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Childhood Socioeconomic Status and Inflammation: Psychological Moderators Among Black and White Americans

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Objective: The current study examined race differences in how childhood socioeconomic status (SES) predicted midlife inflammation. It also tested psychological resources (purpose in life, optimism, and conscientiousness) as moderators of the association between childhood SES and inflammation among Black and White adults. ***Method:*** Data came from the biomarker subsamples of the Midlife in the United States Core and Refresher studies (n = 1,578 White and n = 395 Black participants). Childhood SES was operationalized as a composite of parental education, perceived financial status, and welfare status. Outcomes included circulating IL-6 and CRP. ***Results:*** Childhood SES did not predict IL-6 or CRP among Black or White adults in fully adjusted models. Among Black adults with low optimism, lower childhood SES predicted higher IL-6 and CRP. Among Black adults with low purpose in life, lower childhood SES predicted higher CRP (but not IL-6). Conscientiousness did not moderate childhood SES–inflammation associations among Black adults. Among White adults with low conscientiousness or low optimism, lower childhood SES predicted higher IL-6 (but not CRP). Purpose in life did not moderate associations among White adults. Effect sizes were small (≤1% variance explained) and comparable to effects of clinical risk factors in this sample (e.g., age, chronic conditions). ***Conclusions:*** Race differences in the childhood SES and inflammation association were not apparent. Childhood SES was linked to inflammation more strongly among those with fewer psychological resources across both racial groups. Psychological resources may be important moderators of inflammation in the context of early life SES disadvantage.

Keywords: childhood socioeconomic status, race disparities, psychological well-being, inflammation

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Experiencing socioeconomic disadvantage early in life has enduring negative consequences for adult mental and physical health. Individuals from more disadvantaged backgrounds have higher rates of cardiovascular disease, diabetes, and all-cause mortality, and more adverse risk factor profiles in adulthood (S. Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Galobardes, Lynch, & Smith, 2008; Pollitt, Rose, & Kaufman, 2005). Systemic inflammation is hypothesized to be an important biological mechanism linking childhood socioeconomic adversity to disease outcomes in midlife and old age (S. Cohen et al., 2010; Miller, Chen, & Parker, 2011). Environmental stimuli and psychological states, which vary systematically by socioeconomic status (SES), evoke acute immune system activation, which can lead to chronic, proinflammatory states over time due to frequent activation or breakdowns in the regulation of the immune system response (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). Chronic, systemic inflamma-

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tion has a hypothesized etiological role in age-related risk for chronic illness, frailty, and disability. Chronic inflammation is often assessed by measuring circulating levels of inflammatory proteins, such as proinflammatory cytokine interleukin-6 (IL-6) and acute phase C-reactive protein (CRP).

Life course models emphasize the importance of socioeconomic conditions in childhood and adolescence for biological programming and learned social behaviors that affect health into old age, independent of adult SES (Miller et al., 2011; Wadsworth, 1997). For example, timing models suggest that low SES in childhood has adverse effects on adult health when experienced during specific developmental windows, termed critical or sensitive periods (Cohen et al., 2010). Adverse environmental exposures during prenatal and early life, in particular, shape the structure and function of the immune system across the life course, consistent with the Developmental Origins of Health and Disease framework (Chen, Liu, Yan, Wu, & Ping, 2016). In contrast, accumulation models suggest that the duration and intensity of exposure to low SES across childhood and adolescence and into adulthood, not the developmental period when it occurs, is most important to understanding risks for poor adult health (Cohen et al., 2010). Across the SES hierarchy, resource distribution in childhood sets individuals on trajectories toward stable or deteriorating health via multiple underlying socioeconomic, psychosocial, behavioral, and biological mechanisms (Cohen et al., 2010; Warner & Hayward, 2006). While childhood SES is one determinant of adult SES, adult SES also strongly predicts health via mechanisms independent of childhood status, including access to health care, health behaviors, and stressor exposure (Adler & Stewart, 2010).

Systematic reviews and meta-analyses have found that indicators of lower childhood SES are associated with elevated IL-6 and CRP in adulthood (Liu et al., 2017; Milaniak & Jaffee, 2019; Muscatell, Brosso, & Humphreys, 2018; Nazmi & Victora, 2007), which in turn prospectively predict cardiovascular disease and other chronic conditions (Danesh et al., 2004; Ridker, Hennekens, Buring, & Rifai, 2000). However, few studies have examined racial/ethnic differences in these associations. Studies that have compared the effects of SES on health across racial groups have largely focused on adult, not childhood, SES. Black adults experience poorer health outcomes, including elevated inflammation, relative to White adults at all levels of SES. Importantly, while Black adults have a lower average SES than White adults, the race differences in SES do not fully account for race disparities in health outcomes (e.g., Dowd, Zajacova, & Aiello, 2010; Schmeer & Tarrence, 2018; Williams, Mohammed, Leavell, & Collins, 2010). Notably, there is some evidence that race differences in inflammation are smaller in magnitude, although still significant, at lower levels of SES (Farmer & Ferraro, 2005; Lewis et al., 2005). For example, in the Coronary Artery Risk Development in Young Adults (CARDIA) study, lower education was associated with greater increases in inflammation over a 15-year period, but this association was considerably weaker among Blacks than Whites (Fuller-Rowell, Curtis, Doan, & Coe, 2015). Pollitt and colleagues (2008) also found that greater cumulative exposure to low education and social class across the life course were associated with elevated inflammatory markers, but found that associations were weaker and less consistent among African Americans in the Atherosclerosis Risk in Communities cohort. Somewhat in contrast to these results, a recent meta-analytic study conducted by Muscatell and colleagues (2018) did not find evidence that race moderated the relationship between SES (in childhood and adulthood) and inflammation. However, more than half of the studies included in the meta-analysis either did not provide data on the racial breakdown of their sample or utilized samples with fewer than 10% Black participants. Building on this research, analyses herein examine the association between childhood SES and inflammation separately among Black and White adults and consider whether associations for each group remained significant after adjusting for adult SES.

