# Socioeconomic position in childhood and adult cardiovascular risk factors, vascular structure and function: the Cardiovascular Risk in Young Finns Study

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**Key Words:** socioeconomic status; cardiovascular diseases; atherosclerosis; endothelial function; risk factors

ABSTRACT

**Objective:** To examine the association of childhood socioeconomic position (SEP) with adult cardiovascular risk factors, vascular structure and vascular function in a contemporary population of young adults.

**Design:** A population-based prospective cohort study with baseline assessment in 1980.

**Setting**: Finland.

**Participants:** 856 men and 1,066 women whose childhood SEP was determined by parental occupational status (manual, lower non-manual, upper non-manual) at age 3 to 18.

**Main outcome measures:** Cardiovascular risk factors, carotid artery intima-media thickness and brachial artery flow-mediated vasodilation, assessed at age 24 to 39.

**Results**: After adjustment for age and adult SEP, systolic pressure was 2.3 mmHg higher (P=0.0002), HDL-cholesterol 0.03 mmol/l lower (P=0.02), and the insulin resistance score (HOMA-index) 0.12 units greater (P=0.05) among men; and systolic pressure 1.3 mmHg higher (P=0.02), diastolic pressure 1.1 mmHg higher (P=0.01), and height 1.1 cm less (P<0.0001) among women for each step down the childhood SEP hierarchy. Lower childhood SEP was associated with a 20% increase in the odds of having a waist circumference >102 cm in men and >88 cm in women (overall P=0.05). Childhood SEP was not associated with intima-media thickness, flow-mediated vasodilation, metabolic syndrome, LDL-cholesterol, triglycerides, BMI, alcohol consumption or smoking.

**Conclusions**: In adults under 40, low childhood SEP predicted higher blood pressure and central obesity and, in men, unfavourable HDL-cholesterol and insulin resistance, independent of current SEP. No independent effects were found on adult vascular structure, vascular function or health-related behaviours at this life stage.

Low socioeconomic position (SEP) in childhood is predictive of increased cardiovascular disease (CVD) morbidity and mortality,[1,2] but its association with adult CVD risk factors remains poorly understood. The strongest evidence relates to body mass and blood pressure which have been found to be higher among adults with low childhood SEP.[3-13] A study of postmenopausal women suggests that a low childhood SEP is also predictive of increased risk for metabolic syndrome,[14] a cluster of central obesity, high blood pressure, and impaired lipid and glucose metabolism.[15] However, evidence with regard to lipid metabolism is inconclusive: in some studies, low childhood SEP was associated with less favourable levels of HDL- but not LDL-cholesterol among women,[5,8,16] whereas among men, no association between childhood SEP and cholesterol levels was detected.[5,8,17] Other evidence for men suggests that lower childhood SEP was associated with more favourable cholesterol levels.[3]

Evidence is also ambiguous for markers of atherosclerosis, such as carotid artery intima-media thickness.[18] In one study, an association was found between lower SEP at birth and higher adult arterial intima-media thickness, but this was not replicated for SEP at 10 years of age.[16] Another study reported an association between SEP in childhood and adult intima-media thickness in women, but not in men.[19] Reduced brachial arterial flow-mediated vasodilatation is an established marker of impaired endothelial function, an important step in the atherosclerotic disease process.[20] However, no previous studies have examined whether there is a link between childhood SEP and brachial arterial flow-mediated vasodilatation. Some studies,[3-5,8,12] but not all,[9,12,16,21] have found an association between low childhood SEP and adult smoking, suggesting that early SEP might affect CVD risk through establishment of health-related behaviours rather than through early structural or functional changes in arteries.

Most studies on childhood SEP have included only a limited number of CVD risk factors. Hence, the relative importance of early SEP for risk factors not covered by these studies remains unknown. A further source of potential error is generated by reliance on evidence from older cohorts due to the fact that large-scale studies of contemporary cohorts are scarce. Existing evidence may therefore indicate stronger SEP effects than those seen in later generations born in times of greater prosperity. For these reasons, we investigated the association between childhood SEP and a large variety of established adult risk factors, including metabolic syndrome and markers of atherosclerosis and endothelial functioning, in a contemporary population of young adults.

