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Neighbourhood socioeconomic disadvantage from childhood to midlife and carotid atherosclerosis.

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ABSTRACT

Background: Neighbourhood socioeconomic disadvantage correlates with cardiovascular disease risk. However, its relationship with subclinical atherosclerosis from childhood to midlife is not well-defined. We examined whether cumulative neighbourhood disadvantage is associated with carotid artery plaques, a measure of subclinical atherosclerosis.
Methods: We analysed data from 1998 participants in the Cardiovascular Risk in Young Finns Study, a cohort followed from childhood (mean age 10.7 years in 1980) to adulthood (mean age 48.6 years in 2018–2020). Neighbourhood disadvantage was derived from national grid-based socioeconomic data and computed cumulatively across the life course. The number of carotid artery plaques (mean plaque count) was assessed by standardized ultrasound imaging. Multivariable Poisson regression models were used to evaluate the associations. Mediation analyses were used to assessed the role of ideal cardiovascular health (CVH) metrics.
Results: Higher cumulative neighbourhood disadvantage from childhood to mid-adulthood was associated with a 1.24-fold increase in mean plaque count for every 1 standard deviation increase in cumulative disadvantage. This relationship persisted after controlling for parental carotid artery plaques, polygenic coronary artery disease risk score, and Framingham risk score. The association was partially explained by ideal CVH metrics, particularly smoking and blood pressure, which collectively accounted for almost half of the association.
Conclusions: Long-term exposure to neighbourhood socioeconomic disadvantage beginning in childhood is associated with subclinical atherosclerosis in midlife, independently of achieved socioeconomic position. These findings highlight the importance of cumulative socioeconomic environments across the life course and suggest that behavioural risk factors may partly explain observed neighbourhood-level associations with atherosclerosis.

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1. Introduction

Neighbourhood socioeconomic disadvantage is associated with higher risk of cardiovascular diseases, such as coronary artery disease and stroke [1,2]. In most cases, these cardiovascular disease outcomes are manifestations of the end stages of atherosclerosis - a disease that often begins early in life and progresses asymptomatically for decades [3,4]. Since atherosclerosis develops gradually over the life course, cohort studies focusing on atherosclerosis can provides an opportunity to study the long-term associations of neighbourhood context.

Ultrasound imaging of carotid arteries provides a non-invasive method to measure atherosclerotic plaques in epidemiologic studies. Plaques are markers of generalized atherosclerosis and associate with approximately twofold increased risk of coronary artery disease and stroke [5–7]. Studying the association at the subclinical stage can help elucidate the mechanisms through which neighbourhood socioeconomic disadvantage over the life course influences cardiovascular health. Furthermore, while individual-level socioeconomic status is a strong predictor of cardiovascular outcomes, studying subclinical markers helps determine if neighbourhood socioeconomic disadvantage is independently associated with atherosclerosis after accounting for individual-level socioeconomic factors. The independent role of neighbourhood disadvantage in increasing cardiovascular risk is still uncertain, as many studies have observed that the association between neighbourhood socioeconomic status and cardiovascular disease risk attenuates or loses statistical significance after accounting for individual socioeconomic status [8–10].

Thus, while the association between neighbourhood socioeconomic disadvantage and clinical cardiovascular disease is established, studying asymptomatic carotid artery plaques offers a valuable approach to understanding the early development of atherosclerosis, and elucidating the specific pathways through which neighbourhood disadvantage may be related with cardiovascular health across childhood to midlife. Understanding these complex relationships is essential for developing targeted, effective interventions to reduce cardiovascular health disparities associated with neighbourhood environments [11].

2. Methods

Please see details in the Supplementary Methods. In brief, the longitudinal Cardiovascular Risk in Young Finns Study (YFS) was initiated in 1980 (baseline age, 3–18 years, $N = 3596$) [12]. The latest follow-up was extended to cover the original participants as well as their parents and offspring [13]. In the present analyses, we used data on 1998 original cohort members who participated in the latest follow conducted between February 2018 and February 2020, and had complete data on neighbourhood disadvantage.

2.1. Clinical examination, questionnaires, biochemical and genetic measurements

Anthropometrics and blood pressure were measured. Data on medical conditions, medications, socioeconomic status (SES), life-style habits were collected by questionnaires and national health registries. Standard methods were used to measure serum lipids and lipoproteins. The Framingham risk score [14] and the American Heart Association ideal cardiovascular health score were calculated [15]. Polygenic risk score for coronary artery disease (CAD PRS) was calculated [16].

2.2. Carotid ultrasonography

Carotid ultrasound studies were performed using General Electric (GE) Logiq S8 (GE Vingmed Ultrasound A/S, Horten, Norway) ultrasound mainframes. Carotid arteries were scanned for plaques. The total number of plaques, plaque area and plaque thickness, were measured [17]. As sensitivity analyses, we examined the associations between neighbourhood disadvantage and common carotid artery intima-media thickness (IMT) values that have been measured in the previous follow-up studies in 2001 and 2007 [18], as well as in the most recent follow-up study in 2018–20 [13].

