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# Association of Socioeconomic Status in Childhood With Left Ventricular Structure and Diastolic Function in Adulthood
## The Cardiovascular Risk in Young Finns Study

Tomi T. Laitinen, MD, PhD; Elina Puolakka, BM; Saku Ruohonen, PhD; Costan G. Magnussen, PhD; Kylie J. Smith, PhD; Jorma S. A. Viikari, MD, PhD; Olli J. Heinonen, MD, PhD; Noora Kartiosuo, BSc; Nina Hutri-Kähönen, MD, PhD; Mika Kähönen, MD, PhD; Eero Jokinen, MD, PhD; Tomi P. Laitinen, MD, PhD; Päivi Tossavainen, MD, PhD; Laura Pulkki-Råback, PhD; Marko Elovainio, PhD; Olli T. Raitakari, MD, PhD; Katja Pahkala, PhD; Markus Juonala, MD, PhD

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Supplemental content

**IMPORTANCE** Increased left ventricular (LV) mass and diastolic dysfunction are associated with cardiovascular disease. Prospective data on effects of childhood socioeconomic status (SES) on measures of LV structure and function are lacking.

**OBJECTIVE** To examine whether family SES in childhood was associated with LV mass and diastolic function after adjustment for conventional cardiovascular disease risk factors in childhood and adulthood.

**DESIGN, SETTING, AND PARTICIPANTS** The analyses were performed in 2016 using data gathered in 1980 and 2011 within the longitudinal population-based Cardiovascular Risk in Young Finns Study. The sample comprised 1871 participants who reported family SES at ages 3 to 18 years and were evaluated for LV structure and function 31 years later.

**EXPOSURES** Socioeconomic status was characterized as annual income of the family and classified on a 3-point scale.

**MAIN OUTCOMES AND MEASURES** Left ventricular mass indexed according to height at the allometric power of 2.7 and the E/e' ratio describing LV diastolic performance at ages 34 to 49 years.

**RESULTS** The participants were aged 3 to 18 years at baseline (mean [SD], 10.8 [5.0] years), and the length of follow-up was 31 years. Family SES was inversely associated with LV mass (mean [SD] LV mass index, 31.8 [6.7], 31.0 [6.6], and 30.1 [6.4] g/m$^{2.7}$ in the low, medium, and high SES groups, respectively; differences [95% CI], 1.7 [0.6 to 2.8] for low vs high SES; 0.8 [−0.3 to 1.9] for low vs medium; and 0.9 [0.1 to 1.6] for medium vs high; overall $P = .001$) and E/e' ratio (mean [SD] E/e' ratio, 5.0 [1.0], 4.9 [1.0], and 4.7 [1.0] in the low, medium, and high SES groups, respectively; differences [95% CI], 0.3 [0.1 to 0.4] for low vs high SES; 0.1 [−0.1 to 0.3] for low vs medium; and 0.2 [0 to 0.3] for medium vs high; overall $P < .001$) in adulthood. After adjustment for age, sex, and conventional cardiovascular disease risk factors in childhood and adulthood, and participants' own SES in adulthood, the relationship with LV mass (differences [95% CI], 1.5 [0.2 to 2.8] for low vs high SES; 1.3 [0 to 2.6] for low vs medium; and 0.2 [−0.6 to 1.0] for medium vs high; $P = .03$) and E/e' ratio (differences [95% CI], 0.2 [0 to 0.5] for low vs high SES; 0.1 [−0.1 to 0.4] for low vs medium; and 0.1 [0 to 0.3] for medium vs high; $P = .02$) remained significant.

**CONCLUSIONS AND RELEVANCE** Low family SES was associated with increased LV mass and impaired diastolic performance more than 3 decades later. These findings emphasize that approaches of cardiovascular disease prevention must be directed also to the family environment of the developing child.

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**Author Affiliations:** Author affiliations are listed at the end of this article.

**Corresponding Author:** Tomi T. Laitinen, MD, PhD, Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Kiinamyllynkatu 10, FIN-20520 Turku, Finland (tomi.laitinen@utu.fi).