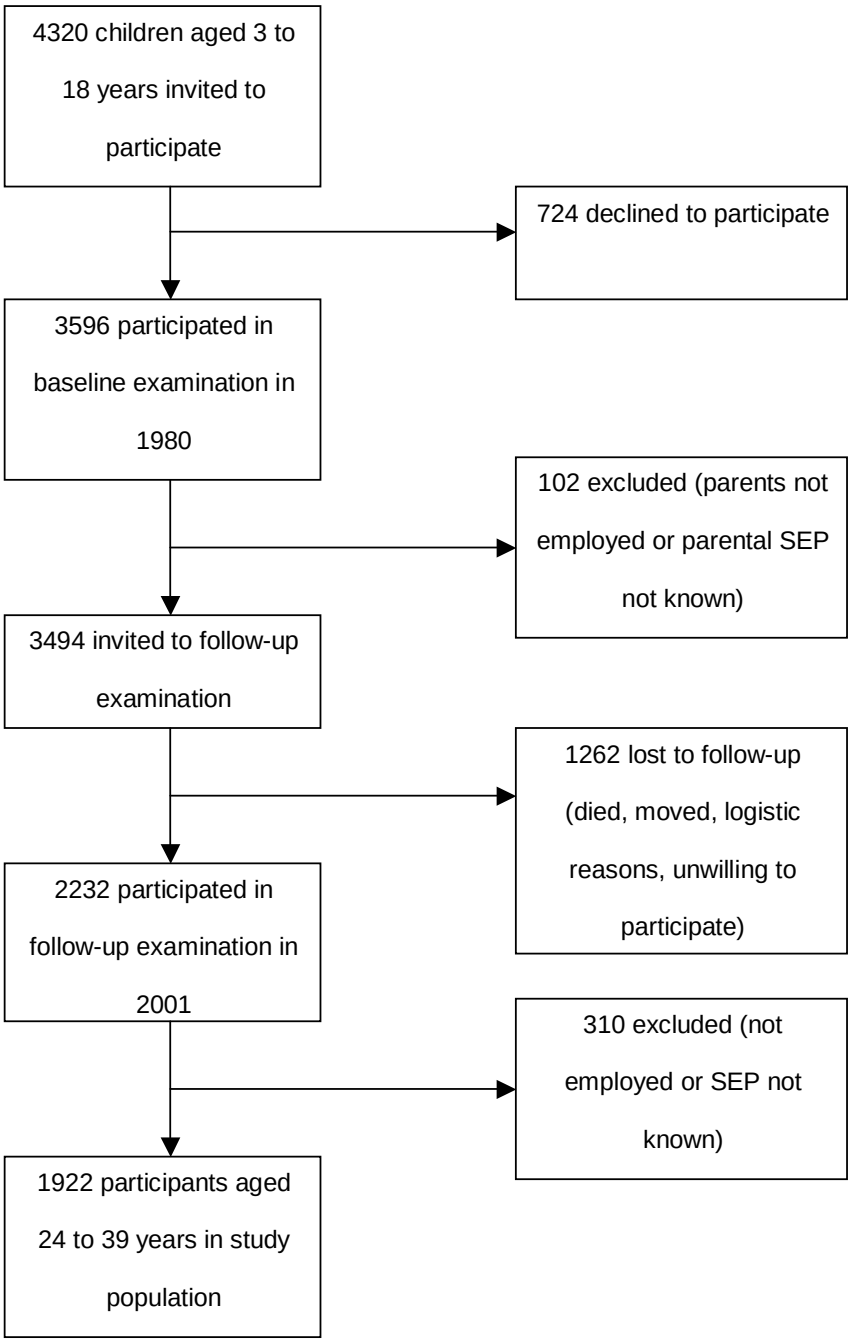
## METHODS

### Participants

The participants were from the Cardiovascular Risk in Young Finns Study, an on-going multicenter follow-up study of CVD risk factors in Finnish children and adolescents.[22,23] The original sample was 4,320 children and adolescents aged 3, 6, 9, 12, 15, and 18 years. The individuals were randomly chosen in five areas of Finland from the national register. Of those invited, 3,596 (83%) participated in the

baseline examination in 1980.[22] In 2001, the participants, who by then had reached 24 to 39 years of age, were re-examined. Participants in the present study were those 1,922 individuals (856 men and 1,066 women) who had data on SEP in 1980 and 2001 and a measurement of CVD risk factors in 2001 (Fig.). The participants did not differ from the population at baseline in terms of age group and SEP (discrepancy in any category 3% or less), but women were slightly over-represented (55% in the study cohort versus 51% in the baseline population).

