

Socioeconomic status and C-reactive protein levels in the US population: NHANES IV

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Abstract

C-reactive protein (CRP), a marker of inflammation, has been identified as a risk factor for cardiovascular disease and mortality. Using data on adults aged 20 and over from the fourth National Health and Nutrition Examination Survey, a nationally representative cross-sectional survey, we examined the association between socioeconomic status and CRP in US adults ($N = 7634$). Socioeconomic variation in CRP occurred only at very high levels of CRP (>10.0 mg/L). There was no significant difference in the prevalence of moderate (1.1–3.0 mg/L) or high values of CRP (3.1–10.0 mg/L) by socioeconomic status; however, among those with family income at or below the poverty level, 15.7% had very high levels of CRP (greater than 10.0 mg/L), compared to only 9.1% of those in families above the poverty level. Logistic regression results indicate that acute illness, chronic conditions, and differential health behaviors account for about two-thirds of this association. African Americans, Hispanics, and women were more likely to have high levels of CRP. Obesity was the largest risk factor for every level of CRP above normal. Results suggest that differences in very high CRP may be due to factors beyond acute illness and may also reflect chronic health, behavioral and disease processes associated with low socioeconomic status.

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1. Introduction

Socioeconomic status (SES) represents one of the most important risk factors for chronic disease, disability, and mortality. Low SES individuals are at greater risk for infectious illness (Cohen, 1999) and have a higher prevalence of sub-clinical markers of disease risk (Seeman et al., 2004) and chronic health conditions (Hayward et al., 2000). All-cause mortality is higher among persons of lower SES, and there is a marked socioeconomic gradient in mortality due to coronary heart disease, stroke, and diabetes (Steenland et al., 2004).

C-reactive protein (CRP) is an acute phase protein produced as part of the immune response to acute infection or injury and is a useful general marker of systemic inflammation. In healthy individuals, CRP levels return to normal after immune activation subsides. However, some persons exhibit chronically elevated levels of CRP (Danesh et al., 2004). This type of chronic inflammation has emerged as an important mechanism in the development of atherosclerosis (Fahdi et al., 2003) and as a risk factor for cardiovascular disease (Danesh et al., 2000; Koenig et al., 1999; Ridker et al., 1998), diabetes mellitus (Pradhan et al., 2001), and mortality (Harris et al., 1999; Reuben et al., 2002). CRP has been shown to be clinically useful, particularly in the prediction of cardiovascular disease and cardiac event outcomes (Smith et al., 2004).

Several studies have found associations between CRP levels and SES. CRP levels have been found to

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be lower among those with more years of education (Koenig et al., 1999; Panagiotakos et al., 2004; Pankow et al., 2001; Wu et al., 2002) and with higher family incomes (Jousilahti et al., 2003). Lower SES, as indexed by job status and job control, has also been inversely related to CRP levels in a British sample, such that those with the lowest status occupations have the highest levels of inflammation (Hemingway et al., 2003).

Socioeconomic differences in levels of inflammation could result from a number of processes. A long history of research shows that socioeconomic status is related to differences in both the physical and social environments and in the capacity to respond to stressful life experiences (Siegrist and Marmot, 2004). Lower SES individuals have greater exposure to infection, are more susceptible to infections (Cohen, 1999), and have a greater risk of developing chronic diseases (Crimmins et al., 1994), but they are also more likely to lack access to health care and treatment (Adler et al., 1993). Thus, differences in susceptibility, exposure, prevalence, and treatment create an environment in which individuals from low SES backgrounds are more likely to experience both acute and chronic health conditions and to suffer from them for longer durations. Individuals from low SES backgrounds are also more likely to engage in risky health behaviors and to experience higher levels of psychological stress, but on average have less information and fewer social resources with which to cope (Pincus and Callahan, 1995).

