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Research Article

Race, Gender, and Socioeconomic Variations in C-Reactive Protein Using the Health and Retirement Study

Heather R. Farmer, PhD,[1,*] Linda A. Wray, PhD,[2] and Steven A. Haas, PhD[3]

[1]Department of Population Health Sciences, Duke University Medical Center, Durham, North Carolina. [2]Department of Biobehavioral Health, The Pennsylvania State University, University Park. [3]Department of Sociology and Demography, The Pennsylvania State University, University Park.

*Address correspondence to: Heather R. Farmer, PhD, Department of Population Health Sciences, Duke University Medical Center, 215 Morris Street, Suite 210 Durham, NC 27701. E-mail: heather.farmer@duke.edu

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Abstract

Objectives: To clarify the relationships among race, gender, and socioeconomic status (SES) with C-reactive protein (CRP).
Method: The present study analyzed data from 6,521 Black and White respondents aged 51 and older in the Health and Retirement Study, a nationally representative sample of midlife and older adults, to address two aims. We sought to (i) assess the independent associations between race, gender, and SES with CRP concentrations and (ii) test whether race, gender, and SES interacted to produce unequal CRP concentrations cross-sectionally and over a 4-year follow-up.
Results: The results demonstrated that race, gender, and SES were each independently associated with baseline CRP, but only SES was associated with CRP at follow-up. Furthermore, race, gender, and education interacted to produce differential CRP levels at baseline. There were incremental benefits for each additional level of education for White men and women, but the relationship between education and CRP was more complicated for Black men and women. Compared with other race/gender groups with less than high school, Black women had the highest and Black men had the lowest levels of CRP. There were no apparent benefits to CRP for Black women with college compared with Black women with high school, while Black men with less than high school and college had similar concentrations of CRP.
Discussion: In clarifying the complexity inherent in CRP disparities, this work contributes to a greater understanding of the biological mechanisms underlying racial disparities in leading causes of morbidity and mortality in the United States.

Keywords: Race, Socioeconomic status, C-reactive protein, Health disparities, Health and Retirement Study

Large and pervasive disparities exist between non-Hispanic Black/African American and non-Hispanic White/Caucasian people for a wide variety of health conditions, especially those that are more common in middle and later life, including cardiovascular disease (CVD), Type 2 diabetes, physical disability, and stroke (Graham, 2015; Kung, Hoyert, Xu, & Murphy, 2008; Ski, King-Shier, & Thompson, 2014). Despite early ideologically based speculations that racial differences in health were due to innate biological differences (see Williams & Sternthal, 2010 for review), racial disparities in health are now recognized as resulting from broader contexts of racialized inequality, which produces the diverging social and economic contexts of Black versus White individuals and contributes to risk factors such as lower socioeconomic status (SES), interpersonal and structural discrimination, and segregation (Chae, Nuru-Jeter, Lincoln, & Francis, 2011; Krieger, Rowley, Herman, Avery, & Phillips, 1993; Williams & Sternthal, 2010).

Emerging research on intersectionality further supports the contention that Black women face added disadvantages

to their health, relative to White men, White women, and Black men, likely as a result of their location at the bottom of multiple social hierarchies (Bauer, 2014; Bowleg, 2012; Howard & Sparks, 2015; Richardson & Brown, 2016). Black women may face constraints due to their race, their gender, and the combination of the two, which might have important implications for their health (Bauer, 2014; Bowleg, 2012; Woods-Giscombé, 2010). This work suggests that examination of race and other social statuses, such as gender and SES, in isolation from one another, may obscure complex multidimensional arrays of inequality that generate unequal risk of poor health outcomes (Bowleg, 2012; Brown & Hargrove, 2013; Richardson & Brown, 2016). This indicates a need for more research to examine how race, gender, and SES interact to stratify health. For example, a high-SES Black woman may have to navigate predominantly White spaces in ways similar to Black men, but they may also navigate spaces occupied more by men than women. Consequently, upward social mobility may result in greater stress and poorer health for Black women compared with Black men (Allen et al., 2019; Nuru-Jeter et al., 2018; Woods-Giscombé, 2010). Based on this work, it is likely that interlocking systems of oppression may give way to greater life-course exposure to racism and other health risks, fewer health-promoting resources, and ultimately, poor health for Black women.

C-reactive protein (CRP) is a measure of systemic inflammation resulting from a coordinated immune response that protects against infection and illness (Miller, Cohen, & Ritchey, 2002). Inflammation represents one component of allostatic load, a measure of multisystem physiological deterioration, that may occur in response to adversity across the life course (Graham, Christian, & Kiecolt-Glaser, 2006; McEwen, 1998; Miller et al., 2002). Research suggests that long-term exposure to inflammation is associated with increased risk of CVD, stroke, and mortality, as well as hypertension and obesity (Choi, Joseph, & Pilote, 2013; Crimmins, Vasunilashorn, Kim, & Alley, 2008; Pearson et al., 2006; Sesso et al., 2003). CVD, stroke, and related cardiometabolic disorders remain leading sources of racial disparities in mortality (Kochanek, Arias, & Anderson, 2013). Furthermore, recent work shows that Black adults have higher levels of CRP compared with White adults and that this association often persists after controlling for a range of characteristics (Nazmi & Victora, 2007). Therefore, CRP may be a useful measure of upstream processes at the social–biological interface that drive racial disparities in health. However, scant research has investigated whether upstream biological markers of disease risk and progression, such as CRP, vary simultaneously by race, gender, and SES.