FIGURE. Study population at the baseline and at follow-up examination



**Socioeconomic position**

SEP in childhood or adolescence was assessed in 1980 from parental occupational status as classified by Statistics Finland[24] and categorised as manual (eg. factory worker, mechanic, security guard, waiter, cleaning lady); lower-grade non-manual (eg. clerical employee, sales representative, nurse, secretary, foreman); and higher-grade non-manual (eg. general manager, lawyer, physician, engineer, secondary school teacher). Where SEP differed between parents, data on the parent with the higher occupational status were used. The participant's own adult SEP was measured by occupational status in 2001 and categorised as for parental SEP. There was a strong association between childhood SEP and adult SEP ($p<0.0001$, $\chi^2$-test). Participants whose childhood SEP was manual were more likely to work in manual or lower non-manual occupations than in upper non-manual occupations (corresponding frequencies 41.1%, 42.7% and 16.2%, respectively) whereas those whose childhood SEP was upper non-manual were more likely to be employed in upper or lower non-manual occupations than in manual occupations (corresponding frequencies 47.4%, 39.1% and 13.5%). Of the participants whose childhood SEP was classified as lower non-manual, most worked in lower non-manual occupations (46.3%) with smaller proportions working in upper non-manual (22.2%) and manual occupations (31.5%).

**Anthropometric measurements and blood pressure**

Physical measurements of weight (kilograms), height (mm), waist circumference (mm, measured in duplicate at the level of the twelfth rib or level with the navel in thin subjects) and hip circumference (mm) were obtained to calculate body mass index (weight in kg/ height$^2$ in m) and waist-to-hip ratio. Systolic and diastolic blood pressure (mm Hg) were measured with a random zero sphygomanometer (Hawksley & Sons Ltd, West Sussex, UK). Blood pressure was measured in a sitting position after at least five minutes rest. Readings were performed at least three times on each participant and the average was used in the statistical analysis.

**Biochemical analyses**

All blood samples were taken after an overnight fast and analyzed in duplicate in the same laboratory. Standard enzymatic methods were used for serum total cholesterol, HDL cholesterol and triglycerides, and plasma glucose concentrations. LDL cholesterol concentration was calculated using the Friedewald formula for subjects with <4 mmol/L triglyserides. Serum insulin was measured by microparticle enzyme immunoassay kit (Abbott Laboratories, Diagnostic Division, Dainabot). Insulin resistance was estimated according to the homeostasis model[25] as the product of fasting glucose and insulin divided by the constant 22.5.

**Assessment of risk behaviours and metabolic syndrome**

Information on smoking and alcohol consumption (units per week) was obtained by questionnaire. One unit of alcohol (12 g) was equal to a glass of wine, a single 4 cl shot of spirits or a 33 cl bottle of beer. The metabolic syndrome was defined according to the Treatment Panel III criteria[15] with 3 or more of the following

conditions: waist circumference >102 cm in men and >88 cm in women; HDL cholesterol <1.0 mmol/L in men and <1.3 mmol/L in women; fasting triglycerides, ≥1.7 mmol/L; blood pressure, >130/85 mm Hg, fasting plasma glucose ≥6.1 mmol/L.

**Ultrasonic measurements**

Ultrasound studies were performed using Sequoia 512 ultrasound mainframes (Acuson, Mountain View, CA, USA) to measure carotid intima-media thickness (IMT) and brachial artery flow-mediated dilation.[23,26] In brief, the image was focused on the posterior (far) wall of the left carotid artery. A minimum of four measurements of the common carotid far wall were taken approximately 10 mm proximal to the carotid bifurcation to derive maximal carotid IMT. The between-visit coefficient of variation for the IMT measurements was 6.4%.[23] To assess brachial artery flow-mediated dilation, the left brachial artery diameter was measured both at rest and during reactive hyperemia. The vessel diameter from scans after reactive hyperemia was expressed as the percentage relative to the diameter from the resting scan. The 3-month between-visit coefficient of variation was 3.2% for the brachial artery diameter measurements, and 26.0% for the flow-mediated dilation measurements.[26]