To date, research has not addressed the extent to which the broad range of factors known to affect CRP (see de Maat and Klufft, 2001, for a review), including acute health problems, chronic conditions, and health behaviors, might account for socioeconomic differences in CRP levels. Furthermore, researchers have not assessed the relationship between CRP and SES in a sample representative of the socioeconomic diversity of the United States, and most previous analyses of socioeconomic variation in CRP have neglected the importance of recognized clinical CRP categories (Pearson et al., 2003), focusing instead on continuous measurement of CRP (Hemingway et al., 2003; Koenig et al., 1999; Panagiotakos et al., 2004; Pankow et al., 2001; Wu et al., 2002). Clinical CRP categories represent different levels of cardiovascular risk and may be related to different sources of inflammation (acute vs. chronic) with varied implications. Blood CRP levels less than 1 mg/L are considered normal; levels between 1 and 3 mg/L indicate moderately increased risk for cardiovascular disease; levels greater than 3 mg/L are considered high risk for cardiovascular disease and cardiac events; and levels greater than 10 mg/L are typically considered indicative of ongoing infection, acute illness, or injury (Pearson et al., 2003). Although these very high levels of CRP are generally considered temporary elevations indicative of acute inflammation (in contrast to levels between 1 and 10 mg/L, which are generally considered indicative of chronic inflammation), levels of CRP above 10 mg/L have also been shown to be associated with increased cardiovascular risk (Ridker and Cook, 2004) and increased risk of mortality following a stroke (Muir et al., 1999).

The purpose of this study was to determine socioeconomic variation in CRP in a US population-based sample, focusing on socioeconomic differences in clinical CRP categories and the mechanisms through which SES may increase CRP levels.

2. Methods

2.1. Participants

Data were drawn from the fourth wave of the National Health and Nutritional Examination Surveys (NHANES), collected between 1999 and 2002. The NHANES are nationally representative, cross-sectional surveys of the non-institutionalized US population, including interview, clinical examination, and laboratory test data. Briefly, NHANES IV surveyed a stratified multi-stage probability sample of US households, with an oversample of older persons, blacks, and Hispanics (predominantly Mexican Americans). When used with sample weights, this probability sample provides estimates for the national community-dwelling population (CDC, 2004).

CRP assays were performed on blood samples from 8874 adults ages 20 and over, or 86% of the total adult sample ($N = 10291$). Among these, 874 persons were missing income data, and 366 pregnant women were excluded. Thus, population distributions of CRP by poverty status were based on a sample of 7634 non-pregnant adults aged 20 or over. Multinomial logistic regressions were based on a sample of 6946, excluding the 688 subjects missing data on one or more covariates: 40 for chronic conditions, 453 for recent illness and immune status, and 195 for health behaviors. Relative to participants with no missing data, subjects with missing data were older, had higher CRP levels, were more likely to be black, female, or in poverty, and were less likely to have exercised in the last month.

2.2. Measures

Serum CRP samples were analyzed by high-sensitivity latex-enhanced nephelometry on a BNII Nephelometer (Dade Behring) (CDC, 2004). The assay used monoclonal anti-CRP antibodies and a calibrator that was traceable to the WHO Reference Material. For the purposes of this analysis, CRP levels were classified into four categories: normal (≤ 1.0 mg/L), moderate (1.1–3.0 mg/L), high (3.1–10.0 mg/L), and very high (>10.0 mg/L).

Sociodemographic characteristics, including age, sex, race/ethnicity, and income, were determined by self-report. SES was operationalized as having a family income above or below the poverty level. Traditionally, SES is operationalized by a wide variety of indicators indicating ability to access societal resources, such as income, education, and occupation. However, the usefulness of these indicators is limited in samples like this one that include a broad age range (ages 20 and over). Because of major increases in average education over the past 70 years, the social and economic implications of education levels are different across age groups. Furthermore, not all adults have or have had an occupation, and the meaning of income changes markedly with age. Thus, poverty was chosen as the main indicator of SES for this analysis because it theoretically provides a comparable measure of SES that applies across age groups. The poverty index score in the NHANES data were calculated by the National Center for Health Statistics, by comparing self-reported household income to national poverty thresholds for a household of similar size, composition, and location. A poverty index score less than or equal to one was used to classify a participant as being in poverty; 14.8% of the weighted sample was in poverty.

