

# Community Socioeconomic Status Is Associated With Carotid Artery Atherosclerosis in Untreated, Hypertensive Men

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**Background:** Individuals of low socioeconomic status (SES) are at increased risk for cardiovascular disease relative to persons of more advantaged social position. Recent evidence suggests that community SES (variation in the material and social resources of communities) also predicts incident cardiovascular disease, and may do so independently of individual level SES. In this study we examined whether community SES is similarly associated with preclinical vascular disease, as measured by carotid artery ultrasonography.

**Methods:** Subjects were 230 untreated hypertensive men without history of coronary heart disease, residing in and around Pittsburgh, PA (80% white, 20% African-American; mean age, 56 years). Community SES was defined by levels of income, economic disadvantage (eg, poverty, unemployment), housing costs, and educational attainment in the US Census tract of residence for each subject. A composite community SES score was calculated for each subject as the average of all extracted census measures.

Individual SES was estimated from subjects' years of education and current annual income.

**Results:** Regression analyses accounting for traditional risk factors showed community disadvantage to be associated with greater intima–medial thickness ( $b = 0.02$ ,  $P < .05$ ) and plaque occurrence (odds ratio [OR] = 1.51,  $P < .01$ ). The latter association persisted on multivariable adjustment for both risk factors and individual markers of social position (SES) (OR = 1.68,  $P < .01$ ).

**Conclusions:** Irrespective of one's own income or educational attainment, untreated hypertensive men living in poorer communities are more likely to exhibit preclinical atherosclerosis than residents of more affluent areas. Am J Hypertens 2006;19:560–566 © 2006 American Journal of Hypertension, Ltd.

**Key Words:** Community, socioeconomic status, SES, carotid artery atherosclerosis, IMT.

Several recent epidemiologic studies show that inequalities in the economic and social resources of communities predict cardiovascular morbidity and mortality.<sup>1–9</sup> Unlike personal income, education, or occupational grade, which likewise predict heart disease, the socioeconomic status (SES) of communities is ordinarily inferred from demographic features of population aggregates, such as census tracts or postal districts.<sup>2,9</sup> Frequently reported indicators of community SES include area rates of unemployment and poverty, distribution of educational attainments, rates of vehicle or home owner-

ship, costs of housing, and median household income. Although reflecting diverse neighborhood attributes, such variables tend to correlate strongly across communities and often reveal a common underlying dimension of neighborhood "quality."<sup>10</sup>

Many characteristics of disadvantaged neighborhoods might plausibly enhance cardiovascular risk, such as restricted access to health care, social norms promoting health-impairing attributes of habit or lifestyle, and the stress of living in areas of crime, physical risk, and uncertainty.<sup>2,10</sup> Neighborhood quality also covaries inversely

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with prominent risk factors for heart disease, including smoking and alcohol consumption, physical inactivity, body weight, blood pressure (BP), and cholesterol concentrations.<sup>4,8,11–13</sup> Yet interestingly, multivariable analyses often show community “influences” on incident cardiovascular disease and mortality to persist after adjusting for standard risk factors or concomitant variability in individual SES (see also refs. 6 and 7).<sup>1–4,8</sup> How the socioeconomic status of communities might contribute to heart disease is also poorly understood. For instance, it is unknown whether community attributes affect disease-relevant pathophysiologic processes, such as the progression of underlying vascular changes or events surrounding symptomatic expression of coronary disease (eg, myocardial ischemia and infarction, arrhythmogenesis). Although one recent study of elderly adults found community quality associated with subclinical atherosclerosis—defined by a composite index that included carotid wall thickness, a positive Rose questionnaire, ankle–arm disparity of BP, and electrocardiogram (ECG) or echocardiographic abnormalities—this finding was no longer statistically significant when adjusted for traditional biologic risk factors and individual markers of SES.<sup>14</sup> It is conceivable, however, that community inequalities promote vascular disease more strongly among people who are already at increased risk for atherosclerosis due to other risk factors or predisposing conditions, such as poorly controlled hypertension. The purpose of the present study was to determine whether variation in the community SES of untreated hypertensive men is associated with preclinical atherosclerosis, as assessed by ultrasound evaluation of carotid artery intima-media thickness (IMT) and plaque occurrence.