2.3. Parental plaque load

Participants were classified as having a high parental plaque load if either parent had carotid plaques above the median for their age and sex-specific age-group.

2.4. Neighbourhood socioeconomic disadvantage

Data on neighbourhood social disadvantage was derived from a grid database established and maintained by Statistics Finland. The database contains socioeconomic information from each residence at a spatial resolution of 250 m by 250 meters [19,20]. The neighbourhood disadvantage score was calculated using data on median household income (coded as additive inverse), education attainment (percentage of people over 18 years old whose highest education level is elementary school) and unemployment rate (unemployed people belonging to the labour force/total labour force) in each grid area. For each of the three variables, we derived a standardized z score based on the total Finnish population (mean = 0, SD = 1). A score for neighbourhood disadvantage was then calculated by taking the mean value across the three z scores. Higher scores on the continuous index denote greater disadvantage. For the statistical analyses, the continuous neighbourhood disadvantage score was also classified into two categories using the national means as the cut-off point. Residential mobility data were obtained from the Population Register Centre for each participant. Data on the residential neighbourhood disadvantage for each time point were linked to the cohort participants' home addresses using latitude and longitude coordinates. A cumulative socioeconomic disadvantage score weighted by residential time at each location in between 1980 and 2018 was calculated for each participant.

2.5. Statistical methods

We determined the exposure to cumulative neighbourhood socioeconomic disadvantage between 1980 and 2018, separately for childhood/adolescence (ages 6 to 20 years), adulthood (ages 21 to 55) and childhood-to-midlife (the life course between ages 6 to 55). Current disadvantage was based on neighbourhood socioeconomic disadvantage in the Jan 1, 2018 residential location. In the analyses on the number of carotid plaques (plaque count), we used Poisson regression analyses and expressed the results as counts (mean number of plaques per individual), and the mean count ratio, as well as their 95% confidence intervals (95% CI).

First, we examined the associations between cumulative neighbourhood socioeconomic disadvantage (treated as a continuous variable) during the three different life stages, as well as current residential neighbourhood and the number of carotid plaques. Second, we examined the associations between CVD risk factors (high or low), including parental plaque load, CAD PRS, Framingham risk score and ideal CVH score – and the number of carotid plaques adjusted for age and sex.

In the main analyses, the continuous neighbourhood disadvantage score was also classified into two categories using the national means as the cut-off point. Thus, we examined the associations of high vs low cumulative neighbourhood disadvantage (standardized z score based on the total Finnish population > 0 or ≤ 0) between 1980 and 2020 among participants based on their CVD risk status.

In these analyses, participants were categorized into groups: (1) low/high neighbourhood disadvantage and low/high parental plaque load, (2) low/high neighbourhood disadvantage and low/high CAD PRS, (3) low/high neighbourhood disadvantage and low/high Framingham risk score and (4) low/high neighbourhood disadvantage and ideal/

intermediate CVH score. From these analyses, we report the mean plaque count (95% CIs) for each group as well as the mean count ratio (95% Cis) of high vs. low neighbourhood disadvantage in the statuses of each low and high CVD within each stratum.

Finally, to examine the extent to which risk indicators account for the associations between neighbourhood disadvantage and carotid plaques, we estimated the proportion of this association mediated by the ideal CVH score components. We decomposed the total association (*total effect*) into an association component operating through the mediator set (*indirect effect*), and a remaining association component not operating through the mediator set (*direct effect*). Despite the nomenclature, these analyses decompose associations under stated modelling assumptions, and should not be interpreted as establishing causal mediation.

3. Results

Of the 2052 cohort members, who participated in the 2018–20 ultrasound examination, 1998 (97%) provided data on their cumulative neighbourhood socioeconomic disadvantage between 1980 and 2020. The mean age was 10.7 years (range 3–18) at baseline in 1980 and 48.6 years (range 41–56) at the end of the 38-year follow-up in 2018–2020. The characteristics table (Table 1) provides descriptive information to assess how strongly each individual characteristic is linked with both carotid atherosclerosis and neighbourhood disadvantage. Higher mean plaque counts were observed in relation to male sex, low education, low parental SES, high parental plaque load, high CAD PRS, high Framingham risk score, poor ideal CVH total score and all non-ideal CVH score components. Participants classified into high and low neighbourhood disadvantage categories based on their cumulative score from 1980 to 2018 did not differ in terms of age, sex, CAD PRS, or parental carotid plaque load.

Individuals exposed to high cumulative neighbourhood socioeconomic disadvantage had on average lower own education level and lower parental socioeconomic position compared to individuals exposed to low disadvantage. They were also less often married or cohabiting, had higher prevalence of type 2 diabetes (10.0 vs. 6.2%, $p = 0.002$) and atherosclerotic cardiovascular diseases (4.5 vs. 3.2%, $p = 0.12$), and (Table 1).

