Socioeconomic Status and Carotid Atherosclerosis

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Background There is a consistent body of evidence that socioeconomic status (SES) is importantly associated with cardiovascular morbidity and mortality. However, little information currently exists on the relationship between SES and early manifestations of atherosclerotic vascular disease.

Methods and Results We investigated the association between education, income, and occupation and intima-media thickness (IMT) in a population-based sample of eastern Finnish men. Data from the Kuopio Ischemic Heart Disease Risk Factor Study were used to estimate mean IMT across levels of SES in 1140 men. The association between SES and IMT was examined in relation to atherosclerotic risk factors and was also stratified by degree of atherosclerotic progression and prevalent cardiovascular disease. There were significant, inverse, graded differences between levels of SES and IMT. For education, the age-adjusted mean IMTs for those with primary schooling or less, some high school, and completed high school or more were 0.96, 0.94, and 0.82 mm, respectively. The difference in mean IMT between the most extreme categories of education corresponds to a 15.4% increase in the risk of myocardial infarction. Similar patterns were found for each measure of SES, although the differences between the highest and lowest levels of SES were attenuated by adjustment for risk factors. In men who had no carotid stenosis or nonstenotic plaque and in men who had no indication of prevalent cardiovascular disease, a graded, inverse association between SES and IMT persisted, even after risk factor adjustment.

Conclusions These findings demonstrate a strong association between SES and atherosclerosis in an unselected population. The results show that this association was mediated by known atherosclerotic risk factors, was evident in the early stages of atherosclerosis, and was apparent in a healthy subgroup. Our findings suggest that the impact of SES is evident early in the natural history of atherosclerotic vascular disease. (***Circulation.* 1995;92:1786-1792.**)

Key Words • atherosclerosis • economics • carotid arteries

There is a large and consistent body of evidence demonstrating that SES is importantly associated with a wide variety of health outcomes.[1-4] Regardless of which measures of SES are used, an inverse gradient, with increased SES associated with lower rates of disease or death, is found. Some of the strongest data on this association come from studies of CVD, in which SES has consistently been shown to predict morbidity and mortality.[5-9]

Although the existence of this association has been reported extensively, there is little understanding of the reasons for the relationship between SES and CVD. Although factors related to smoking, diet, alcohol consumption, and physical activity are often suggested to be the pathways by which SES is related to increased risk of CVD, the available evidence indicates that they provide at best partial explanations.[10-13] Furthermore, as Kaplan and Keil[5] point out, if SES is associated with the acquisition and maintenance of such lifestyle factors, those factors cannot be said to be the causes of the association.

Our understanding of the mechanisms by which SES is associated with CVD is hampered by the analysis of outcomes such as myocardial infarction that occur late in the natural history of the disease.[14] Although greater appreciation of how SES is related to CVD might be gained by examining earlier stages of the disease process, little information of this type is available.

The relatively recent advances in ultrasound scanning of the carotid arteries have provided opportunities to study noninvasively the prevalence, development, and risk factors for atherosclerosis within unselected human populations.[15,16] Ultrasound assessments of IMT of the carotid arteries have been shown to be reliable, to be related to general and coronary atherosclerosis, and to have predictive validity with regard to risk of coronary events.[16-19] The present study examined the association between SES and IMT in a population-based sample of eastern Finnish men.

Methods

Subjects were participants in the KIHD, which was designed to investigate previously unestablished risk factors for ischemic heart disease and carotid atherosclerosis in a population-based sample of eastern Finnish men.[20] Of the 3433 eligible men 42, 48, 54, or 60 years old who resided in the town of Kuopio or its surrounding rural communities, 198 were not included because of death, serious disease, or migration away from the area, and of the remainder, 2682 (82.9%) agreed to participate in the study. Baseline examinations were conducted between March

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Selected Abbreviations and Acronyms

BMI = body mass index
BP = blood pressure
CVD = cardiovascular disease
IMT = intima-media thickness
KIHD = Kuopio Ischemic Heart Disease Risk Factor Study
SES = socioeconomic status

