ORIGINAL RESEARCH ARTICLE

Change in Neighborhood Characteristics and Change in Coronary Artery Calcium

A Longitudinal Investigation in the MESA (Multi-Ethnic Study of Atherosclerosis) Cohort

Editorial, see p 514

BACKGROUND: Although some evidence shows that neighborhood deprivation is associated with greater subclinical atherosclerosis, prior studies have not identified what aspects of deprived neighborhoods were driving the association.

METHODS: We investigated whether social and physical neighborhood characteristics are related to the progression of subclinical atherosclerosis in 5950 adult participants of the MESA (Multi-Ethnic Study of Atherosclerosis) during a 12-year follow-up period. We assessed subclinical disease using coronary artery calcium (CAC). Neighborhood features examined included density of recreational facilities, density of healthy food stores, and survey-based measures of availability of healthy foods, walking environment, and social environment. We used econometric fixed-effects models to investigate how change in a given neighborhood exposure is related to simultaneous change in subclinical atherosclerosis.

RESULTS: Increases in density of neighborhood healthy food stores were associated with decreases in CAC (mean changes in CAC Agatston units per 1-SD increase in neighborhood exposures, −19.99; 95% confidence interval, −35.21 to −4.78) after adjustment for time-varying demographic confounders and computed tomography scanner type. This association remained similar in magnitude after additional adjustment for time-varying behavioral risk factors and depression. The addition of time-varying biomedical factors attenuated associations with CAC slightly (mean changes in CAC per 1-SD increase in neighborhood exposures, −17.60; 95% confidence interval, −32.71 to −2.49). Changes across time in other neighborhood measures were not significantly associated with within-person change in CAC.

CONCLUSIONS: Results from this longitudinal study provide suggestive evidence that greater access to neighborhood healthy food resources may slow the development of coronary atherosclerosis in middle-aged and older adults.

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Sources of Funding, see page 511

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Clinical Perspective

What Is New?

- Neighborhood deprivation is thought to affect subclinical atherosclerosis, but prior studies have not identified which aspects of deprived neighborhoods were driving the association.
- This is the first prospective study to investigate associations of changes in neighborhood factors with the progression of subclinical atherosclerosis.
- Using 5950 adult participants from the MESA (Multi-Ethnic Study of Atherosclerosis) during a 12-year follow-up period, this study investigated whether social and physical neighborhood characteristics are related to the progression of subclinical atherosclerosis measured by coronary artery calcium.

What Are the Clinical Implications?

- Our analysis identified that greater access to neighborhood healthy food resources may slow the development of coronary atherosclerosis in middle-aged and older adults.
- Our findings have important implications for the need for policy changes to support the increase in neighborhood healthy food stores.

Neighborhood deprivation, including high neighborhood poverty level, low education level, high unemployment, poor housing conditions, and other indicators, has been linked to greater prevalence and incidence of coronary heart disease.[1,2] There is some evidence that specific neighborhood features such as fewer neighborhood physical activity resources[3,4] and less safety[5,6] and social connectedness[7] are associated with greater coronary heart disease prevalence, incidence, and mortality, but questions remain as to whether these associations reflect causal processes.

A limitation of focusing on clinical cardiovascular outcomes (especially in the cross-sectional context) is that it is not possible to determine the direction of causality (ie, whether neighborhood characteristics have caused poorer cardiovascular health or poorer cardiovascular health has caused participants to live in certain kinds of neighborhoods). In addition, studies that focus on clinical outcomes are unable to determine whether neighborhood exposures are related to the triggering of cardiovascular events in persons with underlying atherosclerotic disease or to the development of atherosclerosis itself.

A few studies have reported that neighborhood deprivation is associated with greater subclinical atherosclerosis[8–12]; however, prior studies were not able to identify what aspects of deprived neighborhoods were driving the association. Investigation of specific neighborhood exposures beyond neighborhood deprivation is of key public health relevance because this approach provides insight into specific pathways for intervention. Furthermore, the investigation of whether changes in neighborhood characteristics are related to changes in subclinical disease strengthens causal inferences.