Socioeconomic inequalities in cardiovascular disease (CVD) present a major and persistent public health challenge across industrialized nations.[1] Socioeconomic status (SES) is a powerful predictor of incident coronary disease and adverse cardiovascular outcomes.[2] Whether this robust association with SES extends to heart failure (HF) is less certain, as approximately half of cases relate to coronary disease.[3]

Echocardiographically measured left ventricular (LV) mass is associated with incident HF not related to myocardial infarction.[4,5] Left ventricular diastolic dysfunction is also prognostic of incident HF.[6,7] Previous studies have shown that low educational attainment in middle or older age is associated with higher LV mass[8,9] and impaired LV diastolic function.[9] Occupational status at different stages of life course is associated with LV mass and diastolic function at age 60 to 64 years.[10] However, to our knowledge, no studies have prospectively examined whether family SES in childhood, based on the annual income of the family, after adjustment for conventional CVD risk factors in childhood and adulthood, is associated with LV structure and diastolic function.

Using data from the longitudinal Cardiovascular Risk in Young Finns Study cohort, we examined the association of childhood family SES in participants aged 3 to 18 years on echo measures of LV mass and LV diastolic function 31 years later in adulthood when aged 34 to 49 years. We have previously shown that childhood risk factors are associated with subclinical CVD, eg, increased carotid intima-media thickness[11] and coronary artery calcification,[12] even when adjusted with adulthood risk factor levels. Therefore, we also performed analyses taking into account the effects of both childhood and adulthood risk factors.

### Key Points

**Question** Is childhood family socioeconomic status associated with left ventricular mass and diastolic function in adulthood?

**Findings** In this cohort study of 1871 participants, family socioeconomic status in childhood was related to left ventricular mass and diastolic function even after adjustment for age, sex, conventional cardiovascular risk factors both in childhood and adulthood, and participants' own socioeconomic status in adulthood.

**Meaning** These data suggest that adverse childhood socioeconomic environment is associated with higher left ventricular mass and poorer diastolic function in middle age.

## Methods

### Participants

The Cardiovascular Risk in Young Finns Study is an ongoing multicenter follow-up study to assess risk factors underlying CVD.[13] The first cross-sectional survey[13] was conducted in 1980, when 3596 individuals aged 3 to 18 years participated. These participants were randomly chosen from the national register of the study areas in different parts of Finland. Since 1980, several follow-up studies have been conducted. The 31-year follow-up survey was performed in 2011 and included analysis of 2063 of the original participants. Of these 2063 individuals, 1994 (96.7%) participated in the echocardiographic examination. In the present study, the sample comprised 1871 participants who were aged 3 to 18 years at baseline (ie, childhood in 1980) who provided data on family SES in childhood and echocardiographic data at follow-up (ie, adulthood in 2011), when aged 34 to 49 years. All participants provided written informed consent, and the study was approved by the Turku University Hospital ethical committee.

### Family SES

Family annual income was considered an indicator of family SES. Parents of the participants reported the annual income of the family in childhood.[14] The questionnaire included income categories from 1 (lowest) to 8 (highest). Family income in 1980 was converted into its present-day value and 3 income groups were formed: low (response options 1-2, ≤US $14 600), medium (response options 3-5, >US $14 600 to ≤US $32 200), and high (response options 6-8, >US $32 200). As sensitivity analysis, we additionally defined family SES according to parental occupation.[15] Parental occupation was coded from 1 to 5 (1, indicating farmers; 2, lower manual; 3, upper manual; 4, lower nonmanual; and 5, upper nonmanual).

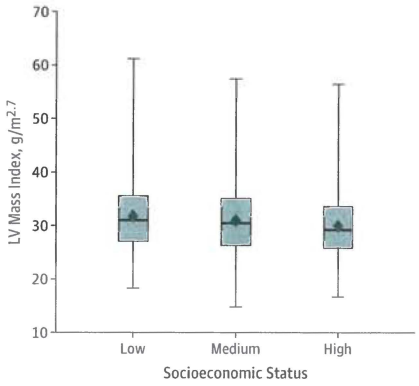
### Cardiovascular Risk Factors

Height and weight were measured, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Blood pressure was measured using a random-zero sphygmomanometer. The average of 3 measurements of blood pressure was used in the analyses. For the determination of serum lipid levels, venous blood samples were drawn after an overnight fast both in childhood and adulthood. These analyses, as well as measurements of adulthood glucose levels, were performed with standard enzymatic methods. Information on smoking habits (participants' and participants' parents') was obtained with a questionnaire. Data on childhood smoking were collected in participants aged 12 to 18 years. In adulthood, a questionnaire was used to gather data on participants' SES (annual income).[14] A questionnaire was also used to assess data on favorable emotional family environment and to subsequently comprise a score consisting of 4 components (absence of diagnosed parental mental disorder, high parental caregiving nurturance, high parental life satisfaction, and reasonable alcohol use).[16] In the score, all components indicated 1 point; thus, the scale range was 0 to 4.