Although investigations into race and gender distributions of CRP in older adults are sparse, existing work shows that Black women have the highest CRP. A study conducted by Khera and colleagues (2005) observed that Black women had the highest levels of CRP, followed by White women, Black men, and White men, respectively. Studies of the interconnections among race, gender, and SES have produced mixed results. Using data from the National Social Life, Health, and Aging Project, Herd, Karraker, and Friedman (2012) found that Black women and men had higher CRP than White adults. After accounting for SES, however, racial differences in CRP for women were reduced to marginal significance, but racial differences in CRP for men were largely unchanged. Most recently, using the Health and Retirement Study (HRS), Farmer and colleagues (2020) found that race differences in elevated CRP (>3.0 mg/L) varied by gender and that the association persisted after accounting for a wide range of factors. Though that study did not explicitly test for race, gender, and SES interactions, the results suggested a possible moderating effect of SES on racial and gender differences in CRP. SES explained race differences in elevated CRP for men, but not for women. These results demonstrate that race, gender, and SES may be associated with CRP in complex ways. The present study further investigates that complexity.

In their seminal paper, Link and Phelan (1995) describe SES as a "fundamental cause" of poor health and mortality because it provides people with differential sets of health-sustaining resources. Subsequent work has shown that compared with Whites, Blacks earn less income for each additional year of education (Williams & Mohammed, 2009). Research also consistently finds that Blacks receive fewer benefits to their health with additional education, a phenomenon referred to as "diminishing returns" (Boen, 2016; Dinwiddie, Zambrana, Doamekpor, & Lopez, 2015; Farmer & Ferraro, 2005; Howard & Sparks, 2015; Walsemann, Goosby, & Farr, 2016). In work by Farmer and Ferraro (2005), there was a strong and consistent educational gradient in self-rated health for Whites. In contrast, increased education was associated with limited improvements in self-rated health for Blacks. Ongoing research shows that there are diminishing socioeconomic returns for inflammation, cardiovascular risk, and allostatic load (Dinwiddie et al., 2015; Howard & Sparks, 2015; Walsemann et al., 2016). In a recent study, income and education were differentially associated with CRP among White compared with Black adults (Dinwiddie et al., 2015). Another study used ADD Health data to show that life course SES contributed to higher cardiovascular risk in Whites, but played a limited role in cardiovascular risk for Blacks (Walsemann et al., 2016). To the best of our knowledge, no studies have examined if race, gender, and SES interact to shape CRP.

To gain a better understanding of how race, gender, and SES are associated with CRP in midlife and older adults, we use HRS data to address two aims: (i) examine the independent associations between race, gender, and SES (measured by education and income) with CRP and (ii) examine whether race, gender, and SES interact to produce unequal levels of CRP. We hypothesize that being Black, a woman, and lower SES will be independently associated with higher CRP. Furthermore, we hypothesize that race,

gender, and SES will interact to produce an unequal distribution of CRP, such that Black women and Black men will not experience the same reductions in CRP with improvements in SES as Whites do, and that Black women will have the highest CRP of all race/gender groups at all levels of SES. In exploring these aims, we address several gaps in the literature on the social patterning of a clinically relevant biomarker and provide insight that may advance understanding how social processes "get under the skin" to produce health disparities in later life.

Method

Data

The HRS is an ongoing nationally representative panel study of more than 20,000 noninstitutionalized midlife and older adults ages 51 and older and their spouses aimed at understanding characteristics of midlife and adults in the United States. The HRS routinely collects information at the individual and household level for demographic background, SES, health risks, and health conditions, among other topics in biennial interviews (Juster & Suzman, 1995). Beginning in 2006, respondents were randomized into one of the two rotating groups for enhanced interviews every other wave to gather additional psychosocial and biomarker data (Smith, Ryan, Fisher, Sonnega, & Weir, 2017). More information on the data collection process is available from the HRS (hrsonline.isr.umich.edu).

The respondents' data for the present study are pooled from the core interview, the tracker file, and the biomarker data. Given the structure of the biomarker collection, respondents were in one of the two groups: their CRP was collected either in 2006 and 2010 or in 2008 and 2012. We constrained the analytic sample to respondents who are non-Hispanic Black or Non-Hispanic White with two CRP observations. This resulted in a final analytic sample of 6,521 unique non-Hispanic Black and White respondents who survived to the 4-year follow-up and who consented to and provided CRP at both waves. The rates of missingness were similar across race, where 1.46% of Whites and 1.54% of Blacks had missing data on an independent variable. To address missingness, we used multiple imputation on 25 data sets via Stata's chained equations routine.

Measures

Outcome

High-sensitivity CRP was ascertained using the dried blood spot (DBS) process. We used a variable released by the HRS that transforms DBS to serum CRP values, which allows us to account for potential measurement differences in CRP collection (Crimmins et al., 2013). Raw CRP values are presented in the descriptive statistics and figure. For all regression analyses, CRP values were log (natural) transformed due to skewness.

Often, respondents with levels of CRP > 10.0 mg/L are excluded from analyses because these concentrations are believed to indicate acute infection or illness (Pearson et al., 2006). However, given the growing evidence on the clinical relevance of very high CRP concentrations, we retained these values in our analyses (Alley et al., 2006; Hamer, Chida, & Stamatakis, 2010; Ishii et al., 2012).

