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# Socioeconomic indices as independent correlates of C-reactive protein in the National Longitudinal Study of Adolescent Health

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## Abstract

**Objectives—**Examine the association between SES and C-reactive protein (CRP) to understand how SES may increase the risk of CVD and thus identify targets for prevention measures.

**Methods—**Path models were used to examine direct and indirect associations of four indices of SES (objective early life built environment ratings, parental and participant education, and income) with CRP measured during early adulthood using data from the Add Health Study (N=11,371, mean age 29, range 24–32 years; 53.8% women, 28.0% black participants). The present study examined potential mediation of the association of SES with CRP by way of body mass index [BMI], smoking, and alcohol consumption within White and Black males and females.

**Results—**BMI was a mediator of the relation between parent education and CRP for White males (path coefficient (γ) = −0.05, $p<0.001$) and females (γ = −0.05, $p<0.001$). Smoking mediated the income-CRP (γ = −0.01, $p< 0.01$) and the education-CRP (γ = −0.07, $p< 0.001$) relation for White males. BMI mediated the relation between all measures of SES and CRP for White females (γ ‘s between −0.02- and −0.05; *p’s* <0.01). None of the risk factors mediated the SES-CRP relation in Black participants.

**Conclusions—**These findings indicate that the association of SES with CRP are influenced by both the timing and type of SES measure examined. In addition, race and sex play a role in how BMI and smoking influence the SES-CRP relationship, such that both factors play a role in white males and BMI in white females.

### Keywords

C-Reactive Protein; Add Health; Race; Sex; Socioeconomic Status

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## Introduction

Cardiovascular disease (CVD) represents the most prevalent cause of mortality and one of the most significant causes of morbidity in industrialized nations (1, 2). Strong links have been reported between socioeconomic status (SES) and CVD (3, 4) and CVD risk factors (5–8). Chronic inflammation is a known risk factor for diabetes (9), hypertension (10), and CVD (11). C-reactive protein (CRP) CRP has been used extensively in research settings as a marker of inflammation in order to study the association between inflammation and CVD. Although a causal role has not been definitively established for inflammation with respect to CVD, some have posited that intervening to reduce CRP levels may in turn reduce the risk of myocardial infarction (12). CRP is also inversely related to SES (13), suggesting that CRP may be one possible causal mechanism at least partly explaining the relation between SES and CVD. Thus, understanding the pathways linking SES and CRP in young adulthood may help to better understand how SES leads to increased development and rates of CVD later in the life cycle.

In addition to SES, race and sex are known to be associated with CPR levels (13) and also with the association between CRP and CVD events (14). Race apparently moderates the association between SES and CRP (15). Results from three large prospective studies (i.e., Coronary Artery Risk Development in Young Adults (CARDIA), NHANES IV, and Atherosclerosis Risk in Communities (ARIC) studies) that have considered race and sex have indicated the necessity to further examine these potential differences. (15,16,17). While not conclusive, effects of SES have generally been stronger among Whites, as compared to Blacks, and some evidence suggests that effects may be stronger in women, as compared to men (15).

In addition to consideration of race and sex, examination of the CVD risk factors obesity, tobacco smoking, and heavy alcohol intake also have informed the SES-CRP relation. In studies using data from CARDIA (15), ARIC (16), and NHANES IV (17) the risk factors of obesity or BMI, smoking, and drinking, among others, were examined as adjustment variables. Generally speaking these studies indicate that these risk factors mediate the relation between SES and CRP, but do not necessarily account for the entire association.

Thus, race, sex, and CVD risk factors are clearly meaningful with regard to the relation between inflammation and SES. In addition, participant age may be important to consider in studying SES and inflammation. In CARDIA, ARIC, and NHANES the baseline age of participants was 18 or older. The National Longitudinal Adolescent Health Study (Add Health) provides the opportunity to examine the SES-CRP relation in a group of individuals who are younger at than those studied in other large prospective studies. Understanding how SES relates to this important biomarker in younger individuals is critical, as this could provide a window of 20 years or more to intervene before major heart disease has time to develop.

Specific indicators of SES, as well as the timing of SES assessment, have also received considerable attention in prior work. Although an individual's current SES is considered important, SES over the life course, especially conditions during early childhood, also may be highly relevant with respect to health outcomes (4). Findings from CARDIA indicate that participant income and education in adulthood, as well as childhood SES and early family environment, are associated with levels of CRP in adulthood and that these associations are partially mediated by CVD risk factors (18). Data from ARIC also indicate that childhood indices of SES, as well as adulthood measures, are related to adult CRP (16). While these studies are longitudinal, they are not nationally representative (representative only in selected locations) and childhood SES is measured retrospectively and therefore subject to

recall and other measurement errors. While nationally representative, NHANES IV, on the other hand, is cross-sectional (i.e., does not follow people over time). Due to the wide range of ages in NHANES IV, and thus the potential of cohort effects with regard to the meaning of education, only income was examined (17). Therefore, examination of a variety of measures of SES, with assessment during childhood and young adulthood, has the potential to meaningfully contribute to the existing literature.

The present study uses longitudinal data from a nationally-representative sample to replicate and extend these prior findings with respect to SES, CRP, CVD risk factors, race, and sex. The Add Health study has sufficient numbers of men and women within Whites and Blacks to examine moderation by both race and sex (19). In addition, the Add Health data set affords us the opportunity to include an objective measure of SES that reflects childhood "built environment" conditions, that is, the state of maintenance of the physical environment (e.g., broken windows, graffiti, etc.), as assessed by a trained study interviewer. Also, the present sample allows examination of the SES-CRP relation in individuals that are younger than those previously examined in large studies, and measures childhood SES at baseline (i.e., not retrospectively as most other studies to date).

Based on the above prior work it was hypothesized that: 1) associations between SES and CRP will be stronger among Whites as compared to Blacks, and stronger in women as compared to men, 2) behavioral factors will partially mediate the association between CRP and SES, with BMI being the strongest mediator, 3) early life SES and current SES indices will be independently associated with CRP in young adulthood. Use of path models to test these hypotheses, and to formally test the extent to which the association between SES and CRP is mediated by BMI, smoking, and alcohol intake will complement and extend, in a younger sample, prior work in this area (20, 21). Based on prior work (22–25) we focus on results for Whites and Blacks in the present study.