A key feature of associations between SES and inflammation is the notable variability in inflammation within socioeconomic strata—not all individuals who experience low SES exhibit elevated inflammation. This observation has led to examination of moderating factors that may account for variability in outcomes. Compensatory factors, such as positive experiences and resource availability and mobilization, may disrupt the link between early life socioeconomic disadvantage and elevated inflammation in midlife. The Reserve Capacity Model and Life Span Biopsychosocial Model of Cumulative Vulnerability and Minority Health both posit that intrapersonal, interpersonal, cultural, and tangible resources may accumulate over the life course and moderate the effects of socioeconomic disadvantage (Gallo & Matthews, 2003; Myers, 2009). Such resources can offset the potential impacts of earlier socioeconomic disadvantage, enabling individuals to better function with or overcome adversity (Ferraro & Kelley-Moore, 2003). In adults, high purpose in life, sense of control, and other psychological resources appear to buffer lower SES persons from elevated inflammation (Elliot & Chapman, 2016; Elliot, Turiano, & Chapman, 2017; Morozink, Friedman, Coe, & Ryff, 2010). However, limited research has assessed whether such psychological resources also moderate links between childhood SES and adult inflammation and whether there are differences by race in these patterns.

The current paper focuses on three psychological resources—purpose in life, conscientiousness, and optimism—as possible moderators (buffers) of the association between childhood SES and inflammation. These factors are all intrapersonal resources with well-developed and standardized scales but are conceptually distinct and have demonstrated independent associations with physical health including morbidity and mortality (Boehm & Kubzansky, 2012; R. Cohen, Bavishi, & Rozanski, 2016; Rasmussen, Scheier, & Greenhouse, 2009; Ryff, 2014; Soto, 2019). Furthermore, these resources have not only been shown to moderate associations between adult SES and inflammation (Elliot & Chapman, 2016; Elliot et al., 2017; Morozink et al., 2010), but multiple lines of inquiry suggest that they may be important in understanding the link between childhood disadvantage and physical health, with some work suggesting these associations may vary by race in the United States (Baldwin, Jackson, Okoh, & Cannon, 2011; Brody, Yu, Miller, & Chen, 2016; Miller, Cohen, Janicki-Deverts, Brody, & Chen, 2016; Williams, Neighbors, & Jackson, 2003). Here we consider the effects of each psychological resource independently, as is standard in the literature on psychological factors and health, and also examine associations with multiple psychological factors in the same model so as to test whether the uniqueness of each psychological factor or the shared variance between psychological factors is most important for predicting inflammatory outcomes.

The current study has two objectives: 1) to examine links between childhood SES and circulating IL-6 and CRP separately among Black and White Americans independently of adult SES, and 2) to examine psychological resources (purpose in life, conscientiousness, and optimism) as moderators of the link between childhood SES and inflammation across race.

Method

Sample

Data came from biological subsamples from two Midlife in the United States (MIDUS) national samples, including two oversamples of African Americans from Milwaukee, Wisconsin. At the second wave of the MIDUS core (M2), a subset of respondents (n = 1,255) provided biological data, which includes a subset of Milwaukee respondents (n = 201). The M2 biological subsample has become an extensive forum of biopsychosocial research (see www.midus.wisc.edu). However, many analyses revealed that the sample was insufficiently large to permit analyses of intersectionality—namely, how patterns differed as a function of multiple demographic factors (age, gender, race, SES). Thus, a second national sample was recruited (MIDUS Refresher; MR) to parallel the age and gender distribution of the baseline sample (Goldman, Glei, & Weinstein, 2018). Biomarkers were collected on a subsample of MR respondents (n = 863, which includes n = 117 Milwaukee respondents) to augment the size of the initial biomarker subsample.

To participate in the biological data collection at M2 and MR, participants were required to complete all waves of survey data and travel to one of three General Clinic Research Centers (GCRC) around the country. At M2, the response rate among eligible participants was 43%, reflecting the demanding protocol and extensive travel for participants (Dienberg Love, Seeman, Weinstein, & Ryff, 2010). The M2 biological subsample was comparable to the M2 survey sample on most demographic and health characteristics (age, gender, race, marital status, personal income, self-rated health, body mass index [BMI], chronic conditions, physician visits), although they were more educated and less likely to smoke than nonparticipants (Dienberg Love et al., 2010). Those who identified as a race other than White or Black and/or African American at M2 were excluded from the present analyses (n = 55). At MR, the response rate for participating in the biological substudy was 42%. The MR biological subsample was comparable to the MR survey sample on gender, race, marital status, BMI, and number of chronic conditions. However, the MR biological subsample was significantly older, had higher educational attainment, higher personal income, better self-rated health, and more physician visits, and was less likely to smoke compared to the MR survey respondents who did not participate in biological data collection. MR respondents who identified as a race other than White or Black and/or African American were excluded (n = 85).

In both the M2 and MR surveys, supplemental city-specific samples of African Americans from Milwaukee, Wisconsin were recruited to increase the representation of African Americans in the MIDUS sample and to facilitate the examination of how psychosocial factors influence health in a highly segregated city. Within census blocks in which at least 40% of residents were Black, door-to-door canvassing was used to screen for individuals on the basis of race, gender, age, and income (to match the MIDUS I survey distribution). Roughly half of the Milwaukee sample resided in census blocks with a median household income below $40,000. The inclusion criteria required that participants self-identified as Black/African American, lived in a noninstitutionalized setting, were able to speak English with sufficient literacy to complete a self-administered questionnaire, and were healthy enough to complete a 40-min interview. Participants were interviewed at home using a Computer Assisted Personal Interview and Audio Computer Assisted Self Interview. The M2 Milwaukee survey sample included 592 individuals, and the MR Milwaukee survey sample included 508 individuals. Although such participants do not represent the national sample of African Americans, they have been valuable in publications on race/ethnicity and health (Slopen et al., 2012, 2010), given that Milwaukee is one of the most highly segregated cities in the U.S. (Frey, 2010), and their life experiences may well reflect the majority of Black adults in the U.S. who likewise reside within segregated urban environments.

The analytic sample combined the M2 and MR biological samples (N = 1,973; n = 1,578 White, n = 395 Black). Sample (M2 vs. MR) was included as a covariate in all analyses to address the possibility that differences in sample representativeness might influence the pattern of findings. Biological and survey data collection were approved by the Institutional Review Boards at Georgetown University, University of California, Los Angeles, and University of Wisconsin—Madison. All participants provided written informed consent. The analytic sample included 382 individuals from MIDUS 2 who were twins or siblings.

