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Background—Low socioeconomic position is known to be associated with cardiovascular events and atherosclerosis. Reasons for these associations remain a topic of research. Inflammation could be an important mediating mechanism linking socioeconomic position to cardiovascular risk.

Methods and Results—This cross-sectional study used data from the baseline examination of the Multi-Ethnic Study of Atherosclerosis (MESA), a study of 6814 men and women 45 to 84 years of age. Race- and ethnicity-stratified regression analyses were used to estimate associations of household income and education with C-reactive protein and interleukin-6 before and after adjustment for infection and medication use, psychosocial factors, behaviors, adiposity, and diabetes mellitus. Low income was associated with higher concentrations of interleukin-6 in all race/ethnic groups. Percent differences associated with 1-SD–lower income were 9% (95% confidence interval [CI], 7 to 11), 6% (95% CI, 1 to 10), 8% (95% CI, 4 to 11), and 8% (95% CI, 3 to 13) for whites, Chinese, blacks, and Hispanics. Low levels of education were associated with higher levels of interleukin-6 only among whites and blacks (percent difference in interleukin-6 associated with 1-SD–lower education: 9% [95% CI, 6 to 12] among Whites, and 7% [95% CI, 3 to 10] among blacks). Similar patterns were observed for C-reactive protein. Adiposity was the single most important factor explaining socioeconomic position associations, especially among blacks and whites. A smaller effect was seen for psychosocial factors and behaviors in all race groups.

Conclusions—Both household income and education are associated with inflammation, but associations vary across race/ethnic groups. Associations likely result from socioeconomic position patterning of adiposity and other factors. **(*Circulation*. 2007;116:2383-2390.)**

Key Words: inflammation ■ race ■ ethnicity ■ risk factors ■ socioeconomic factors

Cardiovascular events and subclinical atherosclerotic disease are more common in lower than in higher socioeconomic groups in industrialized countries.[1,2] Reasons for these differences remain a subject of research. Over the last decade, considerable evidence has accumulated in support of the importance of inflammatory processes in the development of atherosclerosis.[3,4] Multiple pathways exist through which socioeconomic circumstances could be related to systemic inflammation. Inflammatory processes are related to several cardiovascular risk factors that are socioeconomically patterned, including smoking, obesity, alcohol use, and physical activity.[5–8] Socioeconomic differentials in the prevalence of other determinants of inflammation such as chronic and infectious disease and the use of various drugs[9,10] also may play a role. In addition, socioeconomic position (SEP) could affect inflammation through stress-mediated factors and psychosocial processes involving the hypothalamic-pituitary-adrenal axis and the sympathetic and parasympathetic nervous systems.[11,12] The accumulation of these factors could result in large socioeconomic differences in systemic inflammation with important consequences for cardiovascular risk. Thus, inflammation could be a common biological process through which the clustering of behavioral and psychosocial factors in lower socioeconomic groups creates the large and still relatively unexplained social inequalities in cardiovascular disease observed in many populations.

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Associations of low SEP with higher concentrations of inflammatory markers have been reported in European populations,[13–16] but US studies are limited.[17–19] One US study failed to find an association of poverty with the low-grade inflammation that is typical of atherosclerotic risk.[19] The 2 other US studies focused on restricted samples (the mostly white Framingham Offspring Study[17] and a sample of well-functioning adults 70 to 79 years of age[18]). Thus, the range of

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Table 1. Distribution of Covariates by Race/Ethnicity and Composite Measure of SEP: MESA, 2000 to 2002

	Male, %	Mean Age, y	Median Education, y	Median Income, US $	With Psychosocial Stress, %	Current Smokers, %
Entire sample (n=6814)	47	62.1	14	37	35	13
White (n=2624)						
Low SEP (15%)	32	68.6	12	18	47	23
Mid SEP (34%)	43	63.0	14	42	32	13
High SEP (51%)	56	60.5	20	87	25	6
P					<0.0001	<0.0001
Chinese (n=803)						
Low SEP (46%)	36	65.4	10	14	28	6
Mid SEP (28%)	53	61.1	16	27	20	3
High SEP (26%)	65	58.2	20	87	25	2
P					0.708	0.0418
Black (n=1895)						
Low SEP (29%)	38	65.6	12	14	47	24
Mid SEP (39%)	44	61.4	14	37	34	17
High SEP (32%)	51	59.8	20	87	28	11
P					<0.0001	<0.0001
Hispanic (n=1492)						
Low SEP (62%)	43	63.2	4	18	53	12
Mid SEP (28%)	55	58.3	14	42	38	11
High SEP (10%)	62	57.3	16	87	28	5
P					<0.0001	0.0154