## Methods

### Participants

Add Health is a nationally representative sample of approximately 15,000 young adults that was designed to assess the effects of health and health-related behaviors during adolescence and into young adulthood. The study was reviewed and approved by the IRB at The University of North Carolina-Chapel Hill. Written consent was obtained for all data collection. The participants have been followed from grades 7 through 12 in 1995 through early adulthood in 2008–09 in 4 waves of data collection(26). There were 1,159 individuals who did not have CRP data, and further limiting the sample to White and Black participants with post-stratification sample weights resulted in a final analytic sample of 11,371.

### Measures

**Demographic Measures—**Age was assessed at Wave IV. Race was constructed from a series of self-report queries at Wave I.

**C-Reactive Protein—**High sensitivity C-reactive protein was assessed at Wave IV. Capillary whole blood was collected to provide Add Health with the biological specimens necessary to assay and interpret high sensitivity C-reactive protein. UW Lab Med constructed dried blood spot CRP assay calibrators from pooled human plasma with a negligible CRP concentration (negligible CRP plasma; UW Lab Med) spiked with CRP concentrate (Cell Sciences, Canton, MA) and serially diluted with negligible CRP plasma to the desired final CRP concentration. Four dried blood spot quality control samples were constructed from a separate pool of human plasma, either undiluted (high CRP concentration

quality control sample) or diluted with negligible CRP plasma to the desired final CRP concentrations: medium-high, medium, and low. Dried blood spot calibrators and quality control samples were sealed at −70°C in Ziploc bags with desiccant until use (Whitsel, et al. 2012: see "Add Health IV Documentation: Measures of Inflammation and Immune Function" at: http://www.cpc.unc.edu/projects/addhealth/data/guides/add-health-wave-iv-documentation-measures-of-inflammation-and-immune-function). The correlation between dried blood spot and plasma CRP was Pearson R = 0.98.

CRP values were transformed using the natural logarithm prior to analysis. CRP values > 10 mg/dL are typically interpreted as indicative of acute infection or injury (27), and are generally not considered when studying chronic inflammation. Therefore, CRP values > 10 mg/dL before transformation were treated as missing. An additional sensitivity analysis was performed that included the participants with CRP > 10 mg/dL and the conclusions did not differ from the reported analysis.

**BMI—**BMI was calculated as BMI = weight (kg)/height ($m^2$) and was assessed at Wave IV. BMI was modeled in its continuous form in primary analyses, but for descriptive purposes was reported in categories: BMI < 25 = normal weight; 25–29.9 = overweight; 30–34.5 = obese class I; 35–39.5 = obese class II; ≥ 40 = morbidly obese (28).

**Smoking Behavior and Alcohol Consumption—**Alcohol Consumption was defined as follows: 0 = non-drinker; 1 = occasional drinker, drink 2 or fewer days of the week; 2 = light, drink 5–7 days per week and 2 or fewer drinks (1 or fewer if female); 3 = moderate, drink 5 – 7 days per week, 3 drinks for men, 2 drinks for women; 4 = heavy, drink 5 –7 days per week, more than 3 drinks for men, more than 2 for women. Smoking was coded yes/no indicating current daily smoking. These variables were assessed at Wave IV, concurrent with the CRP measurement.

**SES Measures: Built Environment, Parental Education, Household Income, and Respondent Education—**A "built environment" measure (29)(rated by the field interviewer) was the sum of two Likert-type items assessed at Wave I in early adolescence regarding how well the building in which the respondent lived was maintained, and the surrounding buildings were maintained. For the present analyses, we reversed the item responses and summed them such that the total score had a possible range of 2–8 with higher scores reflecting better maintenance. Parent and/or respondent education was coded as the highest level reported (at Wave IV for respondent, Wave I for parent), as follows: 1= some high school or less; 2 = graduated high school; 3 = some college or vo-tech; 4 = bachelor's degree; 5 = some graduate school or more. Parent education was the highest level achieved for mother or father. Participants completing G.E.D.s were grouped in the lower level of education class along with those having some high school or less (30). Annual household income of the young adult respondent was assessed at Wave IV using ordered categories. In order to create an interval-scaled income measure, the midpoint value was assigned to each category, resulting in the 13 values ranging from $2,500 to ≥$150,000.

## Statistical Analyses

Descriptive statistics were calculated using SAS SURVEYMEANS for continuous variables and SURVEYFREQ for categorical variables in SAS v 9.1.2. Race and gender comparisons on mean levels and frequencies of study variables were carried out using PROC SURVEYREG or PROC SURVEYLOGISTIC in SAS 9.2 (Cary, NC), depending on the measurement properties of the dependent variable. For these analyses, we estimated two models for each variable of interest, a first with race and gender main effects and a gender by race interaction term and a second without that term. With the exception of the

comparison on age all other models were adjusted for age. These models incorporated design effects. The primary path analysis was conducted using the Mplus software (31). A single multiple group path model was estimated (stratified by race and sex), which is depicted in Figures 1–4. In this model, CRP is predicted by the three risk factors and the four SES variables, and the risk factors are predicted by the four SES variables. Age (not shown in the figure) was included as a covariate, adjusting all effects on the risk factors and CRP. Thus, the model includes direct effects from SES and risk factors to CRP and also indirect effects from SES to CRP by way of the risk factors. The coefficient for a direct effect from a given SES variable to CRP is adjusted for age, the remaining SES variables, and the risk factors; while the coefficient for a direct path from a given risk variable to CRP is adjusted for age, the remaining risk variables and the SES variables. Indirect, or mediated, effects from the SES variables to CRP via the risk variables are calculated by taking the product of the component paths involved a given indirect effect. The weighted least-squares with mean and variance adjustment (WLSMV) estimation procedure and theta parameterization were used. Missing data were managed in path models using the full information maximum likelihood procedure (FIML). Estimates were weighted using grand sample weights, and the school identification code was specified as a clustering variable. Application of poststratification weights allows the results to be generalized to adults of similar age and background in the U.S. population. A set of null hypothesis tests were also carried out in which each corresponding path from the SES variables to the mediators and CRP and from the mediators to CRP were equivalent across the four groups using the difftest option in Mplus recommended for WLSMV estimation. A separate model comparison was carried out for each path of interest. A set of preliminary analyses was conducted comparing the model results in which the drinking variable was treated as a linear continuous variable to a model in which the drinking variable is treated as a categorical variable. No qualitative difference in the results between the two approaches was observed. For the sake of simplicity, we therefore treated alcohol intake as a continuous variable with a linear effect. Given the large number of parameters estimated we used $p < 0.01$ to define statistical significance.