Measures

Childhood socioeconomic status. Childhood SES was measured as a retrospective self-report of parental education, perceived financial status relative to others, and welfare status. Assessments were obtained during the baseline survey assessment for all participants. Parental education was assessed as the highest level of education that either parent reported from 12 categories ranging from 0 to 6 years of school to completing a doctorate. Parental education was coded into three categories, with 2 = less than a high school education, 1 = high school graduate or GED, and 0 = some college. Financial status relative to others was assessed based on answers to the following question: "When you were growing up, was your family better off or worse off than the average family was at the time? (If your parents lived separately and had different financial situations, answer for the family you lived with for the longest time)." Responses ranged from 1 (*A lot better off*) to 7 (*A lot worse off*). Financial status relative to others was coded into three categories, with 2 = worse off, 1 = about the same as the average family, and 0 = better off. Welfare status in childhood was determined with the question "During your childhood and adolescence, was there ever a period of six months or more when your family was on welfare or ADC [Aid to Dependent Children]?" Affirmative responses ("yes") were scored 2 points and negative responses ("no") scored 0 points. A composite variable summed the above indicators to create a composite ranging from 0 to 6, with higher numbers reflecting greater childhood socioeconomic disadvantage (Gruenewald et al., 2012).

Psychological factors. Purpose in life was based on Ryff's theoretical framework (Ryff, 1989; Ryff & Keyes, 1995). Respon-

dents indicated their agreement with each of seven items on a 7-point scale ranging from strongly agree to strongly disagree (e.g., "I have a sense of direction and purpose in life.") Items were summed to form the purpose in life scale, and higher scores indicate higher purpose in life. Internal consistency was .71 at MIDUS 2 and .74 for the MIDUS Refresher sample. Optimism was assessed with the Life Orientation Test—Revised scale (Scheier, Carver, & Bridges, 1994). This scale has six items (e.g., "In uncertain times, I usually expect the best"), and ratings are made on a 5-point scale from 1 (*a lot agree*) to 5 (*a lot disagree*). Items were summed, and higher scale scores represent higher levels of optimism. Internal consistency was .80 in the MIDUS 2 sample and .82 in the MIDUS Refresher sample. Conscientiousness was assessed by respondents rating the extent to which five adjectives (organized, responsible, hardworking, careless, and thorough) described themselves on a scale from 1 (*not at all*) to 4 (*a lot*). Items were summed after reverse scoring careless. Internal consistency was .68 in the MIDUS 2 sample and .69 in the MIDUS Refresher sample.

Inflammation. Inflammatory markers were determined from a fasting blood sample taken on the morning of the second day of the clinic visit. Assays were conducted in the same laboratories using the same procedures for MIDUS 2 and Refresher participants. Serum IL-6 levels were measured with the Quantikine high-sensitivity enzyme linked immunosorbent assay (ELISA) kit (R&D Systems, Minneapolis, MN). At MIDUS 2, the interassay coefficient of variance (CV) was 12.3% and intraassay CV was 3.3%. For the MIDUS Refresher sample, the interassay CV ranged from 5% to15%, and intraassay CV was 4.7%. Plasma CRP was measured using the BNII nephelometer (Dade Behring, Inc., Deerfield, IL). The intra- and inter-CVs ranged from 2.1% to 5.7% at MIDUS 2. The intra- and inter-CVs ranged from 1.1% to 4.4% for the MIDUS Refresher sample. In all analyses, IL-6 and CRP were natural logged to address skew in the distributions.

Covariates. Covariates included age, gender, sample (M2 v. MR), BMI, chronic health conditions, and adult SES. Height and weight were measured by GCRC staff and used to calculate BMI (weight in kilograms divided by height in meters squared). BMI is a key predictor of inflammation (O'Connor et al., 2009). The chronic health conditions (summed score) were self-reported physician diagnosed diseases out of 30 possible. Inflammation is an important mechanism contributing to many of these chronic illnesses (O'Connor et al., 2009). Adult socioeconomic disadvantage was assessed with a composite similar to the childhood SES measure. Educational attainment was coded into three categories, with 0 = bachelor degree or higher, 1 = some college, and 2 = high school, GED, or less. Perceptions of the current financial situation were assessed with the following question: "Using a scale from 0 to 10 where 0 means 'the worst possible financial situation' and 10 means 'the best possible financial situation,' how would you rate your financial situation these days?" Scores were categorized so that ratings 8, 9, or 10 = 0, 4 thru 7 = 1, and 0 to 3 = 2. Money available to meet needs was coded into three categories, with 0 = more money than you need, 1 = just enough money, and 2 = not enough money. Difficulty paying bills was also coded into three categories, with 0 = not at all difficulty, 1 = not very difficult, and 2 = somewhat or very difficult. A composite variable summed the above indicators to create a composite ranging from 0 to 8, with higher numbers reflecting greater adult socioeconomic disadvantage (i.e., lower adult SES).

Statistical Analyses

Hierarchical ordinary least squares (OLS) regression models were used to test independent associations between IL-6 and CRP, respectively, with childhood SES, conscientiousness, purpose in life, or optimism, and their interaction in race-stratified models (Whitfield, Allaire, Belue, & Edwards, 2008). Race-stratified models were employed due to different sampling frames for the national and Milwaukee sample and because stratifying by race allowed for less biased analysis of within-race variability given different sample sizes for Black and White samples. Model 1 contained main effects of childhood SES, the respective psychological resource, and all covariates except adult SES. Adult SES was entered in Model 2. The interaction between childhood SES and the psychological resources were entered in Model 3. All continuous variables were standardized (i.e., z-scored), and categorical variables were centered. Significant interactions were plotted to show the strength of the association between childhood SES, as a continuous variable, and IL-6 and CRP, respectively, at the mean and plus and minus one standard deviation from the mean on each psychological resource variable (Jaccard & Turrisi, 2003). We note that a significant interaction between two continuous variables indicates that the relationship between childhood SES and inflammation changes significantly across the levels of psychological resources, even if a simple regression slope itself does not significantly differ from zero (Aiken, West, & Reno, 1991). We used results from regression analyses to compute adjusted effect sizes (squared semipartial correlations) of the unique association of each interactive effect with inflammation across models. The squared semipartial correlation is equivalent to the unique change in R^2 estimated for that predictor, over and above the other covariates in a given model, thus providing a clear estimate of the percent of unique variance accounted for by predictors in each model.