Several indicators of health conditions associated with increased inflammation were measured. Participants who reported that they had experienced a head or chest cold, stomach or intestinal illness with vomiting, diarrhea, flu, pneumonia, or ear infection in the last 30 days were coded as having had a recent illness. To explore the effect of other, unreported infection or illness, we also examined blood leukocyte count (Chenillot et al., 2000). Blood leukocyte count reflects current immune

activation, and was significantly but only marginally correlated with indicators of recent illness and with CRP. Leukocyte count was collected as part of the Complete Blood Count using the Beckman Counter MAXM method of counting and sizing, in combination with an automatic diluting and mixing device for sample processing (CDC, 2004) and was coded continuously.

Presence of chronic inflammatory conditions associated with increased CRP, including asthma (Ford, 2003), chronic bronchitis (Mendall et al., 1996), and rheumatoid arthritis (Otterness, 1996), was obtained by self-report. The prevalence of rheumatoid arthritis in NHANES is somewhat higher than reported by other studies of the general population (Gabriel, 2001). This overestimation may be due to misreporting. Subjects who reported that a doctor had told them they had arthritis were asked what type of arthritis it was (rheumatoid arthritis, osteoarthritis, or other).

Finally, several health behaviors that vary by SES have been shown to be important predictors of CRP. Obesity (Visser et al., 1999), smoking (Frohlich et al., 2003), and heavy drinking (Albert et al., 2003; Pankow et al., 2001) are all related to higher CRP levels, whereas exercise is related to lower CRP (Albert et al., 2004; Reuben et al., 2003). Obesity was determined based on standard anthropometry, with subjects with a BMI >30 kg/m² classified as obese. Current smoking status was based on self-report. Respondents who said they drank five or more drinks on their average drinking day were coded as heavy drinkers. Self-reported exercise was dichotomized based on report of engaging in any vigorous exercise in the last 30 days.

2.3. Statistical analysis

First, CRP distributions were examined by poverty status. *T* tests were used to determine whether differences in the prevalence of various CRP levels were significant. Multinomial logistic regression was then used to examine the relative odds of being at CRP levels above normal by poverty status. Regression models provided the odds of having moderate (1.1–3.0 mg/L), high (3.1–10.0 mg/L), and very high CRP levels (>10.0 mg/L) relative to having normal CRP levels (<= 1.0 mg/L). This approach allowed us to examine whether different determinants were associated with varying levels of CRP. Significance of odds ratios was tested using 95% confidence intervals, with significant variables identified if confidence intervals did not include 1.0. While explanatory variables were entered in blocks to examine the effects of mediating variables on the relationship between poverty and CRP, only the final models are shown. All analyses used NHANES IV sample weights, so that results are representative of the US community-dwelling population.

3. Results

3.1. Unadjusted descriptive results

Table 1 provides characteristics of the study population by poverty status. Subjects in poverty had higher mean levels of CRP and blood leukocyte count. They were younger, more likely to be female, and more likely to be non-white. Those in poverty exhibited a higher self-reported prevalence of recent illness, asthma, chronic bronchitis, and rheumatoid arthritis. The prevalence of obesity, current smoking, and heavy drinking were higher among those in poverty, while the prevalence of recent exercise was lower.

Table 2 presents the CRP distribution among all US adults and distributions by poverty status. Differences between those in poverty and those above emerge only in the upper half of the distribution. The upper quartile of CRP values for those in poverty was represented by those with CRP values of 5.6 mg/L and above, while for those above poverty, the top quartile includes those with values