We investigated whether social and physical neighborhood characteristics are related to the progression of subclinical atherosclerosis in healthy older male and female participants of the MESA (Multi-Ethnic Study of Atherosclerosis) during a 12-year follow-up period. Because a range of neighborhood factors could be important, we examined several characteristics previously hypothesized to be linked to the development of coronary heart disease.[13] The study design allowed us to investigate how change in a given neighborhood exposure related to simultaneous change in subclinical atherosclerosis.

METHODS

Study Sample

The study sample consisted of 6814 participants from MESA, a longitudinal study of subclinical cardiovascular disease. Participants who were 45 to 84 years of age and free of clinical cardiovascular disease were enrolled in the study from 6 field sites (Baltimore, MD; Chicago, IL; Forsyth County, North Carolina; Los Angeles, CA; New York, NY; and St. Paul, MN) from 2000 to 2002.[14] The study was approved by the institutional review boards at each site, and all participants gave written informed consent. Our analysis used data from 5 MESA examinations spanning 2000 to 2011 and included only data from those who participated in the neighborhood ancillary study (n=6191). We further restricted our sample to include only those that had multiple coronary artery calcium (CAC) measurements, yielding an analytic sample of 5950 participants.

CAC Outcome

We used CAC as a measure of subclinical disease; it has been shown to independently predict coronary events in diverse ethnic groups. CAC was assessed by chest computed tomography (CT) and quantified with the continuous Agatston score, measured in Agatston units.[15] Cardiac CT was performed on all participants at baseline; a second CAC measurement was performed on half of the cohort at examination 2; and the other half of the participants were scanned at examination 3. Additional CAC measurements were taken on a subset of the MESA cohort at examinations 4 and 5. Four percent of the sample had only 1 CAC measurement (and were excluded); 38% had 2, 45% had 3, and 13% had 4 CAC measurements. The median number of years since baseline for the CAC measurement at each examination is as follows: for examination 2, 1.6 years (interquartile range, 1.4–1.8 years); for examination 3, 3.1 years (interquartile range, 3.0–3.4 years); for examination 4, 4.8 years (interquartile range, 4.6–5.0 years); and for examination 5, 9.4 years (interquartile range, 9.1–9.7 years).

Neighborhood Exposure Measurements

The neighborhood environment was characterized with the use of both commercially available food store and recreational facility data, as well as surveys on healthy food, walking, safety, and social cohesion.

Geographic Information Systems-Based Measures: Recreational and Healthy Food Resources

The availability of healthy food stores and recreational facilities within 1 mile of participants' homes was characterized with ArcGIS 9.3.[16] Normal kernel estimation[17] was used to calculate the densities such that facilities closer to participants' homes were given more weight than those farther away. Densities are expressed in units per square mile (henceforth, density indicates units per square mile). Relevant classifications and locations of stores and facilities were identified from Dun and Bradstreet data as compiled by Walls and Associates in the National Establishment Time Series database.[18] Recreational facilities included 114 standard industrial classification codes that classified an establishment as having potential for physical activity or recreation such as indoor conditioning, dance, bowling, golf, team and racquet sports, and water activities; this classification was derived from previous studies.[19,20] Healthy food stores included fruit and vegetable markets and supermarkets (defined as food stores with at least $2 million in annual sales or at least 25 employees). Additional data were included from the Nielsen TDLinx Service Supermarket Retail Category Database to further capture area supermarkets as described by Auchincloss and coauthors.[21] Densities were created for each calendar year between 2000 and 2010.

Survey-Based Measures

Survey-based neighborhood scales included the availability of healthy food (2 items), walking environment (4 items), safety (2 items), and social cohesion (4 items). The participants were asked to rate their neighborhoods within a mile of their homes (the items are listed in Table I in the online-only Data Supplement). These aspects of the neighborhood environment were selected on the basis of conceptual links to cardiovascular disease[22] and developed from prior work.[23,24] Survey data collected from MESA participants were combined with data from participants in an external survey (the Community Survey) administered to other residents of neighborhoods in which MESA participants lived. Scales were based on a 1-mile buffer around the participant's residence and were created by taking the crude mean of the responses for all respondents living within a 1-mile buffer, excluding themselves. Only respondents who answered all questions within the scale were included. Each score has a total possible range from 1 to 5, with a higher score representing a more favorable environment.