Key predictors

Gender was classified as woman or man. Self-reported education was categorized as less than high school, high school education/GED, or some college or more. Baseline (at first CRP measurement) household income was grouped into tertiles (low: ≤$30,348, middle: $30,349–$65,400, and high: >$65,400).

Covariates

Demographic covariates included baseline measures of respondent's age, current marital status, and tertiles of total household wealth (low: ≤$135,000, middle: $135,001–$483,000, high: >$483,000). Birth cohort was considered for inclusion in the analyses, but preliminary analyses revealed a nonsignificant association with CRP.

Lifestyle characteristics included baseline measures of physical activity, smoking, and drinking. Here, we included whether the respondent never, sometimes, or frequently engaged in moderate or vigorous physical activity, smoking status (never, former, or current), and drinking status (never, moderate, or heavy). Health characteristics included baseline measures for overweight/obesity (using body mass index [BMI], calculated as weight divided by squared height); a measure of depressive symptomatology within the past week using the shortened Center for Epidemiological Studies Depression (CES-D) scale (Radloff, 1977); and an index of comorbid health conditions (range: 0–7), which included arthritis, hypertension, diabetes, stroke, heart conditions, cancer (excluding skin cancer), and lung conditions.

Statistical Analyses

Descriptive analyses were conducted to evaluate the distribution of study characteristics by race, and by gender within race. Analytic models use ordinary least squares (OLS) regression to test the hypotheses on how race, gender, and SES were associated with lnCRP (hereafter referred to as CRP). For Aim 1, we examined the independent associations between race, gender, and SES with CRP at baseline and after a 4-year follow-up. In Model 1, we regressed CRP on race, gender, education, and income, adjusted for age, marital status, and wealth. Model 2 regressed CRP on race, gender, education, and income, adjusted for age, marital status, wealth, overweight/obesity, CES-D, comorbid health conditions, physical activity, smoking status, and alcohol use. Identical models examined CRP after 4 years of follow-up, controlling on CRP at baseline.

To address Aim 2, we assessed whether race, gender, and SES interacted to produce an unequal distribution of CRP by regressing CRP on a three-way race × gender × SES interaction. Separate models were run for each measure of SES (education and income). In Model 1, we regressed CRP on the following interactions separately: Black × woman, Black × woman × education, and Black × woman × income. These models were adjusted for age, marital status, and wealth. Model 2 regressed CRP separately on Black × woman, Black × woman × education, and Black × woman × income, respectively, adjusted for age, marital status, wealth, overweight/obesity, CES-D, comorbid health conditions, physical activity, smoking status, and alcohol use. Identical models examined CRP after 4 years of follow-up, controlling for CRP at baseline. Analyses were conducted using Stata SE, version 14.2. All analyses were weighted and adjusted for the complex design of the HRS using Stata's *svy* commands to produce accurate *SE*.

Results

Descriptive Analyses

Baseline descriptive statistics for the sample overall, stratified by race, and by gender within race, are presented in Table 1. Results from descriptive analyses demonstrated that the sample significantly differed by race across SES, lifestyle characteristics, and health status variables. Blacks had significantly higher median CRP concentrations at baseline, lower SES, more risky lifestyle characteristics, and poorer health than Whites. In addition, nearly all covariates differed by gender within race. At baseline, Black women had a median CRP of 3.94 mg/L (interquartile range [IQR] = 6.81), in contrast to Black men, 2.27 mg/L (IQR = 3.86), White women, 2.06 mg/L (IQR = 3.65), and White men, 1.56 mg/L (IQR = 2.56). Black and White women generally had higher CRP, lower SES, and poorer health than Black and White men, respectively.

Race, Gender, and SES Differences in CRP

Baseline

Model 1 revealed that nearly all proposed social status variables were significantly associated with CRP at baseline (Table 2). Being Black ($b = 0.212$, $SE = 0.064$, $p \leq .01$) and being a woman ($b = 0.251$, $SE = 0.034$, $p \leq .001$) were associated with higher levels of CRP. Compared with some college or more, less than high school ($b = 0.239$ $SE = 0.055$, $p \leq .001$) and high school education ($b = 0.117$, $SE = 0.044$, $p \leq .01$) were also associated with higher baseline CRP. Finally, compared with high income, middle income was associated with higher CRP ($b = 0.117$, $SE = 0.043$, $p \leq .01$).

In Model 2, which full adjusted for demographics, lifestyle characteristics, and health, the relationship between being Black and CRP ($b = 0.138$, $SE = 0.058$, $p \leq .05$) was attenuated but remained significant, and being a woman ($b = 0.308$, $SE = 0.037$, $p \leq .001$) remained positively associated with CRP. Furthermore, the relationship between education and CRP was reduced to nonsignificance. Middle income remained associated with significantly higher CRP than high income ($b = 0.077$, $SE = 0.038$, $p \leq .05$), though the association was attenuated.

Follow-up

In Model 1 of the lagged OLS models, both less than high school ($b = 0.130$, $SE = 0.040$, $p \leq .01$) and high school education ($b = 0.085$, $SE = 0.036$, $p \leq .05$) were associated with higher CRP at follow-up. Both low ($b = 0.084$, $SE = 0.035$, $p \leq .05$) and middle ($b = 0.070$, $SE = 0.031$, $p \leq .05$) income were positively associated with follow-up CRP. After adjustment for demographics, lifestyle characteristics, and health-related factors, only less than high school education ($b = 0.079$, $SE = 0.040$, $p \leq .05$) was associated with higher follow-up CRP.

