Childhood Socioeconomic Status and Arterial Stiffness in Adulthood

The Cardiovascular Risk in Young Finns Study

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Abstract—Increasing evidence supports the importance of socioeconomic factors in the development of atherosclerotic cardiovascular disease. However, the association of childhood socioeconomic status (SES) with arterial stiffness in adulthood has not been reported. Our aim was to determine whether higher childhood family-level SES is associated with lower arterial stiffness in adulthood. The analyses were performed using data gathered within the longitudinal Young Finns Study. The sample comprised 2566 participants who had data concerning family SES at ages 3 to 18 years in 1980 and arterial pulse wave velocity and carotid artery distensibility measured 21 or 27 years later in adulthood. Higher family SES in childhood was associated with lower arterial stiffness in adulthood; carotid artery distensibility being higher (β value±SE, 0.029±0.0089%/10 mm Hg; P=0.001) and pulse wave velocity lower (β value±SE, −0.062±0.022 m/s; P=0.006) among those with higher family SES in a multivariable analysis adjusted with age, sex, and conventional childhood cardiometabolic risk factors. The association remained significant after further adjustment for participant's SES in adulthood (β value±SE, 0.026±0.010%/10 mm Hg; P=0.01 for carotid artery distensibility and β value±SE, −0.048±0.023 m/s; P=0.04 for pulse wave velocity) but attenuated after adjustment for adulthood cardiometabolic risk factors (β value±SE, 0.015±0.008%/10 mm Hg; P=0.08 for carotid artery distensibility and β value±SE, −0.019±0.02 m/s; P=0.38 for pulse wave velocity). In conclusion, we observed an association between higher family SES in childhood and lower arterial stiffness in adulthood. Our findings suggest that special attention could be paid to children from low SES families to prevent cardiometabolic diseases primordially. **(*Hypertension*. 2017;70:729-735. DOI: 10.1161/HYPERTENSIONAHA.117.09718.)**

Key Words: attention ■ cardiovascular diseases ■ risk factors ■ social class ■ vascular stiffness

Stiffness of the central arteries tends to increase with age because elastin becomes replaced by less compliant collagen.[1–3] On account of stiffening, the arteries lose their capacity to function as a physiological buffer during cardiac pulsation and relaxation. Carotid artery distensibility (Cdist), commonly used as a marker of arterial stiffness, is defined as the change in arterial diameter during systole and diastole divided by pulse pressure and describes the ability of the artery to expand during cardiac contraction.[4] Another indicator for arterial stiffening is pulse wave velocity (PWV).[5] When arteries lose their elasticity, the speed of pulse waves is increased and they arrive back to the heart earlier, during systole, which leads to an increased load on the heart.[6] Arterial stiffness is associated with several conventional cardiometabolic risk factors and cardiovascular morbidity.[7–9] In addition, it is shown to be an independent predictor of all-cause and cardiovascular mortality.[10]

The Whitehall studies, which began in 1967 in Britain, were the first to document an inverse association between socioeconomic status (SES) with mortality.[11] The inverse association of SES with cardiovascular disease (CVD) risk

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factor levels and CVD morbidity was later shown for various populations.[12,13] A cross-sectional study showed that lower SES was associated with stiffer carotid arteries in adulthood.[14] However, no study has investigated the association between childhood SES and arterial stiffness in adulthood. This is important because atherosclerosis has been shown to begin in childhood, and socioeconomic disadvantage in childhood has been shown to associate with lifelong risk factors for CVD. Therefore, using data from the prospective longitudinal cardiovascular risk in Young Finns Study, we examined whether family-level SES of children aged 3 to 18 years was associated with their levels of arterial stiffness in adulthood measured 27 years later.

Methods

Participants

Details of the cardiovascular risk in Young Finns Study have been described previously.[15]The present sample comprised 2566 participants aged 3 to 18 years at baseline (1980) who were followed up as adults 21 or 27 years later (2001 and 2007). The mean follow-up period was 25.7 years. The study was performed according to the Declaration of Helsinki and approved by local ethics committees, and written informed consent was given by participants or parents.

