Discrimination and Cardiovascular Health in Black Americans: Exploring Inflammation as a Mechanism and Perceived Control as a Protective Factor

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ABSTRACT

Objective: Inflammation may be an integral physiological mechanism through which discrimination impacts cardiovascular health and contributes to racial health disparities. Limited research has examined psychosocial factors that protect against the negative effects of discrimination on inflammation. Perceived control is a promising possible protective factor, given that it has been shown to moderate the relationship between other psychosocial stressors and physiological outcomes. This study thus tested whether systemic inflammation mediated the link between discrimination and cardiovascular health and whether perceived control moderated this relationship.

Methods: Data for this project included 347 non-Hispanic/Latinx Black adults (mean [standard deviation] age = 51.64 [11.24] years; 33% female) taken from the Midlife in the United States study. Perceived control and daily discrimination were assessed via self-report, and inflammation was measured via circulating levels of C-reactive protein (CRP), interleukin-6 (IL-6), fibrinogen, and tumor necrosis factor α. Cardiovascular health was measured by morbidity of cardiovascular conditions: heart disease, hypertension, and/or stroke.

Results: CRP (indirect effect: $b = 0.004$, 95% confidence interval [CI] = 0.001–0.007) and fibrinogen (indirect effect: $b = 0.002$, 95% CI = 0.0003–0.005) mediated the link between discrimination and cardiovascular conditions. Perceived control moderated the relationship between discrimination and CRP ($F(1, 293) = 4.58$, $\Delta R^2 = 0.013$, $b = -0.02$, SE = 0.01, $p = .033$). CRP mediated the link between discrimination and cardiovascular conditions only for those who reported low levels of perceived control (Index = -0.003, 95% CI = -0.007 to -0.0001).

Conclusion: Findings provide empirical evidence of inflammation as a mechanism linking discrimination to cardiovascular conditions among Black Americans. Additionally, perceived control may be protective. Findings could suggest beliefs about control as a potential intervention target to help reduce the negative effects of discrimination on cardiovascular health among Black Americans.

Key words: discrimination, psychoneuroimmunology, inflammation, control, resilience factors, health disparities.

INTRODUCTION

Black adults in the U.S. experience a greater burden of several cardiovascular conditions including hypertension and stroke, and they are more likely to die of cardiovascular disease relative to White adults (1,2). Interpersonal discrimination has long been considered an individual-level psychosocial stressor contributing to these disparate cardiovascular outcomes for Black Americans (3–5). Indeed, anti-Black racism (present and historical) embedded within our society has created multiple systemic, cultural, and individual-level disadvantages for Black individuals in the U.S., including increased exposure to discrimination. Repeated exposure to discrimination can impact cardiovascular health through multiple mechanisms (6–9), including via chronic activation of regulatory physiological systems (5,10). Such activation can ultimately lead to "wear and tear" on these systems and contribute to risk for and the development of cardiovascular disease (11–13). Critically, many scholars have proposed that the immune system may be a particularly important biological system linking experiences of discrimination to poor cardiovascular-related outcomes

> **CRP** = C-reactive protein, **IL-6** = interleukin-6, **MIDUS** = Midlife in the United States Study, **TNF-α** = tumor necrosis factor α

(10,14–16). This is because the immune system has evolved to not only be responsive to physical threats to the body (e.g., infections, tissue damage, etc.), but also to real or perceived psychological threat (17–20). As such, experiences of psychosocial stress, including discrimination, may result in the activation of the innate immune system, causing elevated levels of proinflammatory cytokines and contributing to systemic inflammation (18,19,21,22). Positive associations between experiences of discrimination and systemic inflammation are well documented in various empirical studies. Such studies have primarily found that greater endorsement of discriminatory experiences is linked with higher levels of circulating inflammatory markers like C-reactive protein (CRP) and interleukin-6 (IL-6) (15,23–28). Moreover, in a recent systematic review summarizing across 28 studies, Cuevas et al. (14) demonstrated that experiencing discrimination is a significant risk factor for elevated systemic inflammation.