Covariates

Time-invariant demographic covariates included highest education level completed, race/ethnicity, and sex, all assessed via questionnaire at the baseline examination. All other covariates were time varying, and for examinations in which a given measure was not assessed, the value from the closest examination was used. Demographic covariates included age, marital status, family income level, and current employment status. Moderate and vigorous physical activity levels were based on summed minutes of reported relevant physical activities multiplied by the metabolic equivalent of the activity, and cigarette smoking status was classified as current, former, or never. Depressive symptoms were assessed with the 20-item Center for Epidemiological Studies Depression Scale.[25] Biomedical factors, measured by physical examination and laboratory tests, included diabetes mellitus, defined as fasting glucose ≥126 mg/dL or the use of hypoglycemic medications; hypertension, defined as a systolic blood pressure ≥140 mm Hg, diastolic blood pressure ≥90 mm Hg, or taking antihypertensive medication; body mass index (BMI), calculated as weight in kilograms divided by height in meters squared; and ratio of total to high-density lipoprotein cholesterol (both measured from blood samples obtained after a 12-hour fast). Self-reported medications for elevated lipids were also included. CT scanner model was also included as a time-varying covariate because scanner type varied across site and time.

Statistical Methods

We examined the distribution of demographic characteristics and risk factors across levels of baseline CAC (participants with baseline CAC equal to 0 [ie, no discernible calcium on the coronary artery], those with baseline CAC between 0 and 86.5 Agatston units [the median of those with CAC >0], and those with CAC ≥86.5 Agatston units). ANOVA was used to compare continuous variables, and χ^2 tests were used for categorical variables. We also examined associations of categories of neighborhood characteristics (based on quartiles) with percent with baseline CAC >0, mean baseline CAC, and mean annual change in CAC after adjustment for age, sex, and race/ethnicity (χ^2 tests, linear tests for trend, and time-interaction terms were used to test these comparisons). On the basis of prior MESA analyses[26] and analytic recommendations and given the known robustness of results to violations of the normality assumption,[27] CAC was treated as a continuous variable on the native scale to facilitate model fitting and interpretation of associations.

The association of CAC with each of the 6 neighborhood measures (density of recreational facilities, density of healthy food stores, and survey-based measures of availability of healthy foods, walking environment, and social cohesion and safety) was initially examined separately. The survey-based social environment measures (ie, safety and social cohesion) were highly correlated and similarly related to CAC and therefore were combined into a summary social environment index (Cronbach α=0.77) by standardizing and then summing their standardized scores. To facilitate comparison across estimates, all neighborhood characteristics were standardized and included in regression models in standard deviation units. Each neighborhood variable was tested in a separate model.

Econometric fixed-effects models[28] were used to estimate associations of within-person changes in neighborhood characteristics with within-person changes in CAC. This approach allowed us to examine whether changes in exposures were related to simultaneous changes in the outcome. Because they condition on the study participant, these models have the important advantage of tightly adjusting for measured and unmeasured time-invariant characteristics of individuals[28]; thus, they were adjusted only for time-varying covariates. We used a sequence of 4 models that are based on the hypothesized causal pathways linking neighborhood exposures to the outcome (the models progressively adjust for variables more proximal to the outcome and hence more likely to be in the causal pathway). Model 1 adjusted for demographic covariates (age, marital status, income, and working status) and CT scanner type; model 2 additionally adjusted for health behaviors (moderate/vigorous physical activity, cigarette smoking status); model 3 additionally adjusted for depressive symptoms (Center for Epidemiological Studies Depression Scale); and model 4 additionally adjusted for biomedical factors (ratio of total to high-density lipoprotein cholesterol, BMI, hypertension, diabetes mellitus, and lipid-lowering medication). Each of the 5 neighborhood characteristics was investigated separately. Because it has been suggested that neighborhood socioeconomic status may confound associations of neighborhood physical and social environments with health outcomes, additional adjustment for neighborhood socioeconomic status using a summary measure[29] was investigated in sensitivity analyses. We also explored effect modification of the neighborhood characteristics by sex and race/ethnicity in an exploratory analysis.

Main analyses included both movers and nonmovers (hence, changes over time in neighborhood exposures may result from moving to a different neighborhood or from changes over time in a neighborhood). In sensitivity analyses, we adjusted for moving by including a time-varying indicator of whether the participant had moved between the prior visit and the current visit and an interaction term between the moving indicator and the neighborhood variable.

