Blood Pressure

ISSN: (Print) (Online) Journal homepage: www.tandfonline.com/journals/iblo20

Association of childhood socioeconomic status with adulthood maximal exercise blood pressure: the Cardiovascular Risk in Young Finns Study

Erika Kähönen, Emilia Kähönen, Kristiina Pälve, Janne Hulkkonen, Mika Kähönen, Olli T. Raitakari, Nina Hutri, Terho Lehtimäki & Heikki Aatola

To cite this article: Erika Kähönen, Emilia Kähönen, Kristiina Pälve, Janne Hulkkonen, Mika Kähönen, Olli T. Raitakari, Nina Hutri, Terho Lehtimäki & Heikki Aatola (2024) Association of childhood socioeconomic status with adulthood maximal exercise blood pressure: the Cardiovascular Risk in Young Finns Study, Blood Pressure, 33:1, 2323987, DOI: 10.1080/08037051.2024.2323987

To link to this article: https://doi.org/10.1080/08037051.2024.2323987

Published online: 11 Mar 2024.

BLOOD PRESSURE
2024, VOL. 33, NO. 1, 2323987
https://doi.org/10.1080/08037051.2024.2323987

Taylor & Francis
Taylor & Francis Group

RESEARCH ARTICLE

OPEN ACCESS
Check for updates

Association of childhood socioeconomic status with adulthood maximal exercise blood pressure: the Cardiovascular Risk in Young Finns Study

Erika Kähönen[a], Emilia Kähönen[a], Kristiina Pälve[b,c], Janne Hulkkonen[d], Mika Kähönen[a,e], Olli T. Raitakari[b,f,g], Nina Hutri[h], Terho Lehtimäki[e,i,j] and Heikki Aatola[a]

[a]Department of Clinical Physiology and Nuclear Medicine, Faculty of Medicine and Health Technology, Tampere University and Tampere University Hospital, Tampere, Finland; [b]Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Turku, Finland; [c]Heart Center, Turku University Hospital, Turku, Finland; [d]Heart Center, Tampere University Hospital, Tampere, Finland; [e]Finnish Cardiovascular Research Center-Tampere, Tampere, Finland; [f]Centre for Population Health Research, University of Turku and Turku University Hospital, Turku, Finland; [g]Department of Clinical Physiology and Nuclear Medicine, Turku University Hospital, Turku, Finland; [h]Tampere Centre for Skills Training and Simulation, Tampere University, Tampere, Finland; [i]Fimlab Laboratories, Tampere, Finland; [j]Department of Clinical Chemistry, Faculty of Medicine and Health Technology, Tampere University and Tampere University Hospital, Tampere, Finland

ABSTRACT

Purpose: Socioeconomic status has been related to resting blood pressure (BP) levels at different stages of life. However, the association of childhood socioeconomic status (SES) and adulthood exercise BP is largely unknown. Therefore, we studied the association of childhood SES with adulthood maximal exercise BP.

Materials and methods: This investigation consisted of 373 individuals (53% women) participating in the Cardiovascular Risk in Young Finns Study who had data concerning family SES in childhood (baseline in 1980, at age of 6–18 years) and exercise BP response data in adulthood (follow-up in adulthood in 27–29 years since baseline). A maximal cardiopulmonary exercise test with BP measurements was performed by participants, and peak exercise BP was measured.

Results: In stepwise multivariable analysis including childhood risk factors and lifestyle factors (body mass index, systolic BP, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, insulin, fruit consumption, vegetable consumption, and physical activity), lower family SES in childhood was associated with higher maximal exercise BP in adulthood (β value ± SE, 1.63 ± 0.77, p = 0.035). The association remained significant after further adjustment with participants SES in adulthood (β value ± SE, 1.68 ± 0.65, p = 0.011) and after further adjustment with adulthood body-mass index, systolic BP, maximal exercise capacity, and peak heart rate in exercise (β value ± SE, 1.25 ± 0.56, p = 0.027).

Conclusions: These findings suggest that lower childhood family SES is associated with higher maximal exercise BP in adulthood.