This link between discrimination and inflammation is important, given that inflammation is implicated in the pathophysiology of many cardiovascular diseases and conditions (29,30). Consequently, it is widely theorized that inflammation may be a mechanism through which discrimination can contribute to worse cardiovascular health among Black Americans (3,4,10,13). Yet, most research exploring this theory has only looked at associations between discrimination and indices of cardiovascular health or inflammatory markers separately (4,14,15). Few studies have empirically tested inflammation as a mediating pathway linking discrimination to cardiovascular conditions. However, some studies have provided evidence linking discrimination and other major health outcomes via inflammatory pathways. For example, a previous study found that the link between exposure to discrimination and prospective number of chronic diseases was mediated by systemic inflammation in a sample of middle-aged Black women in the U.S. (11). Additionally, Zahodne et al. (31) found that CRP partially mediated the relationship between discrimination and indices of neurodegenerative disorders (e.g., dementia) in older adults, regardless of their race. Findings such as these support the idea that inflammation is a pathway through which discrimination may also contribute to poorer cardiovascular health for Black individuals; however, neither of these prior studies specifically focused on cardiovascular outcomes. Also, to our knowledge, these are the only studies that have explicitly tested whether higher inflammation mediates the link between discrimination and negative health outcomes. Further, only Simons et al. (11) explored these relationships among Black Americans exclusively—although, all participants were female. As such, more work is needed to examine whether inflammation links discrimination to worse cardiovascular health for Black individuals.

It is also important to identify possible resilience factors that may buffer against the consequences of discrimination on cardiovascular health. Indeed, not everyone who encounters discrimination will experience the same impact on their inflammatory processes and subsequent cardiovascular health. Intrapersonal resources in particular may play a role in mitigating the impact of discrimination on inflammation (6,14,32). Despite considerable research on the psychological characteristics that moderate the influence of discrimination on cardiovascular health and functioning (33–38), relatively little prior work has examined possible psychological moderators of the discrimination-inflammation link specifically. The few studies that have examined such moderators have primarily focused on racial identity. Along these lines, one study demonstrated that among Black adolescents, racial identity moderated the relationship between discrimination and systemic inflammation, such that discrimination was not related to inflammation among those who reported having a more positive attitude toward their race (i.e., high racial centrality) (39). Another study found that high racial centrality and religiosity *interactively* moderated the relationship between racial discrimination and levels of CRP among healthy Black adults. Specifically, Black individuals who experienced greater racial discrimination, but endorsed high racial centrality *and* high religiosity had lower levels of CRP relative to those who had high centrality but low religiosity (40). Although these studies offer preliminary evidence that individual differences in intrapersonal resources can influence the link between discrimination and inflammation, psychological resilience factors that may buffer against this relationship are still vastly understudied. Thus, identifying additional resilience factors

is a crucial need, especially if such factors could be leveraged in interventions aimed at coping with discrimination and reducing its harmful effects.

One promising psychological resilience factor is perceived control, which refers to an individual's perception that they can influence what happens in their life. This resilience factor has been consistently found to be health-protective, and intervention studies aimed at fostering perceptions of control have resulted in improved health and well-being (41,42). Importantly, perceived control is often conceptualized as having two domains: personal mastery and perceived constraints. Personal mastery reflects one's beliefs about self-efficacy and their ability to attain desired outcomes through their own actions. Perceived constraints reflect the extent to which one believes that external factors negatively influence one's ability to control life circumstances. Many studies have combined these domains together into one composite measure of control (43–46); however, some research has examined the effects of each independently. These studies have found that, although mastery and constraints are overlapping constructs, they may also capture distinct information and have differing effects (47–50). For example, perceiving greater constraints in situations where many things are indeed outside of one's control may actually be adaptive (insofar as it could prevent an individual from attempting to control the uncontrollable), whereas greater perceptions of mastery may be specifically protective in situations that are within one's control (47). Consequently, personal mastery and perceived constraints may have different implications in the context of discrimination, as many individuals may perceive experiences of discrimination as being inherently outside of their own control, as they are experiences inflicted by others.