## Methods

### Participants

Subjects were 40- to 70-year-old men enrolled in the University of Pittsburgh REACT project, a study of biobehavioral correlates of carotid artery atherosclerosis among untreated hypertensives, conducted from 1994 to 1996, and described in several prior reports.<sup>15,16</sup> Subjects were residents of Southwestern Pennsylvania communities who answered mail solicitations or media advertisements seeking volunteers with a currently untreated BP elevation. Respondents were then screened by study staff for BP, exclusionary conditions, and medical history. Exclusions included use of antihypertensive medication within the 2 months before enrollment; hypertension treatment of >1 year during the 5 years preceding enrollment or >2 years ever (85% of participants had no history of antihypertensive therapy; among the remainder, median length of treatment was 2 months). Other exclusions included angina pectoris; myocardial infarction or angioplasty in the preceding 12 months; congestive heart failure, valvular heart disease, atrial fibrillation, renal insufficiency, suspected secondary hypertension, stroke, neurologic disorders, pulmonary disease, clinically apparent coronary artery dis-

ease, or cancer; use of any psychotropic or glucocorticoid medications; coronary revascularization, or carotid or peripheral vascular surgery. Diabetics were excluded if reporting peripheral neuropathy, use of insulin, or a fasting serum glucose concentration >200 mg/dL. Hypertensive status was confirmed by two seated resting BP measurements averaging 140 to 180 mm Hg systolic BP and/or 90 to 110 mm Hg diastolic BP on each of two screening sessions. The study protocol was approved by the University of Pittsburgh Biomedical Institutional Review Board, and informed consent was obtained from study participants.

Volunteers meeting the foregoing criteria included 251 hypertensive men.<sup>15,16</sup> The analyses are based on 230 participants for whom a census tract of residence could be identified retrospectively, as derived from the 1990 US Census.<sup>18</sup> This sample included 47 African American and 183 white men. Subjects averaged 14.8 years of education (SD = 2.8; range, 9 to 25 years) and, at the time of participation (1994 to 1996), reported pretax family income in the ranges of <\$10,000 (coded 1), \$10,000 to \$19,999 (coded 2), \$20,000 to \$34,999 (coded 3), \$35,000 to \$50,000 (coded 4), >\$50,000 (coded 5), with a mean of 3.8 (SD = 1.3). As in prior research,<sup>19</sup> we derived a composite index of SES (here termed individual SES), based on income and education. For this purpose, the two index variables—years of education and family income—were standardized across all participants and the corresponding z-scores averaged for each subject. The resulting distribution was then restandardized and multiplied by –1 so that high scores denote greater socioeconomic disadvantage (ie, less income, fewer years of schooling) (M = 0, SD = 1; range, –2.69 to 2.72).

### Community Socioeconomic Status

Census tracts comprise population aggregates encompassing about 4000 individuals (range, 2500 to 8000 individuals) and 1500 housing units (range, 1000 to 3000 housing units). Subjects resided in 159 unique census tracts; 72% of the tracts contained a single study participant, and all but one tract housed three or fewer subjects. Census variables reflecting area-level educational and material resources were selected for analysis to create an index of community SES analogous to individual-level SES. These variables included: 1) median household income; 2) percentage of households on public assistance; 3) percentage of households beneath the federally designated poverty level; 4) percentage of adults in the work force who were unemployed; 5) median values of owner-occupied housing units; 6) median gross rent, as a percentage of household income; and 7) proportion of residents greater than age 25 years lacking a high school diploma. To normalize raw score distributions, logarithmic transformations were applied to all variables before analysis.

Following Manuck et al,<sup>10</sup> census variables were subjected to Principal Components Analysis, retaining any

factor of eigenvalue  $>1$ . All variables loaded strongly on a single factor (0.57 to 0.96; median factor loading = 0.90), accounting for 74% of total variance. To reflect this common variance in community SES, z-scores for the Census variables (adjusted for direction of factor loading) were then averaged for each participant. As with Individual SES, this continuous index of community SES was then restandardized and multiplied by  $-1$  to yield a distribution in which higher scores denote greater socioeconomic disadvantage ( $M = 0$ ,  $SD = 1$ ; range,  $-2.74$  to  $3.03$ ).

