests for categorical variables.
EEBP: exaggerated exercise blood pressure; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; BP: blood pressure; HR: heart rate; VO_2: oxygen consumption; METs: metabolic equivalents.

of oxygen uptake (VO_2) and other respiratory parameters were performed with computerised analysers (V-max 29C, SensorMedics, Yorba Linda, CA, USA and Jaeger Oxycon Pro, VIASYS Healthcare GmbH, Hoechberg, Germany). VO_2peak was determined as the highest VO_2 during the last 30 s of the test. Maximal metabolic equivalents (METs) were calculated by dividing the VO_2peak by 3.5 [17]. A respiratory exchange ratio >1.10 was used to define maximal exercise. Peak SBP (expressed as mmHg) was measured as close as possible to the end of exercise. BP measurements were performed with the subject in a sitting position [15].

Statistical methods

The comparisons between groups were performed using age- and sex-adjusted linear regression analysis for continuous variables and χ^2 tests for categorical variables. Relative risks and 95% confidence intervals were calculated by using Poisson regression to examine the associations between BP groups and EEBP in adulthood. All analyses were adjusted for age, sex, adulthood BMI, VO_2peak and maximal exercise HR. As an exploratory analysis, analyses were also repeated with further adjustment for adulthood resting BP. Linear trend over BP groups was also tested with a regression analysis. Sex × BP and age × BP interaction effects on EEBP were tested, and there was no significant interaction. Therefore, the results are shown combined. Sensitivity analyses were performed to examine the influence of different childhood BP definition (National High Blood Pressure Education Program tables) [18] as well as exclusion of participants on antihypertensive medication on the magnitude of the associations.

EEBP definition was based on American Heart Association guidelines, which define EEBP during exercise testing as SBP > 210 mmHg for men and > 190 mmHg for women [19]. All analyses were performed with SPSS Statistics (release 26.0.0.0, IBM Corp.). Statistical significance was inferred at a 2-tailed p value <0.05.

Results

The baseline and follow-up characteristics of the study subjects in 1980 and in 2007 by exercise BP status in adulthood are shown in Table 1. There was no difference in height, BMI, or DBP between normal and EEBP groups in the baseline. Those having EEBP as adults were slightly older ($p = 0.044$), more often males ($p = 0.001$), and had higher SBP ($p < 0.001$) and higher prevalence of elevated BP level ($p < 0.001$)

Table 2. Relative risks and 95% confidence intervals of exaggerated exercise blood pressure according to blood pressure group in childhood and adulthood.

	ICBPREC criteria				NHBPEP criteria			
Child-adult BP group*	*n/N*	RR	95% CI	*p*	*n/N*	RR	95% CI	*p*
Control	26/130	1.00	Ref	Ref	42/162	1.00	Ref	Ref
Resolution	53/116	2.17	1.35–3.50	0.001	37/84	1.66	1.05–2.60	0.029
Incident	44/64	3.03	1.77–5.17	<0.001	57/81	2.34	1.51–3.64	<0.001
Persistent	74/96	3.32	2.05–5.40	<0.001	61/79	2.65	1.73–4.06	<0.001

All analyses adjusted for age, sex, adult BMI, VO_2peak, and adult maximum exercise heart rate. BMI indicates body mass index; BP: blood pressure; CI: confidence interval; ICBPREC: International Child Blood Pressure References Establishment Consortium; *n*: number of subjects having exaggerated exercise blood pressure; *N*: number of subjects in the group; NHBPEP: National High Blood Pressure Education Program; RR: relative risk.

*Elevated child BP was defined by ICBPREC Criteria (systolic or diastolic BP were ≥90th percentile by using the ICBPREC tables for age, sex, and height) or by NHBPEP Criteria (systolic or diastolic BP were ≥90th percentile by using the NHBPEP tables for age, sex, and height). Adult BP was classified as elevated if systolic BP ≥120 mm Hg or diastolic BP ≥80 mm Hg. In addition, adult BP status was considered elevated among those self-reporting the use of antihypertensive medications. BP groups were as follows: control group, normal BP in childhood and normal BP in adulthood; resolution group, elevated BP in childhood but not in adulthood; incident group, normal BP in childhood, but elevated BP in adulthood; and persistent group, elevated BP in childhood and adulthood.

in childhood. In adulthood they had higher BMIs ($p < 0.001$), and higher SBP and DPB ($p < 0.001$) and higher prevalence of elevated BP (20.1% versus 59.9%; $p < 0.001$). As expected, there were also clear differences in both peak SBP and DBP of the exercise test ($p < 0.001$), while peak HR did not differ between normal and EEBP response groups ($p = 0.38$). Peak maximal oxygen uptake and exercise capacity were slightly but significantly lower in EEBP group ($p = 0.01$ for both).

Participants with consistently high BP from childhood to adulthood (consistent group) and individuals with normal child but high adult BP (incident group) had increased risk of EEBP response in adulthood (relative risk [95% confidence interval], 3.32 [2.05–5.40] and 3.03 [1.77–5.17], respectively) in comparison with individuals with normal BP both in childhood and adulthood. Interestingly, also individuals with elevated BP in childhood but not in adulthood (resolution group) had increased risk of EEBP (relative risk [95% confidence interval], 2.17 [1.35–3.50]) (Table 2). After further adjustment with adulthood resting BP, relative risks [95% confidence interval; P value] were 2.84 [1.45–5.55, $p = 0.002$], 2.00 [0.96–4.17, $p = 0.065$] and 2.14 [1.28–3.55, $p = 0.003$], respectively.