### Echocardiography

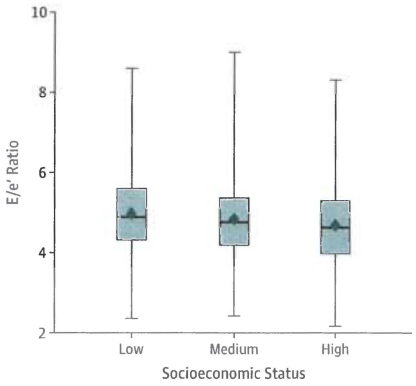
Echocardiographic examinations were performed in adulthood according to American and European guidelines.[17-19] Transthoracic echocardiograms were performed with Sequoia 512 (Acuson) ultrasonography, using a 3.5-MHz scanning frequency phased-array transducer. Analyses of the echo images were performed by a single observer. Both the sonographer and the observer were blinded to the participants' details. Standard echocardiographic examinations were produced from the standardized image planes and modes[17]:

Figure 1. Adult Left Ventricular (LV) Mass Index (LV Mass/Height$^{2.7}$) According to Childhood Family Socioeconomic Status Groups (N = 1845)




Age- and sex-adjusted $P < .001$. Normal values (fifth-95th percentile) for women, 20.3 to 35.9 g/m$^{2.7}$ and men, 21.4 to 41.5 g/m$^{2.7}$.[17]

Figure 2. Adult E/e' Ratio According to Childhood Family Socioeconomic Status Groups (N = 1871)




Age- and sex-adjusted $P < .001$. Normal values (fifth-95th percentile) for women, 3.4 to 6.5 and men, 3 to 6.[17]

parasternal long and short axis in 2-dimensional and M-mode and apical 4-chamber view. Left ventricular mass in grams was calculated from these measurements, as follows: 0.8{1.04[([LV end-diastolic diameter + posterior wall thickness + septal wall thickness]$^3$ - LV end-diastolic diameter$^3$)]} + 0.6.[17] Left ventricular mass was indexed according to height at the allometric power of 2.7 (indexed LV mass = LV mass/height$^{2.7}$) because this indexation performs better in the context of overweight/obesity.[20] Transmitral flow and tissue velocities were measured using continuous and pulsed-wave Doppler to define LV diastolic performance index, E/e' ratio, as previously described.[17] In this study, most of the values of LV mass index and E/e' ratio are within the normal range[17](**Figure 1** and **Figure 2**).

### Statistical Analyses

To examine the associations of childhood cardiovascular risk factors with childhood family SES, we used age- and sex-adjusted linear regression for continuous outcome variables and age- and sex-adjusted logistic regression for binary variables. Associations of family SES in childhood with cardiac structure and function in adulthood were examined using linear regression. Pairwise comparisons of the SES groups were adjusted with the Tukey-Kramer method. The analyses were performed both unadjusted and adjusted with age, sex, and conventional cardiovascular risk factors (BMI, systolic blood pressure, smoking, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides) in childhood and adulthood. To examine age and sex differences in the association of SES with LV mass and E/e' ratio, interaction terms of age-by-SES and sex-by-SES were used. No significant age or sex differences were detected indicating that the effect of SES on LV mass and E/e' ratio was similar between different age groups and between male and female participants. Thus, age groups and sexes were analyzed combined. All statistical tests were performed using SAS version 9.4 (SAS Institute) with statistical significance inferred at a 2-tailed $P$ value < .05.

## Results

Baseline characteristics of the study participants according to childhood family SES status are shown in **Table 1**. The participants were aged 3 to 18 years at baseline (mean [SD], 10.8 [5.0] years), and the length of follow-up was 31 years. Participants with higher family SES tended to be younger at baseline. Significant age- and sex-adjusted trends for childhood family SES groups were observed for baseline measures of parental smoking, systolic and diastolic blood pressure, high-density lipoprotein cholesterol, triglycerides, and favorable emotional family environment score.