Results are presented as the mean difference in CAC for a 1-SD increase in neighborhood score and 95% confidence interval. All statistical tests were 2 sided. Values of $P<0.05$ were considered statistically significant and were not adjusted for multiple testing. All analyses were conducted in SAS version 9.3 (SAS Institute Inc, Cary, NC).

RESULTS

Of the 5950 participants in our sample, 86% contributed to at least 3 time points, with a mean of 2.7 time points (SD=0.7) per participant and a mean follow-up time between CAC measurements of 3.5 years (SD=3.1 years). Average CAC in the total sample at baseline was 135 (SD=380). Just over half of participants (51%) had a CAC=0 at baseline (Table 1). A nonzero CAC at baseline was associated with being older, male, and white; having a lower income and less education; and being a former smoker. Thirty-two percent of participants moved at some point during the follow-up period.

The percent of participants with CAC>0 at baseline was lower (45%) in the highest quartile of healthy food density than in other quartiles (49%–50%) after adjustment for age, sex, and race/ethnicity (Table 2). Residents of neighborhoods with higher levels of social cohesion and safety were slightly more likely to have CAC>0 at baseline than those of neighborhoods with lower social cohesion and safety (51% versus 44% for highest versus lowest quartiles) after adjustment.

Mean baseline CAC was lower in neighborhoods with a higher density of healthy food stores and with higher reported availability of healthy foods after adjustment for age, sex, and race/ethnicity, although trends across quartiles were not statistically significant (Table 2). No clear pattern in mean CAC was apparent across quartiles of recreational facility density, the walking environment, or the social environment. Mean annual CAC progressed fastest in neighborhoods with a lower density of recreational facilities, a lower density of healthy food stores (although no dose response was observed), and lower reported availability of healthy foods after adjustment for age, sex, and race/ethnicity. The patterns of mean annual change were inconsistent across quartiles of other survey-based neighborhood characteristics. Of the total variance in CAC, 17% was within-person variance. Of the total variance in neighborhood characteristics, 10%, 5%, 31%, 21%, and 16% were within-person variances for recreational facility density, healthy food store density, availability of health food, walking environment, and social environment, respectively.

Within-person increases in neighborhood healthy food stores density were associated with within-person decreases in subclinical atherosclerosis (Table 3). In model 1, which adjusted for time-varying demographic confounders and CT scanner type, increases in density of neighborhood healthy food stores were associated with decreases in CAC (mean changes in CAC per 1-SD increase in neighborhood exposures, −19.99; 95% confidence interval, −35.21 to −4.78). This association remained similar in magnitude after additional adjustment for time-varying behavioral risk factors and depression (see models 2 and 3 in Table 3). The addition of time-varying biomedical factors (model 4) attenuated associations with CAC slightly (−17.60; 95% confidence interval, −32.71 to −2.49). Within-person increases in recreational facilities density were associated with decreases in calcium, but associations were not statistically significant. In exploratory analyses, the association of healthy food density with calcium was stronger in women than in men (interaction P=0.02), but no effect modification by race/ethnicity was observed.

When survey-based measures were used, changes in availability of healthy foods, in walking environments, and in the social environment were not significantly associated with changes in CAC over time. In sensitivity analyses, associations of healthy food densities with changes in calcium were robust to additional adjustment for neighborhood socioeconomic status. A sensitivity analysis that included adjustment for moving status did not find heterogeneity in estimates by moving status (all interactions P>0.47).

Table 1. Characteristics of Participants by Levels of CAC at Baseline (n=5950), MESA, 2000 to 2002

Characteristic	CAC*=0, %	CAC >0, %	
		CAC<86.5 (Median Value)	CAC≥86.5 (Median Value)
n	3065	1451	1512
Age, mean (SD), y	57.9 (9.1)	63.6 (9.6)	68.6 (8.6)
46–56	43.4	21.6	8.0
57–66	30.5	30.3	21.1
67–76	21.1	33.4	43.2
77–86	5.0	14.8	27.7
Sex			
Female	63.0	47.7	34.5
Male	37.0	52.3	65.5
Race/ethnicity			
White	34.3	39.8	50.8
Chinese	11.7	13.4	10.5
Black	31.2	25.2	20.1
Hispanic	22.8	21.6	18.6
Annual family income, $			
<12 000	9.3	12.6	12.6
12 000–24 999	18.2	20.2	20.6
25 000–39 999	19.4	18.9	18.5
40 000–74 999	28.6	26.2	25.8
75 000+	24.6	22.2	22.5
Education			
Completed high school/GED or less	32.6	36.4	36.5
Some college, technical or associate degree	29.8	27.4	27.5
Bachelor's degree or higher	37.6	36.3	35.9
Smoking			
Never	56.0	48.2	40.8
Former	31.1	38.9	47.5
Current	12.9	12.9	11.7