Table 1

Characteristics of NHANES IV (1999–2002) study population by poverty status

	In poverty <i>N</i> = 1218	Above poverty <i>N</i> = 5728	<i>P</i>
C-reactive protein (mg/L), mean ± SD	5.3 ± 9.3	4.0 ± 8.0	<.001
<i>Demographic characteristics</i>			
Age (years), mean ± SD	42.4 ± 18.0	46.5 ± 18.6	<.001
Female (%)	58.0	49.3	<.001
White (%)	48.2	77.4	<.001
Hispanic (%)	13.6	5.7	<.001
Black (%)	17.9	8.1	<.001
Other (%)	20.3	8.8	<.001
<i>Illness and immune function</i>			
Recent illness (%)	33.6	28.3	<.001
Leukocyte count (1000 cells/μL), mean ± SD	7.6 ± 2.3	7.1 ± 2.3	<.001
Asthma (%)	15.9	11.5	<.001
Chronic bronchitis (%)	12.1	7.5	<.01
Rheumatoid arthritis (%)	6.6	4.4	<.01
<i>Behavioral risk factors</i>			
Obesity (%)	32.6	29.5	<.05
Current smoker (%)	37.8	22.5	<.001
Heavy drinking (%)	12.8	7.3	<.001
Exercise (%)	25.5	38.5	<.001

Table 2

(A) CRP value (mg/L) by percentile distribution in US adults by poverty status (*N* = 7634); (B) proportion (%) of the US population in clinical CRP categories

	5th	10th	25th	50th	75th	90th	95th
(A) All US adults	0.2	0.4	0.8	2.1	4.7	10.1	16.0
<i>Poverty status</i>							
In poverty	0.3	0.4	0.9	2.3	5.6	13.4	20.6
Above poverty	0.2	0.4	0.8	2.0	4.5	9.1	14.6
	Low	Moderate	High	Very high			
(B) All US adults	32.1	30.7	27.2	10.0			
<i>Poverty status</i>							
In poverty	29.9	28.0	26.4	15.7			
Above poverty	32.4	31.1*	27.4	9.1**			

* *p* < .05.

** *p* < .001.

of 4.5 mg/L or greater. Among those in poverty, the top 10% of CRP values are represented by values of 13.4 mg/L and greater, compared to values of 9.1 mg/L or greater among those with household incomes above the poverty threshold. About a third (30.7%) of the US adult population had moderately high CRP values, another 27.2% had high CRP values, and 10.0% had very high CRP levels. The prevalence of very high levels of CRP was significantly greater among those in poverty, with 15.7% exhibiting CRP values greater than 10 mg/L, compared to 9.1% of those above poverty (*p* < .001). There were only small differences by poverty status in the other three categories.

Because both poverty status and CRP were related to age, we examined whether the differences in very high CRP between those in and above poverty exist at all ages. The prevalence of very high CRP levels generally increased

linearly with age both for those in and above poverty (Fig. 1). A greater percentage of the population in poverty had very high CRP levels (>10 mg/L) at every age group; all differences were statistically significant except among those aged 80 and over. The greatest difference occurred in the 70–79 age group, where the prevalence of very high CRP was 12% greater among those in poverty than those above poverty.

3.2. Adjusted results from regression modeling

Table 3 shows the results of the multinomial logistic regression predicting the odds of having moderate, high, and very high CRP levels relative to normal CRP levels, controlling for covariates. As in the prior bivariate analysis, persons in poverty were more likely to have very high levels of CRP but did not have an increased likelihood of either

moderate or high CRP. Increasing age was associated with increased odds of all CRP levels above normal. Women and blacks were more likely to exhibit high and very high CRP levels.

Both recent illness and chronic diseases were associated with increased likelihood of CRP above normal. Participants who reported a recent infectious illness (cold, flu, pneumonia, and ear infection) had higher odds of having elevated CRP. These odds were greatest for those in the highest CRP category, where a recent illness was associated with 77% increased odds of very high CRP (OR = 1.77, 95% CI: 1.47–2.12). Similarly, elevated blood leukocyte count, reflecting underlying or unreported infection or illness, was associated with all levels of CRP above normal but was more strongly associated with very high CRP. Among inflammatory chronic diseases, rheumatoid arthritis was most closely associated with elevated CRP, increasing the risk of all levels of CRP above normal. Individuals with chronic bronchitis were more likely to have very high CRP, and those with asthma were more likely to have moderate CRP and very high CRP.

