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Short Communication

Assessing the role of socioeconomic status and discrimination exposure for racial disparities in inflammation

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ABSTRACT

Socioeconomic status (SES) and discrimination have been implicated as social determinants of health and health disparities. Yet, very little research has been done to assess their contributing role in Black-White disparities in inflammation. Using data from the Midlife in the United States (2004–2006), we conducted Oaxaca-Blinder decomposition analysis to quantify the extent to which three indicators of SES (i.e., education, household income, and employment status) and three forms of discrimination exposures (i.e., everyday, lifetime, and workplace discrimination) explained Black-White differences in inflammation. Education, particularly having a college degree or more, explained 16.88% of the differences between Blacks and Whites. There was no evidence that household income and employment status explained Black-White inflammation differences. Lifetime discrimination significantly explained 18.18% of Black-White difference in inflammation burden. There was no evidence that everyday and workplace discrimination explained Black-White difference in inflammation burden. Together, the predictors explained 44.16% of inflammation differences between Black and White participants. Education and lifetime exposure to discrimination may play a role in inflammation disparities. Further research is needed to examine other dimensions of SES (e.g., wealth) and discrimination (e.g., racial segregation) that are associated with health to better understand the contributions of these key social determinants of Black-White inflammation disparities.

1. Introduction

Racial disparities in health are pervasive across a broad range of outcomes and have been persistent for decades. Compared to White Americans, Black Americans experience higher chronic disease burden on a wide range of health conditions, including obesity, Type 2 diabetes, hypertension, and cardiovascular disease–with these disparities being significantly pronounced by middle-age (Cunningham et al., 2017; Kochanek et al., 2019; Quiñones et al., 2019). As a result, Black Americans exhibit higher mortality and shorter life expectancy compared to their White counterparts (Arias et al., 2021; Cunningham et al., 2017). Black Americans exhibit mortality risks that are 1.19 times as high as White Americans (Cunningham et al., 2017; Kochanek et al., 2019; Masters et al., 2014). Black Americans also have a life expectancy that is six years shorter than White Americans (Arias et al., 2021), highlighting the public health significance of working to better understand the sources of these disparities.

These health disparities are often theorized to reflect a weathering process, whereby economic and social adversities accumulate over the life course to produce premature biological aging in Black Americans (Forde et al., 2019; Geronimus, 1992). Inflammation is a biological marker of aging and serve as an important marker of physiological dysregulation and aging-related diseases (Singh and Newman, 2011). C-reactive protein (CRP), for instance, is a marker of inflammatory response and immune function and elevated levels of this protein are associated with a wide range of chronic conditions, including heart disease, hypertension, and stroke (Khera et al., 2005; Strang and Schunkert, 2014). Black Americans exhibit higher levels of CRP and other markers of inflammation compared to Whites (Boen, 2020; Stepanikova et al., 2017). While Black-White disparities in inflammation are found across different age cohorts, disparities are most pronounced in middle and older age (Geronimus et al., 2006; Lachman et al., 2015; Levine and Crimmins, 2014; Schmeer and Tarrence, 2018). Therefore, there is a need to identify the economic and social risk factors that produce inflammation disparities in later life and examine the extent those risk factors explain existing disparities.

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The weathering hypothesis posits that existing racial health disparities is partially explained by the disproportionate socioeconomic hardship experienced by Black Americans (Forde et al., 2019; Geronimus, 1992). In 2020, the median Black household income was $46,600, whereas the median White household income was approximately $75,000. In other words, a Black household earned 61 cents for every dollar a White household earned (Wilson, 2020). The 2019 U.S. Census data states that 40.1% of non-Hispanic white Americans held a bachelor's degree or higher, compared to 26.1% of non-Hispanic Black Americans. Finally, in 47 of the past 54 years, Black Americans have faced unemployment rates twice as high as those of their White counterparts (Pew Research Center, 2016).

SES has largely been implicated in explaining racial health disparities as well-established evidence suggests that low SES is associated with more health compromising behaviors and exposure to poor neighborhood conditions. Low SES is associated with higher smoking prevalence, less exercise, poorer diet, and excess weight (Pampel et al., 2010). SES also affects the means to reach healthy goals. All groups may have similar desires for healthy behavior, but low-SES groups have more obstacles in reaching these goals. Low SES also serve as a proxy for exposure to psychosocial stressors. People with low SES are disproportionately exposed to environmental toxins, violent and nonviolent crimes, and air pollution (Williams et al., 2019). As expected, SES has been found to be a strong predictor of inflammation (Muscatell et al., 2020; Stepanikova et al., 2017). Nevertheless, SES does not fully explain racial disparities in inflammation between groups.