Race, Gender, and SES Interactions

Baseline

The first set of columns in Table 3 regressed CRP at baseline on a two-way interaction between race and gender. The Black × woman interaction was not statistically significant. In the next set of columns, CRP was regressed on the three-way Black × woman × education interaction, controlling for age, marital status, income, and wealth. Here, Model 1 revealed a significant Black × woman × high school education interaction ($b = -0.467$, $SE = 0.205$, $p \leq .05$) and a nonsignificant Black × woman × less than high school interaction. An adjusted Wald test demonstrated a statistically significant three-way interaction overall. In the fully adjusted model, however, the Black × woman × education interaction was reduced to nonsignificance.

Figure 1 illustrates the results from the three-way interaction tested in Model 1 of Table 3. Raw (rather than logged) values of CRP are presented for ease of interpretation. This figure indicates that there were differences in the relationship between levels of education and CRP by race and gender. Black women had the highest CRP concentrations compared with all other race–gender groups, at all levels of education. The most striking differences were observed among the least educated, where Black women had over 1.0 mg/L higher than Black men, White women, and White men. In addition, Black men with high school had higher CRP levels at baseline than Black men with less than high school or some college or more. More "traditional" education gradients existed for White women and men: those with the lowest education had the highest CRP, and improvements in education corresponded to lower CRP.

In the final set of columns in Table 3, CRP at baseline was regressed on the Black × woman × income interaction, controlling for age, marital status, education, and wealth. This set of analyses showed a nonsignificant Black × woman × income interaction.

Table 1. Weighted Means (*SD*) and Proportions (%) of Study Variables Among Non-Hispanic White and Black Adults in the Health and Retirement Study (*N* = 6,521)

	White (*n* = 5,742)			Black (*n* = 779)			
	Total	Men (*n* = 2,404)	Women (*n* = 3,338)	Total	Men (*n* = 254)	Women (*n* = 525)	*p*
CRP, median (IQR), mg/L, baseline	1.81 (3.19)	1.56 (2.56)	2.06 (3.65)†	3.28 (5.92)	2.27 (3.86)	3.94 (6.81)†	*
Age	65.11 (8.93)	64.44 (8.16)	65.68 (9.55)†	63.38 (9.82)	63.06 (8.82)	63.57 (10.41)	*
Not married	31.87	22.72	39.72†	60.77	44.44	70.62†	*
SES							
Education							
Less than HS	9.41	8.71	10.01	26.94	24.73	28.27	*
HS education	36.55	32.83	39.75†	34.76	38.18	32.70	
Some college or more	54.04	58.47	50.23†	38.30	37.08	39.03	
Income tertiles							
Low: ≤$30,348	26.62	20.04	32.27†	58.29	41.88	68.19†	*
Middle: $30,349–$65,400	32.04	31.29	32.68	23.73	31.31	19.16†	
High: >$65,400	41.34	48.67	35.05†	17.98	26.81	12.65†	
Wealth tertiles							
Low: ≤$135,000	30.40	27.08	33.26†	71.42	62.65	76.71†	*
Middle: $135,001–$483,000	33.48	33.58	33.40	21.79	29.85	16.92†	
High: >$483,000	36.11	39.34	33.35†	6.80	7.50	6.37	
Health status							
Overweight/obese	72.13	77.99	67.03†	81.43	74.29	85.77†	*
CES-D score	1.17 (1.75)	1.01 (1.53)	1.30 (1.92)†	1.85 (2.64)	1.42 (1.94)	2.11 (3.00)†	*
Comorbid health conditions	1.73 (1.25)	1.66 (1.18)	1.78 (1.30)†	2.14 (1.67)	1.91 (1.58)	2.28 (1.69)†	*
Lifestyle characteristics							
Moderate/vigorous activity							
Never	21.29	21.53	22.26†	28.44	30.69	27.08†	*
Sometimes	12.33	9.27	14.95†	22.99	13.10	28.96†	
Frequent	65.75	69.20	62.79	48.57	56.21	43.96	
Smoking							
Current smoker	11.94	12.92	11.11†	21.11	23.52	19.65	*
Former smoker	43.43	52.31	35.81†	39.48	51.52	32.22†	
Never smoked	44.26	34.77	53.08†	39.42	24.96	48.13†	
Alcohol use							
No alcohol consumption	58.54	49.48	66.32†	78.86	66.38	83.14	*
Moderate consumption	32.08	35.08	29.51†	16.85	20.66	14.56†	
Heavy consumption	9.38	15.44	4.17†	6.29	12.96	2.30†	

Notes: CRP = C-reactive protein; CES-D = Center for Epidemiological Studies Depression short scale; HS = high school; IQR = interquartile range; SES = socioeconomic status. Weighted descriptives using respondents who had biomarker data at both baseline and follow-up.

*Significant difference $p < .05$ between race groups.

†Significant difference $p < .05$ between genders within race.