Composite SEP measure used here is a combination of education and income (sum of *z* scores of education and income); low, mid, and high refer to tertiles of this score. Probability value for trend here is based on regression of covariate against the continuous version of the measure. Psychosocial stress is defined here as being in the highest 10% of cynical distrust, CES-D score, or chronic burden. Physical activity score is a measure incorporating minutes and intensity of physical activity.

socioeconomic experience examined and the diversity of the populations studied are limited, making it difficult to determine the population impact and public health importance of these associations. We investigated associations of 2 markers of SEP, education and household income, with 2 inflammatory markers in the Multi-Ethnic Study of Atherosclerosis (MESA), a large, diverse US cohort of adults 45 to 84 years of age. We also examined heterogeneity in these associations by race and ethnicity. Prior work has shown that the socioeconomic patterning of cardiovascular disease has varied over time and by social group (including race and ethnicity).[20,21] Documenting these heterogeneities and understanding their determinants will contribute to our understanding of the reasons for the social patterning of cardiovascular risk and the cause of atherosclerosis.

Methods

MESA is a longitudinal study of risk factors for subclinical atherosclerosis and its progression. The cohort includes 6814 men and women 45 to 84 years of age who were free of clinical cardiovascular disease at baseline and were recruited from 6 field centers using a variety of population-based approaches. Details of the study design have been given elsewhere.[22] The cohort is 38% white, 28% black, 22% Hispanic, and 12% Chinese. The baseline visit on which these analyses are based took place between July 2000 and September 2002.

Information on education, household income, race/ethnicity, age, behaviors, psychosocial measures, and medical history was obtained from the MESA baseline questionnaire. Education was classified into 3 groups: less than high school, complete high school or equivalent certification, and complete college or more. Household dollar income was bracketed into 3 categories: <20 000, 20 000 to 49 900, and ≥50 000. The first and third categories represent (approximately) the bottom 25% and the top 25% of the income range, respectively, in this population. For some analyses, continuous versions of years of education and household dollar income were rescaled to standardized scores by subtracting each value from the population mean and then dividing by the population standard deviation. For purposes of covariate assessment, a composite SEP measure was constructed by adding standardized income and education scores. Race and ethnicity were characterized on the basis of participants' responses to questions modeled on the Year 2000 US Census.

Behavioral risk factors included smoking, alcohol use, and physical activity. Cigarette smoking and alcohol use were categorized into current, past, or never use and supplemented by information on pack-years of cigarette use and number of drinks per week. Physical activity was assessed with a questionnaire adapted from the Cross-Cultural Activity Participation Study.[23] A composite measure of physical activity based on minutes and intensity (METs) of weekly activities was constructed and categorized into 4 levels. The psychosocial measures examined were cynical distrust, chronic stress, and depressive symptoms, which were previously found to be associated with inflammation in MESA.[24] Cynical distrust was based on an 8-item subset of the full Cook-Medley Hostility Scale that has been linked to atherosclerosis. The chronic stress scale[25] consists of 4 items asking participants to report if they had moderately or severely stressful ongoing difficulties in each of 4 domains: health of others, job or ability to work, finances, and relationships. Depression was assessed with the Center for Epidemiologic Studies–Depression (CES-D) inventory.[26]

Analyses also were adjusted for adiposity, recent infection, and use of medications known to alter the level of inflammation. Body

Table 1. Continued

Current Alcohol Users, %	Log %	Mean BMI, kg/m^2	Mean Waist Circumference, cm	With Diabetes Mellitus, %	With Recent Infection, %	Using Medications, %
55	6.7	28.3	98.2	14	24	49
52	6.6	29.2	102.37	10.2	20	55
66	7.0	28.1	99.14	6.9	19	58
82	7.0	27.1	96.11	5.5	20	60
<0.0001	<0.0001	<0.0001	<0.0001	0.006	0.6578	0.0235
25	5.1	23.8	86.68	16.0	21	25
32	5.4	23.9	86.97	12.7	20	25
34	6.2	24.4	88.08	12.7	20	38
0.0999	<0.0001	0.204	0.858	0.4551	0.4415	0.0099
39	6.5	30.2	102.06	23.7	30	49
48	7.2	30.2	101.54	18.7	28	48
60	7.6	29.7	99.85	15.6	23	46
<0.0001	<0.0001	0.0914	0.0022	0.0034	0.0021	0.1925
40	5.7	29.4	100.77	22.4	29	41
53	7.0	29.6	100.47	16.6	24	46
65	7.2	29.0	100.24	7.4	21	50
<0.0001	<0.0001	0.7982	0.229	0.0001	0.0214	0.0037