To understand how and why these disparities persist, we have to examine the social exposures that are unique to Black Americans. The weathering hypothesis also recognizes that the repeated experience with social adversities lead to early health deterioration for Black Americans. Discrimination, as a stressor, is a prominent risk factor of disease and illness and Black Americans are disproportionately exposed to this stressor. Exposure to discrimination (also known as unfair treatment) can induce negative emotional reaction and activate regulatory physiological systems—cardiovascular, neuroendocrine, and immune function. Chronic activation to these interrelated systems can lead to dysregulation and increased levels of cortisol and inflammation. A recent systematic review found that exposure to discrimination, regardless of attribution (e.g., race, sex, sexual orientation) is a risk factor to chronic, low-grade inflammation (Cuevas et al., 2020). However, the authors point out that very few studies examined the relative contribution of discrimination to racial disparities in inflammation.

Using a decomposition approach allows us to detect how much differences in an outcome would be reduced if one group had the same mean levels of the measured attributes compared to another group. In other words, the approach can be used to quantify the individual and joint contribution of potentially correlated exposures to Black-White inflammation disparities. SES and discrimination are elements within the broader weathering hypothesis. Disentangling these two constructs would provide a better understanding of their contribution to inflammation disparities. In the present study, we applied a decomposition method to examine the relative contribution of three indicators of SES (i.e., household income, education, and employment status) and three measures of discrimination (i.e., everyday discrimination, lifetime discrimination, and chronic job discrimination), in explaining inflammation disparities between Black and White adults in a national probability sample of adults. These findings can inform future interventions as they identify key areas that can be targeted to reduce Black-White disparities in inflammation.

2. Methods

2.1. Data and analytic sample

The Midlife in the United States (MIDUS) is a nationally representative study of noninstitutionalized, English-speaking adults in the US. MIDUS 1 (1995–1996) was designed to be an interdisciplinary examination of the role of sociodemographic, psychosocial, and behavioral factors for mental and physical health (Brim et al., 2004; Ryff and Krueger, 2018). A follow-up (known as MIDUS 2) was conducted approximately a decade later (2004–2006), with repeated baseline assessments and a comprehensive assessment of biomarkers (involving 2-day visits to biomedical clinics) on a subsample of respondents (Ryff and Krueger, 2018). MIDUS 2 was further enhanced by recruiting a new subsample of Black Americans from Milwaukee, Wisconsin. Respondents completed the questionnaires that parallel all the survey and a subsample completed the biomarker assessments. Biomarker data included a participant's complete medical history, physical examination, and the collection of blood, urine, and saliva samples (for additional details, see Dienberg Love et al., 2010). Data collection for the MIDUS, Milwaukee, and biomarker studies were approved by Institutional Review Boards at each participating site, and all participants provided informed consent. Participants in the MIDUS 2 and Milwaukee sample with the psychosocial measures and biomarkers of interest were included in this study. The analytic sample of the current study consisted of 1143 adults aged 28 to 85.

2.2. Measures

Inflammation burden. As performed in previous studies (Glei et al., 2013); (Kang and Marks, 2014); (Yang et al., 2017), quartile values for items assessing inflammation were computed to create dichotomous variables for each item, where 1 = high risk quartile (i.e., high risk = being in the highest quarter of the distribution for IL-6 CRP, E-selectin, ICAM-1, and fibrinogen) and 0 = otherwise). The composite score of inflammation burden, designed to summarize dysregulation across multiple inflammatory markers, was then computed by summing five of the dichotomous inflammatory markers: IL-6, CRP, E-selectin, ICAM-1, and fibrinogen. Details on the measurement and assay of biomarkers are provided as supplementary materials.

Covariates. Socio-demographic and medication use were considered covariates based on the possibility of confounding the associations between discrimination and inflammation. Socio-demographic covariates included age (in years), gender, and use of the following medications: antihypertensive, cholesterol lowering, steroid, and antidepressant medications.

Socioeconomic status. The following variables were selected as indicators of SES: education (less than high school, high school/GED, some college, or college degree or higher), employment status (yes or no), and household income (continuous).