### Carotid Atherosclerosis

Intima-media thickness and carotid plaque were assessed using B-mode ultrasonography at the Department of Epidemiology ultrasound research laboratory at the University of Pittsburgh. A Toshiba SSA-270 (Toshiba, Nasu, Japan) scanner equipped with a 5-Mhz linear-array probe was used to image the right and left common carotid artery (CCA) (1 cm proximal to the carotid bulb), carotid bifurcation (starting at the point where the near and far walls are no longer parallel and ending at the flow divider), and the first centimeter of the internal carotid artery (ICA). Digitized images from each segment were used to assess mean IMT as defined by the adventitia-media and intima-lumen interfaces. This was done for the near wall of the CCA and the far walls of the CCA, bulb, and ICA. The IMT measurements were averaged over all segments to create a mean IMT. Carotid measurements did not differ significantly between white and African-American hypertensives (white IMT  $M = 0.91$  [ $SD = 0.16$ ], African-American IMT  $M = 0.91$  [ $SD = 0.13$ ],  $t_{228} = 0.18$ ,  $P =$  not significant). Subjects' IMT scores were subsequently normalized by reciprocal transformation. For ease of interpretation the sign of test statistics is reversed in analyses reported later.

For each segment, plaque was graded from 0 to 3 based on the number and size of plaques.<sup>20</sup> These grades were summed to create the plaque index, which ranged from 0 to 12. Because of the skewed distribution of this variable, index scores were dichotomized as either "high" (plaque index  $>1$ ) or "not high" (plaque index  $\leq 1$ ). Like IMT, frequency of plaque scores did not differ between whites and African-Americans (51.9% and 51.1%, respectively;  $\chi^2_1 = 0.01$ ,  $P =$  not significant).

### Cardiovascular Risk Factors

Standard cardiovascular risk factors<sup>21</sup> were measured for potential inclusion in multivariable analyses. These included age (years), body mass index (BMI, in kilogram per meters squared), ethnicity, systolic and diastolic BP, smoking status (current versus ex/nonsmoker), fasting serum glucose, and the ratio of total cholesterol-to-HDL-cholesterol concentration (details of assays reported elsewhere<sup>17</sup>).

### Data Analysis

We first present descriptive statistics for predictor variables and covariates, along with correlations among risk factors, socioeconomic indicators, ethnicity, and carotid atherosclerosis (IMT and plaque). Linear and logistic regression analyses were used to determine whether community SES accounted for significant variance in carotid measurements. Because few census tracts contained more than one study participant (and none more than five), hierarchical modeling was not necessary.<sup>22</sup> Variables were entered into the regression models in steps, starting with biological risk factors for IMT and plaque identified in preceding bivariate correlations. We next entered community SES to determine whether it explained additional variance in IMT (in linear regression) or the odds ratio (OR) of having a plaque score  $>1$  (in logistic regression). In separate regression models we entered individual SES (participants' own income and education) after covariates (ie, in the second step) to examine its predictive association with carotid disease in this sample. Community SES was then entered in a final step to determine whether it accounted for additional variance in carotid IMT or plaque (ie, variance not predicted by risk factors or personal socioeconomic indicators). Analyses were conducted using SPSS version 13.0 (SPSS Inc., Chicago, IL).

### Results

In this sample, mean IMT was 0.91 mm ( $SD = 0.16$  mm) and 48.3% of subjects had plaque scores  $>1$ . As shown in Table 1, the mean age of the sample was 55.7 years, and age correlated appreciably with both IMT and plaque, as did BP and fasting glucose concentrations. Smoking status was a significant correlate of IMT as well. Socioeconomic deprivation at the individual level was associated with greater IMT, whereas community SES showed a marginally significant bivariate association with carotid plaque.