mass index was used as a measure of total adiposity, and waist circumference was used as a measure of visceral adiposity. Participants were categorized as having a recent infection if they reported cold/flu, sinus, urinary or tooth infection, bronchitis, or pneumonia in the preceding 2 weeks. Medication use was defined as current use of lipid-lowering medications, nonsteroidal antiinflammatory drugs, or aspirin. C-reactive protein (CRP) analyses additionally adjusted for use of hormone replacement therapy by women. Diabetes mellitus (fasting glucose >6.99 mmol/L [>125 mg/dL] or use of hypoglycemic medication) and impaired fasting glucose (5.60 to 6.99 mmol/L [100 to 125 mg/dL]) were defined according to the 2003 criteria of the American Diabetes Association.

Inflammatory markers examined include interleukin-6 (IL-6) and CRP. Standardized methods were used for blood collection, processing, and shipping, with fasting morning phlebotomy and use of a central laboratory (Laboratory for Clinical Biochemistry Research, University of Vermont, Burlington). IL-6 was measured by ultrasensitive ELISA from R&D Systems (Minneapolis, Minn), and high-sensitivity CRP was measured by nephelometry using the BNII nephelometer (N High Sensitivity CRP, Dade Behring Inc, Deerfield, Ill).

All analyses were stratified to allow investigation of heterogeneity of effects by race/ethnicity. In age-adjusted models, no statistically significant heterogeneity by sex was present in SEP effects on inflammatory markers. Therefore, all models are adjusted for sex. Inflammatory markers were log-transformed for analyses and back-transformed to the original scale for presentation purposes for selected analyses. Gradients of key covariates, including psychosocial factors, behaviors, metabolic factors (adiposity and diabetes mellitus), infection history, and medication use, were examined along tertiles of the composite SEP measure. To assess associations of SEP with inflammation, we estimated age- and sex-adjusted mean levels of inflammatory markers by levels of each of the 2 SEP measures and tested for linear trends across categories.

We investigated the contribution of different sets of covariates to the socioeconomic differences observed by comparing estimates across a series of models. Model 1 (the base model) examined SEP associations adjusted for age and sex. Model 2 adjusted the base model for recent infection and medication use. In model 3, psychosocial factors (cynical distrust, chronic stress, and CES-D score) were added to the base model; models 4 and 5 added health behaviors (smoking, alcohol use, and physical activity) and metabolic factors (adiposity and diabetes mellitus), respectively, to the base model. Models 2 through 5 can be compared to assess the relative contributions of infections/medications, psychosocial factors, behaviors, and metabolic factors to the age- and sex-adjusted associations in model 1 (the base model). Model 6 is a comprehensive model with all covariates included. In these regression analyses, z scores of the continuous education and household income measures were used as predictors. Coefficients from these models can be expressed as percent differences in the inflammatory marker associated with a 1-SD difference in the predictor. Collinearity in these adjusted models was assessed by examining variance inflation factors.

The individual impact of each covariate on the coefficients for education and household income was further examined by means of radar plots.[27] Radar plots graphically illustrate how the associations between a given predictor (in our case, household income and education) and the outcome (each inflammatory marker) change as different covariates are singly added to the model. They also allow comparison of the magnitude of associations across subgroups after adjustment for different covariates.

All probability values reported correspond to 2-tailed tests and were considered significant at the 0.05 level. The study was approved by Institutional Review Boards at all participating institutions. All subjects gave written informed consent.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Of the 6814 MESA participants at baseline, education and household income measures were available for 6791 and