PLAIN LANGUAGE SUMMARY

- Limited data are available about the association of childhood socioeconomic status and adulthood exercise blood pressure.
- We prospectively examined whether childhood socioeconomic status is associated with adulthood exercise blood pressure in 373 participants aged 6–18 years at baseline (1980) from the longitudinal Cardiovascular Risk in Young Finns cohort study.
- In multivariable analysis, including childhood cardiovascular risk factors and lifestyle factors, lower family socioeconomic status in childhood was associated with higher maximal exercise blood pressure in adulthood.
- The association remained significant after further adjustment with participants socioeconomic status in adulthood and also after further adjustment with adulthood body mass index, systolic blood pressure, maximal exercise capacity and peak heart rate in exercise.
- Low childhood socioeconomic status predicted also higher risk of exaggerated exercise blood pressure response in adulthood, although this finding was diluted to non-significant after adjustment with adulthood body mass index and systolic blood pressure.
- These findings suggest that lower childhood family socioeconomic status is associated with higher maximal exercise blood pressure in adulthood.

ARTICLE HISTORY
Received 3 January 2024
Revised 20 February 2024
Accepted 20 February 2024

KEYWORDS
Childhood; socioeconomic status; exercise; blood pressure

CONTACT Erika Kähönen erika.kahonen@gmail.com Department of Clinical Physiology, Tampere University Hospital, P.O. Box 2000, FI-33521 Tampere, Finland.

Introduction

Hypertension is an important public health challenge, with an estimated cause of approximately 13% of deaths globally [1–3]. An exaggerated blood pressure (BP) response to exercise predicts future development of hypertension and the risk of cardiovascular diseases and mortality [4–7], and even exercise systolic BP at moderate workload is linearly associated with coronary disease risk in healthy middle-aged men independently of resting BP [8]. Therefore, identification of individuals at risk for adverse BP reaction to exercise and understanding determinants leading to excessive BP increase in exercise could be helpful in terms of achieving effective prevention.

Low parental socioeconomic position has been shown to be associated with higher BP from childhood to adulthood [9]. Early socioeconomic disadvantage influences later BP in part through BP in early life and in part through adulthood body mass index (BMI) [9].

Even though the association of low socioeconomic status (SES) and lifetime resting BP is well established, the association of childhood SES and adulthood exercise BP remains largely unknown. Therefore, in this study, we sought to determine for the first time the association of childhood socioeconomic status (SES) with adulthood exercise BP in the 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 [10]. The first cross-sectional study was conducted in 1980 with 3,596 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 2,204 participants. During the time period of 2008 to 2009, a cardiopulmonary exercise test was performed on a total of 538 participants at Tampere and Turku study centres. Participants with diabetes ($n = 2$), pregnant women ($n = 2$), patients with a self-reported hypertension medication ($n = 21$), participants with submaximal exercise output ($n = 36$), and participants with undefined maximal oxygen uptake ($n = 8$) were excluded. In addition, participants aged 3 years in 1980 ($n = 79$) were not included in the analyses because BP measurements were performed with an ultrasound device. 17 participants had no available SES data from 1980. In total, 373 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 (ETL-R07100). Informed written consent was obtained from all participants or their closest relatives in the case of youngest participants.

Classification of socioeconomic status

Annual income was considered as an indicator of SES in both childhood and adulthood. The values of annual family income in childhood were corrected for time. Annual income strata were determined on an 8-point scale: at the time of enrolment from 1 (<2,500€) to 8 (>16,800€) and in adulthood in 2007 from 1 (<10,000€) to 8 (>70,000€). In sensitivity analyses, we additionally defined childhood SES according to years of parental education [11–13].

Cardiopulmonary exercise test

Cardiopulmonary exercise was performed as described in detail previously [14]. 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 [15]. The participants performed an incremental test with 1-min intervals, until exhaustion limited maximal power output [14]. 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). Peak heart rate (HR) was obtained from the ECG data. Breath-by-breath measurements of oxygen uptake (VO_2) and other respiratory parameters were performed with computerised analysers (V-max 29 C, 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 [16]. A respiratory exchange ratio >1.10 was used to define maximal exercise. Peak SBP (expressed as mmHg) was measured with the cuff method by means of auscultation as close as possible to the end of exercise. BP measurements were performed with the subject in a sitting position [14].