Classification of SES

Annual income was considered as an indicator of SES in both childhood and adulthood.[16] 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 enrollment from 1 (<2500€) to 8 (>16 800€) and in adulthood in 2007 from 1 (<10 000€) to 8 (>70 000€).[17] In sensitivity analyses, we additionally defined childhood SES according to years of parental education.

Arterial Pulse Wave Velocity

A whole-body impedance cardiography device (CircMon; JR Medical, Ltd) was used to determine PWV. When the pulse pressure wave enters the aortic arch and the diameter of the aorta changes, the whole-body impedance decreases. The CircMon software measures the time difference between the onset of the decrease in the whole-body impedance signal and the distal plethysmographic signal from a popliteal artery at knee-joint level. PWV can be determined from the distance and time difference between the 2 recording sites. A detailed description of the method, a validation study, and reference values have been reported previously.[5,6,18]

Carotid Artery Distensibility

A high-resolution ultrasound system (Sequoia 512, Acuson, CA) with 13.0 MHz linear array transducer was used to study Cdist.[19] From B-mode images, the left common carotid diameter was measured 10 mm proximal to the carotid bifurcation at least twice during end diastole and end systole. Ultrasound and concomitant brachial blood pressure measurements were used to calculate the Cdist, as described previously.[7] The mean of Cdist values examined at the 2001 and 2007 follow-ups was calculated and used in the analyses.

Clinical Characteristics, Laboratory Measures, and Lifestyle Factors

Height, weight, and waist circumference were measured.[15] Body mass index was calculated as weight in kilograms divided by height in meters squared. Systolic and diastolic blood pressures were measured with a standard mercury sphygmomanometer in 1980 and a random-zero sphygmomanometer in 2001 and 2007. The average of 3 measurements for each was used in the analysis. Resting heart rate was recorded as bpm. Venous blood samples were collected after a 12-hour fast. Lipid determinations for triglycerides, total cholesterol, and high-density lipoprotein cholesterol were done with standard methods.[20] Low-density lipoprotein cholesterol was calculated with the Friedewald formula for subjects with triglycerides <4 mmol/L. Plasma glucose concentrations were analyzed enzymatically, and serum insulin was measured by microparticle enzyme immunoassay kit.

Data on dietary habits,[21] alcohol consumption, participant's own smoking, parental smoking, and physical activity[22] in childhood and adolescence were obtained with questionnaires.[20] Parental smoking was considered regular if a parent had smoked daily for at least 12 months and based on the number of regularly smoking parents variable was scaled 0 to 2 (0=either had smoked regularly; 1=mother or father had smoked regularly, and 2=both had smoked regularly). Details of methods have been described previously.[20,23]

Statistical Analyses

To examine differences between characteristics of men and women, we used age-adjusted linear regression, and variables were described as mean (SD) or median (interquartile range). Linear regression was used to study associations of family SES in childhood with PWV and Cdist in adulthood. The models were adjusted for age and sex (model 1) and for age, sex, and independent conventional cardiometabolic risk factors in childhood (model 2), and further for SES in adulthood (model 3). The independent childhood predictors of PWV and Cdist in adulthood were determined using stepwise modeling. Variables in initial stepwise multivariate models included childhood SES, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, insulin, systolic blood pressure, resting heart rate, body mass index, frequency of vegetable and fruit consumption per week, and physical activity. Age and sex were forced into the models. In addition, we adjusted the analyses for conventional risk factors in adulthood (model 4). The effect of each risk factor on diluting the β coefficients for the association between childhood SES and Cdist and PWV was determined. Moreover, we studied the association between childhood SES and Cdist and PWV including in the model 2 parental smoking. Associations between SES and Cdist and PWV were also studied among the subcohort of adolescents aged 12 to 18 years. These analyses were adjusted for age, sex, childhood cardiovascular risk factors, and additionally for adolescent smoking status. Because no significant sex×PWV or Cdist interactions were detected, both sexes were analyzed together.