Prior studies have already shown that perceived control (as a composite of mastery and perceived constraints) can influence the negative impact of discrimination on other, non-inflammatory physiological markers (51,52) and broader health-related outcomes (53–56). For example, higher levels of perceived control were associated with less cortisol release following an acute instance of discrimination among Black adults (51). Relatedly, Xu and Chopnik (56) demonstrated that higher perceived control was negatively associated with number of chronic diseases among individuals who experienced high levels of discrimination. Perceived control has also been shown to weaken the effect of chronic stress (though not discrimination specifically) on levels of inflammation (57). In sum, perceived control is an established resilience factor that has been shown to moderate the impact of discrimination on various indices of health. Further, perceived control has been shown to buffer the effects of other psychosocial stressors on inflammation. Thus, it is that possible perceived control is a protective factor in the relationship between discrimination and inflammation, though to our knowledge, no studies have yet tested this possibility. Likewise, the separable effects of personal mastery and perceived constraints have also not been explored in the context of discrimination and inflammation. Considering the unique effects of these subdomains could be particularly useful in informing future interventions targeting aspects of control in order to cope with discrimination. As such, it may be beneficial to explore how each of these aspects of perceived control may impact the relationship between discrimination and inflammation among Black Americans.

In consideration of the literature reviewed above, the current study aimed to extend the empirical literature establishing inflammation as

a mechanism through which discrimination impacts cardiovascular health among Black Americans *and* to identify potential resilience factors in this context. To address these aims, the current study examined the interrelationships between discrimination, perceived control, inflammation, and cardiovascular morbidity (i.e., being diagnosed with heart disease, stroke, and/or hypertension) in a sample of middle-aged Black adults in the U.S. First, we investigated if levels of systemic inflammation mediated the association between discrimination and cardiovascular morbidity. Specifically, we hypothesized that self-reported discrimination would predict being diagnosed with more cardiovascular conditions indirectly via levels of inflammation. CRP and IL-6 were used as our primary markers of inflammation. These markers were selected because they are the most commonly studied indicators of inflammation in studies using the Everyday Discrimination measure (14). However, as an exploratory aim, we also investigated two additional inflammatory markers, tumor necrosis factor α (TNF-α) and fibrinogen. These markers have been less consistently examined in the literature but are included here in order to explore the sensitivity of the discrimination-inflammation link in our analyses.

Next, we tested whether perceived control was protective in the relationship between discrimination and inflammation—a previously unexplored resilience factor in this relationship. We hypothesized that higher levels of perceived control would weaken the relationship between discrimination and inflammation. As another exploratory aim, we assessed each subdomain of control (i.e., personal mastery and perceived constraints) as a potential moderator of the discrimination-inflammation link. Due to the exploratory nature of this aim, no specific hypotheses were made.