Obesity was the single largest risk factor for CRP above normal levels. Obese individuals were more than three times as likely to have moderately elevated CRP, more than six times as likely to have high CRP, and more than nine times as likely to have very high CRP. Any exercise in the last month reduced the odds of high CRP by 34% (OR = 0.66, 95% CI: 0.54–0.81). Smoking increased odds of both moderate and high CRP by nearly 20%, but heavy drinking was not related to elevated CRP in this sample.

To better understand the mechanisms by which poverty is related to CRP, we report the change in the odds ratio of the poverty variable from regression models with explanatory variables entered in groups (Fig. 2). Controlling for

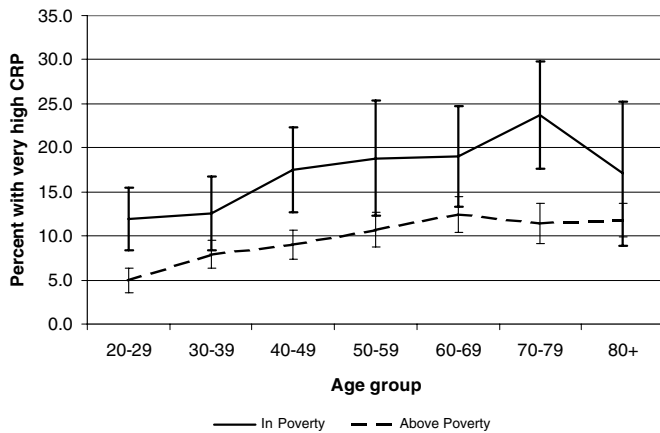


Fig. 1. Prevalence of very high (10+ mg/L) CRP by age group and poverty status with 95% confidence intervals (N = 7634).

Table 3
Odds ratios^a for CRP risk groups relative to low CRP Levels (<= 1.0 mg/L) (N = 6830)

	Odds ratio (95% CI)		
	Moderate CRP (1.1–3.0 mg/L)	High CRP (3.1–10.0 mg/L)	Very high CRP (>10.0 mg/L)
<i>Demographic characteristics</i>			
Poverty	0.95 (0.80–1.13)	0.88 (0.73–1.06)	1.27 (1.01–1.62)
Age	1.02 (1.02–1.02)	1.03 (1.02–1.03)	1.04 (1.03–1.04)
Female	0.99 (0.89–1.11)	1.70 (1.50–1.92)	1.70 (1.50–1.92)
Hispanic	1.10 (0.88–1.38)	1.19 (0.94–1.53)	1.19 (0.94–1.53)
Black	1.18 (0.97–1.45)	1.45 (1.16–1.80)	2.32 (1.76–3.08)
Other	1.02 (0.85–1.23)	1.02 (0.85–1.23)	1.21 (0.91–1.61)
<i>Illness and immune activation</i>			
Recent illness	1.16 (1.02–1.31)	1.16 (1.02–1.31)	1.77 (1.47–2.12)
Leukocyte count	1.13 (1.10–1.17)	1.13 (1.10–1.17)	1.54 (1.47–1.61)
Asthma	1.24 (1.03–1.48)	1.15 (0.94–1.40)	1.85 (1.44–2.36)
Chronic bronchitis	1.16 (0.92–1.47)	1.10 (0.86–1.40)	1.49 (1.11–2.00)
Rheumatoid arthritis	1.48 (1.07–2.05)	1.88 (1.36–2.61)	2.44 (1.66–3.57)
<i>Health behaviors</i>			
Obesity	3.20 (2.76–3.72)	6.83 (5.87–7.95)	9.76 (8.04–11.86)
Smoking	1.18 (1.02–1.35)	1.19 (1.02–1.39)	1.12 (0.91–1.39)
Heavy drinking	0.91 (0.74–1.12)	0.93 (0.74–1.18)	0.98 (0.69–1.39)
Exercise	0.94 (0.84–1.06)	0.76 (0.66–0.86)	0.66 (0.54–0.81)

^a Odds ratios are from multinomial logistic regressions including all variables.