In order to enhance interpretation of path coefficients, the income variable was rescaled such that one unit was equal to $10,000. In addition, in order to facilitate model convergence, BMI was scaled such that one unit was equivalent to 10 $kg/m^2$. When an analysis requires the inclusion of design effects, Mplus produces probit coefficients for paths associated with categorical dependent variables. Probit coefficients assume a latent normal (z) distribution for the categorical variable involved. Therefore, in the present path model, coefficients associated with smoking as a dependent variable can be interpreted as the change in the underlying z-score of the smoking variable for each one unit increase in the predictor. Because CRP is transformed using the natural logarithm in our models, the coefficients associated with CRP can be interpreted in terms of expected percent change in CRP for each unit increase in the independent variable (calculated as $[e^{\gamma}-1]$ [100], where $\gamma$ is the path weight). The remaining paths can be interpreted in the usual manner--the expected change in the dependent variable for every one unit (or one level) increase in the independent variable. For example, the coefficient for the direct effect of respondent education on BMI represents the expected change in BMI for every increase in level of education.

## Results

Table 1 shows the background characteristics of the present sample of White and Black males and females. Mean age of the participants at the time of CRP collection was 29 years, ranging from 24–32. Gender by race interactions were observed for BMI, smoking, income, respondent education, and parent education. Compared to women, men exhibited lower CRP, higher income, slightly older age, greater alcohol intake, higher rates of smoking, and

lower education. Compared to Blacks, Whites had higher alcohol intake, greater rates of smoking, higher income, higher levels of respondent and parent education. The unadjusted correlations presented in Table 2 suggest that, even after applying a more conservative significance level of $p < .01$, most of the SES indicators were modestly associated with CRP among Whites, but very few of these associations were observed among Blacks. In addition, BMI was strongly related to CRP in all groups. Considering the sample as a whole in unadjusted analyses, CRP was associated with all four SES variables: Income ($r = -.25$, $p = .001$); respondent education ($r = -.10$, $p < .0001$); parent education ($r = -.11$, $p < .0001$); and built environment ($r = -.13$, $p < .0001$).

## Model Fit and Comparison

Fit indices indicated adequate fit of the model: Comparative Fit Index = .97, Tucker-Lewis Fit Index = .817, Root Mean Square Error of Approximation = .032, 95%CI = .026–.038. The global null hypothesis that all corresponding paths from the SES variables to the mediators and CRP and from the mediators to CRP were equivalent across the four groups was rejected (chi-square difference = 211.1 [57 df] $p < .001$). Tests of the three-way interactions (independent variable by gender by race) for each individual direct path were statistically significant ($p < .05$) for respondent education, parent education, and income predicting both smoking and BMI, respondent education and parent education predicting alcohol intake, and smoking predicting CRP (see Table e1 in supplement).

## Direct Effects

**SES → CVD risk factors: BMI, Smoking, and Alcohol intake—**Figures 1–4 present significant direct effects of SES on the CVD risk factors for White and Black males and females. There were no significant direct effects of SES measures on BMI for Blacks. In contrast, income, respondent education, parent education, and built environment were negatively associated with BMI for White females; and parent education was negatively associated with BMI for White males. Household income was negatively related to smoking for all participants with the exception of Black males; similarly, with the exception of Black females, respondent education was negatively related to smoking for all groups. Parent education was unrelated to smoking for all groups, and built environment was related only to smoking for White females. Income was positively associated with alcohol intake in White women while respondent education was negatively associated with alcohol intake in White men. Parent education was positively associated with alcohol use for both White groups. There were no significant relations between SES measures and alcohol intake in Blacks.

**SES → CRP—**In the path model, the direct effects of the SES variables on CRP represent the association between the SES variable and CRP after partialling the effects of the mediating variables. The only significant direct relation between SES and CRP after this partialling was a negative relation for respondent education within Black females. This result indicates that the association between respondent education and CRP in Black females was not explained by BMI, smoking, or alcohol intake. However, in analyses of three-way interactions reported above, the respondent education by gender by race interaction was not statistically significant, and therefore this coefficient should be interpreted with considerable caution, if interpreted at all. The lack of direct association between the SES variables and CRP among Whites also suggests that the association between SES and CRP observed in the unadjusted correlations (Table 2) is at least partly explained by the mediating variables.

**CVD risk factors → CRP—**Higher BMI was related to higher CRP in all four groups, with essentially the same magnitude of association, with one unit increase in BMI associated with about a 7.7% increase in CRP. Smoking was related to higher CRP in White males only.

### Mediation

**SES → (BMI, smoking, and alcohol use) → CRP—**Table 3 provides results of the significant indirect effects of SES on CRP, which were only found among Whites in the present sample. For White men, a one unit increase in parental education achievement was related to a 4.9 percent decrease in CRP via BMI. Similarly, in White men a one unit increases in income and education were associated with 1.0 and 7.3 percent decreases in CRP, respectively, via smoking. Among White women, one unit increases in income, respondent education, parent education, and built environment were indirectly associated with 2.0, 3.9, 3.0, and 3.0 percent decreases in CRP, respectively, via BMI. Lastly, there were no indirect effects of the SES variables on CRP via alcohol in any group.

### Additional Analyses

Estimates were recalculated in order to examine whether or not inclusion of medications known to affect CRP (i.e., adrenal cortical steroids, oral contraceptives, antihyperlipidemic agents, inhaled corticosteroids) would alter the findings. Similarly, we examined whether or not the substitution of waist circumference (WC) for BMI would alter the results. Neither inclusion of medication status, nor substitution of BMI with WC resulted in a substantive change with regard to the results.

## Discussion

The present paper examined multiple indices of SES as predictors of CRP in young White and Black males and females, and assessed the extent to which SES might be associated with CRP by way of the mediating variables BMI, smoking, and alcohol consumption. Each of our hypotheses received support in the Add Health sample. Specifically, in Whites only, SES indices were associated with CRP, via mediation with only BMI mediating effects of all four SES indices on CRP in women. Among White men BMI mediated effects of parent education on CRP and smoking mediate effects of HH Income and respondent education. In contrast, among Blacks there were no significant indirect effects and the only significant direct effect was respondent education in Black females.