1984 and December 1989. No marked sociodemographic differences have been found between participants and nonparticipants.[21]

Subjects were recruited in two waves. The first group comprised 1166 men 54 years old, and the second comprised an age-stratified sample of 1516 men 42, 48, 54, or 60 years old. Ultrasonographic assessment of carotid atherosclerosis was conducted between February 1987 and December 1989, on the second wave of participants only. There are no systematic differences between the two waves of recruitment for the study other than the differences in age. Of the group who underwent ultrasonographic examination, information on IMT, SES, and all other covariates was available for between 1116 and 1140 men (depending on which SES measure was used). The numbers of participants in each age cohort were 247, 253, 326, and 314.

Evaluation of Carotid Atherosclerosis

IMT was assessed with B-mode ultrasonographic scanning of the common carotid artery and the carotid bifurcation with the ATL UM4 duplex ultrasound system (Advanced Technology Laboratories). The KIHD B-mode scanning protocol involved locating the site of the most advanced lesion and the projection revealing the greatest distance between the lumen-intima and media-adventitia interfaces in the right and left common carotid arteries and the carotid bifurcation/bulb area. IMT was measured from VCR recordings of the entire scanning process by the same physician. Three measurements of IMT were carried out for the far wall of both the right and left common carotid arteries at the site of the greatest thickening in each recording and in each vessel. The mean of these six measurements was used in all analyses. Details of the protocol for the IMT measures have been described elsewhere.[22-24]

The continuous measures of IMT were also classified into four categories: (1) no atherosclerotic lesion, (2) IMT, (3) nonstenotic plaque, and (4) large, stenotic plaque. IMT was defined as >1.0 mm between the lumen-intima interface and the media-adventitia interface in the common carotid arteries below the carotid bulb. The atherosclerotic lesion (category 3) was defined as plaque when a distinct area could be identified with mineralization or with focal protrusion into the lumen. A plaque was defined as stenotic if it obstructed >20% of the lumen diameter.[16]

Evaluation of SES

A variety of measures of SES were available from questionnaires completed as part of the baseline examination.[25] The present study reports results using three measures: highest educational attainment, current income, and lifetime occupation. The highest level of education was classified into three categories: "primary school" included those who had only part of or who had completed primary school (n=579), "some high school" included those who had some high school or other vocational training beyond primary (n=471), and "completed high school or better" included those who had finished high school or who had tertiary education (n=103). The respondents' current income was divided into quartiles. Subjects reported the occupation that they had held for the longest period in their lives. This was categorized as "blue collar" (n=499), "farmer" (n=129), or "white collar" (n=503).

Evaluation of Risk Factors

Plasma fibrinogen concentration was determined from fresh samples based on clotting of diluted plasma with excess thrombin with the Coagulometer KC4 device (Heinrich Amelung GmbH).[26] Lipoproteins were separated from unfrozen plasma within 3 days of sampling. HDL and LDL fractions were separated from fresh plasma by both ultracentrifugation and precipitation. The cholesterol contents of all lipoprotein fractions were measured enzymatically (CHOD-PAP cholesterol method, Boehringer Mannheim) on the day after the last spin. Serum apolipoprotein B was determined with an immunoturbidimetric method (KONE Corp) using an antiserum (Orion).[27] BP was measured with a random-zero sphygmomanometer after a supine rest of 5 minutes. Three systolic and diastolic BPs were taken with the patient in the supine position and were averaged. Average systolic BP was used in this analysis. BMI was calculated by dividing the subject's weight by the square of his height (kg/m^2). Cardiorespiratory fitness was measured directly on the basis of respiratory gas exchange during a maximal symptom-limited exercise tolerance test on a bicycle ergometer.[28]