Assumptions of the OLS model were verified, including assessments for outliers and other influential data, normality of residuals, homoscedasticity, and independence of errors. Because the MIDUS 2 sample included siblings and twins (19.4% of the analytic sample), supplemental analyses were conducted using generalized estimating equations with an exchangeable within-cluster covariance structure to adjust for biological dependencies in the data (data not shown). Conclusions regarding the results were identical to those presented below.

Results

Sample Descriptives and Bivariate Correlations

Descriptive statistics stratified by race are provided in Table 1. On average, Black respondents were younger, more likely to be female, and had lower childhood and adult SES than White respondents. Black Americans also endorsed less optimism and conscientiousness compared to Whites, but racial groups did not reliably differ on purpose in life. Black Americans evidenced higher IL-6, CRP, and BMI, and more chronic conditions than Whites on average, despite being slightly younger.

Table 1
Descriptive Statistics for Study Participants by Race

Variable	White Americans (n = 1578)		Black Americans (n = 395)		Significant difference by race?
	M (*SD*)	Range	*M* (*SD*)	Range	t or χ^2
Age in years	56.2 (12.7)	25–86	50.7 (11.4)	25–85	7.79*
% female	51.7		68.4		35.36*
Sample (% MIDUS 2)	62.0		56.2		4.42*
Adult SES[a]	3.2 (2.1)	0–8	5.3 (2.1)	0–8	17.21*
Childhood SES[a]	1.7 (1.4)	0–6	2.2 (1.6)	0–6	5.98*
ln IL-6	0.7 (0.8)	−1.8–3.1	1.1 (0.7)	−0.8–3.1	8.71*
ln CRP	0.4 (1.2)	−3.0–4.4	0.9 (1.2)	−2.4–4.1	7.47*
Conscientiousness	3.4 (0.5)	1.4–4.0	3.3 (0.5)	1.6–4.0	3.25*
Purpose in life	39.4 (6.6)	10–49	39.0 (7.5)	16–49	1.03
Optimism	23.6 (4.8)	6–30	22.5 (5.1)	6–30	4.18*
BMI	29.3 (6.3)	15.0–77.6	33.1 (8.7)	16.8–69.7	9.32*
Chronic health conditions sum	2.4 (2.5)	0–29	3.3 (3.1)	0–15	6.29*

Note. CRP = C-reactive protein; SES = socioeconomic status; MIDUS = Midlife in the United States; BMI = body mass index.
[a] Higher numbers reflect greater adult and childhood socioeconomic disadvantage (i.e., lower SES), respectively.
* $p < .05$.

Table 2 reports race-stratified bivariate correlations between primary study variables. For Black Americans, childhood socioeconomic disadvantage was inversely correlated with all three psychological resources, but was not correlated with adult SES or either inflammatory marker. Adult socioeconomic disadvantage was inversely correlated with all three psychological resources and positively correlated with both IL-6 and CRP. Additionally, conscientiousness and purpose in life, but not optimism, were associated with IL-6, while CRP was uncorrelated with all psychological resources. For White Americans, childhood socioeconomic disadvantage was inversely correlated with purpose in life and optimism, but not conscientiousness. Childhood socioeconomic disadvantage was also positively correlated with adult socioeconomic disadvantage and positively correlated with both inflammatory markers. Similar to Black Americans, adult socioeconomic disadvantage was inversely correlated with all three psychological resources and positively correlated with both inflammatory markers. Conscientiousness and purpose in life, but not optimism, were inversely associated with IL-6. Purpose in life was also inversely correlated with CRP.

Is Childhood SES Associated With Inflammation Independent of Adult SES?

Tables 3 and 4 provides race-stratified results of regression analyses examining associations between childhood SES and IL-6 and CRP, respectively, net of age, gender, sample, BMI, and chronic conditions. Adult SES was added as a covariate in Model 2. For Black Americans, there were no significant main effects of childhood SES or adult SES on either IL-6 or CRP. For White Americans, lower childhood SES was associated with higher IL-6 and CRP, although associations were no longer significant when adult SES was added to the model. Lower adult SES significantly predicted higher IL-6 and CRP. Sample (M2 vs. MR) effects were evident within CRP for White Americans; the MR sample had higher CRP than the M2 sample. However, interactions between child SES and sample (M2 vs. MR) did not predict either IL-6 or CRP in race-stratified models. Furthermore, interactions between child SES and gender did not significantly predict either IL-6 or CRP in race-stratified models,

Table 2
Bivariate Correlations for Primary Study Variables by Race

Measure	1	2	3	4	5	6	7
1. Childhood SES[a]		.16*	.13*	.11*	−.04	−.11*	−.11*
2. Adult SES[a]	.10		.11*	.15*	−.16*	−.27*	−.36*
3. ln IL-6	.08	.13*		.54*	−.07*	−.10*	−.03
4. ln CRP	.03	.12*	.52*		−.04	−.07*	−.04
5. Conscientiousness	−.17*	−.23*	−.12*	−.04		.41*	.27*
6. Purpose in life	−.20*	−.34*	−.11*	−.02	.44*		.56*
7. Optimism	−.15*	−.37*	−.08	−.05	.43*	.53*	

Note. CRP = C-reactive protein; SES = socioeconomic status. Values below the diagonal in shaded cells represent correlations in the Black American sample, and values above the diagonal in non-shaded cells represent correlations in the White American sample.
[a] Higher numbers reflect greater childhood and adulthood socioeconomic disadvantage (i.e., lower SES).
* $p < .05$.