CAC indicates coronary artery calcium; GED, General Educational Development; and MESA, Multi-Ethnic Study of Atherosclerosis.
*CAC was measured by the Agatston score. The median value for CAC is 86.5 Agatston units.

DISCUSSION

Our study found that increases in the density of healthy food stores around the home were related to simultaneous decreases in subclinical atherosclerosis as characterized by coronary calcium after adjustment for age, marital status, income, working status, and CT scanner type. These associations were reduced slightly but persisted after additional adjustment for changes in moderate/vigorous physical activity, cigarette smoking status, Center for Epidemiological Studies Depression Scale score, ratio of total to high-density lipoprotein cholesterol, BMI, hypertension, diabetes mellitus, and any lipid-lowering medication. The effect size associated with a 1-SD increase in density was similar to the effect on CAC of a 1-year increase in age (17- to 20-point increase in CAC). Although a number of studies have linked neighborhood characteristics to coronary heart disease incidence and mortality,[6,7,30,31] we are aware of no studies that have investigated associations of changes in neighborhood factors with the progression of subclinical atherosclerosis. Our results suggest that effects of neighborhood food context on the development of subclinical disease may be one of the mecha-

Table 2. Percent of Participants With CAC>0 at Baseline, Mean (SE) at Baseline, and Annual Change in CAC by Neighborhood Characteristic at Baseline (n=5950)

Characteristic	Percent of All Participants With CAC>0* at Baseline†	Mean (SE)‡ CAC at Baseline Among All Participants§	Mean (SE)‡ Annual Change in CAC Among All Participants‖
Density of recreational facilities¶			
Quartile 1 (lowest)	48.2	133.3 (7.5)	3.11 (2.20)
Quartile 2	49.3	169.5 (7.3)	0.89 (2.10)
Quartile 3	49.5	163.7 (7.4)	0.22 (2.15)
Quartile 4 (highest)	47.2	140.4 (7.4)	0.35 (2.17)
P value	0.52	0.67	0.32
Density of healthy food stores¶			
Quartile 1 (lowest)	50.1	160.8 (7.5)	3.31 (2.21)
Quartile 2	49.2	154.5 (7.3)	0.19 (2.11)
Quartile 3	50.0	160.5 (7.7)	0.50 (2.24)
Quartile 4 (highest)	44.7	132.4 (7.4)	1.10 (2.15)
P value	0.008	0.02	0.51
Availability of healthy foods#			
Quartile 1 (lowest)	49.9	158.8 (7.7)	4.09 (2.22)
Quartile 2	45.6	164.9 (7.7)	1.33 (2.12)
Quartile 3	49.8	156.6 (7.4)	0.36 (2.23)
Quartile 4 (highest)	48.8	130.0 (7.6)	–0.97 (2.21)
P value	0.07	0.001	0.09
Walking environment#			
Quartile 1 (lowest)	46.7	141.4 (7.7)	–0.30 (2.24)
Quartile 2	49.7	165.9 (7.5)	1.47 (2.19)
Quartile 3	48.3	160.3 (7.4)	2.28 (2.12)
Quartile 4 (highest)	49.4	143.4 (7.6)	1.54 (2.20)
P value	0.35	0.99	0.54
Social environment#			
Quartile 1 (lowest)	44.2	140.4 (7.9)	1.58 (2.27)
Quartile 2	49.2	161.9 (7.5)	-0.14 (2.15)
Quartile 3	50.0	158.7 (7.6)	0.41 (2.23)
Quartile 4 (highest)	50.7	158.3 (7.6)	3.03 (2.19)
P value	0.002	0.25	0.58

CAC indicates coronary artery calcium.
*CAC was measured by the Agatston score.
†*P* value from χ^2 test.
‡Adjusted for age at baseline, sex, and race/ethnicity.
§*P* value from linear test for trend.
‖*P* value from interaction term between time by neighborhood variable (as quartiles) in a model with CAC as the outcome variable.
¶Measured with Geographic Information Systems.
Measured by questionnaire.

nisms through which these factors affect the incidence of coronary heart disease.