Alcohol consumption was assessed by dietary interview for a 4-day period and also for the previous 12 months by self-administered questionnaire.[29] Smoking was measured by questionnaire and classified for this analysis as "never smoked," "former smoker," "irregular smoker," and "current smoker" (measured in pack-years). Physical activity was assessed from a 12-month leisure-time history. These analyses used the total duration of conditioning physical activity, which has been shown to be predictive of myocardial infarction in this population.[28]

Family history of atherosclerotic disease was assessed by questionnaire. Subjects were considered to have a positive family history if either their parents or any siblings had a record of CVD, hypertension, or stroke. Treatment for hypertension or hyperlipidemia was assessed by a review of medications. Presence of diabetes at baseline was assessed by both self-reported previous physician diagnosis and by fasting blood glucose levels ≥6.7 mmol/L.

Evaluation of Prevalent Disease

Subjects were considered to have prevalent disease if they (1) had any history of prior myocardial infarction, angina pectoris, cardiomyopathy, congestive heart failure, functional heart problems, claudication, stroke, hypertension, or any other CVD; (2) currently used antiangina medication, nitroglycerin, or hypertension medication; (3) had a history of angiography; (4) had positive findings from the Rose questionnaire[30]; or (5) showed evidence of ischemia on exercise or terminated the exercise test because of chest pain.

Statistical Methods

The association between SES and IMT was assessed by estimating mean values of IMT at each level of SES for education, income, and occupation. The analyses were conducted with the GLM procedure in SAS version 6.09 on a Sun Sparc station II.[31] This procedure allows age-adjusted, least-squares mean values of IMT to be estimated and contrasted for each level of SES while potential confounders are simultaneously controlled for.

Results

Table 1 provides mean±SD and prevalence (%) for the potential confounders distributed across categories of SES. Table 2 presents estimated mean IMT values by SES with adjustment for age (model 1) and with additional adjustment for potential confounders (fibrinogen, HDL, serum apolipoprotein B, systolic BP, smoking, BMI, alcohol consumption, cardiorespiratory fitness,

TABLE 1. Mean±SD or Prevalence (%) of Potential Confounders by Level of Highest Educational Attainment, Quartiles of Income, and Longest-Held Occupation

	Highest Education			Income Quartiles			
	≤Primary School (n-52)	Some High School (n=465)	≥High School (n=93)	Lowest (n=274)	Second (n=281)	Third (n=298)	Highest (n=265
Age, y	52.8±6.3	50.7±6.9	50.3±6.4	54.4±6.2	52.0±6.6	50.2±6.6	50.2±6.2
Fibrinogen, g/L	3.06±0.55	3.01±0.58	2.85±0.51	3.17±0.61	3.03±0.56	3.02±0.51	2.88±0.53
ApoB, mg/L×10	104.5±23.5	100.0±24.5	101.1±25.4	104.5±25.2	103.5±23.6	102.3±23.5	98.8±23.7
HDL, mmol/L	1.30±0.31	1.31±0.30	1.22±0.26	1.29±0.32	1.29±0.30	1.29±0.27	1.32±0.30
Physical activity, h/wk	2.2±2.8	2.3±2.5	1.9±1.8	2.6±3.4	2.1±2.7	1.9±1.9	2.2±2.0
BMI, kg/m^2	26.83±3.45	26.71±3.36	26.48±3.86	26.61±3.73	26.92±3.34	26.75±3.38	26.73±3.33
Systolic BP, mm Hg	130.4±17.5	128.5±16.6	128.6±16.3	130.3±18.8	129.9±17.0	128.4±15.7	129.2±16.3
Alcohol consumption, %							
Abstainers	14.9	11.0	6.5	18.6	12.8	11.1	7.5
Lowest 25%	21.8	20.0	23.7	24.1	21.7	19.8	18.5
Second quartile	21.3	21.1	22.6	17.9	19.9	23.2	25.3
Third quartile	18.7	23.7	24.7	19.3	16.7	22.1	25.7
Highest 25%	23.2	24.3	22.6	20.1	28.8	23.8	23.0
Smoking, %							
Never	23.2	30.3	32.3	22.6	21.7	26.8	36.2
Former	40.0	41.1	38.7	38.3	40.6	40.9	41.5
Irregular	3.4	2.8	4.3	3.3	3.2	3.4	3.0
First tertile (pack-years)	7.4	9.0	8.6	7.3	9.6	9.1	6.4
Second tertile	13.4	10.1	8.6	17.5	12.5	10.4	6.8
Third tertile	12.5	6.7	7.5	10.9	12.5	9.4	6.0
Treatment for, %							
Hyperlipidemia	0.9	0.4	1.1	1.8	0.4	0.7	0.4
Hypertension	23.7	20.9	14.0	30.7	22.4	19.1	15.1
Prevalent diabetes, %	2.2	3.9	3.2	3.6	3.9	1.3	3.0
Family history of atherosclerotic disease, %	74.7	79.8	86.0	77.4	71.9	79.5	82.6
Prevalent CVD, %	62.2	58.7	50.5	69.3	61.6	56.7	50.2
Prevalent carotid stenosis or nonstenotic plaque, %	39.8	27.9	12.9	45.6	30.6	28.8	23.8