To study the association of intergenerational mobility of SES on the PWV and mean of Cdist in adulthood, the study population was classified into 4 groups according to their SES status in childhood and adulthood: group 1 (stable low), SES below median in childhood and in adulthood; group 2 (downwardly mobile), SES above median in childhood but below median in adulthood; group 3 (upwardly mobile), SES below median in childhood and above median in adulthood; and group 4 (stable high), SES above median in childhood and adulthood. The difference in means of PWV and Cdist in these SES groups were examined using multiple comparisons adjusted for age and sex. *P* values were controlled for multiple comparison using the Tukey–Kramer method.

All statistical tests were performed using SAS, version 9.4 (SAS institute, Inc, Cary, NC) with statistical significance inferred at a 2-tailed *P* value <0.05.

Results

Baseline Characteristics

The baseline characteristics of the study population are shown in Table 1. At baseline, participants were aged 3 to 18 years with a mean age in women of 10.5 years and in men of 10.4 years. There were slightly more women than men in the study cohort, and there were sex differences noted in systolic blood pressure, lipids, and insulin levels, as well as in dietary and physical activity measures (Table 1).

Table 1. Baseline (1980) Characteristics of the Study Participants*

Variable	Women	Men	*P* Value, Age Adjusted
N	1390	1176	
Age at baseline, y	10.50±4.97	10.44±5.07	0.35
Annual family income, 1000€	26.1±14.2	26.3±14.0	0.62
BMI, kg/m^2	17.8±3.03	17.9±3.11	0.25
Systolic BP, mm Hg	111.7±11.17	113.4±12.92	<0.001
HDL cholesterol, mmol/L	1.57±0.30	1.55±0.31	0.33
LDL cholesterol, mmol/L	3.50±0.82	3.37±0.79	<0.001
Triglycerides, mmol/L	0.61 (0.33)	0.57 (0.32)	0.0002
Fasting insulin, mU/L	10.3±6.20	8.9±5.56	<0.001
Fruit consumption, frequency/wk	7.0±2.73	6.8±2.85	0.03
Vegetable consumption, frequency/wk	6.5±2.79	6.2±2.92	0.02
Physical activity index			
Age, 3–6 y (range, 8–23)	15.7±2.3	16.5±2.4	<0.001
Age, 9–18 y (range, 5–14)	8.6±1.6	9.5±2.0	<0.001

BMI indicates body mass index; BP, blood pressure; HDL, high-density lipoprotein; IQR, interquartile range; and LDL, low-density lipoprotein.

*Data are mean±SD or median (IQR).

Childhood SES and Cdist

SES in childhood was directly associated with Cdist in adulthood when adjusted for age and sex (Table 2, model 1). 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, 0.015±0.0050%/10 mm Hg; P<0.003, adjusted for age and sex). The association remained significant after adjustment for childhood cardiometabolic risk factors that were independently associated with Cdist in adulthood (Table 2, model 2). The results were similar in model 3 (Table 2) when the analysis was also adjusted for participant SES in adulthood. When the analysis was adjusted for conventional cardiometabolic risk factors in adulthood instead of those in childhood (model 4), there were no statistically significant associations between childhood SES and Cdist (β value±SE, 0.014±0.008%/10 mm Hg; P=0.10). Of the individual adulthood cardiometabolic risk factors, systolic blood pressure had the strongest diluting effect because it attenuated the association by 44.2% (from 0.032 to 0.018%/10 mm Hg). High-density lipoprotein cholesterol diluted the effect for childhood SES by 21.9% and triglycerides by 10.8%.