Lifetime discrimination. A total of 11 settings of various lifetime occurrences of reported discrimination were assessed (Kessler et al., 1999), including academic (discouraged from continuing education, denied scholarship), employment (not hired or promoted, fired), financial services (denied a bank loan, prevented from renting or buying a home, given inferior service), and social hostility (forced out of a neighborhood, hassled by the police). Respondents indicated how many times they experienced each event (i.e., 1 = event occurred one or more times, 0 = event never occurred). The summary score ranged from 0 to 11, whereby a higher score indicated greater lifetime discrimination.

Everyday discrimination. Everyday discrimination interactions were assessed with the 9-item Detroit Area Study Everyday Discrimination Scale (Williams et al., 1997). Respondents accounted for the frequency of various forms of interpersonal unfair treatment in their daily lives. Items included: Being treated with less courtesy or respect than others; receiving poorer service than others at restaurants or stores; being called names, insulted, threatened, or harassed; having people act afraid of the respondent; having people act as if the respondent was dishonest, not smart, or not as good as they were. The frequency of each type of everyday discrimination was assessed using a 4-point scale (1 = never; 2 = rarely; 3 = sometimes; 4 = often). Responses were averaged to form a summary score of everyday discrimination (Cronbach α's for the 9-item

index was 0.93). The score ranged from 9 to 36, whereby a higher score indicated greater everyday discrimination.

Chronic job discrimination: Chronic job discrimination was assessed based on responses to six questions. Questions included discriminatory occurrences that resulted in perceived unfair treatment in the workplace. Sample questions included, "How often do your coworkers direct racial or ethnic slurs or jokes at you?"; "How often have you been unfairly humiliated in front of others at work?" Responses were solicited on a five-point scale ranging from 1 (Once a week or more) to 5 (Never). Cronbach's alpha reliability was 0.76. The score ranged from 6 to 30, whereby a higher score indicated greater chronic job discrimination.

Statistical Analyses. We used ANOVA and χ^2 tests to examine distributions of household income, education, and employment status, discrimination measures, and covariates in the whole study sample and by race/ethnicity. We then used the Oaxaca-Blinder decomposition to assess the explanatory effects of the independent variables (covariates [i.e., age, sex, medication use], SES [household income, education, and employment status], and discrimination [i.e., everyday discrimination, lifetime discrimination, and chronic job discrimination) on inflammation disparities between Black and White participants. Household income was positively skewed. Therefore, we square-root transformed household income before applying the decomposition method. The Oaxaca-Blinder decomposition quantifies the proportion of racial/ethnic differences in inflammation with the independent variable, which is referred to as the "explained" portion. It also produces the proportion "unexplained," which is the differences in inflammation that would remain even if the disadvantaged group had the same mean levels on all the independent variables as White participants. A more detailed description of the approach is found elsewhere (Elder et al., 2010).

3. Results

Of the 1,143 participants in the final sample, 82% of participants identified as White/European American and 18% identified as Black/African American. White individuals had significantly higher levels of household income and education than Black participants. Black participants had higher exposure to everyday discrimination, lifetime discrimination, and chronic job discrimination. Black participants also had significantly higher inflammatory levels across four of the five domains, CRP, IL6, E-Selectin, and fibrinogen. Last, Black participants had higher inflammation burden compared to their White counterparts. The distribution of household income, education, and employment status, lifetime discrimination, everyday discrimination, chronic workplace discrimination, and covariates for the overall sample and by race/ethnicity status are shown in Table 1.

3.1. Decomposition of Black–White difference

The "explained" portions of the Oaxaca-Blinder models are presented in Table 2. Having a college degree explained 16.88% of differences between Black and White participants in inflammation burden. There was no evidence that income and employment status explained racial differences in inflammation. Notably, lifetime discrimination explained 18.18% of differences in inflammation burden between Black and White participants. The combined predictors explained 44.16% of the differences between Black and White participants.

3.2. Sensitivity analyses

We examined the proportion explained by the predictors for the individual inflammatory markers. Having a college degree or more explained 39.29% in CRP differences, whereas lifetime discrimination explained 23.21% in CRP differences (see supplemental materials). Lifetime discrimination explained 28.94% in IL6 differences. There was no evidence that household income, education, and employment status explained Black-White differences in IL6. Lifetime discrimination explained 36.55% in fibrinogen differences. There was no evidence that household income, education, and employment status explained Black-White differences in fibrinogen. Household income, education, and employment status nor lifetime discrimination, everyday discrimination, and chronic workplace discrimination explained Black-White differences in E-selectin and ICAM-1.