High neighbourhood socioeconomic disadvantage was also associated with higher cardiovascular risk, as assessed by the Framingham risk score, and inversely associated with the ideal CVH score. The distributions of all individual components of the ideal CVH score differed between the two groups, except for ideal cholesterol level (Table 1).

The associations between cumulative neighbourhood socioeconomic disadvantage across life-stages and the midlife carotid plaques are shown in Table 2. Four distinct exposure periods were examined: 1) cumulative socioeconomic disadvantage between childhood and mid-adulthood; 2) cumulative neighbourhood socioeconomic disadvantage during childhood/adolescence; 3) cumulative neighbourhood socioeconomic disadvantage during adulthood; and 4) current neighbourhood socioeconomic disadvantage, i.e. at the time of the carotid plaques assessment. No cross-sectional associations were observed between the current neighbourhood disadvantage and plaques.

However, cumulative disadvantage exposure was significantly associated with higher mean carotid plaque count in models adjusted for age and sex, and additionally for own and/or parental socioeconomic status, own marital status, as well as the number of moves and population density between 1980 and 2018. The strongest associations between neighbourhood disadvantage and plaques were observed in the life course models spanning the exposure time from childhood to mid-adulthood. The mean count ratio indicated over 20% increase in the mean plaque count by every 1 standard deviation increase in cumulative disadvantage.

We also examined the associations between cumulative disadvantage and plaque features indicating the severity of plaques. In analyses restricted to individuals with one or more plaques ($N = 789$), those with high cumulative life course disadvantage ($N = 306$) had higher mean plaque area (20.7 vs. 17.7 mm^2, $p = 0.017$), and higher plaque maximal thickness (3.5 vs. 3.0 mm, $p = 0.008$) than those with low disadvantage ($N = 483$).

As an additional analyses, we compared common carotid artery IMT values between participants with low vs. high cumulative neighbourhood disadvantage. The IMT values have been measured in previous study years 2001 and 2007, as well as in the most recent follow-up study. In 2001, when the participants were aged between 24 and 39 years, the cumulative neighbourhood disadvantage was not associated with the IMT ($p = 0.23$). However, in 2007 (at ages 30 and 45 years), the group with high disadvantage had 0.01 mm higher IMT than the group with low disadvantage (95%CI 0.001–0.018 mm, age and sex adjusted $p = 0.03$). By 2018–20 (at ages 40 to 58 years), this difference has increased to 0.02 mm (95% CI 0.012–0.033, age and sex adjusted $p < 0.0001$).

Both genetic risk markers (parental plaque load and CAD PRS) and lifestyle risk markers (Framingham score and ideal CVH score) were directly associated with carotid plaque count (Table 3). For example, above the median values for CAD PRS was associated with about 35% higher plaque count as compared to below median values. A high Framingham risk score (over 7.5%) was associated with approximately 45% higher carotid plaque count compared low to a low Framingham risk score. Similarly, having poor ideal CVH score, as opposed to an ideal or intermediate score was also associated with an approximately 45% higher plaque count.

The association between high vs. low cumulative neighbourhood disadvantage and carotid plaques (mean count ratio = 1.26) remained statistically significant and was not substantially attenuated in models additionally adjusted for parental plaque load, CAD PRS and Framingham risk score (measn count ratio after adjustments 1.24–1.25). The association was similar in both sexes: men mean count ratio = 1.25 (1.11–1.42), women 1.27 (1.10–1.47), sex-interaction $p = 0.89$. When the ideal CVH score was included in the model, the association between neighbourhood disadvantage and plaque remained significant, but was attenuated to mean count ratio = 1.15. This suggests that the association between neighbourhood disadvantage and carotid plaques may be partly explained by cardiovascular health captured by the ideal CVH score.

To further examine how the background risk factor profile may influence the link between neighbourhood disadvantage and carotid plaques, we examined this association for the combinations of low/high neighbourhood disadvantage and low/high background risk (Fig. 1). The lowest mean plaque counts were observed in individuals with a low parental plaque load, low CAD PRS, low Framingham risk score, or high ideal CVH score in combination with low cumulative neighbourhood disadvantage. In comparison, the mean carotid plaque count in individuals who had been exposed to high neighbourhood disadvantage was 1.61-fold (95% CI 1.28–2.03) in combination with a high parental plaque load, 1.68-fold (95% CI 1.45–1.95) in combination with a high CAD PRS, 1.82-fold (95% CI 1.56–213) in combination with high Framingham risk score, and 1.72-fold (95% CI 1.49–1.98) in combination with low ideal CVH score.