Across multiple samples that vary by age as well as measurement of SES, the relation between CRP and SES appears to be consistently stronger among Whites as compared to Blacks, e.g. (15). The reason for these differences could be primarily due to both qualitative and quantitative race differences in the social environment, or primarily due to within group differences in the biological mechanisms (e.g., genetic) that may control inflammatory processes, or perhaps a combination of these two potential forces. The results of the present study clearly suggest that the differential operation of mediators of the relation between SES and CRP across race groups that, in part, accounts for the stronger links within Whites. In their review of the SES and racial differences in CRP, Nazmi and Victoria (13) note that none of the studies examined adjusted for genetic markers, thus the inclusion of genetic information in future studies may help shed light on this consistently observed difference.

Although the focus of this study was the examination of CVD risk factors as mediators of the association between SES and CRP, it is also of interest to understand the direct paths between each of the measures in our full path model. These paths represent associations that are independent of one another, and also adjust for age. With regard to the direct relations between the CVD risk factors and CRP, increased BMI was strongly associated with increased CRP, and this relation was virtually identical in all four groups. Circulating CRP is considered to be a marker of obesity-related inflammation (32). Studies of the causal direction between BMI and CRP using Mendelian randomization have concluded that increased BMI is a cause of increased levels of circulating CRP (33,34). The link between

BMI and CRP in the present study is in concordance with prior work from NHANES where CRP was higher in adolescents with BMI at or above the 85th percentile than those with normal BMI (35). As previously reported, levels of obesity in this Add Health sample are high, with incidence over a 5-year period between adolescence and young adult report rising by 12.7% (36). In the present sample, with a mean age of 29, circulating levels of plasma CRP were above 3.0 mg/dL for 20% or more individuals within all groups. Given the evidence that obesity is a cause of elevated CRP, one important consequence of the high rates of obesity is that many of these young adults in the Add Health study also have levels of CRP that are considered abnormally high, which may in turn result in increased risk for early development of CAD.

Smoking has been related to increased levels of CRP in both adult and adolescent samples (37, 38). In the present study results from our adjusted models suggest that smoking was related to increased CRP only among White males. Similar findings have been reported in the MONICA study, where it was shown that serum CRP concentrations were higher in men who were regular smokers than men who reported never smoking (1.92 mg/l vs. 1.03 mg/l, $p < 0.001$), while no such difference was observed in females (1.52 mg/l for regular smokers vs. 1.41 mg/l for never-smokers) (39). These findings may be explained by the observation that females tend to smoke fewer non-filter cigarettes and use smaller and shorter puffs compared to males (40), although it is also possible that there are other factors not examined in the present study that may account for these differences.

Somewhat surprisingly, unlike BMI and smoking, there were no significant direct associations between alcohol and CRP observed in the present Add Health sample. Alcohol has been associated with lower levels of circulating plasma CRP in prior studies, e.g., (41), perhaps providing a beneficial effect with regard to development of cardiovascular disease. One possible reason for the present lack of association may have been that the effects of alcohol were insufficiently large to remain significant in our adjusted path models, but the unadjusted correlations presented in the Table 2 also reveal a lack of association in all groups. Thus, the lack of association in the present sample of young adults therefore warrants further investigation in the possible differences in methodology, including the age of study samples.

With respect to direct associations between SES and the CVD risk factors currently examined, significant effects were observed primarily among White participants, with relations that varied by sex and the specific index of SES. In White males only parental education was a predictor of BMI, with males who had parents with a higher education level more likely to have decreased BMI. In similar work in a German sample, parental education was the SES variable that was most strongly related to childhood obesity, as compared to other measures such as occupation and income (42)—highlighting the importance of consideration of such life-course measures. There are multiple plausible reasons that may underlie the present association between this early life marker of SES and BMI in this young sample. Parents with a lower educational level tend to bottle-feed their children more than parents with a higher educational level, and this may be related to both the association between this early life marker of SES and BMI, as well as CRP, as both obesity and immune functioning have been related to breastfeeding (43, 44). Differences in social norms between parents of high and low education may also account, in part, for the inverse associations between parental education and BMI, as healthy eating and exercise patterns have been show to correspond with higher levels of education (45). Another social consideration is the fact that marital conflict, negative life events, and lack of involvement in the lives of children are more often found in families with lower education levels—factors that may contribute to unhealthy eating patterns (46). The possibility that parental education level shapes behaviors in childhood that may lead to greater level of CRP in young adulthood is a

topic that Add Health is particularly well suited to examine in future work. For example, exploration of whether BMI, smoking, and alcohol use from adolescence to young adulthood (i.e., from Wave I to Wave IV) may underlies SES variations in CRP levels in young adulthood.

For White females all of the four SES indicators were related to lower BMI. Sex and race differences in the relation between BMI and SES levels have been reported in the California Health Interview Study (CHIS), consisting of approximately 37,000 participants. However, their findings suggest that increased respondent education is associated with lower BMI for both Whites and Black females, but the relation for Black males was curvilinear (47). In the present sample household income was negatively related to BMI only for White females, whereas, in CHIS income was negatively related in White and Black females, and Black males, but no significant relation was found in White males. Differences between the ages of the CHIS participants, who were between the ages of 25 and 64, and the Add Health participants who were all below the age of 32, as well as the differences in the methodology used (phone interview in CHIS, as compared to in-home assessments in Add Health) may help account for the discrepancy in findings between these two studies. Finally, more broadly speaking, differences in the measurement of household income may account for variations in results across different studies in this area. For example, some studies adjust for house-hold size or other financial demands, or additional financial resources.

Measures of SES that were concurrent with CRP were related to smoking behavior in the present study. Specifically, smoking was negatively related to current income in all participants, with the exception of Black males, and smoking was negatively related to participant education in all individuals with the exception of Black females. However, early life measures of SES were unrelated to smoking behavior for the majority of participants, a finding that corresponds to results from the national Panel Study of Income Dynamics (48). The only relation between smoking and early life SES was observed for White females, such that those with a better built environment were less likely to smoke later in life. Finally, with regard to direct associations between SES and CVD risk factors, there were few significant links between SES and alcohol consumption, with only income and parental education demonstrating associations. Specifically, higher income was related to greater alcohol consumption for White females, and higher parental education was related to greater amounts of alcohol consumption for both White males and females. Such findings are consistent with epidemiological evidence indicates a positive relationship between income and the prevalence of alcohol abuse in the general population (49), as well as research that suggests higher SES groups drink more frequently as compared to lower SES groups (50).