Because age and BP predicted both community SES and carotid measurements, we reasoned that controlling for correlated variation in age and other risk factors might better reveal any association between carotid atherosclerosis and community-level inequalities. As summarized in Table 2, age, BP, smoking status, and glucose concentrations were entered simultaneously as the first step in the regression model predicting IMT. Age ( $b = 0.002$ ,  $P < .01$ ) and both systolic ( $b = 0.001$ ,  $P < .01$ ) and diastolic ( $b = -0.001$ ,  $P < .01$ ) BP independently predicted IMT, and all covariates together accounted for 33% of the variance in mean IMT ( $P < .0001$ ). When entered in the second step of parallel regression models, both individual and community SES also proved significant predictors of IMT (individual SES:  $P = .009$ ; community SES:  $P = .026$ ). By direction of association, 1) hypertensives possessing less education and lower income had greater mean IMT than did those who ranked higher in these character-

**Table 1.** Correlations among cardiovascular risk factors, carotid atherosclerosis, and community SES

Characteristic	Total sample n = 230 M (SD)	Race r	Mean IMT r	Plaque r <sub>pb</sub>	Community SES r
Age (y)	55.7 (9.0)	-0.14*	0.52**	0.25**	0.20**
SES					
Individual+	0 (1.0)	0.40**	0.19**	0.08	0.50**
Community+	0 (1.0)	0.55**	0.01	0.11+	—
Smoking status, % current	13.5	0.37**	†-0.14*	-0.02	-0.25**
BMI (kg/m <sup>2</sup> )	28.2 (3.2)	-0.08	0.02	-0.10	0.07
Glucose (mg/dL)	94.6 (15.2)	-0.06	0.21**	0.15*	-0.03
Cholesterol:HDL ratio (mg/dL)	4.6 (1.4)	-0.35**	0.07	-0.02	0.14*
Triglycerides (mg/dL)	165.9 (139.6)	-0.20**	-0.03	-0.05	0.11
Systolic BP (mm Hg)	148 (11)	-0.15*	0.34**	0.21**	0.15*
Diastolic BP (mm Hg)	93 (7)	0.17*	-0.37**	-0.21**	-0.13*

+ Individual and community SES indices are expressed as z-scores (M = 0); high SES scores, both individual and community, denote relative socioeconomic disadvantage. Race (20% African-American) coded 1 = White, 2 = Black. BMI = body mass index; Smoking status (0 = not current, 1 = current); Cholesterol: HDL ratio represents the ratio of total serum cholesterol to HDL-cholesterol concentration; IMT = mean carotid artery intima-medial thickness; correlations were conducted on the transformed IMT variable, but signs were reversed in the table for ease of interpretation; carotid plaque was coded as 0 or 1 as described in text; † r<sub>pb</sub> = point biserial correlation.

\*\* P < .01; \* P < .05; +P < .10.

istics; and similarly, 2) mean IMT was greater among residents of less advantaged census tracts than those who lived in more affluent neighborhoods. However, when community SES was entered into the regression model *after* including individual SES as a predictor, the community variable no longer accounted for additional variance

in mean IMT. Hence, area of residence did not explain variability in arterial thickening beyond that associated with personal socioeconomic indicators.

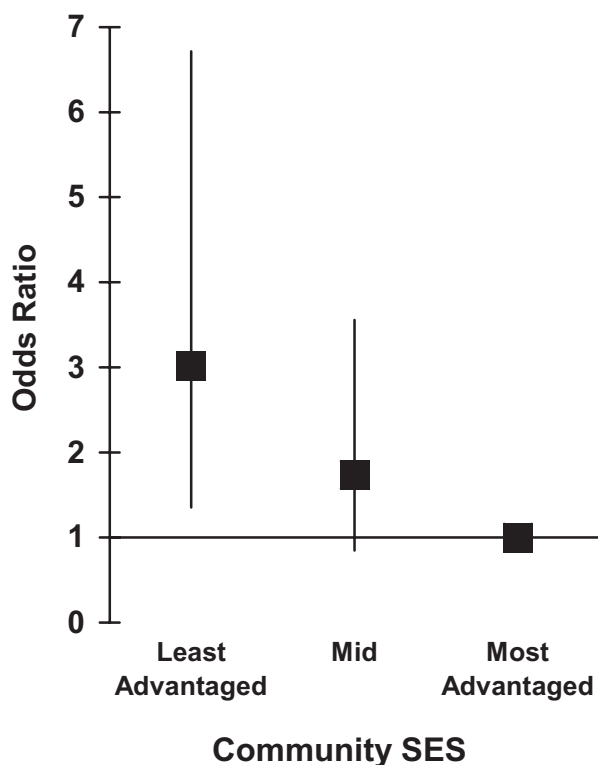
Results differed, however, on multivariable analysis of carotid plaque. In logistic regression, covariates (age, systolic and diastolic BP, fasting glucose) strongly predicted