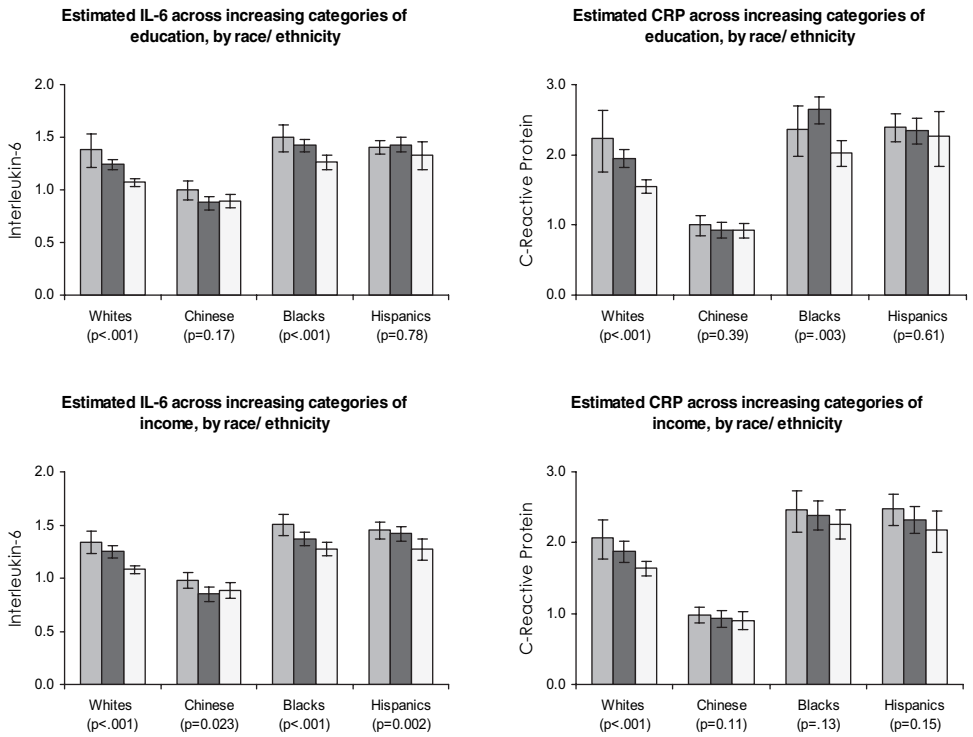

Figure 1. Age- and sex-adjusted geometric means of inflammatory markers by income, education, and race/ethnicity: MESA, 2000 to 2002. Estimates of IL-6 and CRP are geometric means obtained from models, are adjusted for age and sex, and are estimated at the mean age and sex of population. Low, middle, and high education categories refer to less than high school, high school completed, and college completed, respectively. Ranges for income categories are <$20 000 (US), $20 000–49 999, and ≥$50 000, respectively. *P* for trend was obtained from models testing continuous SEP measures in age- and sex-adjusted models.

6541 participants, respectively (<0.5% missing education and 4% missing income values). IL-6 assays were available in 97% of these participants (6599), and CRP was available for >99% (6739). Characteristics of the sample by race/ethnicity and the composite SEP measure are shown in Table 1. Similar patterns were observed for income and education, so only the composite score is shown. The mean age was 62.2 years; 47% were female. The median education and household income of the sample were 14 years and US $37 500, respectively. Means for income and education were 13.6 years (SD, 4.6 years) and US $51 500 (SD, $41 300), respectively. Whites showed the most favorable and Hispanics the least favorable SEP distribution. Psychosocial stress, current smoking, physical activity, and prevalent diabetes mellitus show marked gradation, with lower SEP groups having unfavorable risk profiles in each of the racial/ethnic groups. (The gradients for psychosocial stress and for diabetes mellitus among Chinese, however, are not statistically significant.) Current use of alcohol was more common in higher than in lower SEP groups. Patterning of body mass index and waist circumference by SEP is most marked among whites and, to a lesser extent, blacks. Low SEP is associated with recent infection among blacks and Hispanics. Medication use is associated with high SEP among whites and Chinese.

Age- and sex-adjusted geometric means of IL-6 and CRP across graded categories of household income and education are shown in Figure 1. Higher income was associated with lower concentrations of inflammatory markers in all race/ethnic groups (for heterogeneity in associations of income with inflammatory markers by race/ethnicity, $P=0.19$ for IL-6 and $P=0.23$ for CRP). Higher education was associated with lower levels of inflammatory markers in whites and blacks, but no statistically significant trend by education was observed in Hispanics or Chinese (for heterogeneity in associations of education with inflammatory markers by race/ethnicity, $P=0.0016$ for IL-6 and $P<0.001$ for CRP). Because of the statistically significant heterogeneity in the effects of education by race/ethnicity, all subsequent analyses were stratified by race/ethnicity. Income analyses also were stratified by race/ethnicity for comparison purposes.