High neighbourhood disadvantage was associated with increased mean plaque count both in individuals with a low as well as high risk status. The only exception was a lack of association between neighbourhood disadvantage and mean carotid plaque count among those with ideal CVH score (test of interaction $P < 0.001$). This observation again indicated that the ideal CVH score or its components could play a role in mediating the link between neighbourhood disadvantage and carotid plaque.

To further test this hypothesis, we performed a formal mediation analyses examining the role of each ideal CVH score component separately and in combination as a potential mediator of the association between neighbourhood disadvantage and carotid plaque. The results are shown in Table 4. The column *indirect effect* gives an estimate of the mediation, i.e. how large proportion of the association between

Table 1
Characteristics of the participants.

		Carotid plaques*		Disadvantage**		
Characteristic	All (%)	Mean rate	*P*-value	Low	High	P-value
All, N (%)	1998	0.78		1292	706	
Sex, N (%)						
Women	1108 (55.5)	0.64	<0.0001	718 (55.6)	390 (55.2)	0.8865
Men	890 (44.5)	0.96		574 (44.4)	316 (44.8)	
Age, mean yrs. (SD)	48.6 (5.0)			48.6 (5.1)	48.8 (4.9)	0.3605
<50	960 (48.0)	0.45	<0.0001	636 (49.2)	324 (45.9)	
>50	1038 (52.0)	1.09		656 (50.8)	382 (54.1)	
Education						
Primary/secondary	703 (36.0)	0.91	<0.0001	367 (29.0)	336 (48.8)	<0.0001
Tertiary	1251 (64.0)	0.70		899 (71.0)	352 (51.2)	
Married or cohabiting						
No	461 (24.1)	0.73	0.2662	268 (21.6)	193 (28.7)	0.0005
Yes	1452 (75.9)	0.79		973 (78.4)	479 (71.3)	
Parental SES						
Low	1023 (51.2)	0.90	<0.0001	550 (42.6)	473 (67.0)	<0.0001
High	975 (48.8)	0.65		742 (57.4)	233 (33.0)	
***parental plaque load, N (%)						
Low	517 (50.4)	0.58	0.003	348 (50.4)	169 (50.6)	0.9433
High	508 (49.6)	0.73		343 (49.6)	165 (49.4)	
CAD PRS						
Low (<0)	930 (50.2)	0.64	<0.0001	608 (50.5)	322 (49.6)	0.7167
High (>0)	923 (49.8)	0.93		596 (49.5)	327 (50.4)	
Framingham risk score, N (%)						
Low/intermediate (<7.5)	1391 (73.6)	0.63	<0.0001	922 (75.2)	469 (70.5)	0.0276
High (>7.5)	500 (26.4)	1.18		304 (24.8)	196 (29.5)	
Ideal CVH score						
Ideal/intermediate (3–7)	1028 (61.3)	0.60	<0.0001	724 (65.7)	304 (52.9)	<0.0001
Poor (0–2)	649 (32.6)	1.04		378 (34.3)	271 (47.1)	
Ideal blood pressure						
No	1533 (76.8)	0.86	<0.0001	966 (74.9)	567 (80.4)	0.005
Yes	462 (23.2)	0.52		324 (25.1)	138 (19.6)	
Ideal cholesterol						
No	1055 (53.0)	1.00	<0.0001	675 (52.5)	380 (53.9)	0.5344
Yes	937 (47.0)	0.54		612 (47.5)	325 (46.1)	
Ideal glucose						
No	774 (38.9)	0.90	<0.0001	474 (36.8)	300 (42.6)	0.0122
Yes	1218 (61.1)	0.71		813 (63.2)	405 (57.4)	
Non-smoker						
No	403 (21.2)	1.02	<0.0001	229 (18.6)	174 (26.1)	0.0001
Yes	1497 (78.8)	0.70		1004 (81.4)	493 (73.9)	
Physically active						
No	816 (43.4)	0.86	0.0001	492 (40.0)	324 (49.6)	<0.0001
Yes	1066 (56.6)	0.70		737 (60.0)	329 (50.4)	
BMI <25 kg/m2						
No	1339 (67.0)	0.82	0.008	825 (63.9)	514 (72.8)	<0.0001
Yes	659 (33.0)	0.71		467 (36.2)	192 (27.2)	
Ideal diet						
No	1312 (75.4)	0.80	0.0244	830 (73.1)	482 (79.5)	0.0031
Yes	429 (24.6)	0.69		305 (26.9)	124 (20.5)	
Type 2 diabetes						
No	1837 (92.4)	0.75	<0.0001	1207 (93.8)	630 (90.0)	0.0023
Yes	150 (7.6)	1.19		80 (6.2)	70 (10.0)	
CVD diagnoses						
No	1925 (96.3)	0.76	<0.0001	1251 (96.8)	674 (95.5)	0.12
Yes	73 (3.7)	1.50		41 (3.2)	32 (4.5)	
****Moves 1980–2018, mean (SD)	7.3 (4.3)					
<7	998 (50.0)	0.79	0.751	624 (48.3)	374 (53.0)	0.0456
>7	1000 (50.0)	0.78		668 (51.7)	332 (47.0)	
*****Density 1980–2018, mean (SD)	234.2 (192.7)					
<100	523 (26.2)	0.75	0.6606	255 (19.7)	268 (38.0)	<0.0001
100–299	932 (46.6)	0.80		677 (52.4)	255 (36.1)	
>300	543 (27.2)	0.79		360 (27.9)	183 (25.9)	