Clinical measurements and childhood and adulthood cardiovascular risk factors and adulthood health behaviours as covariates

In childhood and adulthood, height and weight were measured, and BMI was calculated as weight in kilograms divided by height in metres squared. Standard methods were used to determine childhood and adulthood BP, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides and insulin as previously described in detail [17–19]. Health behaviours included physical activity, and fruit and vegetable consumption. Physical activity index was calculated based on the evaluation of self-reported frequency and intensity of physical activity, frequency of vigorous physical activity, hours spent on vigorous physical activity, average duration of a physical activity session, and participation in organised physical activity. Information on dietary habits was obtained with a nonquantitative food frequency questionnaire (FFQ). For subjects aged 6 to 9 years, the data were requested from the parents, and at the ages of 12 to 18 years, study subjects were assisted by their parents when necessary. To examine the frequency of fruit and vegetable consumption, the subjects were asked to fill in a questionnaire on habitual dietary choices for the past month with six response categories: 1 = daily, 2 = almost every day, 3 = a couple of times per week, 4 = about once a week, 5 = a couple of times per month, and 6 = more seldom. The response categories were converted into times of consumption per month (1→35; 2→25; 3→10; 4→4; 5→2; 6→0) [20–22].

Statistical methods

The comparisons between groups were performed using age- and sex-adjusted linear regression analysis for continuous variables and $\chi 2$ tests for categorical variables. Sex × SES and age × SES interaction effects on peak exercise BP were tested, and there was no significant interaction. Therefore, the results are shown combined. To study the independent effects of risk variables on adulthood maximal exercise BP, stepwise multivariable models were constructed. Association of childhood SES (β values are for a 1-unit decrease in family SES) and adulthood maximal exercise BP were performed using stepwise multivariable analysis. Model 1 included childhood risk factors (BMI, SBP, LDL cholesterol, HDL cholesterol, triglycerides, insulin, fruit consumption, vegetable consumption, and physical activity index). Final model 1 consisted of age, sex, and SBP. Model 2 included model 1 plus participant's own SES in adulthood. Model 3 included model 2 plus adulthood BMI, adulthood SBP, maximal METs, and peak HR. Logistic regression analysis was used to predict the adulthood exaggerated exercise BP response with childhood SES. Model 1 included age and sex as covariates and Model 2 included Model 1 plus peak exercise capacity (maximal METs) and peak HR. Model 3 included Model 2 plus adulthood BMI and SBP. Exaggerated exercise BP definition was based on American Heart Association guidelines, which define exaggerated exercise BP during exercise testing as SBP > 210 mmHg for men and > 190 mmHg for women [19]. All analyses were performed with SPSS Statistics (release 29.0.0.0, IBM Corp.). Statistical significance was inferred at a 2-tailed p value of <0.05.

Results

The baseline and follow-up characteristics of the study subjects in 1980 and in 2007 by family socioeconomic status in adulthood are shown in Table 1. There was no difference in sex, age, SBP, LDL cholesterol, fasting insulin, vegetable consumption or physical activity index between SES groups in childhood. Those having low family SES in childhood had lower annual family income ($p < 0.001$), had higher BMI ($p = 0.004$), lower HDL cholesterol ($p = 0.002$), higher triglycerides ($p = 0.007$) and lower fruit consumption frequency ($p = 0.016$) than those with high family SES in childhood. In adulthood, they had higher BMIs ($p = 0.032$), higher SBP ($p = 0.008$) and higher EEBP prevalence ($p = 0.008$). There was also a clear difference in peak SBP of the exercise test ($p = 0.012$), while peak HR did not differ between childhood family SES groups. VO_2peak and maximal METs were lower in the low family SES group than in the high family SES group ($p = 0.042$ for both).

In stepwise multivariable analysis including childhood risk factors (BMI, SBP, LDL cholesterol, HDL cholesterol, triglycerides, insulin, fruit consumption, vegetable consumption, and physical activity index), lower family SES (β values are for a 1-unit decrease in family SES) in childhood was associated with higher maximal exercise BP in adulthood (β value ± SE, 1.63 ± 0.77, $p = 0.035$) (Table 2). In sensitivity analyses that used parental educational years in place of family income as the indicator of childhood SES, the results were essentially similar (β value ± SE, 0.52 ± 0.22; $p = 0.019$). The association of lower family SES in

Table 1. Baseline and follow-up characteristics of study participants according to family socioeconomic status in childhood.