Table 2 shows associations of standardized education in years (SD, 4.6 years) and standardized income in US dollars (SD, $41 300) with inflammatory markers before and after adjustment for sets of covariates. In general, all known risk factors for inflammation were associated with CRP and IL-6 in the expected direction, although associations were sometimes less consistent in Chinese (results not shown). Variance inflation factors did not exceed 3 in any model, indicating no substantial collinearity. In age- and sex-adjusted models (model 1), each 1-SD-lower education level is associated with a 9% higher level of IL-6 and a 14% higher level of CRP among whites; the corresponding effect sizes for blacks are 7% and 10%, respectively. Among Chinese and Hispanics, no association exists between education and IL-6 or CRP. Some reduction of the effect size for education among whites and blacks is seen after adjustment for behaviors (model 4), particularly for CRP, but it is only in the comprehensive model 6, which includes all covariates, that the association of education with either inflammatory marker disappears.

Table 2. Percent Difference in Inflammatory Markers Associated With 1-SD-Lower Education and Income After Adjustment for Covariates: MESA, 2000 to 2002

	Whites	*P*	Chinese	*P*	Blacks	*P*	Hispanics	*P*
Education								
IL-6 models, n	2562	...	792	...	1794	...	1451	...
Model 1 (age and sex)	9 (6 to 12)	<0.0001	3 (−1 to 7)	0.187	7 (3 to 10)	<0.001	0 (− to 3)	0.790
Model 2 (model 1+infection, medications)	9 (6 to 12)	<0.0001	3 (−1 to 7)	0.197	6 (3 to 10)	0.001	0 (−3 to 3)	0.775
Model 3 (model 1+psychosocial)	8 (4 to 11)	<0.0001	2 (−3 to 6)	0.393	5 (1 to 9)	0.014	−2 (−5 to 1)	0.268
Model 4 (model 1+behaviors)	6 (3 to 10)	<0.001	2 (−2 to 6)	0.365	5 (1 to 8)	0.015	0 (−3 to 3)	0.938
Model 5 (model 1+metabolic)	4 (1 to 8)	0.007	2 (−2 to 6)	0.237	4 (1 to 8)	0.016	0 (−3 to 2)	0.887
Model 6 (all covariates)	2 (−2 to 5)	0.371	0 (−5 to 5)	0.964	1 (−3 to 5)	0.525	−3 (−6 to 0)	0.073
CRP models, n	2599		800		1854		1486	
Model 1 (age and sex)	14 (9 to 19)	<0.0001	3 (−4 to 9)	0.393	10 (3 to 15)	0.004	1 (−4 to 6)	0.625
Model 2 (model 1+infection, medications)	16 (11 to 21)	<0.0001	4 (−3 to 10)	0.290	9 (3 to 15)	0.004	1 (−4 to 6)	0.615
Model 3 (model 1+psychosocial)	14 (8 to 19)	<0.0001	6 (−1 to 12)	0.120	10 (3 to 16)	0.008	1 (−5 to 6)	0.844
Model 4 (model 1+behaviors)	11 (5 to 17)	<0.001	2 (−5 to 9)	0.509	8 (1 to 14)	0.024	1 (−4 to 6)	0.732
Model 5 (model 1+metabolic)	7 (2 to 12)	0.011	3 (−4 to 9)	0.374	6 (0 to 12)	0.065	1 (−4 to 5)	0.763
Model 6 (all covariates)	6 (0 to 11)	0.039	5 (−1 to 11)	0.125	4 (−3 to 11)	0.279	−1 (−6 to 5)	0.792
Income								
IL-6 models, n	2502		787		1659		1417	
Model 1 (age and sex)	9 (7 to 11)	<0.0001	6 (1 to 10)	0.022	8 (4 to 11)	<0.0001	8 (3 to 13)	0.002
Model 2 (model 1+infection, medications)	9 (7 to 11)	<0.0001	6 (1 to 10)	0.017	7 (4 to 11)	<0.0001	8 (3 to 13)	0.002
model 3 (MODEL 1+psychosocial)	8 (6 to 11)	<0.0001	5 (1 to 10)	0.029	5 (1 to 9)	0.010	6 (1 to 11)	0.033
Model 4 (model 1+behaviors)	8 (6 to 10)	<0.0001	5 (0 to 10)	0.045	6 (3 to 10)	0.001	6 (1 to 11)	0.024
Model 5 (model 1+metabolic)	6 (4 to 8)	<0.0001	6 (2 to 10)	0.008	6 (3 to 10)	<0.001	6 (2 to 11)	0.009
Model 6 (all covariates)	5 (3 to 8)	<0.0001	5 (0 to 10)	0.039	3 (−1 to 6)	0.192	3 (−3 to 8)	0.313
CRP models, n	2537		795		1711		1451	
Model 1 (age and sex)	9 (5 to 13)	<0.0001	6 (−1, 13)	0.106	5 (−2 to 11)	0.139	7 (−2 to 15)	0.106
Model 2 (model 1+infection, medications)	11 (7 to 14)	<0.0001	7 (0 to 14)	0.066	5 (−2 to 11)	0.130	6 (−3 to 14)	0.157
Model 3 (model 1+psychosocial)	8 (3 to 12)	0.001	7 (0 to 14)	0.061	5 (−3 to 11)	0.219	5 (−4 to 14)	0.283
Model 4 (model 1+behaviors)	8 (4 to 11)	<0.001	6 (−2 to 13)	0.128	3 (−3 to 10)	0.313	6 (−3 to 14)	0.187
Model 5 (model 1+metabolic)	4 (0 to 7)	0.048	8 (1 to 14)	0.033	3 (−3 to 9)	0.306	5 (−3 to 13)	0.215
Model 6 (all covariates)	4 (0 to 8)	0.051	9 (2 to 15)	0.015	1 (−6 to 8)	0.717	1 (−9 to 9)	0.875