Table 3
Results of Regression Analyses Examining Associations Among Childhood Socioeconomic Disadvantage, IL-6, and the Potential Moderating Effects of Psychological Resources by Race

	Black Americans (n = 375)						White Americans (n = 1556)					
	Model 1		Model 2		Model 3		Model 1		Model 2		Model 3	
Variable	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2
Moderator 1: Conscientiousness												
Sample (1 = MR)	−.10*	.009	−.10	.008	−.09	.007	.002	<.001	.003	<.001	.002	<.001
Age	.14*	.016	.15*	.018	.15*	.020	.32*	.095	.33*	.100	.34*	.100
lnBMI	.31*	.087	.31*	.088	.30*	.083	.34*	.109	.33*	.101	.33*	.102
Gender (1 = female)	.09	.007	.08	.006	.08	.006	.04	.001	.03	.001	.03	.001
Chronic conditions	.10	.008	.07	.004	.06	.003	.10*	.010	.09*	.007	.09*	.007
Childhood SES[a]	.003	<.001	−.001	<.001	−.02	<.001	.05*	.002	.03	.001	.04	.001
Conscientiousness	−.12*	.013	−.10*	.009	−.09	.006	−.03	.001	−.02	<.001	−.02	.001
Adult SES[a]			.01	.008	.10	.008			.07*	.005	.07*	.004
Childhood SES × Conscientiousness					−.07	.004					−.07*	.004
R^2 for model	.18		.19		.19		.26		.27		.27	
Moderator 2: Purpose in life												
Sample (1 = MR)	−.10*	.009	−.09	.008	−.09	.007	.003	<.001	.01	<.001	.004	<.001
Age	.14*	.018	.16*	.020	.16*	.022	.32*	.096	.33*	.099	.34*	.100
lnBMI	.34*	.103	.34*	.103	.33*	.102	.34*	.110	.33*	.102	.33*	.102
Gender (1 = female)	.08	.005	.08	.005	.07	.005	.04	.001	.03	.001	.03	.001
Chronic conditions	.06	.003	.04	.002	.04	.001	.01*	.008	.09*	.007	.09*	.006
Childhood SES[a]	.01	<.001	.01	<.001	−.01	<.001	.04	.002	.03	.001	.03	.001
Purpose in life	−.12*	.013	−.10	.008	−.08	.005	−.04	.002	−.03	.001	−.03	.001
Adult SES[a]			.07	.004	.08	.005			.07*	.004	.07*	.004
Childhood SES × Purpose in Life					−.08	.006					−.03	.001
R^2 for model	.19		.19		.20		.26		.27		.27	
Moderator 3: Optimism												
Sample (1 = MR)	−.11*	.011	−.10*	.009	−.09	.007	.003	<.001	.01	<.001	.004	<.001
Age	.15*	.017	.16*	.020	.17*	.023	.33*	.093	.33*	.096	.34*	.097
lnBMI	.31*	.090	.31*	.090	.30*	.084	.34*	.111	.34*	.103	.33	.102
Gender (1 = female)	.07	.005	.07	.005	.07	.005	.04	.001	.03	.001	.03	.001
Chronic conditions	.10	.008	.08	.005	.07	.003	.10*	.008	.09*	.007	.09*	.007
Childhood SES[a]	.004	<.001	<.001	<.001	−.04	.001	.04	.002	.03	.001	.04	.001
Optimism	−.09	.007	−.06	.003	−.02	<.001	−.03	.001	−.01	<.001	−.01	<.001
Adult SES[a]			.09	.006	.10	.007			.07*	.004	.07*	.004
Childhood SES × Optimism					−.13*	.013					−.05*	.002
R^2 for model	.18		.18		.20		.26		.27		.27	

Note. All continuous predictor variables are z-scored. MR = MIDUS Refresher; BMI = body mass index; SES = socioeconomic status; ΔR^2 = squared semi-partial correlation indicating the unique change attributable to that predictor variable.
[a] Higher numbers reflect greater adult and childhood socioeconomic disadvantage (i.e., lower SES), respectively.
* $p < .05$.

and the three-way interaction (race × gender × childhood SES) was also not significant (data not shown).

Do Psychological Resources Moderate Associations Between Childhood SES and Inflammation?

Tables 3 and 4 also present tests of whether psychological resources moderate the associations between childhood SES and IL-6 and CRP, respectively. The tables are organized by moderator, and analyses were conducted in race-stratified models. Figure 1 shows a graphical representation of significant interactions predicting IL-6 and CRP, respectively. The significance of simple main effects of childhood SES on the inflammation at the mean and plus or minus one standard deviation on psychological resources is noted in the figure.

Conscientiousness. For Black Americans, interactions between childhood SES and conscientiousness predicting IL-6 or CRP were not significant. Conscientiousness was associated with lower IL-6 in main effect models (p = .045). For White Americans, the interaction between conscientiousness and childhood SES predicting IL-6 was significant (p = .003). As shown in Figure 1 (Panel B), the inverse association between childhood SES and IL-6 was apparent at low levels of conscientiousness, but the association became more positive as conscientiousness increased. There were no main effects of conscientiousness on IL-6 or CRP, and conscientiousness did not interact with childhood SES to predict CRP.

Purpose in life. For Black Americans, the interaction between childhood SES and purpose in life significantly predicted CRP (p = .013), but not IL-6. As shown in Panel A in Figure

Table 4
Results of Regression Analyses Examining Associations Among Childhood Socioeconomic Disadvantage, CRP, and the Potential Moderating Effects of Psychological Resources by Race