Apo B indicates serum apolipoprotein B; Physical activity, total duration of conditioning physical activity.

physical activity, history of diabetes, family history of atherosclerotic disease, and treatment for hyperlipidemia and hypertension) in model 2.

SES and IMT in the Whole Sample

The mean age-adjusted IMT within categories of education was 0.96 mm for those who had only primary education, 0.94 mm for those with some high school, and 0.82 mm for those who had completed high school or continued with tertiary studies. The age-adjusted mean IMT for those who had completed high school was 15% lower than those with only primary education and 13% lower than those with only part of high school ($P<.01$). After controlling for potential confounders, the mean IMTs were 0.95, 0.95, and 0.83 mm for the three levels of education, respectively. The mean IMT for the highest level of education remained significantly lower than the others ($P<.01$).

When income was used as the measure of SES, a 9% difference in age-adjusted mean IMT was observed between the highest (0.97 mm) and lowest (0.89 mm) quartiles. This difference was attenuated by adjustment for potential confounders. For occupation, a 6% difference between age-adjusted mean IMT was found between blue-collar (0.97 mm) and white-collar (0.91 mm) workers, which was also reduced after adjustment for potential confounders.

SES and IMT in Those Without Advanced Thickening

Table 3 shows the results of similar regression models of the relationship between mean IMT, education, income, and occupation in a subgroup that excluded 374 men with ultrasonographic evidence of ≥20% carotid stenosis or nonstenotic atherosclerotic plaque (categories 3 and 4). The mean IMT was 0.84 mm for those who had only primary education, 0.81 mm for those with some high school, and 0.75 mm for those who had completed high school or gone on to tertiary studies. Contrasts between these age-adjusted mean IMTs showed that those with a high school education or better had an 11% lower mean IMT compared with those who had only a primary school education ($P<.001$). After potential confounders were accounted for, the mean IMTs were 0.83, 0.81, and 0.75 mm for the three levels of education, respectively. The difference observed in the age-adjusted model between the highest and lowest levels of educational attainment was maintained ($P<.01$).

When income was used as the measure of SES, an 11% difference in age-adjusted mean IMT was observed between the highest (0.84 mm) and lowest (0.76 mm) quartiles ($P<.005$). The magnitude of this difference was somewhat reduced after adjustment for potential confounders ($P<.05$). For occupation, a 7% difference be-

TABLE 1. (Continued)