Variable	Low SES	Mid SES	High SES	*p* Value
n (males/females)	79 (31/48)	210 (107/103)	84 (37/47)	0.634
Childhood				
Age, y	12.3 (4.4)	11.9 (4.0)	11.4 (4.1)	0.172
Annual family income, 1000 €	15.0 (3.6)	33.4 (6.6)	57.6 (8.8)	<0.001
BMI, kg/m^2	18.6 (3.2)	18.1 (3.0)	17.4 (2.8)	0.044
SBP, mmHg	112 (12)	110 (10)	109 (11)	0.282
HDL cholesterol, mmol/l	1.52 (0.29)	1.57 (0.32)	1.67 (0.3)	0.002
LDL cholesterol, mmol/l	3.48 (0.79)	3.29 (0.73)	3.40 (0.74)	0.315
Triglycerides, mmol/l	0.69 (0.53–0.86)	0.60 (0.44–0.79)	0.53 (0.41–0.71)	0.007
Fasting insulin, mU/l	10.8 (6.4)	10.4 (5.7)	9.5 (5.4)	0.429
Fruit consumption, frequency/wk	6.2 (2.5)	6.8 (2.8)	7.3 (2.6)	0.016
Vegetable consumption, frequency/wk	6.5 (2.7)	6.3 (3.0)	6.9 (2.5)	0.325
Physical activity index, points	8.7 (1.7)	9.2 (1.9)	9.1 (1.7)	0.561
Adulthood				
Age, y	39.3 (4.4)	38.9 (4.0)	38.4 (4.2)	0.172
BMI, kg/m^2	25.6 (4.1)	26.3 (4.4)	24.2 (3.8)	0.032
SBP, mmHg	116 (12)	117 (14)	111 (12)	0.008
Exercise test				
Peak SBP, mmHg	202 (27)	203 (27)	193 (25)	0.012
EEBP prevalence, %	54.4	51.9	33.3	0.008
Peak HR, 1/min	174 (12)	176 (13)	176 (13)	0.339
Peak VO_2, mL·kg^{-1}·min^{-1}	29.5 (7.6)	30.0 (8.5)	32.3 (7.9)	0.042
Maximal METs, 1 MET = 3.5 mL*kg^{-1}*min^{-1}	8.4 (2.2)	8.6 (2.4)	9.2 (2.3)	0.042

Definition: Childhood family socioeconomic status was categorised as quintiles in three categories. Values are mean (SD) for continuous variables or median (25th–75th percentile) unless stated otherwise. *p* values are from comparisons between Low and High groups and were performed using age and sex adjusted linear regression analyses for continuous variables and and χ2 tests for categorical variables.
Abbreviations: BMI: body mass index; EEBP: exaggerated exercise blood pressure; HDL, high-density lipoprotein; HR: heart rate; LDL, low-density lipoprotein; METs: metabolic equivalents; SBP: systolic blood pressure; SES: socioeconomic status; and VO_2: oxygen consumption.

childhood with higher maximal exercise BP in adulthood remained significant after further adjustment with participants SES in adulthood (β value ± SE, 1.68 ± 0.65, $p = 0.011$) and after further adjustment with adulthood BMI, SBP, maximal METs and peak HR in exercise (β value ± SE, 1.25 ± 0.56, $p = 0.027$) (Table 2).

In logistic regression analysis, low childhood SES predicted a higher risk of exaggerated exercise BP response in adulthood when adjusted with age and sex (1-point decrease in SES; OR = 1.15; 95% CI 1.02–1.29, $p = 0.018$) and also after further adjustment with maximal METs and peak HR (1-point decrease in SES; OR 1.14; 1.01–1.28, $p = 0.030$). The association was diluted to non-significant after further adjustment with adult BMI and SBP (OR = 1.14; 95% CI 0.99–1.32, $p = 0.077$).

Table 2. Association of childhood SES on peak exercise systolic blood pressure.