Follow-up

As shown in Table 4, the Black × woman interaction was not statistically significant at follow-up. In the next set of columns, Model 1 revealed a significant Black × woman × less than high school interaction ($b = -0.475$, $SE = 0.235$, $p \leq .05$). A Wald test revealed a nonsignificant three-way interaction overall. In Model 1, less than high school ($b = 0.169$, $SE = 0.067$, $p \leq .05$), high school ($b = 0.133$, $SE = 0.055$, $p \leq .05$), low income ($b = 0.082$, $SE = 0.035$, $p \leq .05$), and middle income ($b = 0.065$, $SE = 0.031$, $p \leq .05$) were associated with higher CRP. In the fully adjusted model, only less than high school ($b = 0.134$, $SE = 0.067$, $p \leq .05$) and high school education ($b = 0.116$, $SE = 0.054$, $p \leq .05$) were significantly associated with baseline CRP.

The final set of columns in Table 4 presents the results for the three-way Black × woman × income interaction on follow-up CRP, controlling for baseline CRP, age, marital status, education, and wealth. Model 1 shows that neither of the Black × woman × income interaction terms was statistically significant. This association was unchanged in the fully adjusted model.

Table 2. Linear Regression Models Testing Independent Relationship Between Race, Gender, and SES With C-Reactive Protein at Baseline and Over 4 Years in the HRS (n = 6,521)

	Baseline				Follow-up			
	Model 1		Model 2		Model 1		Model 2	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.449**	0.158	−0.330*	0.137	0.176	0.129	−0.048	0.131
Black[a]	0.212**	0.064	0.138*	0.058	0.015	0.049	−0.008	0.048
Woman[b]	0.251***	0.034	0.308***	0.037	−0.019	0.028	0.008	0.029
Education[c]								
Less than HS	0.239***	0.055	0.098	0.055	0.130**	0.040	0.079*	0.040
HS education	0.117**	0.044	0.057	0.043	0.085*	0.036	0.063	0.036
Income tertile[d]								
Low	0.060	0.054	0.018	0.048	0.084*	0.035	0.061	0.035
Middle	0.117**	0.043	0.077*	0.038	0.070*	0.031	0.049	0.033

Notes: HRS = Health and Retirement Study; HS = high school; SES = socioeconomic status. Data are pooled from the 2006 and 2008 waves of the Health and Retirement Study (HRS). Analyses were weighted to adjust for differential selection into study. Model 1 adjusted for age, marital status, and wealth. Model 2 adjusted for age, marital status, wealth, alcohol use, smoking status, physical inactivity, overweight/obesity, CES-D, and comorbid chronic conditions.

[a]Non-Hispanic White is the reference category. [b]Men are the reference category. [c]Some college or more is the reference category. [d]High income is the reference category.

Significance levels for two-tailed tests of coefficients: *p < .05, **p < .01, ***p < .001.

Discussion

The purpose of this article was to determine how race, gender, and SES—as measured by education and income—were linked to a clinically meaningful marker of inflammation at baseline and over a 4-year follow-up in a nationally representative sample of midlife and older adults. Our study sought first to test the independent associations between race, gender, and SES with CRP, and second to test whether race, gender, and SES interacted to produce unequal levels of CRP.

The study findings demonstrated significant independent effects of race, gender, education, and income on baseline CRP. Consistent with the larger body of literature, Blacks had higher CRP than Whites (Khera et al., 2005; Nazmi & Victora, 2007); women had higher CRP than men (Khera et al., 2005; Lakoski et al., 2006); and lower SES was associated with higher CRP (Nazmi & Victora, 2007). In analyses examining the relationship between these social status variables and CRP after 4 years, only education and income were associated with increased follow-up CRP.

Race was not associated with CRP at follow-up, contradicting work showing greater increases in inflammatory markers over 15 years for Blacks than Whites (Fuller-Rowell, Curtis, Doan, & Coe, 2015). Based on the literature documenting temporal variation in inflammation (Ockene et al., 2001), a 4-year follow-up may be too short to capture gradual changes in CRP. Thus, it is unclear if racial disparities in CRP grow, remain stable, or decline over time because there is limited longitudinal data on the association between race and CRP.

Education and income were negatively associated with CRP at baseline and at follow-up, as expected. The literature consistently demonstrates that higher SES is associated with lower CRP (Nazmi & Victora, 2007); however, many studies examine education as the sole measure of SES. When studied concurrently (Herd et al., 2012; Janicki-Deverts, Cohen, Kalra, & Matthews, 2012), studies show that both education and income are inversely associated with CRP. The present study's findings indicate a strong association between education and CRP. Our analyses indicated a significant association between reporting less than high school and higher CRP, above and beyond SES, lifestyle characteristics, and health status. This implies that other unaccounted for factors may link education to CRP.

Moreover, we found no support for a two-way interaction between race and gender on baseline or follow-up CRP. This is inconsistent with the available literature: a recent paper by Farmer and colleagues used HRS data from 2006 to 2014 and found that race differences in elevated CRP significantly varied by gender, similar to work by Khera and colleagues (2005). That the present study was unable to replicate this finding may be due to the small sample size of Black women and men or the operationalization of CRP. In both of the aforementioned studies, CRP was dichotomized into elevated (>3.0 mg/L) versus nonelevated. This may reflect possible threshold effects. It is important to note that the race × gender interaction on baseline CRP was marginally significant (p < .10) and in the expected direction. Thus, it is possible that baseline CRP may differ by race and gender, whereas changes in CRP are monotonic across all groups.