Table 1
Sample characteristics.

	Stratified by Race Status				
	Non-Hispanic White (N = 941)		Non-Hispanic Black (N = 202)		
Discrimination measures					
	Mean	SD	Mean	SD	p-value
Everyday discrimination (9–36)	12.45	4.05	15.16	6.54	<0.001
Lifetime discrimination (0–10)	0.87	1.43	2.97	2.78	<0.001
Job discrimination (6–30)	10.30	4.26	12.30	5.54	<0.001
Socioeconomic indicators					
	Mean	SD	Mean	SD	p-value
Income	77500.04	60910.40	40149.11	37125.13	<0.001
	n	%	N	%	
Education					<0.001
Less than high school	29	3.1	32	15.8	
High school	195	20.8	59	29.2	
Some college	268	28.5	70	34.7	
College degree and above	447	47.6	41	20.3	
Employment status					0.27
Has a job	435	46.3	85	42.3	
Does not have a job	504	53.7	116	57.7	
Covariates					
Gender					<0.001
Male	432	45.9	67	33.2	
Female	509	54.1	135	66.8	
Hypertension medication					0.001
No	629	77.7	134	67.0	
Yes	181	22.3	66	33.0	
Cholesterol medication					0.009
No	605	73.5	161	81.7	
Yes	218	26.5	36	18.3	
Hormone therapy					0.10
No	689	89.2	184	94.8	
Yes	83	10.8	10	5.2	
Anxiety/depression medication					0.80
No	649	82.3	170	86.7	
Yes	140	17.7	26	13.3	
Inflammatory markers					
	Mean	SD	Mean	SD	p-value
CRP	2.79	2.84	4.58	6.54	<0.001
IL6	2.71	4.37	3.99	3.53	<0.001
Fibrinogen	339.17	83.11	390.50	96.02	<0.001
ICAM1	290.03	100.45	272.24	157.72	0.58
E-Selectin	41.23	20.54	50.69	27.52	<0.001
Age (years)	55.60	11.82	51.14	10.60	<0.001
Number of high inflammatory levels (0–5)	1.07	1.18	1.90	1.31	<0.001

Note: Independent sample t-tests (for continuous variables) and Chi-square tests (for categorical variables) were conducted to evaluate racial differences.

Table 2
Proportion of Black-White differences in inflammation attributable to socioeconomic status, discrimination, and covariates.

Predictors	Black vs. White	Percent explained
	Coeff (95% CI)	
Income	0.01 (-0.0.06, 0.07)	1.30 %
Education		
Less than high school (reference)		
High school/GED	−0.01 (-0.03, 0.02)	−1.30%
Some college	−0.02 (-0.05, 0.02)	−2.6 %
College degree and above	0.13 (0.02, 0.24)	16.88%
Employment status		
Has a job (reference)		
Does not have a job	−0.002 (-0.01, 0.01)	0.00 %
Everyday discrimination	0.02 (-0.06, 0.10)	2.6 %
Lifetime discrimination	0.14 (0.0, 0.27)	18.18 %
Job discrimination	0.04 (-0.01, 0.10)	5.19%
Sex	0.01 (-0.01, 0.03)	1.30 %
Age	−0.02 (-0.04, 0.01)	−2.60 %
Medication use		
Hypertension medication	0.03 (-0.00, 0.07)	3.90 %
Cholesterol medication	−0.01 (-0.03, 0.01)	−1.30 %
Hormone therapy	0.02 (-0.01, 0.04)	2.60 %
Anxiety/depression medication	−0.01 (-0.02, 0.01)	−1.30 %
Explained total	0.34 (0.18, 0.49)	44.16 %
Unexplained	0.43 (0.19, 0.67)	55.84 %
Total predicted gap	0.77 (0.56, 0.98)	

4. Discussion

We used the Oaxaca-Blinder decomposition method to examine the explanatory effects of SES (i.e., household income, education, and employment status) and three indicators of discrimination exposure Black-White disparities in inflammation. Having a college degree or more explained 16.88% of racial inflammation disparities. In other words, if Black and White participants had the same level of college degree attainment, the differences in inflammation would be reduced by 16.88%. There was no evidence that other indicators of SES (i.e., household income and employment status) explained racial differences in inflammation. Lifetime discrimination explained 18.18% of racial disparities in inflammation. There was no evidence that everyday discrimination and chronic job discrimination played a statistically significant role in explaining inflammation disparities. In examining the individual inflammatory markers, we found that having a college degree or more and lifetime discrimination explained 39.29% and 23.21%, respectively, in CRP differences. Lifetime discrimination explained 28.94% of IL6 differences and 37.03% in fibrinogen differences.