CAD PRS; polygenic risk score for coronary artery disease.
CVD diagnoses; clinical cardiovascular disease diagnoses with atherosclerotic aetiology.
* Carotid plaque rate indicates the mean plaque count (number of plaques).
** Cumulative disadvantage between mean ages 10.7 (range 3–18) - 48.7 (range 41–56) years in relation to the national mean: Low <0, High >0.
*** Participants whose either parent had carotid plaques above the median in the corresponding sex-specific age-group.
**** Number of times moved between 1980 and 2018.
***** Number of residents in the neighbourhood.

Table 2
Cumulative neighbourhood disadvantage at different life stages and the number of carotid plaques (rate ratio / 1 SD increase in disadvantage)

	Residential time	Model 1		Model 2	
Disadvantage (continuous)*	Mean years (SD)	Rate ratio	95%CI	Rate ratio	95%CI
Over the lifecourse	38.3 (3.9)	1.26	(1.15–1.39)	1.24	(1.12–1.38)
Childhood/ adolescence	9.0 (5.3)	1.19	(1.10–1.29)	1.16	(1.07–1.26)
Adulthood	29.3 (4.6)	1.21	(1.11–1.32)	1.19	(1.08–1.32)
Current	–	1.02	(0.96–1.09)	0.99	(0.92–1.07)

Model 1, adjusted for age and sex.
Model 2, adjusted additionally for parental SES (for childhood/adolescence exposure) or parental SES, own education and marital status (for life course, adulthood and current exposure).
Mean current age 48.7 years (range 41–56).
* Standardized in relation to the national mean.

neighbourhood disadvantage and carotid plaque is accounted by ideal CVH health. Adjusted for age and sex, the ideal CVH score components in combination mediated almost half of the total effect and more than one third after further adjustment for parental SES, own education and marital status. Of the individual components, smoking (15%) and blood pressure (6%) were the strongest mediators.

4. Discussion

Our study demonstrates that cumulative exposure to neighbourhood socioeconomic disadvantage, over 40 years, beginning in childhood, is associated with an increased risk of atherosclerotic carotid artery plaques in mid-adulthood. This association was independent of own or parental socioeconomic position. We additionally found evidence that the association between neighbourhood socioeconomic disadvantage and the mean plaque count was substantially mediated by cardiovascular risk factors, such as smoking and elevated blood pressure. These observations suggest that long-term exposure to neighbourhood socioeconomic disadvantage links to vascular pathology partly through behavioural pathways, such as initiation of smoking during childhood/ adolescence and/or inability to stop smoking during adulthood, and elevated blood pressure. Our findings are in line with the few existing studies that suggest an association of high neighbourhood socioeconomic disadvantage across the life course with cardiovascular disease risk and events in later life [20–23].

Thus, our results indicated that smoking behaviour and elevated blood pressure partly explained the association between neighbourhood disadvantage and carotid atherosclerosis. Neighbourhood-targeted strategies that may specifically impact smoking and blood pressure include reduced tobacco retail access [24], enhanced greenness [25] and increased neighbourhood safety [26]. The previous literature on neighbourhood-targeted strategies with the most direct relevance to atherosclerotic cardiovascular diseases include reducing tobacco retail access [24] and enhancing neighbourhood greenness [27]. Because we observe associations from childhood through midlife, earlier and sustained neighbourhood interventions are justified. Tobacco-retailer caps near schools, safe routes engouraging active commuting, greening around youth amenities, and access to affordable healthy foods are likely to accumulate benefits across developmental stages. Therefore, it could be hypothesised that policy changes initiated in childhood and maintained into adulthood could modify trajectories of subclinical atherosclerosis before clinical disease emerges.

Neighbourhood socioeconomic disadvantage and individuals' own socioeconomic status have been linked to markers of subclinical atherosclerosis, such as greater carotid IMT and coronary artery calcification (recently reviewed by Hajj et al. [28]). In many previous studies, however, this association has been attenuated when information on individual socioeconomic status was considered simultaneously, suggesting that the association between neighbourhood disadvantage and cardiovascular risk may reflect compositional influences, as opposed to contextual effects of neighbourhood environments per se. For example, the Atherosclerosis Risk in Communities Study (ARIC) found that lower cumulative neighbourhood socioeconomic status across the life course was associated with greater mean carotid IMT among white participants. This association, however, became statistically non-significant when individual-level socioeconomic was considered simultaneously [8]. Similarly, the Cardiovascular Health Study found that while neighbourhood socioeconomic disadvantage was inversely related to prevalent subclinical disease, these associations did not remain statistically significant after adjustment for own socioeconomic position [9]. In contrast, we found that association between cumulative neighbourhood socioeconomic disadvantage and carotid atherosclerosis was only slightly attenuated after adjustment for individual-level socioeconomic position. The strong independent associations between neighbourhood socioeconomic disadvantage and subclinical atherosclerosis observed in

Table 3
The associations of CVD risk factors and cumulative neighbourhood disadvantage with carotid artery plaques.