The use of fixed-effects models allowed us to investigate whether a within-person change in the neighborhood attribute was related to a within-person change in CAC while tightly adjusting for the time-invariant person characteristics that could confound associations of neighborhood characteristics with CAC. This is a major advance over prior work, which can be subject to between-person confounding. Although residual confounding by time-varying

Table 3. Mean Within-Person Differences in CAC Associated With a 1-SD Within-Person Increase in Selected Neighborhood Characteristics*

Parameter	Mean Difference (95% CI)			
	Model 1†	Model 2‡	Model 3§	Model 4‖
Geographic information systems measures				
Density of recreational facilities¶	−8.42 (−17.36 to 0.52)	−8.39 (−17.32 to 0.54)	−8.43 (−17.34 to 0.49)	−7.68 (−16.57 to 1.21)
Density of healthy food stores¶	−19.99 (−35.21 to −4.78)	−18.99 (−34.19 to −3.79)	−19.41 (−34.59 to −4.23)	−17.60 (−32.71 to −2.49)
Survey-based measures				
Availability of healthy foods#	6.84 (−0.46 to 14.15)	6.79 (−0.51 to 14.09)	6.84 (−0.45 to 14.13)	6.44 (−0.77 to 13.65)
Walking environment#	5.99 (−2.01 to 14.00)	5.92 (−2.07 to 13.92)	5.82 (−2.16 to 13.80)	4.95 (−2.96 to 12.86)
Social environment#	7.86 (−0.74 to 16.45)	8.00 (−0.59 to 16.59)	7.94 (−0.63 to 16.51)	6.88 (−1.62 to 15.37)

CAC indicates coronary artery calcium; and CI, confidence interval.

*All estimates are derived from fixed-effects models. Each neighborhood characteristic is investigated in a separate model.

†Model 1: adjusted for age, marital status, income, working status, and CT scanner type.

‡Model 2: adjusted as for model 1 plus moderate/vigorous physical activity and cigarette smoking status.

§Model 3: adjusted as for model 2 plus Center for Epidemiological Studies Depression Scale score.

‖Model 4: adjusted as for model 3 plus ratio of total to high-density lipoprotein cholesterol, body mass index, hypertension, diabetes mellitus, and any lipid-lowering medication.

¶Measured with geographic information systems. Higher scores represent more density: 1 SD of density of recreational facilities=8.4 U/sq mile, and 1 SD of density of healthy food stores=4.3 U/sq mile.

#Measured via questionnaire; higher scores represent a more favorable environment: 1 SD of the availability of healthy foods score=0.54, 1 SD of the walking environment score=0.33, and 1 SD of the social environment score=1.65.

characteristics (including socioeconomic factors) cannot be completely ruled out in our study, we adjusted for several time-varying confounders in the models. If moving status is related to changes in calcium and to changes in neighborhood exposures, it could confound the associations of interest. However, adjusting for moving status and allowing associations to differ by moving status did not materially affect our conclusions. A limitation of fixed-effects models is that they rely exclusively on within-person variability and can be inefficient when within-person variability in exposures or outcomes is very low. As expected, within-person variability in neighborhood characteristics was substantially smaller than between-person variability, possibly limiting our ability to detect statistically significant associations with some neighborhood exposures.

Neighborhood food environments may affect cardiovascular risk by shaping dietary intake. Studies have linked food environments with limited choices or a high prevalence of fast food restaurants to poorer diet quality and greater fast food consumption.[32,33] Greater neighborhood availability of healthier foods has also been linked to higher fruit and vegetable consumption.[34] Diets higher in fruits, vegetables, antioxidants, whole grains, and fish have been shown to reduce the progression of subclinical heart disease in prospective observational studies[35] and randomized trials,[36,37] and some evidence from randomized trials shows a reversal of carotid atherosclerosis through adherence to a heart healthy diet.[38] In our study, greater access to healthier foods may have promoted healthier diets and less coronary plaque formation in neighborhood residents. The limited dietary data available in MESA at the time of these analyses limited our ability to examine the mediating effects of diet.