Table 2. Association of Childhood SES on Carotid Artery Distensibility

Model	β Value±SE (%/10 mm Hg)	*P* Value
1 (N=2562)	0.033±0.0090	0.004
2 (N=2526)	0.029±0.0089	0.001
3 (N=2026)	0.026±0.010	0.01

Results from linear regression. β values are for a 1-unit increase in family SES in childhood. Model 1: adjusted for age (3–18 y at baseline) and sex. Model 2: stepwise multivariable analysis, including childhood risk factors of age, sex, systolic blood pressure, and resting heart rate. Initial model also included LDL cholesterol, HDL cholesterol, triglycerides, insulin, BMI, frequency of vegetable and fruit consumption per week and physical activity. Model 3 included model 2 covariates plus participant SES in adulthood. BMI indicates body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; and SES, socioeconomic status.

Table 3. Association of Childhood SES on Pulse Wave Velocity

Model	β Value±SE (m/s)	*P* Value
1 (N=1803)	−0.069±0.022	0.002
2 (N=1771)	−0.062±0.022	0.006
3 (N=1704)	−0.048±0.023	0.04

Results are for linear regression. β values are for a 1-unit increase in family SES in childhood. Model 1 was adjusted for age (3–18 y at baseline) and sex. Results for model 2 are from stepwise multivariable analysis, including childhood risk factors. Final model 2 consisted of age, sex, BMI, systolic blood pressure, and vegetable consumption. Initial model also included LDL cholesterol, HDL cholesterol, triglycerides, insulin, frequency of fruit consumption per week, and physical activity. Model 3 included model 2 plus participant's own SES in adulthood. BMI indicates body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; and SES, socioeconomic status.

Childhood SES and PWV

Childhood SES was inversely associated with adult PWV (Table 3). The association was significant when adjusted for age and sex (model 1) and also in sensitivity analysis when parent years of education was used as the indicator of childhood SES (β value±SE, −0.046±0.012 m/s; P<0.001, adjusted for age and sex). The association remained after adjustment for conventional cardiometabolic risk factors in childhood that were independently associated with PWV in adulthood (model 2). Further, the association persisted when adulthood SES was added to the model (model 3). There was no statistically significant association between childhood SES and PWV in adulthood when adjustment was made for conventional cardiometabolic risk factors in adulthood (β value±SE, −0.019±0.02 m/s; P=0.38). Of the individual adulthood cardiometabolic risk factors, systolic blood pressure had the strongest diluting effect, attenuating the β value for childhood SES by 54% (from −0.069 to −0.032 m/s). Of the other adulthood cardiometabolic risk factors, triglycerides decreased the β value for childhood SES by 18.0%, and plasma glucose decreased the β value for childhood SES by 10.1%.

Effect of Smoking

At baseline, data on smoking (current/noncurrent smoker) was available only from adolescents aged 12 to 18 years (N=1282). In this subcohort, the association between childhood SES and Cdist in adulthood was not found when the analysis was adjusted for the conventional cardiometabolic risk factors in childhood used in model 2 (childhood SES β value±SE,

0.020±0.013%/10 mm Hg; *P*=0.13) or when smoking in childhood was added to the model (*β* value±SE, 0.017±0.013%/10 mm Hg; *P*=0.21). When PWV was used as the outcome, the observed effect for childhood SES remained in this subcohort after adjustment for conventional cardiometabolic risk factors (*β* value±SE, −0.078±0.035 m/s; *P*=0.02) and when additionally adjusted for smoking (*β* value±SE, −0.079±0.036 m/s; *P*=0.03).

In line, when we included in model 2 shown in Table 2 (N=2526) and in Table 3 (N=1771) parental smoking, the results remained essentially similar both between the childhood SES and Cdist in adulthood (childhood SES *β* value±SE, 0.031±0.010%/10 mm Hg; *P*=0.01) and between the childhood SES and PWV in adulthood (*β* value±SE, −0.068±0.025 m/s; *P*=0.006).

Intergenerational Mobility in SES

Cdist and PWV in adulthood were compared between the 4 childhood–adulthood SES groups (Figure 1 and Figure 2). Participants with high SES both in childhood and adulthood (stable high) had higher Cdist compared with participants in all other childhood–adulthood SES groups (Figure 1, other pairwise comparisons nonsignificant). For PWV, participants with stable high SES (group 4) had significantly less stiff arteries compared with those with stable low SES and those with downwardly mobile SES. However, participants with stable high SES did not deviate from those with upwardly mobile SES (Figure 2).