Our initial hypotheses *in toto* predicted that, with regard to the mediating factors that would account for the relation between SES and CRP, BMI would be the strongest mediator, and that effects would be most consistent among Whites and females. Indeed, BMI was the only significant mediator of the relation between SES and CRP for all four indicators of SES and this pattern of relations was observed only for White females. Importantly, the effects of these SES indicators were independent of one another in our path analytic approach. Thus, for example, the effects of built environment remained after adjustment for the other SES indicators, i.e., independent of one's education, income level, or parents' education level, the built environment in which these female participants lived as a child predicted higher BMI, which in turn predicted higher CRP at about 29 years of age. In addition to these striking effects in White females, BMI was also a mediator of the SES-CRP relation in White males such that higher parental education, adjusted for all other measures of SES, was indirectly related to higher CRP via BMI. The mediated paths that met our significance criterion of $p < .01$ ranged in effect size from an expected 2 to nearly 7 percent decreases in CRP for each one unit increase in a given SES variable. These effects are independent and thus also

potentially additive. In other words, the greater the number of SES disadvantages, the greater the risk of CRP. For example, if we compared a White woman at relatively high levels of personal and parental education, household income, and built environment to a person one level lower on each of those variables at a given time point, the statistical model suggests that the person with the more advantaged SES status would be expected to have a CRP level 12 percent lower than that of the person with the lower levels of SES.

As for the other two potential mediators considered in the present study, smoking mediated relations between respondent education and CRP, and income and CRP, but only among White males; and alcohol was not involved in any direct or indirect relations to CRP in any of the eight groups. In unadjusted correlations smoking was significantly associated with CRP in both White and Black males, and approached significance in White and Black females, indicating that the effects of smoking may have been confounded with other measures in our path models. Unlike smoking, alcohol was not significantly related to CRP in any of the groups examined using our path models, nor when examining unadjusted correlations, and therefore was not a viable measure with regard to mediation.

As noted earlier, the only direct effect between one of our four SES variables and CRP was observed for education among Black women. Although speculative, certain social determinants of health such as discrimination and or racism might be might be contributing to this SES-CRP relationship that did not occur through our hypothesized mediating pathways. It is known that depressive symptoms and hostility can modulate inflammatory immune responses and Mwendwa, et al. (51) have shown, in an all-Black sample, depressive symptoms and CRP were more strongly associated among participants with greater hostility and lower educational attainment. Thus, the chronic stress of racism may add to the existing stress that may accompany low levels of education thereby contributing to increased levels of CRP via mechanisms that have little or no relation to our examined mediators.

Pollitt et al. (16) examined the SES-CRP association using data from 12,681 participants enrolled in the ARIC study, and included both retrospective childhood and prospective adult measures of SES, as well as potential for mediation by biobehavioral risk factors. It is worth noting that although our modeling strategy, as well as some of the measures considered, differed from that used by Pollitt, et. al., (16) the general conclusions reached by the two studies are similar. For example, Pollitt, et. al., found that both childhood and adult measures of SES were associated with CRP, and furthermore that findings were more consistent among White as compared to Black participants, and finally that BMI and smoking were mediators of the SES-CRP relation. Given ARIC studies older adults, and Add Health studies younger adults, the similar findings suggest that these relationships are operate similarly across the life course.

It is important not only to understand these relationships in a younger cohort, but to consider the long range ramifications with regard to health and longevity for these individuals. At a mean age of roughly 29, many of these Add Health participants are just realizing their “own” achieved level of SES, and as a cohort they have likely been affected by the recent recession that occurred in 2008. It is unclear how these factors may influence future SES-health relations in this cohort, however, observed SES-CRP relationships that occur this early in life have the ability to impact health over a very prolonged period of time, adding to the importance of examining the impact that different measures of SES may have for different race and sex groups of this age.

To date, CRP has only been assessed at one point in time in the Add Health sample, thus the effects of SES on changes in levels of CRP over time cannot be addressed until the Add Health sample is followed up at Wave V. Due to the complexity of the models examined and

the number of groups considered, we did not consider additional potential mediators of the relation between SES and CRP and it is likely that other factors, such as psychosocial factors (e.g., stress, depressive symptoms) might have had an impact on the present results. Consideration of additional mediators, as well as other potential biological confounders will be a topic of future research in this sample. In addition, the methodological differences regarding blood sources for CRP measurement (e.g., dried blood spot vs. plasma from a standard blood draw via antecubital vein) may account for variations in findings with other studies, however, this is unlikely given the high correlation between CRP measured via both methods in the present study (r= 0.98).

As with all observational studies, the associations observed in the present study are potentially subject to unobserved confounding with unmeasured variables. Despite our adoption of the conventional terminology of “effect” when referring to our associations, we hasten to note that no statistical technique is capable of confirming causation. The current study is therefore limited to describing associations that, only with further work, may ultimately be shown to be causal. Nevertheless, path modeling can be a useful means of decomposing correlations into putative causal paths, identifying where the data may be consistent, or more importantly, inconsistent with those proposed paths.

In sum, we hypothesized that both current and early life measures of SES would be associated with CRP in the present sample. This hypothesis was supported, but primarily only for Whites, emphasizing the need to consider race. Our results highlight the fact that the association of SES with CRP may vary in important ways that are dependent upon both the timing and type of SES measure examined, as well as dependent upon the sex and race of the person. Sociological theories suggest that income may reflect direct access to material goods, whereas education levels may indicate access to non-material goods (52), and the specific pathways through which different measures of SES effect health outcomes such as CRP is an important area for continued work. Importantly, our findings add support for the existing theory that SES likely has a negative effect on cardiovascular disease due in part to its relation with CRP via higher levels of BMI, particularly within White populations. A major implication of the present finding is that we would predict in follow-up data collection White females with lower income and education, with parents’ having lower education and who lived in a tougher build environment during adolescence (and males with lower parent education, personal education and house hold income) will have higher BMI (BMI and smoking for males) leading to higher CRP and higher CHD incidents. Such findings may provide the ability to identify specific race and sex groups in whom lower SES will have stronger effects on CRP and hence CHD risk. Thus, interventions that target reduced BMI in White women, and reduced smoking and BMI in White men may lead to lower levels of CRP and ultimately reduced risk of CVD in these individuals.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## Abbreviations

| | |
|---|---|
| **CVD** | Cardiovascular disease |
| **SES** | Socioeconomic Status |
| **CRP** | C-Reactive protein |
| **CARDIA** | Coronary Artery Risk Development in Young Adults |
| **ARIC** | Atherosclerosis Risk in Communities |
| **BMI** | Body Mass Index |