Model 1 (the base model) controls for age and sex. Model 2 is base model plus recent infection and use of medication in which models for CRP add use of hormone replacement therapy to medication use. Model 3 is base model plus cynical distrust, chronic burden, and CES-D score. Model 4 is base model plus behavioral risk factors (smoking status, pack-years of cigarette use, number of alcoholic drinks per week for current and former drinkers, and quartiles of physical activity). Model 5 is base model plus metabolic factors (body mass index, waist circumference, and diabetes/impair glucose tolerance. Model 6 includes all covariates. Values are expressed as n or % difference (95% confidence interval).

In contrast, lower household income is associated with higher levels of IL-6 in all race/ethnic groups after adjustment for age and sex, although associations are weaker in Chinese than in the other groups. This association is largely unchanged across multiple model specifications. The magnitude of income associations with IL-6 is slightly reduced in the comprehensive model 6 among blacks and Hispanics. Similar patterns across models are seen in the CRP analyses, but effect sizes are smaller and associations are usually not statistically significant, except for whites in models 1 through 6 and for Chinese in models 5 and 6.

Figure 2 shows results for each covariate separately in the form of radar plots. Radar plots use polar coordinate systems to represent multivariate data. The radius in the twelve o'clock position in these plots represents the size of the SEP effect (household income or education standard deviation units) when adjusted for age and sex in a baseline model. Each of the other radii in these plots represents 1 covariate, and the length of the radius represents the estimate of the same SEP effect after adjustment of the baseline model for the corresponding covariate. The ends of radii are connected to form a polygon. Irregularities in the shape of the polygon (when the SEP in the base model serves as a reference point) reflect a strong contribution of the covariates to the SEP effect. Plots in Figure 2A show race- and ethnicity-specific associations of household income and education with IL-6; plots in Figure 2B are for CRP. The same scale is used across plots within each panel to facilitate comparison across race/ethnic groups and SEP measure for each inflammatory marker.

Education associations are similar to or larger than income associations among whites and blacks, whereas among His-