Adult LV mass index and E/e' ratio among the childhood family SES groups are depicted in Figure 1 and Figure 2, respectively, showing an inverse association of family SES with LV mass and E/e' ratio (adjusted for age and sex). In analyses that adjusted for risk factors both in childhood and adulthood, and participants' own SES (annual income) in adulthood, the inverse relation between childhood SES and both adult LV mass and E/e' ratio remained significant (**Table 2** and **Table 3**). Concerning other risk variables, childhood BMI, adulthood BMI, and childhood low-density lipoprotein cholesterol were directly associated with LV mass in this age- and sex-adjusted multivariable analysis. Childhood favorable emotional family environment score, triglycerides, and adulthood low-density lipoprotein cholesterol were inversely associated with LV mass. Accordingly, adulthood BMI and systolic blood pressure were directly associated with E/e' ratio (eTable 1 in the Supplement). The associations of family SES with LV mass and E/e' ratio were similar when parental occupation was used as an indicator of family SES (eTable 2 in the Supplement). For LV mass, the results were also similar in participants aged 12 to 18 years at baseline (n = 588) in a model that further adjusted for each participant's own smoking status in childhood (differences [95% CI], 2.8 [0.8 to 4.9] g/m$^{2.7}$ for low vs high SES; 2.7 [0.6 to 4.7] g/m$^{2.7}$ for low vs medium; and 0.2 [−1.1 to 1.6] for medium vs

**Table 1. Baseline Characteristics of the 1871 Cardiovascular Risk in Young Finns Study Participants**

| Characteristic | Childhood Family SES, Mean (SD)[a] | | | P Value for Age- and Sex-Adjusted |
|---|---|---|---|---|
| | Low (n = 262) | Medium (n = 779) | High (n = 830) | |
| Age at baseline, y | 11.7 (5.0) | 10.5 (5.0) | 10.7 (4.9) | .003 |
| Boys, No. (%) | 115 (43.9) | 362 (46.5) | 379 (45.7) | .83 |
| Smokers, No. (%)[b] | 42 (27.6) | 76 (20.1) | 95 (23.6) | .93 |
| Parental smoking, No. (%) | 175 (79.2) | 540 (74.3) | 571 (69.6) | .002 |
| BMI | 18.4 (3.3) | 17.9 (3.1) | 17.8 (2.9) | .32 |
| BP, mm Hg | | | | |
| Systolic | 115.3 (12.7) | 112.5 (12.1) | 112.0 (11.5) | .03 |
| Diastolic | 68.3 (10.0) | 69.3 (9.3) | 67.8 (9.3) | .008 |
| Cholesterol, mg/dL | | | | |
| HDL | 57.92 (11.58) | 61.78 (11.58) | 61.78 (11.58) | .01 |
| LDL | 135.14 (30.89) | 135.14 (34.75) | 131.27 (30.89) | .15 |
| Triglycerides, mg/dL | 61.95 (26.55) | 61.95 (26.55) | 53.10 (26.55) | .002 |
| Favorable emotional family environment score | 2.6 (1.0) | 2.5 (0.9) | 2.4 (0.9) | .05 |

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SES, socioeconomic status.

SI conversion factors: To convert cholesterol and triglycerides to millimoles per liter, multiply by 0.0259.

[a] Low family SES indicates that the family annual income was ≤US $14 600; medium, >US $14 600 to ≤US $32 200; and high, >US $32 200.

[b] Data collected only in participants aged 12 to 18 years (n = 934).

**Table 2. Association Between Family SES in Childhood With LV Mass 31 Years Later in Adulthood**

| Family SES Model | LV Mass, g/m$^{2.7}$[a] | | | | | | |
|---|---|---|---|---|---|---|---|
| | SES[b] | | | SES, Difference (95% CI)[b] | | | |
| | Low | Medium | High | Low vs High | Low vs Medium | Medium vs High | P Value |
| Unadjusted model, mean (SD) (n = 1845) | 31.8 (6.7) | 31.0 (6.6) | 30.1 (6.4) | 1.7 (0.6 to 2.8) | 0.8 (−0.3 to 1.9) | 0.9 (0.1 to 1.6) | .001 |
| Adjusted model 1, mean (n = 1712)[c] | 32.4 | 31.1 | 30.3 | 2.1 (1.0 to 3.3) | 1.3 (0.2 to 2.4) | 0.8 (0.1 to 1.6) | <.001 |
| Adjusted model 2, mean (n = 1214)[d] | 32.1 | 30.8 | 30.6 | 1.5 (0.2 to 2.8) | 1.3 (0 to 2.6) | 0.2 (−0.6 to 1.0) | .03 |

Abbreviations: LV, left ventricular; SES, socioeconomic status.