Risk factor	Adjustment	N	Plaque count	(95% CI)	RR	(95% CI)
Parental plaque load	Age and sex					
Low		517	0.48	(0.43–0.54)	1.00	
High		508	0.67	(0.60–0.74)	1.38	(1.19–1.61)
CAD PRS	Age and sex*					
Low (<0 SD)		930	0.63	(0.57–0.70)	1.00	
High (>0)		923	0.86	(0.77–0.95)	1.36	(1.22–1.51)
Framingham risk score	Age and sex					
Low (<7.5)		1391	0.61	(0.57–0.65)	1.00	
High (>7.5)		500	0.88	(0.80–0.97)	1.45	(1.28–1.64)
Ideal CVH score	Age and sex					
Ideal or intermediate (3–7)		1028	0.57	(0.52–0.62)	1.00	
Poor (0–2)		649	0.83	(0.76–0.90)	1.45	(1.30–1.63)
Neighbourhood disadvantage**	Age and sex					
Low (<0)		1292	0.63	(0.59–0.68)	1.00	
High (>0)		706	0.80	(0.73–0.87)	1.26	(1.14–1.40)
High	+parental plaque load				1.20	(1.021.40)
High	+polygenic CAD risk score				1.25	(1.12–1.38)
High	+Framingham risk score				1.24	(1.11–1.38)
High	+ideal CVH score				1.15	(1.02–1.29)

CAD PRS; polygenic risk score for coronary artery disease.
* Adjusted additionally for genetic principal components.
** Cumulative disadvantage between mean ages 10.7–48.7 years in relation to the national mean: Low <0, High >0.