Occupation		
Blue Collar (n-491)	Farmer (n=137)	White Collar (n-488)
51.4±6.7	53.7±6.1	51.04±6.5
3.04±0.56	3.09±0.50	2.99±0.58
103.9±24.0	102.9±23.7	100.4±24.4
1.29±0.30	1.33±0.32	1.30±0.30
2.2±2.8	1.4±1.9	2.4±2.5
26.74±3.44	26.76±3.78	26.71±3.38
129.4±16.3	131.5±19.0	128.8±17.0
14.1	23.4	8.0
18.3	25.5	22.0
19.6	24.8	22.5
20.4	15.3	23.8
27.7	10.9	23.6
22.6	33.6	29.3
39.5	33.6	42.2
2.0	4.4	4.3
8.1	7.3	8.8
15.7	10.9	8.0
12.0	10.9	7.4
0.2	2.9	0.8
23.4	19.7	20.5
2.6	2.9	3.5
74.5	79.6	80.9
58.9	62.8	59.0
33.8	45.8	27.1

tween mean age-adjusted IMT was found between blue-collar (0.84 mm) and white-collar (0.78 mm) workers, which maintained statistical significance even after adjustment for potential confounders ($P<.01$).

SES and IMT in Those Without Prevalent CVD

Table 4 shows the results of regression models of the relationship between mean IMT, education, income, and occupation in a subgroup that excluded 679 men with prevalent CVD. The mean IMT was 0.90 mm for those who had only primary education, 0.86 for those with some high school, and 0.77 for those who had completed high school or gone on to tertiary studies. Contrasts between these age-adjusted mean IMTs showed that those with a high school education or better had a 14% lower mean IMT than those who had only a primary school education ($P<.02$). After potential confounders were accounted for, the difference in mean IMTs was slightly reduced but did not reach statistical significance ($P<.07$).

When income was used as the measure of SES, an 8% difference in age-adjusted mean IMT was observed between the highest (0.91 mm) and lowest (0.84 mm) quartiles. The magnitude of this difference was unchanged after adjustment for potential confounders. For occupation, a 9% difference between mean age-adjusted IMTs was found between blue-collar (0.91 mm) and white-collar (0.83 mm) workers ($P<.02$). This difference was unaffected by adjustment for potential confounders ($P<.04$). The Figure shows the age-adjusted mean IMTs across categories of education within the whole sample and in the subsamples of men without advanced atherosclerotic thickening and those who were free of prevalent CVD.

TABLE 2. Mean±SEM IMT and Education, Income, and Occupation in Finnish Men

		Model 1, Age Adjusted		Model 2, Age and Risk Factor Adjusted†	
	n	Mean±SEM	[Diff]*	Mean±SEM	[Diff]*
Education					
a. Primary	1140	0.96±0.016	[c]	0.95±0.016	[c]
b. Some high school		0.94±0.018	[c]	0.95±0.018	[c]
c. Completed high school		0.82±0.040		0.83±0.039	
Income					
a. Lowest 25%	1118	0.97±0.024	[d]	0.94±0.024	
b. Second quartile		0.95±0.023		0.95±0.023	
c. Third quartile		0.94±0.022		0.95±0.022	
d. Highest 25%		0.89±0.024		0.91±0.024	
Occupation					
a. Blue collar	1116	0.97±0.017	[c]	0.96±0.017	
b. Farmer		0.94±0.033		0.94±0.033	
c. White collar		0.91±0.017		0.92±0.017	

*[Diff] indicates differences between mean IMTs, which are significant at $P<.05$; eg, [c] indicates that the corresponding level of SES is significantly different from level c.

†Adjusted for fibrinogen, HDL, apolipoprotein B, smoking, alcohol, BMI, systolic BP, physical activity, cardiorespiratory fitness, family history of atherosclerotic disease, prevalent diabetes, treatment for hypertension, or hyperlipidemia.