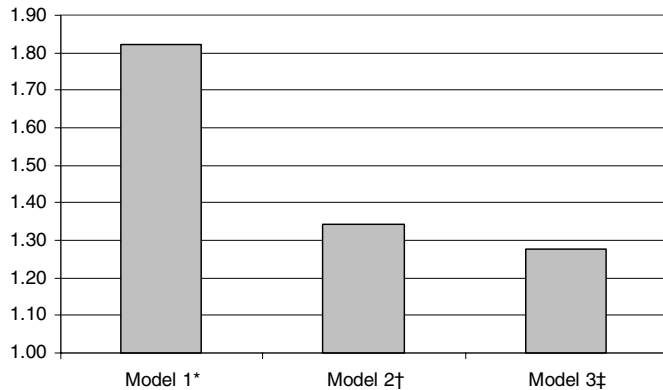


Fig. 2. Odds ratio for poverty status predicting very high CRP (10+ mg/L). *Model 1 controlled for demographic characteristics. †Model 2 controlled for demographic characteristics, recent illness, leukocyte count, and chronic inflammatory conditions. ‡Model 3 controlled for demographic characteristics, recent illness, leukocyte count, chronic inflammatory conditions, and health behaviors.

age, sex, and race/ethnicity, living in poverty was associated with 82% elevated odds of having very high CRP levels (Model 1) relative to those above poverty. Because those in poverty had a higher incidence of infection, higher levels of immune activation, and a greater prevalence of inflammatory disease, adding controls for these factors reduced the odds for of very high CRP by 59% (Model 2). Further controlling for behavioral risk factors reduced the odds by another 21% (OR = 1.27, 95% CI: 1.01–1.62) (Model 3). Thus, about two-thirds (67%) of the poverty-CRP association is explained by recent illness and immune activation, chronic conditions, and behavioral risk factors, but poverty remained a significant risk factor for very high CRP even after controlling for these covariates.

In an effort to consider broader implications of socioeconomic status beyond poverty, we also performed the above analysis using a dichotomous indicator of educational status (less than high school versus high school or more) and a continuous indicator of household income. These analyses generally supported the above results; low education was associated with increased odds of having both high and very high CRP, but this relationship was strongest in those with very high levels of CRP (OR = 1.51, 95% CI: 1.11–2.05). Furthermore, high income was protective against having moderate CRP levels. Taken together, these results support an inverse relationship between CRP and SES in the United States, with the effects of low SES strongest at very high CRP values.

4. Discussion

The purpose of this analysis was to examine socioeconomic variation in CRP with a focus on clinical CRP categories and the mechanisms through which SES may increase CRP levels. Three main findings deserve further consideration. First, results suggest that the relationship between socioeconomic status and inflammation is not lin-

ear. Rather, differences are evident only at very high levels of CRP (>10 mg/L). People in poverty were significantly more likely to have very high levels of CRP often considered clinically indicative of infection (>10 mg/L), but there were no significant socioeconomic differences in the prevalence of moderate or high CRP levels generally considered indicative of cardiovascular risk in clinical practice. Additional analysis confirmed similar relationships using other measures of socioeconomic status. Future research might consider whether additional factors associated with relative wealth might contribute to risk at lower levels.

A second finding was that much of the variation by poverty status was accounted for by a higher incidence of acute illness, a greater prevalence of inflammatory chronic conditions, and poorer health behaviors. The greatest contributor to increased risk of very high CRP among those in poverty was acute and chronic illness, which accounted for 59% of the initial poverty effect. People in poverty were more likely to have recently experienced a cold, stomach illness, flu, or pneumonia. They also had significantly higher blood leukocyte counts, indicating an ongoing immune response, and a higher prevalence of chronic inflammatory conditions. Additionally, those in poverty were more likely to be obese and less likely to have exercised in the last 30 days, contributing to a higher risk of very high CRP. Yet, controlling for acute and chronic conditions and health behaviors did not fully account for the effect of poverty on CRP. Future research should consider other potential pathways, including issues related to access to care and psychosocial variables, such as social support and stress.