	Black Americans (n = 375)						White Americans (n = 1556)					
	Model 1		Model 2		Model 3		Model 1		Model 2		Model 3	
Variable	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2
Moderator 1: Conscientiousness												
Sample (1 = MR)	<.001	<.001	.01	<.001	.02	<.001	.06*	.003	.06*	.003	.06*	.003
Age	.01	<.001	.02	<.001	.03	.001	.10*	.009	.11*	.011	.11*	.011
lnBMI	.43*	.170	.43*	.171	.42*	.163	.45*	.190	.44*	.179	.44*	.180
Gender (1 = female)	.13*	.014	.13*	.014	.13*	.014	.18*	.031	.18*	.029	.18*	.029
Chronic conditions	.12*	.012	.09	.007	.08	.005	.02	<.001	.01	<.001	.01	<.001
Childhood SES[a]	.01	<.001	.004	<.001	−.02	<.001	.05	.002	.04	.001	.04	.001
Conscientiousness	−.04	.002	−.03	.001	−.01	<.001	−.003	<.001	.01	<.001	.004	<.001
Adult SES[a]			.09	.007	.08	.006			.07*	.004	.07*	.004
Childhood SES × Conscientiousness					−.09	.006					−.03	.001
R^2 for model	.26		.26		.27		.24		.24		.25	
Moderator 2: Purpose in life												
Sample (1 = MR)	.001	<.001	.01	<.001	.02	<.001	.06*	.003	.06*	.003	.06*	.003
Age	.003	<.001	.01	<.001	.03	.001	.10*	.010	.11*	.011	.11*	.011
lnBMI	.45*	.185	.45*	.185	.44*	.182	.45*	.190	.44*	.180	.44*	.180
Gender (1 = female)	.11*	.011	.11*	.011	.11*	.010	.18*	.032	.18*	.030	.18*	.030
Chronic conditions	.09	.007	.07	.004	.06	.003	.01	<.001	.003	<.001	.003	<.001
Childhood SES[a]	.02	<.001	.01	<.001	−.01	<.001	.05*	.002	.04	.001	.04	.001
Purpose in life	−.04	.001	−.02	<.001	.01	<.001	−.02	<.001	−.01	<.001	−.01	<.001
Adult SES[a]			.07	.004	.08	.005			.06*	.003	.06*	.003
Childhood SES × Purpose in Life					−.12*	.012					−.01	<.001
R^2 for model	.26		.26		.28		.24		.25		.25	
Moderator 3: Optimism												
Sample (1 = MR)	<.001	<.001	.01	<.001	.02	<.001	.06*	.003	.06*	.003	.06*	.003
Age	.02	<.001	.03	.001	.04	.002	.10*	.009	.11*	.010	.11*	.011
lnBMI	.44*	.175	.44*	.175	.43*	.167	.45*	.192	.44*	.181	.44*	.180
Gender (1 = female)	.12*	.013	.12*	.013	.12*	.013	.18*	.032	.18*	.029	.17*	.028
Chronic conditions	.11*	.010	.09	.006	.08	.005	.01	<.001	.01	<.001	.01	<.001
Childhood SES[a]	.004	<.001	.001	<.001	−.04	.001	.05*	.002	.04	.001	.04	.002
Optimism	−.05	.002	−.02	.001	.01	<.001	−.01	<.001	.01	<.001	.01	<.001
Adult SES[a]			.08	.004	.09	.006			.07*	.004	.07*	.004
Childhood SES × Optimism					−.13*	.013					−.04	.002
R^2 for model	.26		.26		.28		.24		.25		.25	

Note. All continuous predictor variables are z-scored. CRP = C-reactive protein; MR = MIDUS Refresher; BMI = body mass index; SES = socioeconomic status; ΔR^2 = squared semi-partial correlation indicating the unique change attributable to that predictor variable.
[a] Higher numbers reflect greater adult and childhood socioeconomic disadvantage (i.e., lower SES), respectively.
* $p < .05$.

1, the inverse association between childhood SES and CRP was apparent at low levels of purpose in life and became more positive as purpose increased. There were no main effects of purpose in life on IL-6 or CRP. For White Americans, there were no main effects of purpose in life on IL-6 or CRP, and purpose in life did not interact with childhood SES to predict IL-6 or CRP.

Optimism. For Black Americans, interactions between childhood SES and optimism predicting both IL-6 and CRP were significant (*p*s < .016). Childhood SES and IL-6 and CRP, respectively, were inversely related at low levels of optimism, and the association became more positive as optimism increased (Figure 1, Panel A). Main effects of optimism on either IL-6 or CRP were not significant. For White Americans, the interaction between childhood SES and optimism significantly predicted IL-6 (p = .023) but not CRP (p = .071). Both interaction effect sizes were small in magnitude (both $R^2 < .01$). Childhood SES and IL-6 were inversely related at low levels of optimism, and the relationship became more positive as optimism increased (Figure 1, Panel B). The pattern of simple effects was similar for the interaction predicting CRP. There were no main effects for optimism on IL-6 or CRP.

Additional Analyses

Are psychological resources differentially protective across racial groups? Despite similar patterns of associations across race, moderators, and outcomes, statistical significance and effect sizes of interactions often differed among Black and White adults. Thus, we applied a statistical test to determine if regression coefficients could be considered equal across race (Paternoster, Brame, Mazerolle, & Piquero, 1998). All tests failed to reject the null