Discussion

This prospective study showed that low family SES in childhood predicts arterial stiffness, defined by 2 established markers, >25 years later in adulthood. The association was independent of conventional cardiometabolic risk factors in childhood and also of participant's own SES in adulthood but was attenuated after adjustment for risk factors in adulthood. Participants who managed to maintain SES above the median from childhood to adulthood had the least stiff arteries, indicated by both Cdist and PWV.

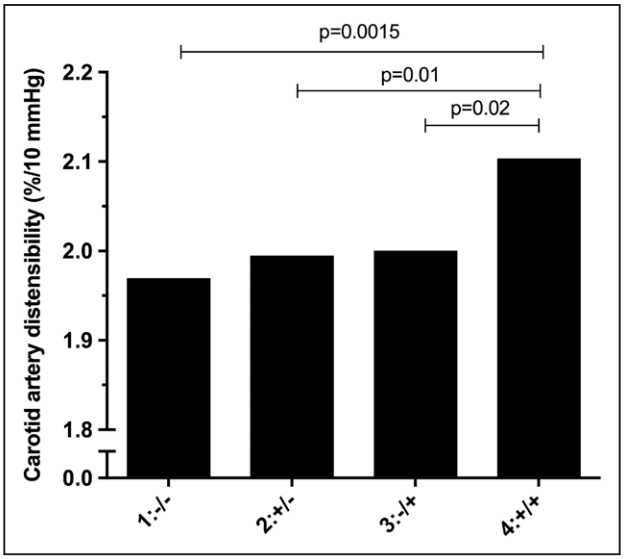

Figure 1. Mean±SEM for carotid artery distensibility in different childhood/adulthood socioeconomic status (SES) groups (N=2223). Group 1, stable low (N=502; SES under median in childhood and in adulthood); group 2, downwardly mobile (N=535; SES above median in childhood and below it in adulthood); group 3, upwardly mobile (N=409; SES below median in childhood and above it in adulthood); and group 4, stable high (N=777; SES above median in childhood and in adulthood). *P* values are shown for significant differences between the groups using multiple comparisons adjusted for age and sex. *P* values were multiplicity adjusted for the Tukey–Kramer.

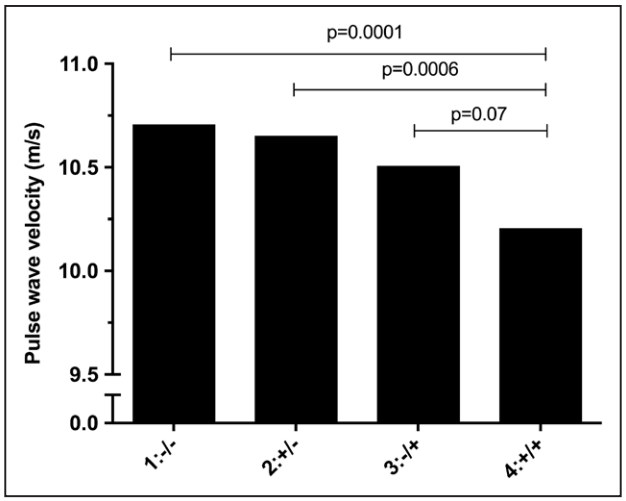

Figure 2. Mean±SEM for pulse wave velocity in different childhood/adulthood socioeconomic status (SES) groups (N=1759). Group 1, stable low (N=405; SES under median in childhood and in adulthood); group 2, downwardly mobile (N=430; SES above median in childhood and below it in adulthood); group 3, upwardly mobile (N=326; SES below median in childhood and above it in adulthood); and group 4, stable high (N=598; SES above median in childhood and in adulthood). *P* values are shown for significant differences between the groups and additionally between difference of groups 3 and 4 using multiple comparisons adjusted for age and sex. *P* values were multiplicity adjusted for the Tukey–Kramer.