Discussion

These results show that those who have less education or lower incomes or are employed in blue-collar or farming work have greater carotid atherosclerosis. For each measure of SES, an inverse, graded age-adjusted

TABLE 3. Mean±SEM IMT and Education, Income, and Occupation in a Subsample of Finnish Men Without Carotid Stenosis or Nonstenotic Atherosclerotic Plaque

		Model 1, Age Adjusted		Model 2, Age and Risk Factor Adjusted†	
	n	Mean±SEM	[Diff]*	Mean±SEM	[Diff]*
Education					
a. Primary	766	0.84±0.012	[c]	0.83±0.012	[c]
b. Some high school		0.81±0.012	[c]	0.81±0.012	[c]
c. Completed high school		0.75±0.025		0.75±0.025	
Income					
a. Lowest 25%	758	0.84±0.018	[d]	0.83±0.019	[d]
b. Second quartile		0.83±0.016	[d]	0.83±0.016	[d]
c. Third quartile		0.83±0.015	[d]	0.83±0.015	[d]
d. Highest 25%		0.76±0.016		0.77±0.016	
Occupation					
a. Blue collar	753	0.84±0.012	[c]	0.83±0.012	[c]
b. Farmer		0.86±0.026	[c]	0.85±0.026	[c]
c. White collar		0.78±0.012		0.79±0.11	

Definitions as in Table 2.

TABLE 4. Mean±SEM IMT and Education, Income, and Occupation in a Subsample of Finnish Men With No Prevalent CVD

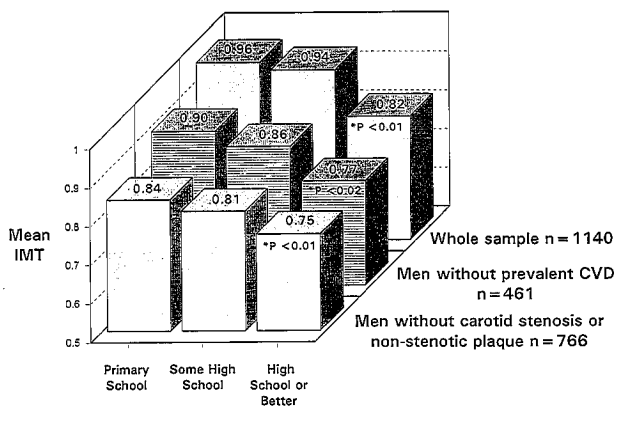
		Model 1, Age Adjusted		Model 2, Age and Risk Factor Adjusted†	
	n	Mean±SEM	[Diff]*	Mean±SEM	[Diff]*
Education					
a. Primary	461	0.90±0.023	[c]	0.89±0.023	
b. Some high school		0.86±0.024		0.86±0.024	
c. Completed high school		0.77±0.049		0.79±0.050	
Income					
a. Lowest 25%	453	0.91±0.037		0.91±0.039	
b. Second quartile		0.87±0.032		0.87±0.033	
c. Third quartile		0.88±0.030		0.87±0.030	
d. Highest 25%		0.84±0.030		0.84±0.030	
Occupation					
a. Blue collar	453	0.91±0.023	[c]	0.90±0.023	[c]
b. Farmer		0.88±0.046		0.90±0.048	
c. White collar		0.83±0.023		0.83±0.024	

Definitions as in Table 2.

relationship existed between the level of SES and the extent of IMT.

In the analysis that used the entire sample, when potential confounding variables were included, the mean IMT values were attenuated and the graded relationship between SES and IMT was reduced. Variables that remained significant predictors of IMT in multivariate models were age, systolic BP, fibrinogen, smoking, serum apolipoprotein B, treatment for hyperlipidemia, prevalent diabetes, and education.

This observation suggests that the association between SES and IMT may be mediated by known risk factors. According to this line of argument, the relationship between SES and IMT would therefore be "explained" by these risk factors, and SES would not be "independently" associated with IMT. However, it is also important to recognize that these risk factors are differentially distributed in the population according to SES (Table 1).



Bar graph showing age-adjusted mean levels of IMT and highest level of education in the whole sample of Finnish men (n=1140), in a subgroup without prevalent CVD (n=461), and in a subgroup without carotid stenosis or nonstenotic plaque (n=766). *Comparison between mean IMTs for primary school and high school or better.