	β Value ± SE (mmHg)	*p* Value
Model 1		
SBP childhood	0.42 ± 0.13	<0.001
SES childhood	1.63 ± 0.77	0.035
Model 2		
SBP childhood	0.32 ± 0.12	0.010
SES childhood	1.68 ± 0.65	0.011
Model 3		
SBP adulthood	0.90 ± 0.08	<0.001
BMI adulthood	1.11 ± 0.27	<0.001
SES childhood	1.25 ± 0.56	0.027

Results are for age- and sex-adjusted linear regression, and statistically significant associations are presented. β values are for a 1-unit decrease in family SES. Results for model 1 are from stepwise multivariable analysis, including childhood risk factors (BMI, SBP, LDL cholesterol, HDL cholesterol, triglycerides, insulin, fruit consumption, vegetable consumption and physical activity index). Final model 1 consisted of age, sex, and SBP. Model 2 included model 1 plus participant's own SES in adulthood. Model 3 included model 2 plus adulthood BMI, SBP, maximal METs, and peak HR. BMI indicates body mass index; HR, heart rate; METs, metabolic equivalents; SBP, systolic blood pressure; and SES, socioeconomic status.

Discussion

The current study showed that multivariable analysis including childhood cardiovascular risk factors and lifestyle factors, lower family SES in childhood was associated with higher maximal exercise BP in adulthood. The association remained significant even after further adjustment with participants SES in adulthood and also after further adjustment with adulthood BMI, SBP, maximal METs and peak HR in exercise. Low childhood SES predicted also higher risk of exaggerated exercise BP response in adulthood, although this finding was diluted to non-significant after adjustment with adult BMI and SBP.

Exaggerated exercise BP appears to be related with the development of hypertension [6,7], and increase the incidence of cardiovascular diseases and mortality [4]. High BP response to exercise appears to predict future development of hypertension in young athletes even after adjustment with resting BP [7], and exercise systolic BP at moderate workload is linearly associated with coronary disease risk in healthy middle-aged men independently of resting BP [8]. Therefore, understanding and modification of the lifetime determinants of exaggerated exercise BP response in adulthood may help in the prevention of adverse adulthood outcomes. The present findings expand current understanding of lifetime determinants of exercise BP response and highlight the significance of targeting preventive efforts, especially on children in low SES families, in the prevention of cardiovascular adverse outcomes.

In the current study, the association between childhood SES and adulthood exercise BP remained significant in the multivariable model, including several childhood cardiovascular risk factors and lifestyle factors, suggesting that other mediators at least partially underlie the association. Early socioeconomic disadvantage has been shown to influence adulthood resting BP, in part through BP in early life and in part through adulthood BMI [9]. In the present study, the association of childhood SES and exaggerated exercise BP responses diluted to non-significant after adjustment with adult BMI and SBP, suggesting that adulthood BMI and SBP at least partially underlie this association. Interestingly, the association of childhood SES with maximal exercise BP value remained also after adulthood BMI and SBP, suggesting that other mechanisms at least partially mediated the association. Since SES has been associated with levels of stress hormones [23], one potential pathophysiological mechanism behind the current finding could be stronger activation of neurohumoral sympathetic responses during exercise in participants with lower childhood SES, leading to higher BP in exercise. On the other hand, childhood SES has been found to predict arterial stiffness in adulthood [11]. Arterial stiffness is associated with exercise pressure response in adulthood [24,25]. Thus, another pathophysiological link between the association of childhood SES and adulthood exercise BP could be changes in arterial stiffness mediating the effect of childhood BP on cardiovascular reactivity in adulthood.

One of the strengths of the present work is that this study was based on a large randomly selected and well in baseline and in follow-up phenotyped cohort of young adults who were followed prospectively for 27 years since childhood. Some limitations, however, need to be taken into consideration. The exercise test was performed on average 14 months later than the main follow-up visit. However, since the focus in the current study was on the association of childhood SES on adulthood exercise BP with a very long follow-up, this may not be a major limitation. Although non-invasive BP measurement is the most useful method for exercise BP measurement, there remains uncertainty in the precise measurement of BP values during dynamic exercise. Additionally, the study cohort was homogenous ethnically, consisting solely of white European subjects. Therefore, the results may not be directly generalisable to other populations, and the findings should be confirmed in racially more mixed populations.