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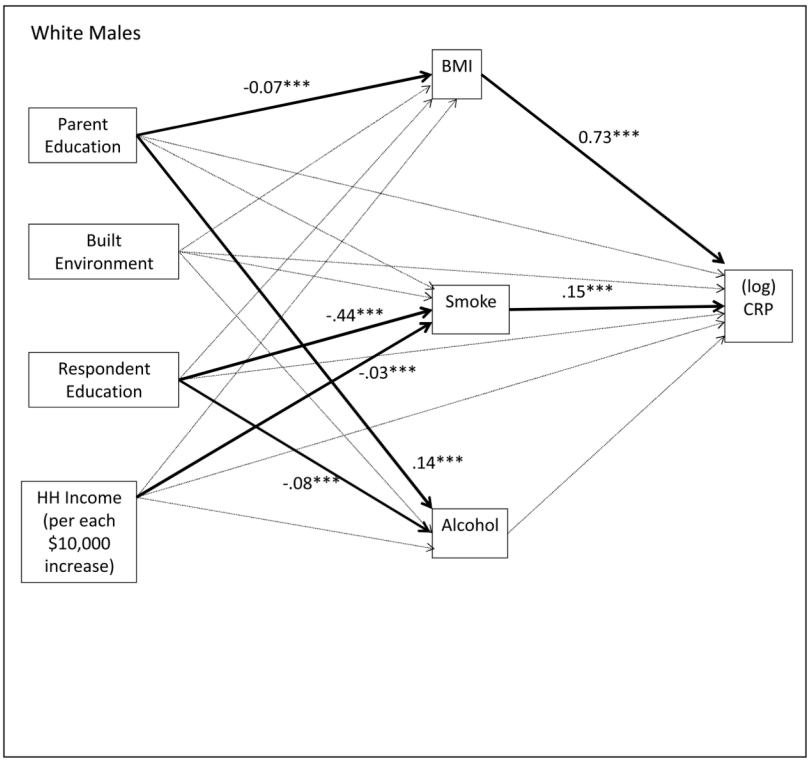