[a] Left ventricular mass was normalized by dividing with height in meters at the allometric power of 2.7.

[b] Low family SES indicates that the family annual income was ≤US $14 600; medium, >US $14 600 to ≤US $32 200; and high, >US $32 200.

[c] Adjusted model 1 included age (3-18 years at baseline), sex, and childhood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, and parental smoking).

[d] Adjusted model 2 included age (3-18 years at baseline), childhood risk factors as in adjusted model 1, childhood favorable emotional family environment score, and adulthood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, smoking, glucose, and participants' own SES [annual income] in adulthood).

**Table 3. Association Between Family SES in Childhood With Diastolic Function (E/e' Ratio) 31 Years Later in Adulthood**

| Family SES Model | E/e' Ratio | | | | | | |
|---|---|---|---|---|---|---|---|
| | SES[a] | | | SES, Difference (95% CI)[a] | | | |
| | Low | Medium | High | Low vs High | Low vs Medium | Medium vs High | P Value |
| Unadjusted model, mean (SD) (n = 1871) | 5.0 (1.0) | 4.9 (1.0) | 4.7 (1.0) | 0.3 (0.1 to 0.4) | 0.1 (0 to 0.3) | 0.2 (0 to 0.3) | <.001 |
| Adjusted model 1, mean (n = 1733)[b] | 4.9 | 4.8 | 4.7 | 0.2 (0.1 to 0.4) | 0.1 (−0.1 to 0.3) | 0.1 (0 to 0.3) | .001 |
| Adjusted model 2, mean (n = 1222)[c] | 4.9 | 4.8 | 4.7 | 0.2 (0 to 0.5) | 0.1 (−0.1 to 0.4) | 0.1 (0 to 0.3) | .02 |

Abbreviation: SES, socioeconomic status.

[a] Low family SES indicates that the family annual income was ≤US $14 600; medium, >US $14 600 to ≤US $32 200; and high, >US $32 200.

[b] Adjusted model 1 included age (3 to 18 years at baseline), sex, and childhood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, and parental smoking).

[c] Adjusted model 2 included age (3 to 18 years at baseline), childhood risk factors as in adjusted model 1, childhood favorable emotional family environment score, and adulthood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, smoking, glucose, and participants' own SES [annual income] in adulthood).

high; overall $P = .004$). The effect of family SES on E/e' ratio was not statistically significant in participants aged 12 to 18 years (n = 593) when participants' own smoking status in childhood was added to the model (differences [95% CI], 0.3 [−0.1 to 0.6] for low vs high SES; 0.2 [−0.2 to 0.5] for low vs medium; and 0.1 [−0.1 to 0.3] for medium vs high; overall

$P$ = .18). In eTable 3 and eTable 4 in the Supplement, we have repeated the analyses shown in Table 2 and Table 3, respectively, using data on only the participants who had complete data on all childhood and adulthood risk factors. In addition, a sensitivity analysis for unmeasured confounding has been provided in the eAppendix in the Supplement.

## Discussion

These prospective data suggest that adverse childhood socioeconomic environment is associated with higher LV mass and poorer diastolic function more than 3 decades after the baseline evaluation. Family SES in childhood, characterized on the basis of family annual income, was related to LV mass and diastolic function even after adjustment for age, sex, and conventional cardiovascular risk factors in childhood. Moreover, the inverse association of family SES with LV mass and diastolic function was independent of adulthood conventional cardiovascular risk factors and participants' own SES in adulthood.

Left ventricular hypertrophy is associated with CVD morbidity in adulthood.[4] Of cardiovascular risk factors, childhood obesity is linked with eccentric LV hypertrophy in adulthood.[21] Here, we observed that childhood family SES was associated with adult LV mass after adjustment for BMI and other conventional CVD risk factors measured both in childhood and adulthood. Moreover, the association was independent of participants' own SES in adulthood. These results suggest that family SES in early life is an important determinant of subsequent LV mass.