Greater neighborhood availability of recreational resources has been associated with a greater physical activity level in residents,[39,40] and longitudinal studies have shown that greater physical activity reduces the progression of subclinical coronary disease.[41,42] Other studies have shown that favorable neighborhood physical activity environments are associated with better cardiometabolic risk factors,[43] lower BMI and smaller waist size,[44] lower risk of type 2 diabetes mellitus,[45] and decreased risk of coronary events.[30,31] Although the associations of changes in recreational facilities with changes in CAC were in the hypothesized direction, they were not statistically significant. It could be that recreational facility densities are not important drivers of overall physical activity (which may be more relevant to CAC change than only leisure activity).

Survey-based measures of the neighborhood food, physical activity, and social environments were not significantly related to CAC progression. It has been argued that survey-based measures of neighborhoods may capture information that is not reflected in objective locational data.[46] The social environment in particular is more readily measured with survey measures. In earlier work, we documented associations of baseline survey-based measures of the physical environment (especially the food environment) with the incidence of diabetes mellitus and obesity.[47–49] An important limitation of survey-based measures in this study, however, is that their time-varying nature was much more limited than it was for the density measures (which were updated every year). In contrast, the survey-based measures relied on substantial interpolation to create time-varying measures. This added measurement error and reduced within-person variability may have seriously limited our ability to detect associations of change with change in the fixed-effects models.

We hypothesized that a favorable social environment would improve stress and depressive symptoms, favorably affecting CAC. Although only 1 identified cross-sectional study has evaluated neighborhood social characteristics in relation to CAC, reporting that better social environment was related to lower CAC,[11] other studies have found that a better social neighborhood environment is associated with reduced myocardial infarction incidence.[6,7,31] We also hypothesized that an improvement in the walking environment would be related to reduced progression of subclinical disease. It has been shown that changes in walking environments are related to change in physical activity and changes in BMI over time.[50,51] However, these studies relied on objective measures of the built environment features rather than survey measures, as used in the analyses reported here. Studies with improved measurement of the social and walking environments are needed to draw firmer conclusions on the possible effects of these domains on changes in subclinical disease.

Our study has several strengths. It is the first study to examine whether changes in neighborhood characteristics influence the progression of CAC. It included a diverse sample from 6 different sites across the United States. It included detailed time-varying measures of neighborhood environments and state-of-the-art assessments of subclinical atherosclerosis. The study also has several limitations. We used a 1-mile buffer size for our neighborhood measures; however, relevant buffer sizes could be different for different exposures, for example, healthy food versus physical activity versus social engagement, and different distances may be relevant for different individuals.[52] Additionally, the study does not have information on whether participants actually use the nearby healthy food or physical activity resources, and we did not include other neighborhood environment variables such as green space, esthetic quality, or objectively measured crime rates. Workplace environments may have been more relevant for those participants who worked, and we did not characterize the neighborhood environment around participant workplaces. We explored a range of neighborhood-level variables; however, correlations between variables and power limitations inherent in the fixed-effects approach (which relies only on within-subject variability) precluded meaningful analyses of their independent effects. We also did not account for multiple testing in our models and comparisons.

The fixed-effects models that we used adjust for measured and unmeasured person-specific covariates related to residential location, but residual confounding resulting from unmeasured time-varying covariates cannot be ruled out. Changes in densities may also be proxying other environmental changes related to subclinical disease. Prior work has documented changes in CAC associated with aging comparable to the strength of the associations that we observed between a 1-SD increase in healthy food availability and changes in CAC.[53] Thus, our estimates of effects seem plausible. However, caution should be used in the interpretation of our results as causal, and further replication is needed before major public health implications can be drawn from this study.

The results from this longitudinal study provide new evidence that greater access to neighborhood healthy food resources may slow the development of coronary atherosclerosis in middle-aged and older adults. Our findings support the need to consider neighborhood and environmental interventions in the prevention of cardiovascular disease. Future research should examine the impact on cardiovascular risk of specific interventions such as promoting the location of healthy food stores and how neighborhood characteristics may interact with individual-level factors, including genetic predispositions.

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DISCLOSURES

None.

AFFILIATIONS

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FOOTNOTES

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