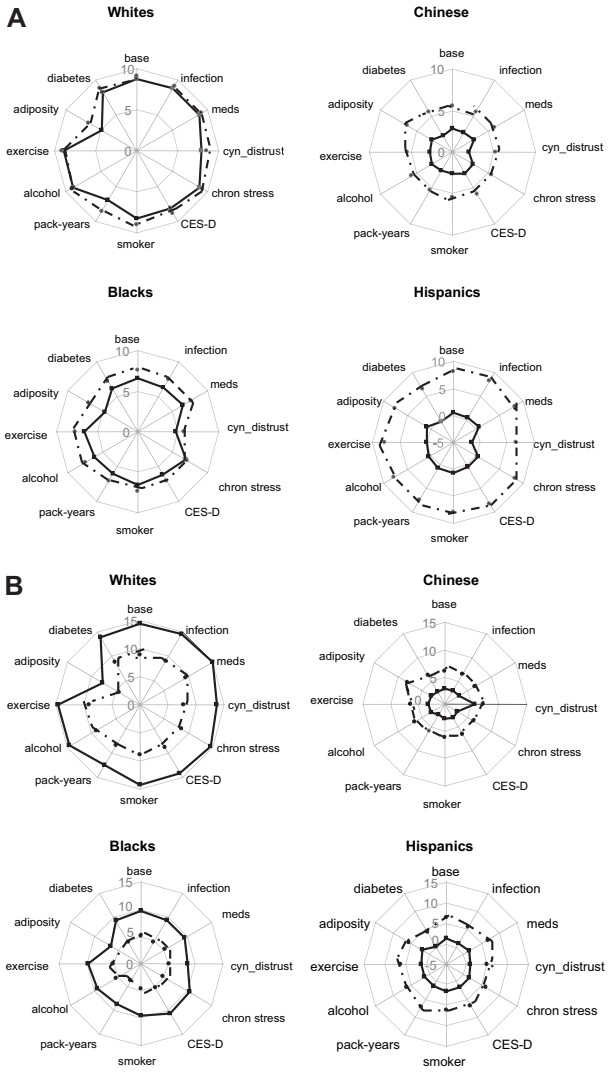

Figure 2. Size of income and education coefficients in models for IL-6 (A) and CRP (B) after adjustment for each covariate: MESA, 2000 to 2002. Income and education coefficients are represented by dotted and solid lines, respectively. Radar plots show changes in size of age-and sex-adjusted socioeconomic associations ("base" model) after adjustment for each of several covariates. To facilitate comparison across race/ethnic groups, the same scale is used across all plots within a given panel. The effect size displayed on each radius corresponds to the percent difference in the inflammatory marker associated with a 1-SD–higher SEP measure. For example, a unit increase in education is associated with a 15%-lower CRP level among whites (B; solid polygon, "Base" model, 12 o'clock), compared to 10% among blacks, <5% among Chinese, and close to 0% among Hispanics. Further, among whites, the baseline education effect of 15% is decreased to 8% after adjusting for adiposity, and to 12% after adjusting for pack-years of cigarette use. Cigarette and alcohol use, body mass index, waist circumference, cynical distrust, depression, and physical activity were entered in these models as standardized variables, whereas categorical measures were used for the remaining variables.

panics and Chinese, income associations are larger than education associations. Education associations are clearly stronger in whites and blacks than in Chinese and Hispanics. A considerable fraction of the education and income associations among whites and blacks is explained by adiposity; in contrast, among Chinese and Hispanics, less evidence exists of confounding/mediation of the income and education effect by adiposity. Cynical distrust appears to contribute to the associations of education with IL-6 among all race/ethnic groups, whereas both smoking status and extent of smoking (pack-years of cigarettes) appear to contribute to the association of education with IL-6 among blacks and whites. In general, radar plots are more symmetric among Chinese and Hispanics, indicating that among these groups, SEP associations result from the combined small effects of multiple factors.

Estimates remained largely unaffected by use of race/ethnicity-specific z scores for household income and education, by adjustment of income models by the education measure (and vice versa), by use of a per-capita income measure, and by restriction of the sample to adults self-rating their overall health as better than "fair." We also examined whether age- and sex-adjusted SEP associations with inflammation in the Hispanic sample differed by level of acculturation as indexed by language spoken at home. When stratified by language spoken at home (English versus all others), clear differences in the education coefficients were seen, with the estimated IL-6 and CRP effects at ≈10% among English-speaking Hispanics compared with 0% and 3%, respectively, among all other Hispanics. Similar direction of effects was seen when the Hispanic sample was stratified by US nativity. Lack of variability in the language measure among Chinese (<6% spoke English at home, and <4% were born in the United States) precluded these analyses in the Chinese sample.

Discussion

In this cross-sectional analysis of adults 45 to 84 years of age who were free of clinical cardiovascular disease, lower SEP was associated with higher concentrations of the inflammatory markers IL-6 and CRP, although these associations varied by race/ethnicity. In age- and sex-adjusted models, household income was inversely associated with levels of inflammatory markers in all race/ethnic groups, with 1-SD-unit-lower income (approximately US $40 000) corresponding to 6% to 9% higher IL-6 and CRP (depending on race/ethnic group), with the largest association seen among whites. In contrast, associations of education with these markers were inconsistent across race/ethnic groups. Among whites and blacks, each 1-SD-unit-lower level of education corresponded to 6% to 14% higher levels of IL-6 or CRP, whereas among Chinese and Hispanics, no associations of education with inflammation were observed. The associations of SEP with inflammatory markers may reflect multiple pathways. Only in models simultaneously controlling for numerous factors, including use of medication and infection, psychosocial measures, behaviors, and metabolic factors, were these associations attenuated to nonsignificance. When covariates were examined singly for each race/ethnic group, SEP measure, and inflammatory markers, adiposity, smoking, and cynical distrust emerged as important covariates in these associations, although their importance varied across race/ethnic groups. Adiposity was consistently the most important mediator of associations among blacks and whites for both SEP measures.