Left ventricular diastolic function is recognized as an important marker of hemodynamic status. Here, we examined LV diastolic function by using the E/e' ratio, where E wave in the pulsed Doppler registration describes the early mitral inflow in diastole, and e' in the tissue Doppler registration measures the mitral annular longitudinal motion in early diastole. The E/e' ratio is considered the best echocardiographic measurement of diastolic function in the estimation of LV filling pressure.[19] In this study, we observed that children with lower family SES had poorer LV diastolic performance in adulthood. This finding is important because LV diastolic performance is associated with primary cardiac events[22] and incident HF.[7]

Socioeconomic deprivation is associated with HF development.[3,23] The effect is only partly explained by established CVD risk factors measured in adulthood.[3] To our knowledge, this study is the first that prospectively examined the association of childhood SES with subsequent cardiac structure and function also taking into account the role of childhood risk factors. This is important because differences in risk factor levels related to SES have been observed in the life course already in children and adolescents.[24] However, even though we also found differences in several childhood CVD risk factors according to family SES, the association of family SES on adult measures of LV mass and diastolic function remained significant after adjustment of CVD risk factors measured both in childhood and adulthood. These findings suggest that differences in childhood CVD risk factors are not necessarily the principal pathway linking low childhood SES and adverse changes in cardiac structure and function. Other potential mechanisms may underlie the findings observed in this study. Previously it has been suggested that childhood SES and early family environment contribute to metabolic functioning through pathways of depression, hostility, and poor quality of social contacts.[25] Adverse childhood experiences, including abuse, neglect, and household dysfunction, have also been associated with many health risk behaviors and diseases.[26,27] A model introduced in 2012 suggests that positive psychological experiences may at the same time increase restorative processes (eg, healthy behaviors) and decrease deteriorative processes (eg, inflammation), leading to better cardiovascular health.[28] We have previously reported that higher SES and positive psychosocial factors in childhood are associated with subsequent cardiovascular health, as defined by the American Heart Association.[15,16] In this study, however, the association of family SES in childhood with adult LV mass and diastolic function was independent of the number of positive emotional factors in the childhood family.

### Limitations and Strengths

This study had limitations. First, cardiac magnetic resonance imaging may be a more sensitive way to measure ventricular mass and volumes than transthoracic echocardiography.[29] However, echocardiography is still the most used application particularly in clinical practice. Second, echocardiography was not assessed in childhood in this cohort, and thus we were not able to determine at what stage of the life course childhood family SES begins to associate with cardiac structure and function. Third, some limitations in the classifications of SES are also noteworthy. For example, unemployment, poor health, and other life circumstances may have affected the family's income and subsequent classification of SES. Fourth, even though we showed that family SES in childhood was associated with LV mass and diastolic function after adjustment for conventional CVD risk factors in childhood and adulthood, we were not able to separate the factors affecting these covariates from the factors affecting the long-term outcomes. Fifth, because our study cohort was racially homogeneous, the generalizability of our results is limited to white populations. During the extensive study period of the Cardiovascular Risk in Young Finns Study, it is inevitable that loss to follow-up occurs. However, the study group has been dynamic, and we have previously reported that baseline risk factor levels were essentially similar among participants and nonparticipants.[30] Thus, the present study population is likely representative of the original population. Major strengths of this study include the prospective study design and the long follow-up of participants who were well phenotyped both in childhood and adulthood.

## Conclusions

Low family SES in childhood, after adjustment for conventional CVD risk factors in childhood and adulthood, was asso-

ciated with increased LV mass and poorer diastolic performance 3 decades later in adulthood. These findings further emphasize that approaches of CVD prevention must be directed also to the family environment of the developing child. Particularly, support for families with low SES may pay off in sustaining cardiovascular health to later life.