The results further demonstrate a significant three-way interaction between race, gender, and education on baseline CRP. Black women with less than high school had the

Table 3. Linear Regression Models Testing Interactive Relationship Among Race, Gender, and SES With C-Reactive Protein at Baseline in the HRS (n = 6,521)

	Black × woman				Black × woman × education				Black × woman × income			
	Model 1		Model 2		Model 1		Model 2		Model 1		Model 2	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.452**	0.158	−0.328*	0.138	0.464**	0.157	−0.333*	0.137	0.410**	0.161	−0.403**	0.138
Black[a]	0.081	0.103	0.113	0.098	0.029	0.159	0.101	0.164	0.087	0.184	0.134	0.190
Woman[b]	0.234***	0.035	0.305***	0.039	0.210***	0.050	0.317***	0.048	0.295***	0.055	0.409***	0.061
Education[c]												
Less than HS	0.240***	0.054	0.098	0.055	0.272***	0.079	0.201**	0.072	0.238***	0.054	0.096	0.054
HS education	0.119**	0.044	0.057	0.043	0.075	0.065	0.054	0.060	0.117**	0.044	0.055	0.043
Income tertile[d]												
Low	0.058	0.054	0.018	0.048	0.057	0.054	0.017	0.048	0.126	0.078	0.151*	0.071
Middle	0.118**	0.043	0.077*	0.038	0.119**	0.043	0.076*	0.038	0.175**	0.064	0.153*	0.061
Black × woman	0.215	0.115	0.041	0.113	0.322	0.172	0.092	0.181	0.554**	0.212	0.321	0.225
Black × less than HS					−0.259	0.225	−0.300	0.226				
Black × HS education					0.296	0.191	0.181	0.204				
Woman × less than HS					−0.026	0.102	−0.166	0.095				
Woman × HS education					0.076	0.071	0.006	0.063				
Black × woman × less than HS					0.267	0.245	0.383	0.257				
Black × woman × HS education					−0.467*	0.205	−0.343	0.219				
Black × low income									0.158	0.206	0.084	0.215
Black × middle income									−0.288	0.255	−0.285	0.255
Woman × low income									−0.140	0.091	−0.255**	0.090
Woman × middle income									−0.081	0.086	−0.131	0.084
Black × woman × low income									−0.480	0.256	−0.351	0.273
Black × woman × middle income									−0.274	0.252	−0.185	0.262

Notes: HRS = Health and Retirement Study; HS = high school; SES = socioeconomic status. Data are pooled from the 2006 and 2008 waves of the Health and Retirement Study (HRS). Analyses were weighted to adjust for differential selection into study. Model 1 adjusted for age, marital status, and wealth. Model 2 adjusted for age, marital status, wealth, alcohol use, smoking status, physical inactivity, overweight/obesity, CES-D, and comorbid chronic conditions.

[a]Non-Hispanic White is the reference category. [b]Men are the reference category. [c]Some college or more is the reference category. [d]High income is the reference category.

Significance levels for two-tailed tests of coefficients: *$p < .05$, **$p < .01$, ***$p < .001$.

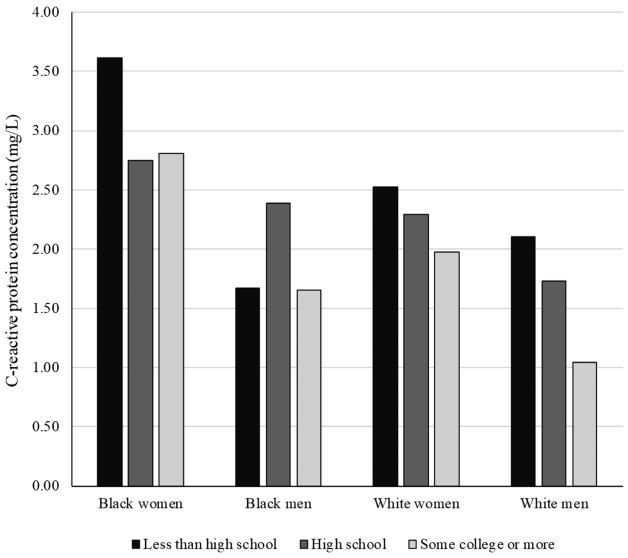

Figure 1. C-reactive protein (mg/L) by level of educational attainment across race/gender subgroups in the Health and Retirement Study (N = 6,521). Analyses were based on black × woman × education interaction tested in Model 1 of Table 3. Analyses were weighted to adjust for differential selection into the study. Analyses adjusted for age, marital status, income, and wealth.

highest CRP of all race/gender groups at all levels of SES. Black men with a high school education had higher CRP compared with those with less than high school, suggesting that increases in SES may not always protect Black men. However, compared with a high school education, at least some college education was protective against higher CRP for Black men. For both White men and women, there was an incremental decrease in CRP with improvements in education. The race × gender × education interaction was reduced to nonsignificance after adjusting for demographic, lifestyle, and health indicators, suggesting that these characteristics might be pathways by which combinations of race, gender, and education are linked to CRP. To date, no studies have examined the simultaneous relationships among race, gender, and SES on CRP. The extant literature examining race, gender, and SES together have largely conceptualized SES as a mediating, rather than moderating, factor (Farmer et al., 2020; Herd et al., 2012). Based on the existing literature on intersectionality and diminishing returns, we hypothesized that compounding lower social statuses (e.g., being Black, women, and lower SES) would result in fewer opportunities and resources and yield more health-related risks, resulting in greater inflammation.