**Statistical analysis**

Age-adjusted sex differences in risk factors were tested with logistic regression analysis for dichotomous variables (smoking, metabolic syndrome) and analysis of variance for continuous variables (all other risk factors). Insulin, glucose and the HOMA-score exhibit slightly skewed distributions so we replicated analyses with these variables after log transformation. As this did not materially alter the associations the untransformed data are presented. Age- and current SEP-adjusted associations between childhood SEP and risk factors were estimated separately for men and women with regression models (logistic models for dichotomous risk factors) with childhood SEP fitted as an ordinal variable (1=upper non-manual; 2=lower non-manual; 3=manual) to assess trend. Logistic regression analyses were performed to examine the associations of childhood SEP with some common, clinically defined conditions: central obesity (waist circumference >102 cm in men and >88 cm in women),[27] hypertension (systolic blood pressure >135 mm Hg or diastolic blood pressure >80 mm Hg or antihypertensive medication),[15] low HDL cholesterol concentration (≤1.0 mmol/L in men and ≤1.3 mmol/L in women),[28] hyperinsulinemia (insulin >9.0 mU/L, top quartile of the study population), insulin resistance (HOMA index >2.05, top quartile of the study population), and metabolic syndrome.[15] These models were adjusted for age and current SEP in men and women and, additionally, for sex in the total sample. All P values are two-sided. All analyses were performed with the use of SAS software, version 8.2 (SAS Institute, Cary).

## RESULTS

The characteristics of the participants are shown in table 1. Sex differences in age, SEP in childhood, adult insulin levels and insulin resistance were small. The proportions of men in the highest and lowest adult SEP categories were greater than for women, and men had a less favourable pattern of socioeconomic mobility ($p<0.001$). Men were taller and they had a greater carotid artery intima-media thickness and lower flow-mediated dilation. They also had a poorer cardiovascular risk profile indicated by: higher blood pressure; higher levels of total cholesterol, LDL cholesterol, triglycerides and glucose; lower HDL cholesterol; higher body mass index, waist circumference and waist-to-hip ratio; a higher prevalence of smoking; and higher alcohol consumption (all $p<0.001$).

**Table 1** Sample characteristics by sex

| Variable | Men | | Women | |
|---|---|---|---|---|
| | N | Mean±SE or % | N | Mean±SE or % |
| Mean age, yrs | 856 | 31.8±0.2 | 1066 | 31.8±0.1 |
| Childhood (parental) SEP | | | | |
| Manual | 359 | 42 | 420 | 39 |
| Lower non-manual | 345 | 40 | 471 | 44 |
| Upper non-manual | 152 | 18 | 175 | 16 |
| Own adult SEP | | | | |
| Manual | 369 | 43 | 252 | 24 |
| Lower non-manual | 234 | 27 | 605 | 57 |
| Upper non-manual | 253 | 30 | 209 | 20 |
| BMI, kg/m$^2$ | 850 | 25.7±0.1 | 1032 | 24.6±0.1 |
| Waist circumference, cm | 850 | 89.8±0.3 | 1034 | 79.7±0.3 |
| Waist-to-hip ratio | 850 | 0.90±0.002 | 1032 | 0.79±0.002 |
| Height, cm | 850 | 179.6±0.2 | 1032 | 166.0±0.2 |
| Systolic pressure, mmHg | 848 | 121.6±0.4 | 1052 | 112.6±0.4 |
| Diastolic pressure, mmHg | 848 | 73.1±0.4 | 1052 | 68.8±0.3 |
| Total cholesterol, mmol/L | 852 | 5.28±0.03 | 1064 | 5.10±0.03 |
| HDL-cholesterol, mmol/L | 850 | 1.16±0.01 | 1064 | 1.40±0.01 |
| LDL-cholesterol, mmol/L | 828 | 3.44±0.03 | 1060 | 3.17±0.03 |
| Triglycerides, mmol/L | 852 | 1.52±0.03 | 1064 | 1.19±0.03 |
| Glucose, mmol/L | 851 | 5.20±0.02 | 1064 | 4.92±0.02 |
| Insulin, mU/L | 852 | 7.45±0.20 | 1064 | 7.89±0.18 |
| Insulin resistance (HOMA index*) | 844 | 1.70 (0.05) | 1060 | 1.74 (0.04) |
| Smoking | | | | |
| No | 579 | 69.2 | 852 | 81.5 |
| Yes | 258 | 30.8 | 193 | 18.5 |
| Alcohol consumption, units‡/week | 846 | 8.8±0.3 | 1060 | 3.5±0.2 |
| Metabolic syndrome† | | | | |
| No | 751 | 90.4 | 925 | 93.3 |
| Yes | 80 | 9.6 | 67 | 6.7 |
| Intima-media thickness, mm | 844 | 0.594±0.003 | 1053 | 0.574±0.003 |
| Brachial artery flow-mediated dilation, % | 767 | 6.80±0.16 | 993 | 8.82±0.14 |

*The product of fasting glucose and insulin divided by the constant 22.5.