In conclusion, the current study showed that lower family SES in childhood was associated with higher maximal exercise BP in adulthood independently of childhood risk factors such as BMI, SBP, LDL cholesterol, HDL cholesterol, triglycerides, insulin, fruit consumption, vegetable consumption and physical activity. The association remained significant after further adjustment with participants SES in adulthood and after further adjustment with adulthood BMI, SBP, maximal METs and peak HR in exercise. Furthermore, low childhood SES predicted higher risk of exaggerated exercise BP response in adulthood, although this finding was diluted to non-significant after adjustment with adulthood BMI and SBP. These findings help in the understanding of the role of childhood family SES as a lifetime determinant of adulthood exercise BP response and through early lifestyle advice especially for children in low SES families may help in the prevention of adverse adulthood outcomes.

Disclosure statement

The authors report no conflicts of interest.

Funding

The Young Finns Study has been financially supported by the Academy of Finland: grants 356405, 322098, 286284, 134309 (Eye), 126925, 121584, 124282, 129378 (Salve), 117797 (Gendi), and 141071 (Skidi); the Social Insurance Institution of Finland; Competitive State Research Financing of the Expert Responsibility area of Kuopio, Tampere and Turku University Hospitals (grant X51001); Juho Vainio Foundation; Paavo Nurmi Foundation; Finnish Foundation for Cardiovascular Research; Finnish Cultural Foundation; The Sigrid Juselius Foundation; Tampere Tuberculosis Foundation; Emil Aaltonen Foundation; Yrjö Jahnsson Foundation; Signe and Ane Gyllenberg Foundation; Diabetes Research Foundation of Finnish Diabetes Association; EU Horizon 2020 (grant 755320 for TAXINOMISIS and grant 848146 for To Aition); European Research Council (grant 742927 for MULTIEPIGEN project); Tampere University Hospital Supporting Foundation; Finnish Society of Clinical Chemistry; the Cancer Foundation Finland; pBETTER4U_EU (Preventing obesity through Biologically and bEhaviorally Tailored inTERventions for you; project number: 101080117); and the Jane and Aatos Erkko Foundation.

ORCID

Heikki Aatola http://orcid.org/0000-0001-9172-5063

References

[1] Kearney PM, Whelton M, Reynolds K, et al. Global burden of hypertension: analysis of worldwide data.

Lancet. 2005;365(9455):1–7. doi: 10.1016/S0140-6736(05)17741-1.

[2] Mendis S, Puska P, Norrving B, ed. Global atlas on cardiovascular disease prevention and control. Geneva: World Health Organization, Geneva; 2011.

[3] World Health Organization. Global status report on noncommunicable diseases 2014. Available at: http://www.who.int/nmh/publications/ncd-status-report-2014/en/.

[4] Schultz MG, La Gerche A, Sharman JE. Blood pressure response to exercise and cardiovascular disease. Curr Hypertens Rep. 2017;19(11):89. doi: 10.1007/s11906-017-0787-1.

[5] Schultz MG, Otahal P, Cleland VJ, et al. Exercise-induced hypertension, cardiovascular events, and mortality in patients undergoing exercise stress testing; a systematic review and meta-analysis. Am J Hypertens. 2013;26(3):357–366. doi: 10.1093/ajh/hps053.

[6] Singh JP, Larson MG, Manolio TA, et al. Blood pressure response during treadmill testing as a risk factor for new-onset hypertension. The Framingham heart study. Circulation. 1999;99(14):1831–1836. doi: 10.1161/01.cir.99.14.1831.

[7] Caselli S, Serdoz A, Mango F, et al. High blood pressure response to exercise predicts future development of hypertension in young athletes. Eur Heart J. 2019;40(1):62–68. doi: 10.1093/eurheartj/ehy810.

[8] Mariampillai JE, Liestøl K, Kjeldsen SE, et al. Exercise systolic blood pressure at moderate workload is linearly associated with coronary disease risk in healthy men. Hypertension. 2020;75(1):44–50. doi: 10.1161/HYPERTENSIONAHA.119.13528.

[9] Kivimäki M, Lawlor DA, Davey Smith G, et al. Early socioeconomic position and blood pressure in childhood and adulthood: the Cardiovascular Risk in Young Finns Study. Hypertension. 2006;47(1):39–44. doi: 10.1161/01.HYP.0000196682.43723.8a.

[10] Raitakari OT, Juonala M, Rönnemaa T, et al. Cohort profile: the cardiovascular risk in Young Finns Study. Int J Epidemiol. 2008;37(6):1220–1226. doi: 10.1093/ije/dym225.