## ARTICLE INFORMATION

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**Author Affiliations:** The Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Turku, Finland (T. T. Laitinen, Puolakka, Ruohonen, Magnussen, Smith, Kartiosuo, Raitakari, Pahkala, Juonala); Paavo Nurmi Centre, Sports, & Exercise Medicine Unit, Department of Physical Activity and Health, University of Turku, Turku, Finland (T. T. Laitinen, Heinonen, Pahkala); Menzies Institute for Medical Research, University of Tasmania, Hobart, Australia (Magnussen); Department of Medicine and Division of Medicine, Turku University Hospital, Turku, Finland (Viikari, Juonala); Department of Pediatrics, University of Tampere, Tampere University Hospital, Tampere, Finland (Hutri-Kähönen); Department of Clinical Physiology, University of Tampere, Tampere University Hospital, Tampere, Finland (Kähönen); Department of Pediatric Cardiology, Hospital for Children and Adolescents, University of Helsinki, Helsinki, Finland (Jokinen); Department of Clinical Physiology and Nuclear Medicine, Kuopio University Hospital, University of Eastern Finland, Kuopio, Finland (T. P. Laitinen); Department of Pediatrics, Oulu University Hospital, PEDEGO Research Unit and Medical Research Center Oulu, University of Oulu, Oulu, Finland (Tossavainen); Helsinki Collegium for Advanced Studies, University of Helsinki, Helsinki, Finland (Pulkki-Råback); Unit of Personality, Work, and Health, Institute of Behavioural Sciences, University of Helsinki, Helsinki, Finland (Pulkki-Råback, Elovainio); Department of Clinical Physiology and Nuclear Medicine, University of Turku, Turku University Hospital, Turku, Finland (Raitakari).

**Author Contributions:** Dr T. T. Laitinen had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.
*Study concept and design:* T. T. Laitinen, Ruohonen, Hutri-Kähönen, Jokinen, Tossavainen, Elovainio, Raitakari, Pahkala, Juonala.
*Acquisition, analysis, or interpretation of data:* T. T. Laitinen, Puolakka, Magnussen, Smith, Viikari, Heinonen, Kartiosuo, Kähönen, T. P. Laitinen, Pulkki-Råback, Raitakari, Pahkala, Juonala.
*Drafting of the manuscript:* T. T. Laitinen, Ruohonen, Magnussen, Jokinen, Pulkki-Råback, Raitakari.
*Critical revision of the manuscript for important intellectual content:* T. T. Laitinen, Puolakka, Magnussen, Smith, Viikari, Heinonen, Kartiosuo, Hutri-Kähönen, Kähönen, T. P. Laitinen, Tossavainen, Elovainio, Raitakari, Pahkala, Juonala.
*Statistical analysis:* T. T. Laitinen, Puolakka, Kartiosuo, Elovainio.
*Obtained funding:* Kähönen, T. P. Laitinen, Raitakari, Juonala.
*Administrative, technical, or material support:* Ruohonen, Smith, Kartiosuo, Hutri-Kähönen, Kähönen, Pulkki-Råback, Raitakari, Juonala.
*Study supervision:* Ruohonen, Magnussen, Viikari, Heinonen, Raitakari, Pahkala, Juonala.

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## REFERENCES

1. Mackenbach JP, Cavelaars AE, Kunst AE, Groenhof F. Socioeconomic inequalities in cardiovascular disease mortality: an international study. *Eur Heart J*. 2000;21(14):1141-1151.

2. Sundquist K, Malmström M, Johansson SE. Neighbourhood deprivation and incidence of coronary heart disease: a multilevel study of 2.6 million women and men in Sweden. *J Epidemiol Community Health*. 2004;58(1):71-77.

3. Ramsay SE, Whincup PH, Papacosta O, Morris RW, Lennon LT, Wannamethee SG. Inequalities in heart failure in older men: prospective associations between socioeconomic measures and heart failure incidence in a 10-year follow-up study. *Eur Heart J*. 2014;35(7):442-447.

4. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med*. 1990;322(22):1561-1566.

5. de Simone G, Gottdiener JS, Chinali M, Maurer MS. Left ventricular mass predicts heart failure not related to previous myocardial infarction: the Cardiovascular Health Study. *Eur Heart J*. 2008;29(6):741-747.

6. Drazner MH, Rame JE, Marino EK, et al. Increased left ventricular mass is a risk factor for the development of a depressed left ventricular ejection fraction within five years: the Cardiovascular Health Study. *J Am Coll Cardiol*. 2004;43(12):2207-2215.

7. Kane GC, Karon BL, Mahoney DW, et al. Progression of left ventricular diastolic dysfunction and risk of heart failure. *JAMA*. 2011;306(8):856-863.

8. Rodriguez CJ, Sciacca RR, Diez-Roux AV, et al. Relation between socioeconomic status, race-ethnicity, and left ventricular mass: the Northern Manhattan study. *Hypertension*. 2004;43(4):775-779.

9. Christensen S, Mogelvang R, Heitmann M, Prescott E. Level of education and risk of heart failure: a prospective cohort study with echocardiography evaluation. *Eur Heart J*. 2011;32(4):450-458.