Our findings are consistent with work on fundamental causes of health for White men and women, whereas they support the diminishing returns framework for Black men and women. For example, having less than high school was associated with the highest CRP for Black women, with little difference in CRP between those with high school versus those with some college or more. A growing line of work demonstrates that SES does not yield equal advantages to the health of Blacks and Whites: there may be diminishing returns to higher SES for Black relative to White adults (Assari, 2018; Farmer & Ferraro, 2005; Hayward, Hummer, & Sasson, 2015; Montez, Hummer, Hayward, Woo, & Rogers, 2011). That is, compared with Whites, Blacks experience much smaller marginal returns to health from higher SES. The seemingly paradoxical association between high SES and poor health for Blacks is often referred to as "John Henryism," a phenomenon frequently documented among Black men (Felix et al., 2019; Hudson, Neighbors, Geronimus, & Jackson, 2016; Hudson et al., 2012; James, Strogatz, Wing, & Ramsey, 1987). John Henryism posits that upward mobility requires that Blacks engage in higher-effort coping, which ultimately results in the accumulation of stressors and maladaptive coping responses to buffer the effects of such stressors (Hudson et al., 2016).

Strengths

Race is associated with life course exposure to health-promoting resources and risks to health, including but not limited to early adversity, SES, residential segregation, interpersonal and structural discrimination, exposure to stress, and poorer living and working conditions (Chae et al., 2011; Williams, 2012; Williams & Jackson, 2005; Williams & Mohammed, 2009). This study adds to the existing literature in several important ways. First, it provides insight into an underexplored area in gerontology: the social patterning of inflammation. This study examines CRP as an objective marker of health underlying leading causes of morbidity and mortality. Given that CRP is less frequently studied in older adults, we believe this study makes an important contribution to understanding how social processes "get under the skin." Furthermore, this work addresses a major gap in the literature by providing an understanding of how social statuses interact to produce unequal distributions of CRP. Building on recent work by Farmer and colleagues (2020) who examined racial and gender differences in CRP, we explicitly tested three-way interactions between race, gender, and SES. Indeed, the results highlight substantial heterogeneity in the impact of socioeconomic resources on inflammatory processes across racial and gender groups. Extant work demonstrates the importance of examining CRP in isolation for assessing future risk for health problems, such as CVD and CVD-related mortality (Pearson et al., 2006). This makes CRP a useful biomarker for understanding how social processes relate to poor health through physiological dysregulation.

Limitations

Despite the potentially noteworthy findings, this study was limited in several ways. First, although there were two waves of data, this is insufficient to infer causality among the study variables, particularly because the data were collected over 4 years, and this may not have been enough time to observe gradual changes in CRP over time. The

Table 4. Lagged Effect Linear Regression Models Testing Interactive Relationship Among Race, Gender, and SES With C-Reactive Protein Over 4 Years in the HRS (n = 6,521)

	Black × woman				Black × woman × education				Black × woman × income			
	Model 1		Model 2		Model 1		Model 2		Model 1		Model 2	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.177	0.128	−0.047	0.130	0.164	0.128	−0.064	0.132	0.153	0.128	−0.084	0.132
Black[a]	−0.038	0.092	−0.039	0.093	−0.133	0.125	−0.126	0.125	−0.104	0.104	−0.103	0.107
Woman[b]	−0.026	0.028	0.005	0.028	0.005	0.034	0.041	0.036	0.035	0.036	0.076*	0.038
Education[c]												
Less than HS	0.130**	0.040	0.079*	0.040	0.169*	0.067	0.134*	0.067	0.134**	0.040	0.082*	0.040
HS education	0.086*	0.035	0.063	0.036	0.133*	0.055	0.116*	0.054	0.086*	0.036	0.063	0.036
Income tertile[d]												
Low	0.083*	0.035	0.061	0.035	0.082*	0.035	0.060	0.035	0.166**	0.056	0.161**	0.056
Middle	0.070*	0.031	0.049	0.033	0.065*	0.031	0.043	0.033	0.110*	0.047	0.090	0.049
Black × woman	0.087	0.108	0.050	0.111	0.299	0.164	0.250	0.163	0.363*	0.152	0.305	0.158
Black × less than HS					0.098	0.200	0.090	0.193				
Black × HS education					0.163	0.234	0.144	0.238				
Woman × less than HS					−0.006	0.079	−0.038	0.079				
Woman × HS education					−0.086	0.061	−0.095	0.062				
Black × woman × less than HS					−0.475*	0.235	−0.437	0.234				
Black × woman × HS education					−0.222	0.265	−0.206	0.270				
Black × low income									0.041	0.195	0.026	0.200
Black × middle income									0.091	0.148	0.097	0.149
Woman × low income									−0.137*	0.063	−0.168**	0.065
Woman × middle income									−0.088	0.072	−0.095	0.072
Black × woman × low income									−0.302	0.239	−0.262	0.251
Black × woman × middle income									−0.205	0.199	−0.182	0.208

Notes: HRS = Health and Retirement Study; HS = high school; SES = socioeconomic status. Data are pooled from the 2006 and 2008 waves of the Health and Retirement Study (HRS). Analyses were weighted to adjust for differential selection into study. Model 1 adjusted for age, marital status, and wealth. Model 2 adjusted for age, marital status, wealth, alcohol use, smoking status, physical inactivity, overweight/obesity, CES-D, and comorbid chronic conditions.