**Table 2.** Predictive analyses of IMT and plaque occurrence by covariates, individual SES and community SES

	r <sub>p</sub>	Unstandardized b	P
Linear regression			
DV: 1/Mean IMT*			
Step 1: (F = 22.35, P = .00)			
Age	0.31	0.002	.000
Smoking Status	-0.03	-0.003	.682
Average SBP	0.23	0.001	.001
Average DBP	-0.19	-0.001	.004
Glucose	0.08	0.000	.210
Step 2:			
Individual SES	0.17	0.006	.009
Step 3:			
Community SES	0.07	0.003	.282
	B	Odds ratio (95% CI)	P
Logistic regression			
DV: Plaque (0,1)			
Step 1: (X <sup>2</sup> = 24.26, P = .00)			
Age	0.02	1.03 (0.99, 1.06)	.202
Average SBP	0.03	1.03 (1.01, 1.06)	.019
Average DBP	-0.04	0.96 (0.92, 1.00)	.063
Glucose	0.01	1.01 (0.99, 1.03)	.203
Step 2:			
Individual SES	0.09	1.09 (0.83, 1.44)	.526
Step 3:			
Community SES	0.52	1.68 (1.18, 2.38)	.004

r<sub>p</sub> = partial correlation coefficient.

\* Dependent variable is the reciprocal transformation of mean IMT; signs are reversed for ease in interpretation.



**FIG. 1.** Odds ratio of plaque index >1 for low and moderate tertiles of community socioeconomic status (SES) compared with the highest tertile (referent category). Odds ratio adjusted for age, systolic and diastolic blood pressure, glucose, and individual SES.

plaque ( $P < .001$ ), although only systolic BP did so independently of other risk factors (OR = 1.03; 95% confidence interval [CI]: 1.01–1.06). Community SES subsequently predicted carotid plaque (OR = 1.51; 95% CI = 1.13–2.02), as it had IMT, but individual-level SES showed no relation to plaque measurements when similarly added to the regression model (OR = 1.09; 95% CI = 0.83–1.44). Accordingly, community SES continued to predict plaque when entered after both covariate risk factors and individual SES (OR = 1.68, 95% CI = 1.18–2.38) (Table 2). To further illustrate this association, we partitioned the sample by tertiles of community SES. Thus, Fig. 1 depicts the change in odds of having a plaque score >1 among hypertensive men comprising the most disadvantaged and intermediate tertiles of the distribution of community SES, relative to the most advantaged (referent), after adjusting for age, other risk factors, and individual SES. The odds of “high plaque” among subjects in the poorest tertile of community SES were about two-thirds higher than in the most affluent tertile. For comparison, it may be noted that median household incomes in the least and most advantaged tertiles of community SES averaged \$20,122 and \$48,131, respectively. Corresponding proportions of households in poverty were 21.3% and 3.9%; and for adults lacking a high school diploma, 29.4% and 9.9%.

## Secondary Analysis

Although mean IMT and plaque scores did not vary by ethnic group, African American participants lived disproportionately in poorer census tracts than whites, as reflected in our composite index of community SES (z-score) (African Americans:  $M = 1.08$  [SD = 1.04]; whites:  $M = -0.29$  [SD = 0.78]  $t_{228} = -9.93$ ,  $P < .0001$ ). Because inequalities among communities were thus partly confounded by ethnicity, we next asked whether community SES predicted carotid atherosclerosis in regression analyses restricted to whites only. Results of these analyses were virtually identical to those based on the full sample, and in particular, community SES again predicted carotid plaque after adjustment for covariate risk factors and individual SES (OR = 1.81; 95% CI = 1.14–2.90,  $P = .013$ ). Owing to insufficient statistical power, parallel analyses were not conducted on data of African American hypertensives alone.