If lower SES acts as a powerful force in the adoption of poor diet or influences behaviors such as smoking and physical activity, then this explanation for the relationship between SES and IMT is limited to understanding the mechanisms through which SES affects IMT. Although it is important to discover how SES is related to atherosclerotic vascular disease, it still does not address why SES is implicated in the adoption and maintenance of the risk factors that mediate the association between SES and IMT. In addition, it should be noted that drawing conclusions about the independence of particular risk factors from multivariate models is at best difficult.[32,33] When multiple predictors are measured with error, estimates of the association between any single predictor and the outcome can be biased in either direction.[34]

To test whether the impact of SES was evident early in the progression of atherosclerosis, we excluded all men who had evidence of carotid stenosis or nonstenotic plaque. Graded, inverse relationships were observed in this subgroup. The magnitude of the differences between extreme categories was also similar to that observed in the whole sample, and these differences remained statistically significant after adjustment for risk factors.

To examine whether the association between levels of SES and IMT was only a reflection of the greater likelihood that those who had lower SES would have higher rates of prevalent CVD, we repeated the analysis in a healthy subsample. All men who had any indication of prevalent CVD were excluded from the analysis. In this healthy subsample, the same graded, inverse relationship between SES and IMT was observed, and the magnitude of the differences associated with the most extreme categories of SES was similar to those obtained within the whole sample.

Although we believe that these results demonstrate an important relationship between SES and the early signs of atherosclerotic vascular disease, it is important to consider their relationship to coronary atherosclerosis. There are a number of reasons to believe that carotid atherosclerosis is a good model for coronary atherosclerosis.[35] Carotid atherosclerosis as measured by B-mode ultrasound imaging of the intima-media has been shown to be strongly associated with coronary atherosclerosis measured angiographically,[36] to be correlated with the degree of coronary atherosclerosis in autopsy studies,[37] to share the same risk factors as coronary atherosclerosis,[38,39] and to be predictive of future coronary events.[16]

These findings are important for at least two reasons. First, our results show a strong relationship between SES and the early signs of atherosclerotic vascular disease in an unselected sample, and they point to the significant role that SES plays in the early development of atherosclerosis, in addition to its better-known association with the clinically relevant and distal manifestations of CVD. The results suggest that SES is associated with the early stages of atherosclerotic vascular disease. Furthermore, the IMT differences between extreme categories of SES observed in these data are of public health significance. Salonen and Salonen[16] demonstrated within the same population that for a 0.1-mm increase in IMT, the risk of acute myocardial infarction rose by 11% (95% CI, 6% to 16%).

Second, these results demonstrate that the relationship between lower SES and increased IMT is graded across levels of SES, both early in the development of atherosclerosis and among healthy individuals. These findings, therefore, do not support the "drift" or "selection" hypothesis that has been proposed as one explanation for the reported relationships between SES and health outcomes.[40,41] This argument contends that any observed association between SES and health reflects the fact that sick individuals "drift down" the social hierarchy, so that measures of SES are really only proxies for levels of illness. Against this argument, we found significant, graded differences among levels of education, income, and occupation in a healthy subgroup of this population who had no indicators of prevalent CVD.

Finally, because the analyses presented here are cross-sectional in nature, it is important to consider potential biases associated with such analyses. The most important bias relates to the possibility that existing disease might result in lowered SES. Certainly, for the case of income this is tenable. However, the fact that SES-IMT associations were also found for education and lifetime occupation and that strong associations were found early in the manifestation of atherosclerosis and in the healthy subsample would seem to indicate that such a bias is not significant in the overall pattern of these results.

In summary, the evidence presented indicates that the impact of SES is evident early in the natural history of atherosclerotic vascular disease and is not only limited to affecting more distal manifestations of CVD. Precisely how early these influences begin is not known, but other evidence from the KIHD indicates that low SES early in life does not invariably lead to increased risk of CVD in middle age.[25] Kaplan and Keil,[5] in their review of the literature on the association between SES and CVD, argue that studies that examine the impact of SES over the entire lifetime will be necessary to identify and understand the ways in which SES may lead to increased risk of adult CVD.

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