[11] Puolakka E, Pahkala K, Laitinen T, et al. Childhood socioeconomic status and arterial stiffness in adulthood. The Cardiovascular Risk in Young Finns Study. Hypertension. 2017;70(4):729–735. doi: 10.1161/HYPERTENSIONAHA.117.09718.

[12] Liu RS, Burgner DP, Sabin MA, et al. Childhood infections, socioeconomic status, and adulthood cardiometabolic risk. Pediatrics. 2016;137(6):e20160236. doi: 10.1542/peds.2016-0236.

[13] Elovainio M, Pulkki-Råback L, Jokela M, et al. Socioeconomic status and the development of depressive symptoms from childhood to adulthood: a longitudinal analysis across 27 years of follow-up in the Young Finns Study. Soc Sci Med. 2012;74(6):923–929. doi: 10.1016/j.socscimed.2011-12-017.

[14] Hulkkonen J, Aatola H, Pälve K, et al. Determinants of exercise peak arterial blood pressure, circulatory power, and exercise cardiac power in a population based sample of Finnish male and female aged 30 to 47 years: the Cardiovascular Risk in Young Finns Study. BMC Cardiovasc Disord. 2014;14(1):35. doi: 10.1186/1471-2261-14-35.

[15] American Thoracic Society, American College of Chest Physicians. ATS/ACCP statement on cardio- pulmonary exercise testing. Am J Respir Crit Care Med. 2003;167:211–277.

[16] Ainsworth BE, Haskell WL, Herrmann SD, et al. Compendium of physical activities. Med Sci Sports Exerc. 2011;43(8):1575–1581. doi: 10.1249/MSS.0b013e31821ece12.

[17] Juhola J, Magnussen CG, Berenson GS, et al. Combined effects of child and adult elevated blood pressure on subclinical atherosclerosis: the International Childhood Cardiovascular Cohort Consortium. Circulation. 2013;128(3):217–224. doi: 10.1161/CIRCULATIONAHA.113.001614.

[18] Aatola H, Koivistoinen T, Tuominen H, et al. Influence of child and adult elevated blood pressure on adult arterial stiffness. The Cardiovascular Risk in Young Finns Study. Hypertension. 2017;70(3):531–536. doi: 10.1161/HYPERTENSIONAHA.117.09444.

[19] Raiko JRH, Viikari JSA, Ilmanen A, et al. Follow-ups of the Cardiovascular Risk in Young Finns Study in 2001 and 2007: levels and 6-year changes in risk factors. J Intern Med. 2010;267(4):370–384. doi: 10.1111/j.1365-2796.2009.02148.x.

[20] Aatola H, Koivistoinen T, Hutri-Kähönen N, et al. Lifetime fruit and vegetable consumption and arterial pulse wave velocity in adulthood: the Cardiovascular Risk in Young Finns Study. Circulation. 2010;122(24):2521–2528. doi: 10.1161/CIRCULATIONAHA.110.969279.

[21] Mikkilä V, Räsänen L, Raitakari OT, et al. Consistent dietary patterns identified from childhood to adulthood: the cardiovascular risk in Young Finns Study. Br J Nutr. 2005;93(6):923–931. doi: 10.1079/bjn20051418.

[22] Mikkilä V, Räsänen L, Raitakari OT, et al. Major dietary patterns and cardiovascular risk factors from childhood to adulthood. The Cardiovascular Risk in Young Finns Study. Br J Nutr. 2007;98(1):218–225. doi: 10.1017/S0007114507691831.

[23] Cohen S, Doyle WJ, Baum A. Socioeconomic status is associated wit stress hormones. Psychosom Med. 2006;68(3):414–420. doi: 10.1097/01.psy.0000221236.37158.b9.

[24] Haarala A, Kähönen E, Koivistoinen T, et al. Pulse wave velocity is related to exercise blood pressure response in young adults. The Cardiovascular Risk in Young Finns Study. Blood Press. 2020;29(4):256–263. doi: 10.1080/08037051.2020.1750944.

[25] Thanassoulis G, Lyass A, Benjamin EJ, et al. Relations of exercise blood pressure response to cardiovascular risk factors and vascular function in the Framingham heart study. Circulation. 2012;125(23):2836–2843. doi: 10.1161/CIRCULATIONAHA.111.063933.