## Discussion

In this study, an area-based measure of socioeconomic inequalities predicted extracranial carotid artery atherosclerosis in multivariable analyses adjusted for other cardiovascular risk factors. Untreated hypertensives living in less affluent census tracts showed more carotid IMT and plaque by duplex ultrasonography than residents of more advantaged neighborhoods. When further adjusted for concomitant variability in individual SES (personal income and education), communities still accounted for significant variation in atherosclerosis. Although African-American participants lived disproportionately in poorer neighborhoods, community SES predicted carotid plaque equally well in secondary analyses restricted to data of white hypertensives only.

These findings add to growing evidence that socio-demographic characteristics of areas of residence predict cardiovascular disease morbidity and mortality, even after adjustment for common variation in traditional risk factors or individual markers of SES.<sup>1–4,8</sup> Extending these observations, our findings suggest that the socioeconomic attributes of communities > also confer risk for preclinical vascular disease, at least among hypertensives in this sample. This finding implies that the relation of community SES with incident cardiovascular disease may reflect influences other than those commonly linked to symptomatic expressions of disease, such as plaque rupture and thrombus formation, myocardial ischemia, or cardiac arrhythmia. More specifically, our data are consistent with the hypothesis that living in disadvantaged communities accelerates the development of vascular changes underlying later clinical manifestations of atherosclerotic cardiovascular disease. Although mechanisms by which community attributes may affect risk for atherosclerosis are unclear, obvious candidates include exposure to environmental factors promoting risk-related

behaviors (eg, density of fast-food restaurants, targeted advertising of tobacco products), differential access to health resources (eg, medical care, recreational facilities), or the chronic stress of living in neighborhoods marked by crime, noise, or crowding. Of course, these observations do not exclude the possibility that community variables also affect pathophysiologic processes triggering acute clinical events or contextual factors that influence patient outcomes, such as the availability of emergency care or variability in symptom labeling and treatment-seeking behaviors.

We note that community SES correlated only weakly with carotid plaque (and not at all with IMT) in bivariate analyses (Table 1), whereas these associations emerged more prominently in the multivariable models. Participants varied in age from 40 to 70 years, and age is perhaps the strongest predictor of carotid disease.<sup>23</sup> Blood pressure also covaries strongly with age, and both age and BP correlated significantly with community SES. Thus, participants who lived in census tracts of higher “rank” tended to be older and have more elevated systolic and lower diastolic BPs, but did not show the greater carotid changes these risk factors otherwise predict. When influences of age and BP were controlled by multivariable adjustment, moreover, the adverse effect of poorer community quality on carotid plaque was strengthened and a similar association with IMT unmasked.

Although high community status may mitigate the pathogenicity of age and BP, the correlation between increasing age and more affluent community SES raises the possibility that our community variable reflects individual wealth (which typically accumulates with age), as well as variation in the material and social resources of more and less wealthy neighborhoods. Insofar as people do not randomly select the communities in which they live, individual and community attributes are necessarily entwined. Hence, associations between markers of neighborhood quality and disease risk cannot be interpreted as direct evidence of socioenvironmental influences on health. Still, controlling for personal education and income did not attenuate the association between community status and carotid plaque in this study. This finding is consistent with speculation that neighborhood qualities influence cardiovascular risk over and above commonly reported socioeconomic attributes of individuals.<sup>1–4,8</sup>

Including only untreated hypertensives in these analyses eliminated potential confounding by concomitant antihypertensive medication, but also prevents generalization of our findings to a broader population, including medicated hypertensives, normotensives, and women. Conclusions are limited, too, by sample size, the cross-sectional nature of our data, and a lack of information regarding participants’ duration of neighborhood residence. It should also be noted that census-derived estimates of status only crudely reflect the sociodemographic features of communities and offer little insight into proximal mechanisms—be they behavioral or biological—that affect disease risk and

progression. Future research may benefit from examining microenvironmental features of disparate neighborhood settings, including variation in preventive health care services, recreational facilities, dietary habits, and psychological stress (eg, exposure to violence and personal risk) to better elucidate how communities may shape the health experiences of individuals.

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