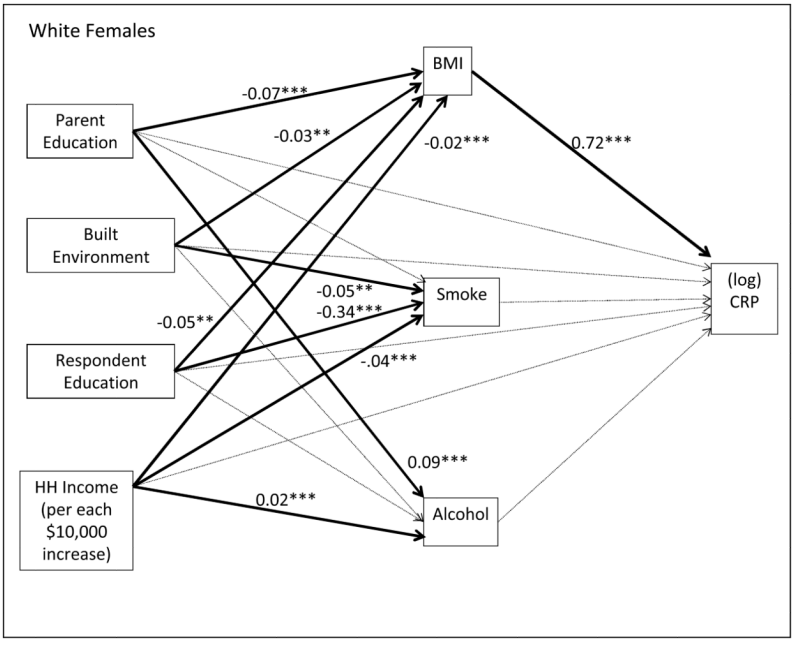

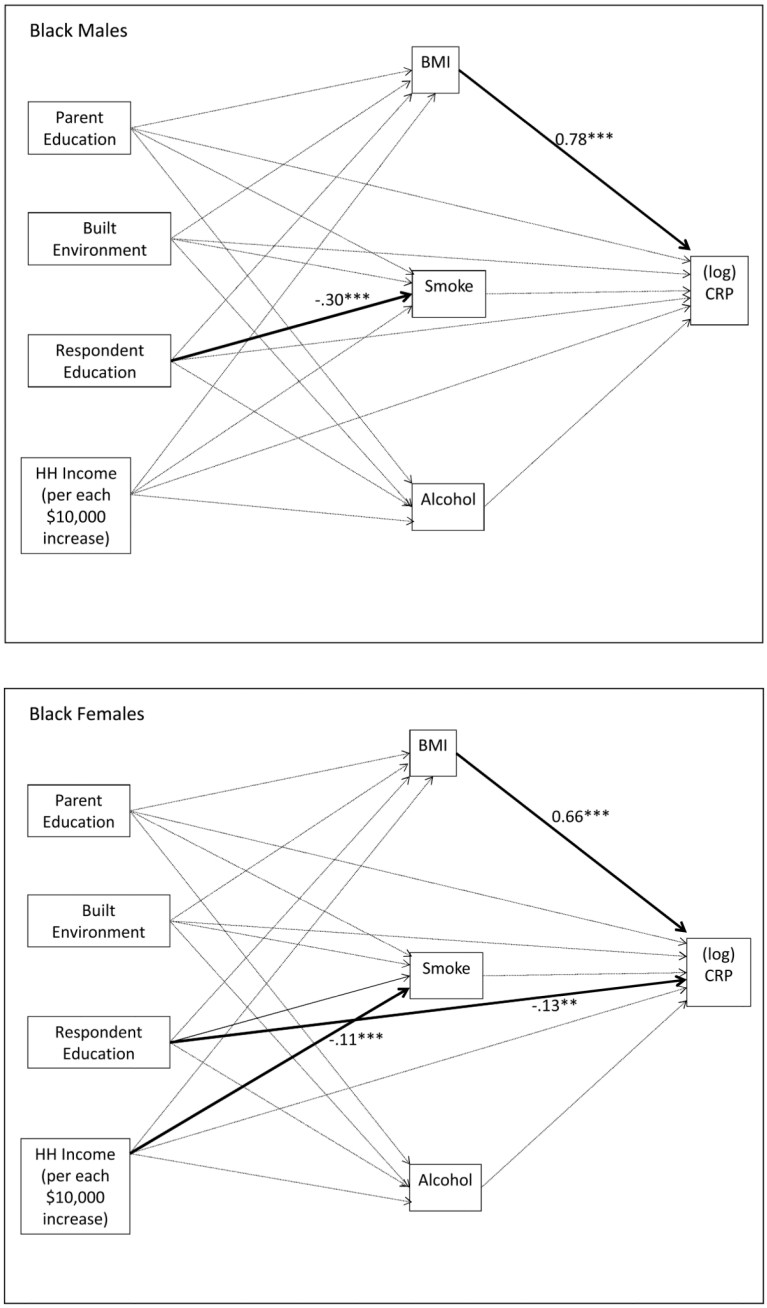


**Figure 1 – 4.**

Observed associations from path models (gray dashed lines indicate estimated paths that were not statistically significant at $p < 0.01$; solid lines are estimated paths that were statistically significant ** $p < 0.01$, and *** $p < 0.001$). Models were estimated using the mean- and variance-adjusted least squares (WLSMV) algorithm in Mplus. Parameter values from the SES-variables to Smoking are unstandardized probit coefficients. The remaining path values are unstandardized linear coefficients. The global null hypothesis of equivalent corresponding coefficients across the four groups was rejected ($p < .001$).

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**Table 1**

Sample Characteristics by Sex and Race

| | White Men N=3883 | White Women N=4304 | Black Men N=1371 | Black Women N=1813 | Gender Main Effect *p*-value | Race Main Effect *p*-value | Gender by Race interaction *p*-value |
|---|---|---|---|---|---|---|---|
| CRP, mg/dL, geometric mean (95% CI) | 1.30 (1.22, 1.37) | 1.73 (1.64, 1.81) | 1.36 (1.22, 1.52) | 1.77 (1.58, 1.97) | < .0001 | .50 | .78 |
| CRP > 3.0 mg/dL, % | 21.8 | 29.4 | 21.3 | 28.5 | < .0001 | .57 | .95 |
| BMI, kg/m$^2$, mean (95% CI) | 28.8 (28.5, 29.0) | 28.4 (28.0, 28.9) | 29.1 (28.5, 29.6) | 32.4 (31.8, 33.1) | .053 | < .0001 | < .0001 |
| Age, years, mean (95% CI) | 28.9 (28.6, 29.1) | 28.7 (28.4, 28.9) | 29.2 (28.8, 29.7) | 29.0 (28.5, 29.4) | < .0001 | .16 | .77 |
| Smoker, % | 30.1 | 26.9 | 23.5 | 12.0 | < .0001 | < .0001 | .0003 |
| Alcohol Intake, % | | | | | < .0001 | < .0001 | .43 |
| None | 19.7 | 24.4 | 37.5 | 44.6 | | | |
| Light | 61.5 | 67.6 | 51.2 | 51.3 | | | |
| Moderate | 4.1 | 3.2 | 3.1 | 1.9 | | | |
| Heavy | 3.1 | 1.8 | 2.1 | 1.4 | | | |
| Very Heavy | 11.5 | 3.0 | 6.1 | 0.8 | | | |
| Income X $1000, median (25$^{th}$, 75$^{th}$ quartile) | 51.1 (32.1, 74.9) | 49.9 (30.7, 72.9) | 36.7 (15.9, 58.5) | 29.7 (12.0, 50.5) | .009 | < .0001 | .001 |
| Respondent Ed, % | | | | | < .0001 | .0004 | .065 |
| < HS | 8.9 | 6.6 | 16.5 | 10.1 | | | |
| HS | 20.2 | 13.2 | 26.1 | 16.8 | | | |
| Some College | 41.5 | 43.2 | 40.2 | 48.0 | | | |
| 4 year degree | 19.7 | 22.3 | 10.3 | 13.1 | | | |
| Graduate/Professional | 9.8 | 14.7 | 6.9 | 12.0 | | | |
| Parent Education, % | 8.9 | 6.6 | 16.5 | 10.1 | .35 | < .0001 | .032 |
| < HS | 9.9 | 9.7 | 18.2 | 20.7 | | | |
| HS | 23.8 | 25.2 | 29.9 | 33.5 | | | |
| Some College | 32.9 | 30.7 | 28.0 | 26.6 | | | |
| 4 year degree | 18.9 | 18.8 | 13.9 | 11.8 | | | |
| Graduate/Professional | 14.5 | 15.6 | 10.1 | 7.4 | | | |
| Built Environment, median (25$^{th}$, 75$^{th}$ quartile)[a] | 7.05 (5.46, 7.52) | 7.11 (5.54, 7.56) | 5.77 (5.03, 7.21) | 5.69 (4.65, 7.21) | .30 | < .0001 | .15 |

[a] Higher scores indicate better maintained environment.

Note. Tests of group differences were carried out using PROC SURVEYREG for continuous response variables and PROC SURVEYLOGISTIC for categorical response variables. With the exception of the case where age is the response variable, all analyses were adjusted for age. Gender and race main effects terms were derived from a model that did not include the two-way interaction term.