Past work in US samples has reported graded associations of CRP and IL-6 with income categories in a sample of healthy black and white adults 70 to 79 years of age[18] with no evidence of racial heterogeneity. These findings are validated in the present study with a larger age range (45 to 84 years); moreover, we also show similar associations among Chinese and Hispanics. A recent National Health and Nutrition Examination Survey (NHANES) report, however, showed an association of income level (defined as above or below poverty level) only with very high levels of CRP (>10 mg/L).[19] This failure to detect associations across the continuum of CRP may reflect limited resolution of the income measure used in that study and the lack of analysis of CRP as a continuous variable.

The strong association of education with both IL-6 and CRP among whites that we report is consistent with findings from the largely white Framingham Offspring cohort.[17] However, we found no association of education with inflammatory markers in Hispanic and Chinese participants. Among Hispanics, the weaker educational patterning of inflammation was restricted to Hispanics who did not report English as the primary language spoken at home and were presumably less acculturated. Among English-speaking Hispanics, the observed effect size for education was comparable to that observed in whites and blacks in the sample. This differential patterning of inflammation by education in less acculturated Hispanics could reflect measurement issues or differences in the behavioral, psychosocial, and metabolic correlates of education, depending on the country of origin.[28] In contrast to income, which may change over time, education is acquired early in life and is unlikely to change in adulthood. Thus, education patterns may reflect risks factors acquired earlier in life following the social patterning in the country of origin, whereas income patterning may reflect SEP patterning in the country of residence.

We also explored possible pathways contributing to the SEP associations with inflammation. Overall results from regression analyses and inspection of radar plots underscore the fact that factors explaining SEP associations vary across race groups. Among whites and blacks, adiposity and, to a lesser extent, smoking play an important role in explaining associations of SEP with inflammation. The contribution of adiposity to the socioeconomic patterning of inflammation has been previously noted.[15,16,18] Cynical distrust, an additional important covariate in these associations, appears to play some role across all race/ethnic groups, particularly with respect to associations with education. This finding adds to previous MESA data reporting an association of cynical distrust with inflammation.[24] Although we found no evidence of statistically significant interactions between sex and SEP with respect to inflammatory markers, future work will need to examine whether the relative importance of mediators varies by sex.

In cross-sectional analyses such as these, causal pathways between SEP and inflammation cannot be inferred. However, it is unlikely that inflammation causes lower SEP in healthy people. The fact that our sample comprised generally healthy adults (and that findings were similar when restricted to those reporting good or better health) suggests that these patterns are not the result of the presence of chronic diseases in the lower SEP groups. Although the size of the associations may appear small, a 14% higher CRP and an 8% higher IL-6 (corresponding to the observed effect of a 1-SD increase in education and income, respectively, among whites) are equivalent to the difference in CRP associated with an $\approx$1.7-kg/m^2-higher body mass index. In other work, a CRP difference of 14% is approximately equivalent to the CRP reduction seen with statin therapy.[29]

Our results suggest that persons of lower SEP have greater inflammatory burden than those of high SEP as a result of the cumulative effects of multiple behavioral psychosocial and metabolic characteristics. If the role of inflammation in the origin of multiple chronic diseases[30] is confirmed, inflammation may represent a common element through which SEP is related to cardiovascular disease and other chronic disease common in aging.

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Disclosures

None.

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CLINICAL PERSPECTIVE

Persons of low socioeconomic position are known to be at higher risk of cardiovascular disease, but the reasons remain unknown. This article examined whether levels of systemic inflammation (including C-reactive protein and interleukin-6) are related to socioeconomic position and hence could mediate socioeconomic differences in cardiovascular risk. The study used data from the baseline examination of the Multi-Ethnic Study of Atherosclerosis (MESA), a study of 6814 men and women 45 to 84 years of age. Low income was associated with higher concentrations of interleukin-6 in whites, blacks, Chinese, and Hispanics, with 6% to 9% higher levels of interleukin-6 seen for each 1-SD–unit increase in income. Low education also was associated with higher levels of inflammatory markers in blacks and whites but not in the other race/ethnic groups. Similar patterns were seen with C-reactive protein. Adiposity was the single largest factor explaining these associations, especially among blacks and whites. Smoking and psychosocial factors played a smaller role in these associations. Persons of low socioeconomic position may be at higher risks of cardiovascular disease because of higher inflammatory burden resulting from adiposity and other behavioral and psychosocial factors.