†3 or more of the following conditions: waist >102 cm in men and >88 cm in women, serum triglycerides >1.7 mmol/L (150 mg/dl), HDL cholesterol <1.04 mmol/L (40 mg/dl) in men and <1.29 mmol/L (50 mg/dl) in women, blood pressure >130/85 mmHg or treated, and plasma glucose >6.1 mmol/L (110 mg/dl).

‡One unit (12 g) was equal to a glass of wine, a single 4 cl shot of spirits or a 33 cl bottle of beer.

Table 2 presents the age- and adult-SEP-adjusted associations between childhood SEP and adult risk factors in men. Low SEP in childhood was associated with higher waist-to-hip ratio, higher systolic blood pressure, lower HDL cholesterol, higher insulin concentration, and higher insulin resistance. Exclusion of participants treated for hypertension (n=24), hypercholesterolemia (n=5) or diabetes (n=4) had little effect on these findings.

**Table 2** Age-adjusted levels of risk factors by childhood SEP and age- and adult SEP-adjusted beta coefficients (B) per parental SEP category in men (N = 767 to 852).

| | Category of childhood SEP | | | Change per descending childhood SEP category | |
|---|---|---|---|---|---|
| Risk factor | Upper non-manual | Lower non-manual | Manual | Adjusted* B (SE) | P |
| BMI, kg/m$^2$ | 25.2 | 25.6 | 25.8 | 0.3 (0.2) | 0.13 |
| Waist circumference, cm | 88.1 | 89.5 | 90.2 | 1.0 (0.5) | 0.06 |
| Waist-to-hip ratio | 0.88 | 0.89 | 0.90 | 0.01 (0.00) | 0.006 |
| Height, cm | 180.0 | 179.9 | 179.1 | -0.5 (0.3) | 0.10 |
| Systolic blood pressure, mmHg | 118.1 | 121.2 | 123.1 | 2.3 (0.6) | 0.0002 |
| Diastolic blood pressure, mmHg | 72.2 | 72.7 | 73.1 | 0.4 (0.5) | 0.45 |
| Total cholesterol, mmol/L | 5.20 | 5.27 | 5.27 | 0.02 (0.05) | 0.69 |
| HDL-cholesterol, mmol/Ll | 1.21 | 1.15 | 1.15 | -0.03 (0.01) | 0.02 |
| LDL-cholesterol, mmol/L | 3.38 | 3.42 | 3.45 | 0.02 (0.04) | 0.60 |
| Triglycerides, mmol/L | 1.35 | 1.60 | 1.49 | 0.06 (0.05) | 0.20 |
| Glucose, mmol/L | 5.10 | 5.21 | 5.21 | 0.03 (0.03) | 0.29 |
| Insulin, mU/L | 6.68 | 7.29 | 7.93 | 0.68 (0.28) | 0.02 |
| Insulin resistance (HOMA index†) | 1.54 | 1.69 | 1.77 | 0.12 (0.06) | 0.05 |
| Smoking, % of smokers | 23.5 | 30.4 | 35.0 | -1.0 (2.2) | 0.67 |
| Alcohol consumption, units†/week | 11.2 | 8.1 | 8.7 | -0.81 (0.52) | 0.12 |
| Intima-media thickness, mm | 0.597 | 0.587 | 0.595 | -0.000 (0.005) | 0.96 |
| Brachial artery flow-mediated dilation, % | 6.80 | 6.89 | 6.76 | -0.12 (0.21) | 0.55 |

*Adjusted for age and adult SEP except height which is adjusted for age. †For definition, see Table 1

Table 3 shows the age- and adult-SEP-adjusted associations between childhood SEP and adult risk factors in women. Those with lower SEP in childhood were shorter and had higher waist circumference, higher waist-to-hip ratio, and higher systolic and diastolic blood pressure. There was no material change in these findings after the exclusion of treated participants (29 for hypertension, 1 for hypercholesterolemia and 3 for diabetes).

**Table 3** Age-adjusted levels of risk factors by childhood SEP and age- and adult SEP-adjusted beta coefficients (B) per parental SEP category in women (N = 992 to 1064).