**Table 2**

Correlations (unadjusted) among study variables

| Variable 1 | Variable 2 | White Men | | White Women | | Black Men | | Black Women | |
|---|---|---|---|---|---|---|---|---|---|
| | | r | *p* | r | *P* | r | *p* | r | *p* |
| CRP | AGE | .06 | .035 | −.01 | .51 | .10 | .033 | .06 | .27 |
| CRP | SMOKING | **.08** | **.003** | −.04 | .046 | **.16** | **.001** | −.07 | .086 |
| CRP | ALCOHOL | −.05 | .088 | −.04 | .12 | −.03 | .53 | −.01 | .83 |
| CRP | BMI | **.43** | **<.001** | **.46** | **<.001** | **.44** | **<.001** | **.50** | **<.001** |
| CRP | RESP. ED. | **−.14** | **<.001** | **−.06** | **.003** | −.10 | .025 | −.08 | .041 |
| CRP | PAR. ED. | **−.12** | **<.001** | **−.06** | **.009** | .02 | .71 | −.07 | .091 |
| CRP | INCOME | −.06 | .011 | −.06 | .010 | −.02 | .85 | .04 | .28 |
| CRP | BLT. ENV. | **−.10** | **<.001** | −.06 | .011 | −.06 | .17 | −.01 | .83 |
| AGE | SMOKING | −.05 | .073 | .01 | .66 | −.01 | .89 | −.01 | .81 |
| AGE | ALCOHOL | −.04 | .14 | **−.08** | **.004** | −.06 | .18 | −.03 | .55 |
| AGE | BMI | .04 | .099 | .04 | .065 | −.05 | .39 | −.05 | .26 |
| AGE | RESP. ED. | .01 | .80 | −.02 | .59 | −.08 | .36 | −.02 | .66 |
| AGE | PAR. ED. | −.05 | .22 | −.04 | .19 | −.16 | .063 | −.01 | .84 |
| AGE | INCOME | .06 | .046 | .07 | .013 | .02 | .78 | .08 | .11 |
| AGE | BLT. ENV. | −.05 | .087 | −.03 | .32 | −.13 | .14 | −.01 | .85 |
| SMOKING | ALCOHOL | **.09** | **.006** | .02 | .50 | **.17** | **<.001** | .01 | .91 |
| SMOKING | BMI | **−.11** | **<.001** | .02 | .44 | −.01 | .80 | −.06 | .14 |
| SMOKING | RESP. ED. | **−.35** | **<.001** | **−.31** | **<.001** | **−.24** | **<.001** | −.12 | .018 |
| SMOKING | PAR. ED. | **−.16** | **<.001** | **−.21** | **<.001** | −.08 | .11 | −.01 | .89 |
| SMOKING | INCOME | **−.18** | **<.001** | **−.18** | **<.001** | −.07 | .23 | **−.14** | **<.001** |
| SMOKING | BLT. ENV. | **−.15** | **<.001** | **−.17** | **<.001** | −.14 | .012 | −.05 | .27 |
| ALCOHOL | BMI | **−.09** | **.008** | **−.10** | **<.001** | −.07 | .101 | −.04 | .37 |
| ALCOHOL | RESP. ED. | .01 | .82 | **.14** | **<.001** | .05 | .31 | .09 | .12 |
| ALCOHOL | PAR. ED. | .07 | .011 | **.17** | **<.001** | .03 | .55 | .11 | .011 |
| ALCOHOL | INCOME | .06 | .024 | **.12** | **<.001** | .03 | .62 | **.13** | .001 |
| ALCOHOL | BLT. ENV. | .05 | .019 | **.09** | **.003** | .07 | .11 | .06 | .13 |

| Variable 1 | Variable 2 | White Men | | White Women | | Black Men | | Black Women | |
|---|---|---|---|---|---|---|---|---|---|
| | | r | *p* | r | *P* | r | *p* | r | *p* |
| BMI | RESP. ED. | −.05 | .047 | **−.17** | **<.001** | .06 | .26 | −.05 | .15 |
| BMI | PAR. ED. | **−.09** | **<.001** | **−.14** | **<.001** | .09 | .13 | **−.09** | .007 |
| BMI | INCOME | .03 | .28 | **−.15** | **<.001** | .06 | .34 | −.02 | .71 |
| BMI | BLT. ENV. | **−.06** | **.006** | **−.16** | **<.001** | .00 | .97 | −.03 | .41 |
| RESP. ED. | PAR. ED. | **.44** | **<.001** | **.45** | **<.001** | **.35** | **<.001** | **.48** | <.001 |
| RESP. ED. | INCOME | **.26** | **<.001** | **.32** | **<.001** | **.25** | **<.001** | **.47** | <.001 |
| RESP. ED. | BLT. ENV. | **.31** | **<.001** | **.32** | **<.001** | **.25** | **<.001** | **.25** | <.001 |
| PAR. ED. | INCOME | **.18** | **<.001** | **.21** | **<.001** | **.22** | **<.001** | **.36** | <.001 |
| PAR. ED. | BLT. ENV. | **.30** | **<.001** | **.28** | **<.001** | **.36** | **<.001** | **.27** | <.001 |
| HHINC | BLT. ENV. | **.17** | **<.001** | **.21** | **<.001** | **.21** | **<.001** | **.19** | <.001 |

Note: Correlations with *p*-values < .01 are in bold type. Correlations involving categorical variables correspond to polychoric correlations.

BLT. ENV: Built environment; BMI: Body Mass Index; CRP: C-Reactive Protein; Income: Annual Household Income; PAR. ED.: Highest level of parent education; RESP. ED.: Highest level of respondent education; Smoking: Current smoker; ALCOHOL: Average weekly alcohol intake.

## Table 3

Summary of significant indirect effects from path model

| INDIRECT Effects: estimate(CI) | White Men | | White Women | |
|---|---|---|---|---|
| | Unstandardized path coefficient | % change in CRP | Unstandardized path coefficient | % change in CRP |
| Income->BMI->CRP | .006 (−.002, .014) | 0.6 | −.02 (−.03, −.01) ** | −2.0 |
| Education ->BMI->CRP | .006 (−.03, .04) | 0.6 | −.04 (−.07, −.01) ** | −3.9 |
| Parent Education->BMI->CRP | −.05 (−.08, −.02) *** | −4.9 | −.05 (−.09, −.02) *** | −5.0 |
| Built Environment->BMI->CRP | −.01 (−.03, .009) | −1.0 | −.02 (−.05, −.01) ** | −2.0 |
| Income->Smoking->CRP | −.01 (−.01, −.001) ** | −1.0 | .003 (−.001, .008) | 0.3 |
| Education -> Smoking ->CRP | −.07 (−.11, −.02) *** | −7.3 | .02 (−.006, .05) | 2.0 |

** indicates $p < 0.01$;

*** indicates $p < 0.001$.

Notes: Only effects with $p$-values < 0.01 displayed. Income was scaled to units of $10,000 US; Percent change is the expected percent change in CRP levels for each one unit increase in the SES variable by way of the mediator; CRP is logged in analyses.