We found that college degree attainment explained a substantial proportion of Black-White inflammation disparities. The weathering hypothesis posits that exposure to adversity across the life course leads to an accelerated decline in health (Forde et al., 2019; Geronimus, 1992). A systematic review found evidence of weathering in socioeconomically disadvantaged groups, marked by greater physiological dysregulation compared to socioeconomically advantaged groups (Forde et al., 2019). While socioeconomic status typically encompasses education, household income, and employment status, educational attainment is antecedent to employment and income (Zajacova and Lawrence, 2018). Higher education predicts employment stability and higher wages and income; thus, allowing individuals and families to accumulate wealth that can be used to improve health (Zajacova and Lawrence, 2018). There are other pathways by which education improves health, independent of income and employment status, including engagement in health promoting behaviors and better access to social support (Zajacova and Lawrence, 2018). The overrepresentation of Black Americans at low levels of educational attainment suggests that Black Americans experience greater adversity as a result of lower education, such as job instability, lower income, and lower access to social support and healthcare (Zajacova and Lawrence, 2018). Thus, education represents a major source of the explanatory power of Black-White inflammation disparities. Nevertheless, education alone does not completely explain existing disparities. Discrimination is a distinct exposure that Black Americans experience at notably higher levels than White Americans and could explain racial differences in weathering patterns (Forde et al., 2019). Both acute and chronic forms of discrimination can operate like other psychosocial stressors, in that they can induce negative emotional reactions and activate physiological systems (Cuevas et al., 2020). A recent review found that continual exposure to discrimination increase the risk of low-grade inflammation (Cuevas et al., 2020). Adding to this body of literature, we found that lifetime discrimination explained a substantial proportion of racial differences, above and beyond education. In other words, the disproportionate exposure to acute forms of discrimination across the life course may lead to a disparate inflammation burden in Black Americans. More research is needed to identify psychosocial resources that can mitigate the biological impact of lifetime discrimination. It is important to note, however, that although everyday and chronic job discrimination did not explain a significant proportion of inflammation disparities, they are still health risk factors that disproportionately affects Black Americans (Williams et al., 2019). Further research should replicate these findings using other physiological indicators of health.

4.1. Limitations

We focused on one indicator of physiological dysregulation and aging-related disease. Future studies should examine other indicators, including cortisol, body mass index, blood pressure, that would help comprehensively capture of physiological impact of SES and discrimination. Additionally, this study only focused on Black/white disparities. However, other groups are overrepresented at the lower end of SES and disproportionately exposed to discrimination as well, including Hispanic/Latino populations. Future research should examine the explanatory effects of SES and discrimination across other racial/ethnic groups. In addition, future studies should consider other aspects of socioeconomic position. Black Americans have higher rates of underemployment and lower wealth compared to White participants. These SES indicators were not included in this study, but can help further explain burdens of chronic disease. Finally, self-reported discrimination is only a minute part of a much larger, more complex system of racism. Other forms of discrimination, mostly unseen by people–mortgage lending discrimination, employment discrimination, racial segregation, historical redlining, and Jim Crow laws–would provide a clearer understanding of discrimination's contribution to Black-White inflammation disparities.

5. Conclusion

Our study examined the role of three SES indicators (i.e., household income, education, and employment status) and three indicators discrimination exposure (i.e., lifetime discrimination, everyday discrimination, and chronic workplace discrimination) in explaining racial disparities in inflammation. Using a decomposition method, we found that having a college degree or more explained approximately 16.88% of inflammation burden differences between Black and White participants, and lifetime discrimination explained an additional 18.18% of differences. There was no evidence that other indicators of SES (household income and employment status) and discrimination (i.e., everyday and chronic job discrimination) explained racial differences in inflammation burden. Further research is needed to examine other dimensions of SES (e.g., wealth) and discrimination (e.g., racial segregation) that are associated with health to better understand the contributions of these key social determinants of health and health disparities.

6. Ethics declaration

Funding

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Ethical approval

Tufts University Institutional Review Board (IRB) approved the study protocol. This study was performed in line with the principles of the Declaration of Helsinki.

Informed Consent

Informed consent was obtained from all participants included in the study.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbi.2022.03.005.

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