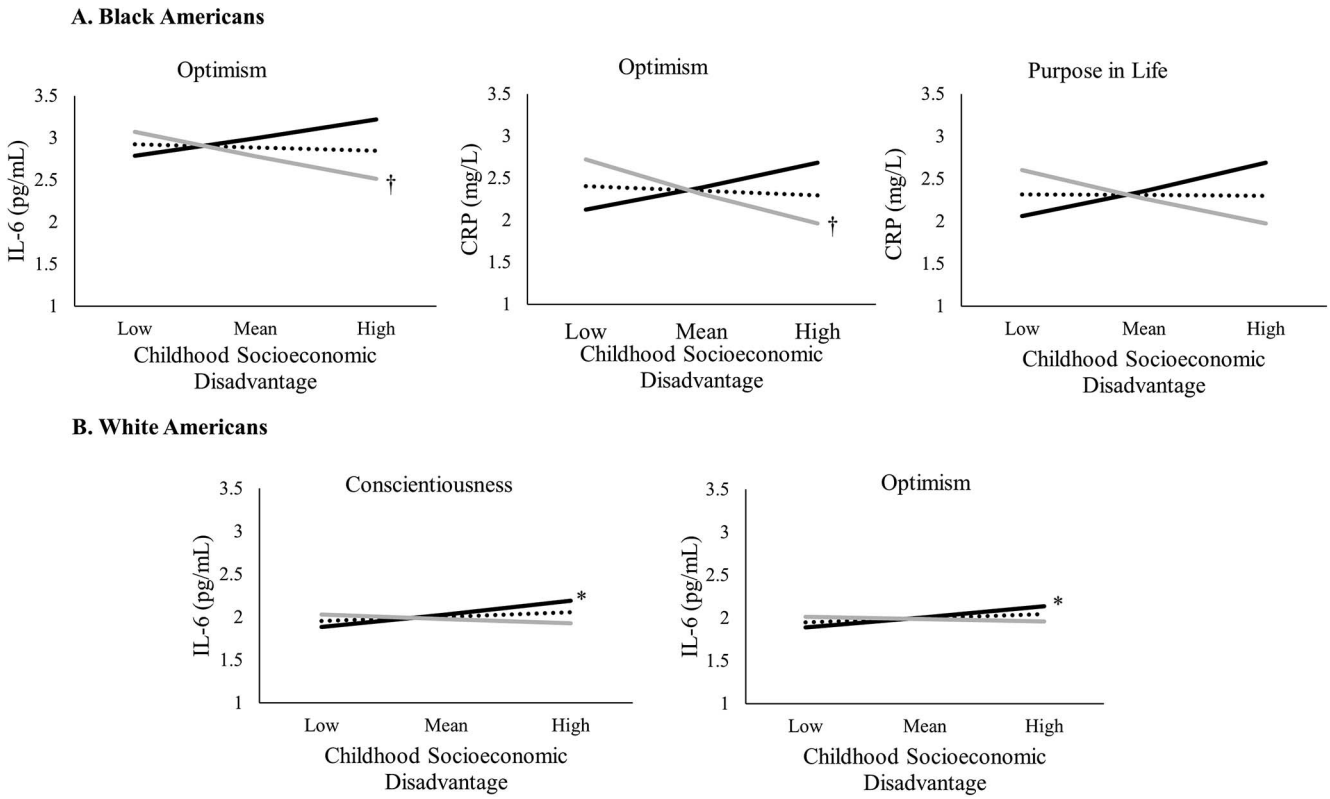

Figure 1. Moderation of childhood SES on adult IL-6 and CRP by psychosocial resources controlling for age, gender, sample, BMI, chronic conditions, and adult SES. For all figures solid black lines represent scores below −1 SD on the moderator, dotted lines represent scores around the mean on the moderator, and solid gray lines represent scores above +1 SD on the moderator. Note that models were run with ln(IL-6) and ln(CRP) as the outcome, but the values have been exponentiated for visual purposes only. * simple slope $p < .05$; † simple slope $p < .10$.

hypothesis, indicating that coefficients between childhood SES, psychological resources, and their interaction and IL-6 and CRP, respectively, were equivalent across race. We also statistically examined whether psychological resources were similarly protective across racial groups by testing three-way interactions among race, childhood SES, and psychological resources on IL-6 and CRP, respectively. According to G*Power (Faul, Erdfelder, Buchner, & Lang, 2009), the required sample size to detect a small effect size ($f^2 = .02$) in a model with 13 predictors (as in the fully adjusted models) and alpha of .05 was 1,339, below our sample size. Results are provided in supplemental Tables 1 and 2. We found no evidence that the interaction between childhood SES and psychological resources on IL-6 differed by race (all $ps > .28$). However, the interaction between childhood SES and purpose in life on CRP was significantly moderated by race ($p = .048$). As described above, purpose in life buffered the association between lower childhood SES and higher CRP for Black Americans but showed no effect for White Americans. Consistent with results for IL-6, we found no evidence of race moderation for interactions of childhood SES with conscientiousness or optimism on CRP ($ps > .20$).

Independence among psychological moderators. Given correlations among the psychological resources examined here ($rs > .27$), we examined whether any significant interaction effects between childhood SES and resources remained similar in magnitude and significance when regression models included the main effects for the other two resources not previously in the model (supplemental Tables 3 and 4). There were no significant differences in moderation results when controlling for other psychological resources, suggesting that each psychological factor operates independently as a moderator of the association between childhood SES and inflammation.

Discussion

Given limited knowledge regarding how childhood SES relates to inflammation among both Black and White Americans, a key objective of the current paper was to test such associations in the MIDUS cohort. We explicitly considered whether such associations were independent of adult SES. Furthermore, we considered three distinct psychological resources, namely conscientiousness, purpose in life, and optimism, as moderators of the childhood SES and inflammation link. Below, we first discuss the lack of associations between childhood SES and inflammation across race after accounting for adult SES. We then review the findings on psychological resources moderating the association between childhood

SES and inflammatory markers. Limitations of the study are considered, including sampling issues and the measurement of SES. We conclude by considering the practical significance of the results and the future directions toward which they point, such as identifying antecedents to psychological resources.

Initial findings revealed that there were no significant associations between childhood SES and IL-6 and CRP, independent of age, gender, sample, BMI, chronic conditions, and adult SES in either racial group. Adult SES was inversely associated with both IL-6 and CRP among White Americans, suggesting that concurrent SES is more closely associated with inflammation at midlife in this group than childhood SES. Notably, previous results from the MIDUS 2 biomarker subsample found that early life adversity was associated more strongly with IL-6 and CRP among African Americans compared to Whites (Slopen et al., 2010). However, early life adversity in that investigation was not operationalized as SES, but rather as a composite of stressful experiences in childhood and adolescence, relationships with parents during childhood, and frequency of verbal and physical assault by parents. Together, these findings suggest that the mechanisms for potential race differences in inflammation may differ between adverse childhood experiences and childhood SES.

There was modest evidence supporting interactions between childhood SES and psychological resources on inflammation in fully adjusted models. Moderation patterns (childhood SES by psychological resources on inflammation) appeared similar in Black and White adults and were statistically indistinguishable, supported by the lack of significant effects (i.e., $p < .05$) for three-way interactions with race as well as tests of regression coefficient equivalence. However, the specific psychological resources that emerged as significant moderators differed slightly in the Black and White samples. Due to methodological differences based on race, potential clinical significance, and guidelines for examining within-group variability by race (Whitfield et al., 2008), we present race-stratified results despite the lack of statistically reliable differences by race in the current sample. Generally, inverse associations between childhood SES and IL-6 and CRP were apparent among those with low psychological resources, suggesting that low levels of psychological resources may strengthen the relationship between childhood SES and adult inflammation. Graded associations between childhood SES and inflammation were not present among those with higher conscientiousness and purpose in life. These results support psychological resources as a buffer against elevated inflammation among both Black and White adults from socioeconomically disadvantaged backgrounds.