The association of both child and adult SES with CVD risk factors and increased risk for cardiovascular morbidity have been shown.[13] For instance, the ARIC study (Atherosclerosis Risk in Communities) showed the cross-sectional association of adulthood SES with arterial stiffness defined by pulsatile arterial diameter change of the common carotid artery.[14] To date, however, the association between childhood SES and adult arterial stiffness has not been indicated. Our findings are important because differences in CVD mortality between socioeconomic groups are growing,[24] and the socioeconomic disparities in the risk factors start to accumulate already in childhood.[25] Because arterial stiffness is an independent predictor of cardiovascular events and all-cause mortality, these data highlight the need to target preventive efforts to families with low SES to support cardiovascular health.[10,26]

The mechanisms by which lower SES in childhood affects arterial stiffness in adulthood could be partially explained by the tracking of conventional risk factors from childhood to adulthood or the interrelation between SES, risk factors, and markers of arterial stiffness. Previous studies have shown the correlation between several conventional cardiometabolic risk factors in childhood, associated with lower SES,[27] and carotid artery stiffness in adulthood.[7] Of conventional cardiometabolic risk factors in childhood, elevated blood pressure has been shown to be the most effective predictor of adulthood

arterial stiffness.[28] In concert, we found that adult blood pressure had the strongest diluting effect on the association between childhood SES and arterial stiffness in adulthood. In addition to the inverse association with clinical risk factors, a higher childhood SES is associated with multiple lifestyle-related factors that favor cardiometabolic health, such as increased consumption of vegetables and fruits[29] and higher levels of physical activity.[30] These lifestyle factors have been shown to associate with the artery stiffness later in life and might, therefore, partly explain the association between childhood SES and arterial stiffness in adulthood.[31,32]

On the contrary, in this study, the association between childhood SES with later arterial stiffness remained after adjustment for conventional childhood cardiometabolic risk factors, in line with our prior reports.[33] Because the association was independent of these conventional risk factors in childhood, there might be some other childhood mediators which modify this association. One possible underlying mechanism is the infection-mediated pathway because the earlier infection-related hospitalization has shown to associate with lower Cdist in adulthood and the association between earlier infection-related hospitalizations in childhood and worse cardiovascular risk factor profile in adulthood is shown to exist, especially among low-SES families.[16,34] Additionally, lower SES has shown to associate with higher levels of stress hormones, which by activating the neurohumoral sympathetic responses might affect arterial stiffening.[35–37]

However, in this study, the association of childhood SES with arterial stiffness in adulthood was attenuated after adjustment for adulthood risk factors. This suggests that the effect of SES in childhood on adult arterial stiffness is partially mediated by the cardiometabolic risk factor profile in adulthood. One risk factor in adulthood which might affect arterial stiffness is alcohol intake, as indicated by a J-shaped relationship between alcohol consumption and PWV.[38] Another lifestyle factor strongly associated with higher arterial stiffness is smoking. Smoking has adverse acute and chronic effects on arterial structure and function, with similar effects found also among those exposed to tobacco smoke.[39,40] Because these arterial changes are long lasting and passive smoking is nearly as dangerous as active smoking,[23] the smoking habits of one's parents in childhood might affect arterial stiffness later in adulthood.[41] Alcohol consumption and smoking habits vary according to SES, described by an inverse association of SES and smoking and greater tobacco smoke exposure in low-SES families.[42,43] In this study, we showed that among participants aged 12 to 18 years at baseline with data on smoking habits, the association between childhood SES and arterial stiffness in adulthood persisted after adjustment for early life smoking, indicating that the observed association was not modified by higher levels of smoking among the socioeconomic disadvantaged. In line, when we adjusted the models for parental smoking, the results remained essentially similar indicating that the childhood exposure to secondhand smoke did not affect the association between childhood SES and arterial stiffness in adulthood. However, the mechanisms underlying the association between childhood SES and arterial stiffness are likely numerous, and further studies on that are needed.