10. Murray ET, Jones R, Thomas C, et al. Life course socioeconomic position: associations with cardiac structure and function at age 60-64 years in the 1946 British birth cohort. *PLoS One*. 2016;11(3):e0152691.

11. Raitakari OT, Juonala M, Kähönen M, et al. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study. *JAMA*. 2003;290(17):2277-2283.

12. Hartiala O, Magnussen CG, Kajander S, et al. Adolescence risk factors are predictive of coronary artery calcification at middle age: the Cardiovascular Risk in Young Finns Study. *J Am Coll Cardiol*. 2012;60(15):1364-1370.

13. 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.

14. Liu RS, Burgner DP, Sabin MA, et al. Childhood infections, socioeconomic status, and adult cardiometabolic risk. *Pediatrics*. 2016;137(6):e20160236.

15. Laitinen TT, Pahkala K, Venn A, et al. Childhood lifestyle and clinical determinants of adult ideal cardiovascular health: the Cardiovascular Risk in Young Finns Study, the Childhood Determinants of Adult Health Study, the Princeton Follow-Up Study. *Int J Cardiol*. 2013;169(2):126-132.

16. Pulkki-Råback L, Elovainio M, Hakulinen C, et al. Cumulative effect of psychosocial factors in youth on ideal cardiovascular health in adulthood: the Cardiovascular Risk in Young Finns Study [published correction appears in *Circulation*. 2015;131(14):e403]. *Circulation*. 2015;131(3):245-253.

17. Ruohonen S, Koskenvuo JW, Wendelin-Saarenhovi M, et al. Reference values for echocardiography in middle-aged population: the Cardiovascular Risk in Young Finns Study. *Echocardiography*. 2016;33(2):193-206.

18. Lang RM, Bierig M, Devereux RB, et al; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2005;18(12):1440-1463.

19. Nagueh SF, Smiseth OA, Appleton CP, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association

of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2016;29(4):277-314.

**20**. de Simone G, Daniels SR, Devereux RB, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. *J Am Coll Cardiol*. 1992;20(5):1251-1260.

**21**. Toprak A, Wang H, Chen W, Paul T, Srinivasan S, Berenson G. Relation of childhood risk factors to left ventricular hypertrophy (eccentric or concentric) in relatively young adulthood (from the Bogalusa Heart Study). *Am J Cardiol*. 2008;101(11):1621-1625.

**22**. Sharp AS, Tapp RJ, Thom SA, et al; ASCOT Investigators. Tissue Doppler E/E' ratio is a powerful predictor of primary cardiac events in a hypertensive population: an ASCOT substudy. *Eur Heart J*. 2010;31(6):747-752.

**23**. Hawkins NM, Jhund PS, McMurray JJ, Capewell S. Heart failure and socioeconomic status: accumulating evidence of inequality. *Eur J Heart Fail*. 2012;14(2):138-146.

**24**. Khoury PR, Morrison JA, Laskarzewski P, et al. Relationships of education and occupation to coronary heart disease risk factors in school children and adults: the Princeton School District Study. *Am J Epidemiol*. 1981;113(4):378-395.

**25**. Lehman BJ, Taylor SE, Kiefe CI, Seeman TE. Relation of childhood socioeconomic status and family environment to adult metabolic functioning in the CARDIA study. *Psychosom Med*. 2005;67(6):846-854.

**26**. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: the Adverse Childhood Experiences (ACE) Study. *Am J Prev Med*. 1998;14(4):245-258.

**27**. Anda RF, Croft JB, Felitti VJ, et al. Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA*. 1999;282(17):1652-1658.

**28**. Boehm JK, Kubzansky LD. The heart's content: the association between positive psychological well-being and cardiovascular health. *Psychol Bull*. 2012;138(4):655-691.

**29**. Grothues F, Smith GC, Moon JC, et al. Comparison of interstudy reproducibility of cardiovascular magnetic resonance with two-dimensional echocardiography in normal subjects and in patients with heart failure or left ventricular hypertrophy. *Am J Cardiol*. 2002;90(1):29-34.

**30**. Nuotio J, Oikonen M, Magnussen CG, et al. Cardiovascular risk factors in 2011 and secular trends since 2007: the Cardiovascular Risk in Young Finns Study. *Scand J Public Health*. 2014;42(7):563-571.