| | Category of childhood SEP | | | Change per descending childhood SEP category | |
|---|---|---|---|---|---|
| Risk Factor | Upper non-manual | Lower non-manual | Manual | Adjusted* B (SE) | P |
| BMI, kg/m$^2$ | 23.9 | 24.6 | 25.0 | 0.4 (0.2) | 0.07 |
| Waist circumference, cm | 77.2 | 79.2 | 80.7 | 1.2 (0.5) | 0.02 |
| Waist-to-hip ratio | 0.78 | 0.79 | 0.80 | 0.01 (0.00) | 0.0002 |
| Height, cm | 167.7 | 166.0 | 165.3 | 1.1 (0.3) | <0.0001 |
| Systolic blood pressure, mmHg | 110.3 | 112.9 | 113.4 | 1.3 (0.6) | 0.02 |
| Diastolic blood pressure, mmHg | 67.2 | 68.7 | 69.4 | 1.1 (0.5) | 0.01 |
| Total cholesterol, mmol/L | 5.06 | 5.07 | 5.14 | 0.04 (0.04) | 0.34 |
| HDL-cholesterol, mmol/L | 1.42 | 1.40 | 1.39 | -0.01 (0.01) | 0.59 |
| LDL-cholesterol, mmol/L | 3.13 | 3.13 | 3.22 | 0.05 (0.03) | 0.16 |
| Triglycerides, mmol/L | 1.15 | 1.23 | 1.18 | -0.00 (0.03) | 0.88 |
| Glucose, mmol/L | 4.95 | 4.87 | 4.95 | 0.01 (0.03) | 0.81 |
| Insulin, mU/L | 7.72 | 8.00 | 8.05 | 0.09 (0.27) | 0.74 |
| Insulin resistance (HOMA index†) | 1.62 | 1.78 | 1.76 | 0.05 (0.07) | 0.42 |
| Smoking, % of smokers | 11.2 | 19.6 | 20.9 | 1.7 (1.7) | 0.33 |
| Alcohol consumption, units†/week | 4.2 | 3.3 | 3.7 | -0.1 (0.3) | 0.72 |
| Intima-media thickness, mm | 0.573 | 0.573 | 0.571 | -0.000 (0.004) | 0.99 |
| Brachial artery flow-mediated dilation, % | 8.57 | 8.88 | 8.72 | -0.04 (0.21) | 0.86 |

*Adjusted for age and adult SEP except height which is adjusted for age. †For definition, see Table 1

Table 4 presents the age- and adult-SEP-adjusted associations between childhood SEP and common clinical conditions. Low childhood SEP was associated with increased prevalence of central obesity, hypertension, hyperinsulinemia and insulin resistance in the total sample with slightly stronger effects among men than women. Among men, low childhood SEP was additionally associated with adverse HDL cholesterol levels. No robust association was found with metabolic syndrome in this low-risk population.

**Table 4** Age- and sex-adjusted prevalence (%) for some clinically defined risk factors* by childhood SEP and age-, sex- and adult SEP-adjusted odds ratio per childhood SEP category.

| | Category of childhood SEP | | | Change per descending childhood SEP category | |
|---|---|---|---|---|---|
| Risk factor* | Upper non-manual | Lower non-manual | Manual | Adjusted† odds ratio (95% CI) | P |
| All participants (n = 1823 to 1884) | | | | | |
| Central obesity | 12.3 | 17.4 | 19.1 | 1.20 (1.00 to 1.43) | 0.05 |
| Hypertension | 15.3 | 19.0 | 22.0 | 1.23 (1.03 to 1.46) | 0.02 |
| Low HDL-cholesterol | 32.9 | 33.6 | 36.3 | 1.09 (0.95 to 1.26) | 0.20 |
| Hyperinsulinemia | 16.4 | 22.9 | 24.7 | 1.23 (1.05 to 1.44) | 0.01 |
| Insulin resistance | 17.9 | 25.7 | 27.2 | 1.24 (1.07 to 1.45) | 0.005 |
| Metabolic syndrome | 5.3 | 9.1 | 8.1 | 1.14 (0.89 to 1.47) | 0.31 |
| Men (n = 831 to 850) | | | | | |
| Central obesity | 8.2 | 13.7 | 15.6 | 1.29 (0.95 to 1.74) | 0.10 |
| Hypertension | 22.7 | 26.3 | 32.6 | 1.27 (1.02 to 1.59) | 0.04 |
| Low HDL-cholesterol | 26.4 | 29.6 | 33.0 | 1.25 (1.00 to 1.54) | 0.05 |
| Hyperinsulinemia | 16.0 | 21.7 | 25.3 | 1.34 (1.06 to 1.71) | 0.02 |
| Insulin resistance | 19.3 | 25.6 | 28.2 | 1.29 (1.03 to 1.62) | 0.03 |
| Metabolic syndrome | 5.2 | 10.3 | 10.0 | 1.28 (0.90 to 1.82) | 0.17 |
| Women (n = 987 to 1034) | | | | | |
| Central obesity | 15.7 | 20.5 | 22.0 | 1.14 (0.91 to 1.43) | 0.25 |
| Hypertension | 9.4 | 13.2 | 13.4 | 1.16 (0.88 to 1.53) | 0.28 |
| Low HDL-cholesterol | 41.0 | 38.5 | 42.3 | 1.03 (0.86 to 1.23) | 0.76 |
| Hyperinsulinemia | 16.8 | 23.8 | 24.0 | 1.15 (0.93 to 1.42) | 0.21 |
| Insulin resistance | 16.7 | 25.7 | 26.4 | 1.21 (0.98 to 1.49) | 0.08 |
| Metabolic syndrome | 5.2 | 7.8 | 6.1 | 0.99 (0.69 to 1.43) | 0.97 |