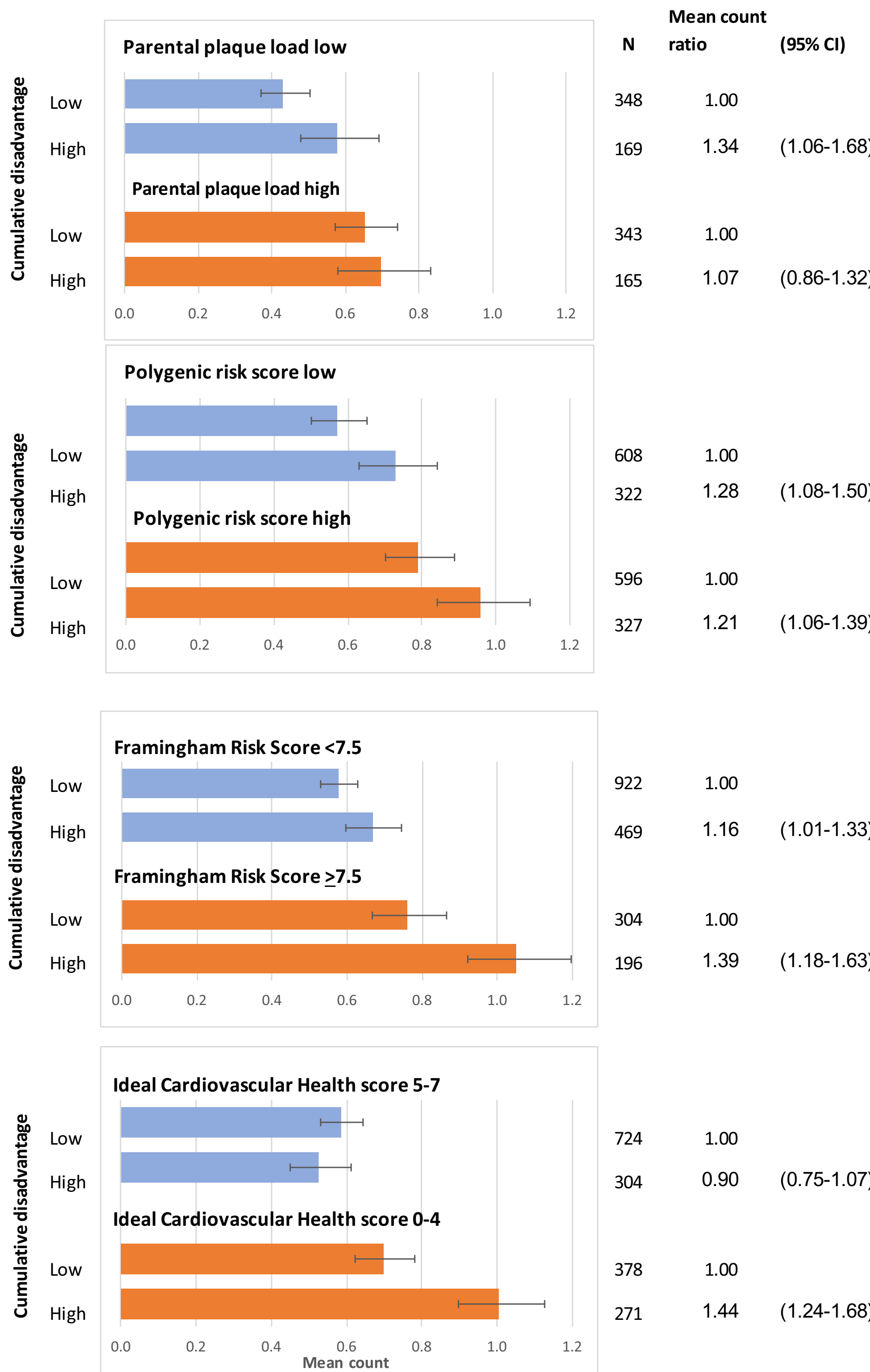

Fig. 1. Life course cumulative neighbourhood disadvantage between mean ages 10.7 (SD 5.0, range 3–18) and 48.7 (SD 5.0, range 41–56) years in relation to the national mean (low <0, high >0) and the mean plaque count by the level of cardiovascular risk factors: parental plaque load, CAR PRS (polygenic risk score for coronary artery disease), Framingham risk and Ideal Cardiovascular Health risk scores. The bars denote plaque counts (mean number of carotid plaques in each subgroup) and error bars their 95% confidence intervals. Numeric table on the right panel shows the mean count ratio.

our study are likely attributable to our ability to capture cumulatively exposures across a nearly 40-year prospective follow-up. However, it is important to acknowledge limitations when comparing our findings with those from U.S.-based cohorts. In studies such as ARIC and the Cardiovascular Health Study, individual-level socioeconomic indicators—particularly educational attainment—may not be directly comparable across due to historical and regional differences in educational access and quality. For example, a proportion of ARIC participants completed their education in regions where racial segregation substantially influenced educational rigor, such that lower educational

Table 4
Total, direct and indirect (through Ideal Cardiovascular Health score components separately and in combination) effect of neighbourhood disadvantage* on the the development of carotid plaques.

		Total effect			Direct effect			Indirect effect			
Mediator	N	RR	Lower	Upper	RR	Lower	Upper	RR	Lower	Upper	% total**
Ideal blood pressure	1995	1.28	1.15	1.41	1.26	1.14	1.39	1.01	1.00	1.03	5.8
Ideal cholesterol	1992	1.28	1.16	1.41	1.27	1.15	1.41	1.01	0.98	1.03	2.1
Ideal glucose	1992	1.28	1.16	1.41	1.28	1.16	1.41	1.00	0.98	1.01	−1.3
Non-smoker	1900	1.26	1.14	1.40	1.22	1.11	1.34	1.04	1.01	1.07	15.2
Physically active	1882	1.24	1.12	1.37	1.22	1.11	1.35	1.01	0.99	1.04	6.2
BMI <25 kg/m2	1998	1.28	1.15	1.41	1.27	1.15	1.40	1.01	0.99	1.03	3.3
Ideal diet	1741	1.23	1.11	1.37	1.22	1.10	1.35	1.01	0.99	1.04	5.6
Ideal score components in combination	1677	1.23	1.10	1.37	1.12	1.03	1.23	1.09	1.02	1.17	43.1
Ideal blood pressure	1903	1.21	1.09	1.35	1.20	1.08	1.33	1.01	1.00	1.03	6.5
Ideal cholesterol	1901	1.21	1.09	1.35	1.20	1.08	1.34	1.01	0.98	1.04	5.5
Ideal glucose	1901	1.21	1.09	1.35	1.22	1.09	1.35	1.00	0.98	1.01	−1.4
Non-smoker	1884	1.22	1.09	1.36	1.19	1.08	1.32	1.02	1.00	1.05	9.5
Physically active	1866	1.21	1.08	1.35	1.20	1.08	1.33	1.01	0.99	1.03	3.6
BMI <25 kg/m2	1905	1.21	1.09	1.35	1.20	1.08	1.34	1.01	0.99	1.03	3.8
Ideal diet	1721	1.19	1.07	1.34	1.18	1.06	1.32	1.01	0.99	1.03	4.8
Ideal score components in combination	1666	1.19	1.06	1.34	1.12	1.01	1.23	1.07	1.00	1.14	36.1

* Cumulative disadvantage between mean ages 10.7–48.7 years in relation to the national mean: Low <0, High >0.
** Percentages mediated of the total effect by the mediator.

attainment may capture structural disadvantage beyond individual socioeconomic position. In contrast, Finland has a relatively uniform and high standard of education, which may reduce heterogeneity in the meaning of educational categories and limit their capacity to attenuate neighbourhood-level associations.