There was a significant trend in adulthood peak exercise SBP over BP groups in linear regression analyses ($p < 0.001$). The absolute values of peak SBP were comparable between individuals with consistently high BP from childhood to adulthood (consistent group) and individuals with normal child but high adult BP (incident group) (219 mmHg and 216 mmHg, respectively), while peak SBP of the control group was clearly lower (188 mmHg) ($p < 0.001$). There was significant difference in peak SBP also between participants with elevated BP in childhood but not in adulthood and the control group (195 mmHg and 188 mmHg, respectively, $p = 0.048$).

In order to test the robustness of the results, the analysis was performed using another standardised cut point. Results remained essentially similar when National High Blood Pressure Education Program tables were used to define elevated BP in childhood, and ≥120/80 mmHg BP in adulthood, particularly among participants who had elevated BP in adulthood (incident and consistent groups) (relative risk [95% confidence interval], 2.34 [1.51–3.64] and 2.65 [1.73–4.06], respectively). Again, the relative risk was higher for participants whose elevated BP in childhood resolved by adulthood (relative risk [95% confidence interval], 1.66 [1.05–2.60]) when compared with the individuals with normal BP both in childhood and adulthood (Table 2).

As a sensitivity analysis, analyses were also performed after exclusion of participants on antihypertensive medication. The findings remained essentially similar (Supplementary Table).

Discussion

The current study showed that individuals with consistently high BP from childhood to adulthood and individuals with normal child, but high adult BP had increased risk of EEBP response in adulthood in comparison with individuals with normal BP both in childhood and adulthood. Interestingly, also individuals with elevated BP in childhood but not in adulthood had increased risk of EEBP.

Previous reports have shown that BP is tracked from childhood to adulthood [8,9], with an average correlation coefficients being 0.38 for systolic BP and 0.28 for diastolic BP in meta-analysis [8]. However, to the best of our knowledge, the influence of BP

changes from childhood to adulthood on adult exercise BP response has remained unexplored. Since exaggerated exercise BP appears to relate with the development of hypertension [6,7], and increase the incidence of cardiovascular diseases and mortality [4], the current findings highlight the importance of lifetime BP control from childhood to adulthood in the prevention of adverse adulthood outcomes.

Previous studies have shown that favourable change in BP profile from childhood to adulthood reduce the risk of carotid atherosclerosis and high arterial stiffness in adulthood [10,11]. These findings suggest that BP resolution from childhood to adulthood can lead to decrease or reversal of the risk of structural arterial changes in adulthood. Interestingly, since individuals with elevated BP in childhood but not in adulthood had still significantly increased risk of EEBP, this seems not to be fully the case regarding the response of cardiovascular system to maximal physical exercise.

We and others have shown that childhood SBP is an independent predictor of arterial pulse wave velocity in adulthood [20–22], and that arterial pulse wave velocity is also associated with exercise pressure response in adulthood [23,24]. Thus, one pathophysiological link between the association of childhood BP and adulthood EEBP could be subtle lifetime changes in arterial stiffness mediating the effect of childhood BP on cardiovascular reactivity in adulthood. On the other hand, since hypertensive subjects have been found to have blunted microvascular reactivity in skeletal muscles [25], another possible pathophysiological mechanism could be alterations in microvasculature caused by early life burden of elevated BP on small vessels. Other mechanisms could include endothelial dysfunction, high sympathetic tone or dysregulation of sympathetic vasoconstriction in exercising muscles caused by hypertension [26,27].

Currie et al. has recently shown that EEBP in well-trained individuals do not display resting cardiovascular state typically observed in untrained individuals with EEBP, suggesting that EEBP response in athletes is likely a compensatory mechanism to satisfy peripheral blood flow demand rather than indicative of latent dysfunction [28]. In addition, different BP measurement methods and measurements in different conditions such as in rest and during exercise provide BP values that are correlated. Despite that, high BP response to exercise appears to predict future development of hypertension in young athletes even after adjustment with resting BP [7]. Interestingly, Mariampillai et al. recently reported that exercise systolic BP at moderate workload is linearly associated with coronary disease risk in healthy middle-aged men independently of resting BP [29]. Altogether, it appears that even though EEBP may be a physiological compensatory mechanism at least in well-trained athletes, it may also reveal failing compensatory mechanisms or hypertensive tendency beyond resting BP. Future experiments with long follow-up of young individuals will be warranted to answer the question whether or not EEBP also in younger individuals is a predictor for later cardiovascular disease and mortality.

One of the strengths of the present work is that this study was based on a large randomly selected cohort of young adults who were followed for 27 years since childhood. Some limitations, however, need to be taken into consideration. The rather high prevalence of elevated BP in childhood might be partially explained by white coat syndrome. However, such high prevalence has also been previously reported in some other cohorts [10]. The cardiopulmonary exercise test was performed on average 14 months later than the main follow-up visit. However, since focus was on lifetime BP through childhood to adulthood, we do not consider this as a major limitation in the current study. Although non-invasive BP measurement is probably the most feasible method for exercise BP measurement, it can be also considered as a study limitation due to its uncertainty in the precise measurement of BP values during exercise. Additionally, the study cohort was rather homogenous ethnically, consisting solely of white European (Finnish) subjects. Therefore, the results may not be directly generalisable to other populations.

In conclusion, this study has demonstrated that individuals with elevated adult BP have significantly increased risk of EEBP response rather independently of BP status in childhood, in comparison with individuals with persistently normal BP. The relative risk remained still increased if elevated childhood BP resolved by adulthood when compared with persistently normal BP. These findings reinforce the importance of achieving and sustaining normal BP since childhood through adulthood.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

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