However, among Black adults with higher optimism, higher childhood SES was associated with higher IL-6 and CRP. That is, the interactions revealed that the direction of the association between childhood SES and inflammation flips at high, relative to low, optimism for Black adults. These patterns are consistent with prior research demonstrating that optimism is associated with worse immunity outcomes in the context of prolonged stressors, perhaps due to continued engagement with difficult circumstances enacting a physiological toll (Segerstrom, 2005). Given the segregated urban context from which most of the Black adults were drawn, it may be that Black optimists from higher childhood SES backgrounds continually engage with more difficult situations that highlight and reinforce social inequalities (e.g., racism) than Black optimists from lower childhood SES backgrounds. Future research should consider how daily interpersonal interactions differ across race, SES, and psychological characteristics and how social mobility affects opportunities to develop and maintain psychological resources along with effects on physical health across racial groups.

The unique effect sizes of psychological factors on IL-6 and CRP were small in terms of the percentage of variance accounted for after adjusting for adult SES and demographic and health covariates (i.e., $\leq 1\%$). Despite this small effect size, it is important to underscore that the predicted difference in IL-6 for an individual with low childhood SES and high conscientiousness, relative to low conscientiousness, was 0.3 pg/mL for Whites and 0.8 pg/mL for Blacks. Similarly, the predicted CRP for an individual with low childhood SES and high optimism, relative to low optimism, was 0.2 μg/mL for Whites and 0.9 μg/mL for Blacks. These effects are comparable to the effect sizes for age and chronic conditions on IL-6 and CRP (see Tables 3 and 4), thus supporting the relative importance of psychological resources as key factors affecting physiological markers.

The current study extends the literature on socioeconomic and psychological predictors of inflammation by demonstrating that psychological buffering was present among both Black and White adults and that such buffering applies to childhood SES, net of adult SES and multiple other covariates, including health status. Because three respective psychological resources were tested, results provide a nuanced understanding of how childhood SES and psychological resources independently and jointly predict adult inflammatory biology. Investigating multiple psychological resources is a key strength of the study given that most prior research examines only a single psychological factor at a time. Including multiple psychological factors in the same study allows for comparison of effects across psychological factors and investigation of the overlap among psychological factors in predicting inflammatory markers. When other psychological resources were included in interaction models as main effects, none of the observed interactions changed in magnitude or significance, consistent with the idea that how resources buffer associations between childhood SES and inflammation is independent of the main effects of the other resources. As such, developing and maintaining any such resources may be protective, especially in contexts of social disadvantage. Religion and spirituality, supportive role models, particularly in childhood and adolescence, and community level factors may be fruitful targets to pursue as antecedents to high psychological resources later in life. We previously found that parenting practices in adolescence (i.e., consistency, supervision, communication) were important mediating factors linking childhood SES to psychological resources, health behaviors, and cardiovascular health in adulthood within a community sample of Black and White men (Boylan, Cundiff, Jakubowski, Pardini, & Matthews, 2018; Matthews et al., 2017). Future research should continue interrogating diverse pathways through which individuals come to have high psychological resources, including across different socioeconomic contexts.

Results and conclusions drawn from this study should be interpreted in light of several limitations. First, the race-based comparison involved a national sample of White Americans and a city-specific sample of Black Americans from Milwaukee, Wisconsin. Although both samples were recruited with proba-

bility sampling designs, a city-specific sample does not represent Blacks in the U.S. nationally. Milwaukee does, however, offer an informative context for studying race disparities given that the majority of Black adults in the U.S. reside within segregated urban environments, and the Milwaukee sample may well represent these life experiences. Second, the biological subsamples, especially the Refresher sample, were significantly healthier and better educated than the MIDUS survey samples, potentially limiting representation at the lower end of the social hierarchy. It is possible that stronger effects would be evident in more representative samples with increased heterogeneity. Third, childhood SES was assessed via retrospective report, which may be biased due to recall bias or memory problems, especially for the subjective measures of SES. In a systematic review and meta-analysis, Senese, Almeida, Fath, Smith, and Loucks (2009) examined whether the association between childhood SES and obesity varied based on timing of child SES measurement (concurrently in childhood vs. retrospectively). Effect sizes were smaller when childhood SES was measured retrospectively, suggesting that recall bias in the measurement of childhood SES led to an underestimation, not an inflation, of the association with obesity. Additionally, our measures of SES did not include occupational status, a common metric of childhood and adult SES (Galobardes, Smith, & Lynch, 2006). While data on respondent and parental occupational status were available in MIDUS, we opted not to include occupational status given inherent difficulties in categorizing this measure. For instance, there are not clear guidelines regarding cut-points for high, mid, and low status occupations, and there are also challenges in characterizing individuals who are retired and/or homemakers. In the MIDUS 2 core survey sample, for example, 47.5% reported that they were not currently working for pay. Nonetheless, the measures of childhood and adult SES followed precedent from the prior literature (e.g., Gruenewald et al., 2012), appropriately captured the multidimensional nature of SES, and captured dimensions of both access to resources as well as social rank. Fourth, the measure of conscientiousness had relatively low internal consistency, likely due to the small number of items used to assess the trait. National survey research necessitates balancing among comprehensive coverage of individual psychological constructors with participant burden and scope of psychological questionnaires assessed. Mroczek and Kolarz (1998) have demonstrated that the personality measures in MIDUS have high test–retest reliability and good construct validity. The present measure also correlates highly ($r > .8$) with more comprehensive personality assessments (Lachman & Weaver, 1997). Finally, several interaction effects were tested (three moderators and two outcomes across race), perhaps leading to concerns of spurious findings. However, interactions were predicted a priori and patterns were largely consistent across moderators and outcomes.

In conclusion, this study contributes further evidence linking childhood SES and adult psychological resources to inflammation in both Black and White Americans. The integration of childhood SES and psychological functioning within a diverse sample demonstrates that childhood SES is differentially associated with inflammation across levels of psychological resources in both Black and White Americans. Given the potential importance of childhood in shaping health trajectories across the life course, future research should continue investigating the interplay between early life SES and psychosocial factors as contributors to health risks in adulthood. So doing could point to possible strategies to reduce such disparities. Overall, the new findings add to a growing literature documenting physiological dysregulation following early socioeconomic disadvantage and point to the protective role of psychological resources vis-à-vis such disadvantage across racial/ethnic groups.

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