High SES is associated with several indicators of better cardiovascular health. Previously, the effect of intergenerational mobility in SES has been investigated in terms of healthy lifestyle showing that parental education was not associated with having a high healthy lifestyle score among adult offspring after adjustment for the offspring's own education.[43] Our results, however, suggest that those with a stable high SES from childhood to adulthood have the least stiff arteries when using the Cdist as a marker of arterial stiffness. On the contrary, when using the PWV as a marker, the difference between those whose SES remained high and those whose SES was increased from childhood to adulthood was not statistically significant. This might indicate that the risk of greater arterial stiffening is at least partially diminished among those who improved their SES from childhood to adulthood. Our results indicate that despite changes being dynamic and additionally influenced by current risk factors in adulthood, arterial stiffening starts at a young age[44] and is influenced by SES in childhood.

We acknowledge that our study has limitations. First, the major limitation of this study is that the calculation of Cdist used pulse pressure measured from the brachial artery rather than from the carotid artery itself. Use of brachial pulse pressure may overestimate pulse pressure in central arteries. However, the difference between central and peripheral pulse pressure is likely to be similar between study subjects within a narrow age range, as is the case in the present study.[2,7] Further, we also used measurements of PWV to define arterial stiffness and the results of these 2 markers were in line. Second, the measurement of PWV was made using the CircMon software, which uses the time difference between the decrease in the whole-body impedance signal and the distal plethysmographic signal from a popliteal artery at knee-joint level. Thus, the majority of the path includes muscular arteries, which can be modified by the neurogenic and geometric factors, and these might affect the interpretation of our results. Furthermore, aortic PWV has conventionally been more strongly associated with cardiovascular risk than peripheral PWV. However, the inclusion of the femoral artery in the measurement protocol could prove important because some studies have proposed that the deleterious effects of cardiovascular risk factors on local arterial stiffness are more pronounced in the more muscular than the more elastic arteries.[45,46] Furthermore, the longer pathway (aorta-poplitea) could provide a more global index of vascular health. Third, correlations between repeated distensibility measures are relatively weak, as we have reported previously.[47]This may be linked to physiological fluctuation, instead of measurement error. Fourth, the generalizability of our study is limited to white populations because our study cohort was racially homogenous. A fifth limitation of this study includes the possibility of bias because of differential loss of follow-up. However, we have previously reported baseline risk factor levels of participants and nonparticipants in this cohort were similar and, thus, the present cohort is representative of the original study population.[48] Finally, it is possible that residual confounding may explain our observation of child SES remaining an independent predictor of adult elasticity after adjustment for lifestyle-related risk factors. The major strengths of this study include the prospective study

design, availability of 2 measures of elasticity, a long follow-up period, and the availability of data from participants who were well phenotyped in childhood and adulthood.

Perspectives

Our findings demonstrate that high SES in childhood associates with lower arterial stiffening later in adulthood defined by 2 widely used markers of arterial elasticity, PWV and Cdist, independently of conventional childhood risk factors. These findings emphasize the importance of identifying children of low-SES families, who are prone to stiffer arteries in adulthood with the aim to enhance long-term cardiovascular health from early life. These people might profit from more intensive preventive lifestyle advice already in the childhood. On the contrary, because the factors related to higher SES are associated with better cardiovascular outcome, these data can be used in the promotion of cardiometabolic health. Because our study population consisted of ethnically homogenous white population, the analyses need to be repeated in more racially mixed population. We also need further investigation on methods to determine low SES because more accurate definition would help identify those higher-risk people.

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Disclosures

None.

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Novelty and Significance

What Is New?

- This study is the first to determine the association of childhood socioeconomic status with arterial stiffness in adulthood.

What Is Relevant?

- Arterial stiffness is a prognostic index and possible therapeutic target in hypertensive patients. It is closely linked to the raised blood pressure, but has associations with cardiometabolic outcomes also independently.

Summary

We observed an association between higher family socioeconomic status in childhood and lower arterial stiffness in adulthood, suggesting that special attention could be paid to children from low–socioeconomic status families to enhance long-term cardiovascular health.

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