We have previously demonstrated that childhood smoking and dyslipidaemia predict the development of carotid plaque in adulthood [29]. Furthermore, growing evidence supports an association between early risk factor exposure and increased risk of subsequent cardiovascular events later in life [3,4]. Therefore, the harmful effects of neighbourhood socioeconomic disadvantage likely begin to operate early in life. In a cohort of Canadian children, males from disadvantaged neighbourhoods during childhood were more than 2-times more likely to develop a cardiovascular event compared to males from more advantaged neighbourhoods [30]. These observations indicate that living in socioeconomically disadvantaged areas begin to shape health from childhood onwards. Life-style related behaviours (such as smoking initiation, physical activity, and diet) likely play a major role in explaining these links. We have previously shown in this cohort that high neighbourhood socioeconomic disadvantage is characterised by decreased fruit and vegetable intake as early as age 6 years, as well as decreased physical activity, and increased prevalence of daily smoking from adolescence onwards [20]. Many life-style behaviours are moulded early in the life course, as evidences by significant tracking of physical activity [31] and dietary patterns [32] from childhood to adulthood.

The strengths of our study included prospective design with a follow-up of over 38 years from childhood to adulthood and detailed assessment of atherosclerotic carotid artery plaques in adulthood. The possibility to measure neighbourhood socioeconomic disadvantage with objective high-resolution data of all residential locations with dates of moves linked to neighbourhood socioeconomic disadvantage by place and time over the whole follow-up is unique. In other cohorts, neighbourhood disadvantage has been measured either at baseline [33] or with addresses at different age periods [8,21,22,30,34]. In addition, the area unit in our study, a 250 m*250 m grid, is likely to capture the variation in the characteristics of the residential location more accurately than large areas, such as counties1.

A key contribution of this study is the demonstration that neighbourhood-level socioeconomic disparities are associated with the development of subclinical atherosclerosis even in Finland, a country characterised by consistently narrow income inequality. Despite a strong social welfare system and relatively compressed income distributions, meaningful spatial socioeconomic differences remain and are relevant for cardiovascular health. These findings suggest that neighbourhood disadvantage reflects cumulative and contextual exposures not fully captured by national-level measures of inequality. This underscores the importance of socioeconomic environments influencing cardiovascular risk even in comparatively egalitarian societies.

There are some limitations. Since our measurement of neighbourhood socioeconomic disadvantage was limited to three features (education, unemployment, and mean household income), we do not know, what characteristics of neighbourhoods associating with disadvantage could explain the linkage. Potential explanations include, e.g. access to tobacco retail outlets, traffic noise, exposure to peer influence in schools, lack of usable green space and pollution [11,35–37]. The cohort was racially homogeneous (all white Europeans) and done in a single country, which potentially restrict the generalisability of the findings. Finally, non-participation is an inherent problem of longitudinal studies. Despite this, the population who has remained in the study seems to be relatively well representative of the original cohort. In the latest follow-up study, we observed that those who participated were on average 6 months older and more often females than those who did not participate, but similar in terms of baseline (year 1980) cardiovascular risk markers [13].

In conclusion, these longitudinal data show that exposure to neighbourhood socioeconomic disadvantage beginning in childhood is associated with subclinical atherosclerosis in midlife independently of individual socioeconomic status. Given the observational design, causal interpretation is limited. Nonetheless, the findings highlight the potential importance of prevention strategies beginning in childhood that address both neighbourhood environments and health behaviours.

CRediT authorship contribution statement

Olli Raitakari: Writing – original draft, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization. **Jaana Pentti:** Writing – review & editing, Writing – original draft, Formal analysis. **Juhani S. Koskinen:** Writing – review & editing, Methodology, Investigation, Data curation. **Juha Mykkänen:** Writing – review & editing, Project administration, Methodology, Investigation. **Suvi Rovio:** Writing – review & editing, Project administration, Methodology, Investigation, Funding acquisition. **Katja Pahkala:** Writing – review & editing, Project administration, Investigation. **Markus Juonala:** Writing – review & editing, Methodology. **Terho Lehtimäki:** Writing – review & editing. **Mika Kähönen:** Writing – review & editing. **Ichiro Kawachi:** Writing –

review & editing. **Mika Kivimäki:** Writing – review & editing. **Jorma Viikari:** Writing – review & editing, Project administration, Investigation, Funding acquisition. **Jussi Vahtera:** Writing – original draft, Investigation, Formal analysis, Conceptualization.

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Declaration of competing interest

The authors have nothing to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2026.134216.

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