[a]Non-Hispanic White is the reference category. [b]Men are the reference category. [c]Some college or more is the reference category. [d]High income is the reference category.

Significance levels for two-tailed tests of coefficients: *p < .05, **p < .01, ***p < .001.

study findings are instructive, but must be interpreted with caution.

Second, several respondents were not eligible for the study due to missing CRP data (e.g., did not provide consent, did not participate in both waves of data collection). This could present problems in interpreting and generalizing the results of the study. For instance, excluded respondents were more likely to be Black. Despite the wide variation in wealth across race, we were unable to look at the relationship between wealth and CRP across race/gender subgroups due to small cell sizes. Furthermore, we retained cases with very high (>10.0 mg/L) CRP levels but were unable to control for recent acute infection or illness. There is limited consensus on whether these cases should be included in analyses. Existing work suggests that very high CRP may represent chronic rather than acute inflammation (Ishii et al., 2012) and that very high CRP is associated with increased risk of adverse health outcomes (Alley et al., 2006; Hamer et al., 2010). Thus, we decided to keep these cases in our sample. It is important to note that the substantive results do not change when those with very high CRP are excluded.

With the exception of CRP and BMI, the variables in the study were based on self-report, which could create problems due to recall bias. Nevertheless, existing work indicates high reliability of the HRS data (Hayward, 2002). Finally, when studying disparities in later life, there is the possibility of bias due to mortality selection. Those who are of greatest analytic interest in the study—Blacks, those of lower SES—are often in the poorest health, and more likely to die prior to the study initiation or to be lost to follow-up. If these groups have higher levels of CRP as suggested by the results, then the present findings are likely to be underestimates of the true associations.

Future Research

Few studies have examined the simultaneous effect of race, gender, and SES on health, and none to date have investigated the intersection of race, gender, and SES on CRP. Although we collapsed some college education and a college degree due to small cell sizes, preliminary analyses showed no distinguishable differences in CRP for Black men who had some college versus a college degree, but did show differences for Black women and White men and women. This is an area in need of more examination. Another fruitful area for research would be to examine the relationships among social status and inflammation longitudinally. The results from this study suggest that race, gender, and education interacted to produce unequal levels of baseline CRP, but not changes in CRP. This indicates a need for additional investigations into the long-term relationship between education and CRP across race and gender.

Future research is warranted and crucial to further address and clarify the psychosocial and biological mechanisms linking social status to negative health outcomes. The distinct life experiences of Black adults might be key to understanding why the education–CRP relationship was less graded in Black women and men. Work on John Henryism shows that Black adults report more depressive symptoms when they were frequently exposed to racial discrimination and of higher SES (Hudson et al., 2012). The Superwoman Schema is a framework that explains how stress and coping may influence Black women's health (Allen et al., 2019; Chae et al., 2011; Versey & Curtin, 2016; Woods-Giscombé, 2010). Thus, disproportionate exposure to chronic stress and racial discrimination are important avenues for research seeking to examine the mechanisms underlying the complex relationships among race, gender, SES, and CRP, particularly given the burgeoning literature linking discrimination to biological outcomes, including inflammation and allostatic load (Allen et al., 2019; Beatty Moody, Brown, Matthews, & Bromberger, 2014; Brody et al., 2014; Cobb, Parker, & Thorpe, 2020; Goosby, Cheadle, & Mitchell, 2018; Kershaw et al., 2016; Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010; Ong, Williams, Nwizu, & Gruenewald, 2017; Thomas et al., 2019; Van Dyke et al., 2017).

In conjunction with lower rates of marriage for Black women, the increased likelihood of being in single-parent families, and the greater responsibilities that Black women may face as head of the household, Black women of higher SES, particularly as they age, may face different life experiences that translate to fewer to no decreases in inflammation with improvements in SES compared with other groups, who show graded decreases in CRP with higher SES (Allen et al., 2019; Belgrave & Abrams, 2016; Felix et al., 2019; Higginbotham & Weber, 1992; Lichter, McLaughlin, Kephart, & Landry, 1992; Woods-Giscombé, 2010).

Conclusion

Many studies have documented the social patterning of CRP, but fewer have examined the potential intersectional role of race, gender, and socioeconomic inequality in stratifying CRP. The results from this study found that the relationship between education and CRP varied by race and gender. Black men and women did not experience the same benefits associated with improvements in education that White men and women did. Black women with less than high school had higher CRP than all other race, gender, and education subgroups, and at all levels of education, Black women had the highest levels of CRP. This suggests Black women bear a disproportionate burden of inflammation. Next, Black men had higher CRP with high school education compared with less than high school and any college education. In clarifying the complexity inherent in CRP, this work will contribute to a greater understanding of the biological mechanisms underlying racial disparities in leading causes of morbidity and mortality in the United States.

Supplementary Material

Supplementary data are available at *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences* online.

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Conflict of Interest

None reported.

Author Contributions

H.R.F. planned the study, performed the statistical analyses, takes full responsibility for the accuracy of the data analysis, and wrote the paper. L.A.W. and S.A.H. contributed to interpretation of the data and contributed to critical revision of the manuscript for intellectual content.

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