*Central obesity refers to waist circumference >102 cm in men and >88 cm in women; hypertension to systolic blood pressure >130 mmHg or diastolic pressure >85 mmHg or antihypertensive medication; low HDL cholesterol to high-density lipoprotein cholesterol concentration ≤1.0 mmol/L in men and ≤1.3 in women; hyperinsulinemia to insulin level >9.0 mU/L (top quartile of the study population); and insulin resistance to HOMA-index >2.05 (top quartile of the study population). For definition of metabolic syndrome, see Table 1.

†Adjusted for age and adult SEP in men and women and additionally for sex in the total sample.

## DISCUSSION

Findings based on a contemporary, population-based sample of Finns at age 24 to 39 show that low SEP in childhood is associated with elevated adult blood pressure and central obesity for both sexes, independent of adult SEP. Low childhood SEP is also associated with less favourable lipid and glucose metabolism in men and with shorter height in women. All these effects were relatively small in magnitude and no robust evidence was found for adverse effects of low childhood SEP on adult LDL cholesterol, triglycerides, behavioural risk factors (smoking and alcohol consumption), metabolic syndrome, carotid intima-media thickness or brachial artery flow-mediated dilation.

One hypothesized underlying mechanism for the association between childhood SEP and adult CVD is the sustained influence of early socioeconomic circumstances on behavioural risk factors, such as smoking and diet.[14] Another is that low childhood SEP is a marker of suboptimal nutrition during the intrauterine period or early childhood, which programs later elevated blood pressure, insulin resistance and shorter height,[14] all risk factors for CVD. Our findings suggest that neither smoking nor alcohol consumption are key mediating factors in a contemporary population, although an association between childhood SEP and adult smoking has been reported in some older cohorts.[3-5,8,12,14] In contrast, our results on the effect of childhood SEP on adult blood pressure and obesity accord with those observed for various other populations and age ranges in the US,[21] New Zealand,[9] Finland,[7] and the UK.[4,10] Socioeconomic differences in blood pressure and obesity may start early, as they tend to track from young ages into adulthood.[29] Our findings are also in line with previous studies that have found a stronger association between childhood SEP and height among women than men,[5] although reasons for this sex difference remain unknown.

The observed SEP differences in blood pressure and central obesity in young adulthood could result in differential morbidity risk for our study population in the future. The difference in systolic blood pressure was 3.4 to 5.0 mm Hg between participants whose childhood SEP was manual and those whose childhood SEP was upper non-manual. In addition, lower childhood SEP was associated with a 30% greater risk of hypertension among men. An 18 mm Hg increase in systolic blood pressure at age 60-69 has been estimated to be associated with a 100% increase in the risk of stroke[30,31] and with more than a 50% increase in coronary risk, but in younger adults the relative risks associated with a given degree of blood pressure elevation may be much higher.[31] Obesity is a significant contributor to many health problems including type 2 diabetes mellitus, coronary heart disease, certain forms of cancer, and osteoarthritis of large and small joints.[32] In our population, the relative risk of a level of central obesity that should trigger therapeutic action increased by 20% at each step down SEP hierarchy.

Insulin resistance is a central feature of the metabolic syndrome and has been linked with development of CVD. The association found between low childhood SEP and higher insulin resistance in men is consistent with the early programming hypothesis, as insulin resistance is associated with correlates of low childhood SEP

such as suboptimal maternal nutrition and catch-up growth in early childhood.[33] An association between low childhood SEP and insulin resistance has previously been reported for postmenopausal women participating in the British Women's Heart and Health Study.[8,14] The absence of an association between childhood SEP and insulin resistance among young women in this study may indicate a protective role for pre-menopausal status or younger age.