Third, determinants of serum CRP vary by CRP level. Clearly, poverty was related to very high CRP but not to moderate and high CRP levels, but several other factors were also uniquely related to the odds of specific CRP levels. For instance, asthma was related to an increased risk of moderate and very high CRP. In asthmatics, moderate CRP could reflect ongoing lung problems, while very high CRP could be related to a recent acute asthma attack. Being female or black were both associated with an increased risk of high and very high CRP, but were not related to moderate CRP risk. Smoking appeared to exert greater effects at lower CRP levels, being related to higher odds of moderate and high CRP, whereas exercise appeared to exert greater effects at higher CRP levels.

These findings suggest that very high CRP may be related not only to acute infection, but also to chronic conditions (asthma and bronchitis) and health behaviors (obesity and exercise). More research on stability and change in these very high CRP levels over time is needed. Very high levels are often assumed to indicate acute infection in clinical practice, but the influence of chronic conditions and health behaviors suggests that some of the variance at even these very high levels could be related to more long-term processes. Additionally, CRP levels greater than 10 mg/L may be clinically relevant as predictors of cardiovascular risk and mortality (Muir et al., 1999; Ridker and Cook, 2004).

In interpreting these findings, it is important to note the limitations of this study. First, a significant number (22.2%) of potential participants were missing CRP or income data, and additional participants were missing data for covariates. However, if anything, these missing participants likely attenuated observed observations as missing subjects had higher CRP levels and were more likely to be in poverty. Second, CRP was available at only one time point, neglecting intra-individual variability. In an effort to address this issue, we attempted to control for a wide range of covariates known to affect variability in CRP and to use leukocyte count as a proxy for acute immune response. However, data collection on infectious illness and autoimmune diseases associated with elevated CRP was limited and relied on self-reported measures that may be subject to bias. For instance, African Americans had a lower prevalence of self-reported recent illnesses, suggesting potential underreporting. It is also possible that persons of lower SES suffer from unrecognized or unreported low-grade infections. For example, sexually transmitted diseases and periodontal diseases were not controlled in these analyses.

Because of the cross-sectional nature of the data, making causal inferences about the relationships observed here is difficult. For instance, it is possible that high levels of inflammation relating to ongoing illness prevent people from working and result in higher levels of poverty among this group. Another potential concern is the direction of the relationship between CRP and chronic conditions. Individuals who have experienced a heart attack or stroke or who have diabetes have higher levels of CRP as a result of these conditions, but CRP has also been implicated in the pathogenesis of these conditions. To address this issue, regressions were run excluding subjects reporting a diagnosis of heart attack, stroke, or diabetes. Exclusion of these individuals did not change the direction or significance of any coefficients. However, when we controlled for these health conditions by adding them to Model 3 in the full sample, we found that a history of heart attack or stroke was related to an increased probability of elevated CRP levels. Including these variables did not affect the significance or direction of other variables except poverty, which was not significant after controlling for previous diagnosis of diabetes, heart attack, and stroke. Thus, it does not appear that the inclusion of subjects with a history of heart attack, stroke, or diabetes biased results related to recent illness, asthma, bronchitis, arthritis, or health behaviors, but this finding does suggest that there is a link between the prevalence of cardiovascular conditions and the high CRP of those in poverty. Longitudinal studies are needed to better consider the interrelationships between socioeconomic status, chronic conditions, and inflammation.

Finally, the inclusion of individuals with a recent illness may limit our ability to isolate chronically elevated CRP. In an attempt to address this issue, regressions were run excluding subjects reporting an episode of cold, flu, or pneumonia in the last 30 days. In general, excluding indi-

viduals reporting recent acute illness also did not affect the results.

5. Conclusion

In conclusion, socioeconomic status is related to higher CRP, but this effect is greatest at very high CRP levels (>10 mg/L). Recent illness and immune activation, chronic conditions, and health behaviors explain much, but not all, of this association. This research provides another piece of information about the relationships between socioeconomic status and inflammation, an important risk factor for poor health outcomes in late life.

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