In an historical study of British men, lower childhood SEP was associated with lower total cholesterol, an association in the opposite direction to cardiovascular risk.[3] This finding reflected less heart-healthy dietary patterns in higher SEP families, but the situation has subsequently changed. Some later studies found no association between childhood SEP and adult cholesterol levels among men,[5,16,17] but in the present study of young adults, low childhood SEP was associated with increased risk of dyslipideamia, as indicated by a less favourable level of HDL cholesterol.

In spite of the adverse levels of many metabolic syndrome factors among men with low childhood SEP, no robust association was seen between early SEP and adult metabolic syndrome.[15] However, the present data may be underpowered to observe associations with this relatively rare syndrome in young adults and our findings in men indicate that a longer follow-up may reveal such an association. To our knowledge, no previous prospective study has examined childhood SEP and adult metabolic syndrome.

Major changes in vascular function and structure come relatively late in the continuum from distal risk factors to manifest CVD and death.[20] According to one possible pathway, iterative exposure of endothelium to risk factors leads sequentially to endothelial dysfunction, followed by intimal-medial thickening, overt manifestations of atherosclerosis, development of arterial stenosis, and ultimately to plaque rupture and endovascular thrombosis.[20] In our cohort of young adults, there was no association between childhood SEP and the adult subclinical risk markers, carotid arterial intima-media thickness and brachial artery flow-mediated vasodilation. This absence of association may be due to insufficient lag time or relatively high prosperity in childhood. Indeed, in older cohorts born in less favourable circumstances, a weak association between childhood SEP and carotid atherosclerosis has been seen. The Newcastle thousand families birth cohort study found an independent effect of SEP at birth but not at age 10 on intima-media thickness in women aged 49 to 51.[19] Irrespective of the indicator of early SEP, no effect was observed for men. A retrospective study of Swedes aged 46 to 68 reported an association between childhood SEP and carotid atherosclerosis for women, but not for men.[16] In our study, imprecise measurement is an unlikely explanation for the absence of socioeconomic differences in intima-media thickness because biological and geographical factors in childhood and adult risk factors have consistently predicted intima-media thickness in this cohort.[23,26] Greater body size among participants with higher SEP cannot explain the negative finding as additional adjustment for height did not strengthen the association between childhood SEP and adult intima-media thickness.

## Study limitations

At least four potential limitations are noteworthy. First, 45% of the baseline cohort was lost during the 21-year follow-up, the loss being slightly greater among men than women. This may not be a major source of bias because all analyses were carried out separately for men and women and the loss was not differential between age groups or socioeconomic groups. Second, we performed 17 tests of associations between childhood SEP and CVD risk factors and found 6 with $p<0.05$ in men and 5 with $p<0.05$ in women; only one in 20 would have been expected by chance at this level of statistical significance. Although multiple comparisons can increase the overall type I error rate in statistical testing, associations observed in this study were all in the expected direction and findings for blood pressure and waist-to-hip ratio were similar for both sexes. Third, early SEP can be measured by using information about parental occupational group, income or education, but the present results were reported only for parental occupational group assessed at age 3 to 18. In our cohort, parental occupational status is strongly associated with parental education (weighted Kappa=0.42, $p<0.0001$). Analyses using parental education as the marker of childhood SEP generally replicated the main findings, although effects were slightly weaker. For example, after adjustment for age and sex, waist-to-hip ratio was 0.006 higher ($p=0.003$), systolic blood pressure was 1.4 mm Hg higher ($p=0.002$), and, among men, insulin resistance score was 0.11 greater ($p=0.09$) for each step down the educational hierarchy. These figures correspond to 0.009 ($p=0.0001$), 1.8 mm Hg ($p<0.0001$) and 0.12 ($p=0.05$) respectively for parental occupational group. Fourth, corresponding to the vast majority of the Finnish population in 1980, our sample was ethnically homogenous, thus, results cannot be generalized to other ethnic groups.

## Conclusions

Data from a contemporary cohort of young adults, born in times of greater prosperity than preceding generations, suggest that childhood SEP remains a predictor of adult CVD risk factors, such as waist circumference, waist-to-hip ratio, blood pressure and, in men, HDL cholesterol and insulin resistance. At this life stage – 24 to 39 years of age – the contribution of childhood SEP to adult vascular function and structure, LDL cholesterol and triglyceride concentrations, prevalence of metabolic syndrome and behavioural risk factors seems to be substantially smaller.

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**Competing interests:**

No competing interests declared.
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**Ethics Approval**

This study was conducted according to the guidelines of the Helsinki declaration and the study protocol was approved by local ethics committees. All participants gave their informed consent.

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