Finally, we examined whether the proposed mediation model linking discrimination to cardiovascular conditions through inflammation was moderated by perceived control (and/or its subdomains,
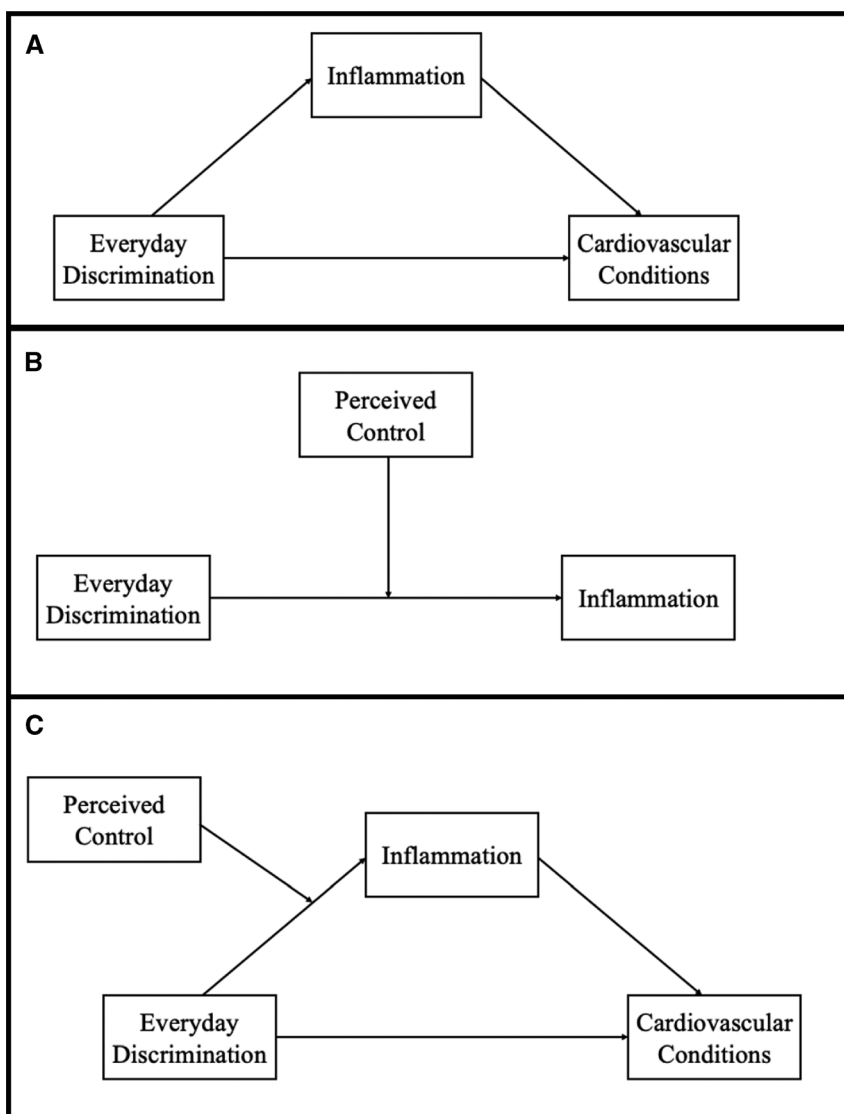

FIGURE 1. Models depicting hypothesized relationships between discrimination, inflammation, perceived control, and cardiovascular outcomes. Note. Panel A depicts the proposed mediation pathway via inflammation. Panel B depicts the moderating relationship, and panel C models the proposed moderated mediation linking discrimination to cardiovascular conditions through inflammation, but dependent upon perceived control.

i.e., moderated mediation). Ultimately, we predicted that the mediating effect of inflammation on the relationship between discrimination and cardiovascular morbidity would be mitigated by greater perceptions of control (see Figure 1 for a depiction of these proposed hypotheses). In investigating these hypotheses, we aimed to further clarify the biological mechanisms through which discrimination may impact cardiovascular health for Black Americans, and to identify a possible resilience factor that may buffer Black individuals from the insidious health impacts of discrimination.

METHODS

Analytic Sample and Procedures

Data for this project were taken from the Midlife in the United States (MIDUS) study, which is a national longitudinal study of English-speaking adults over the age of 24 in the U.S. This large-scale study comprises multiple project phases (i.e., MIDUS 1, MIDUS 2, MIDUS 3, MIDUS Refresher, etc.) and subprojects (i.e., MIDUS 2 Project 1, MIDUS 2 Project 4). The current study utilized data from MIDUS 2 Projects 1 and 4 and MIDUS Refresher 1 Projects 1 and 4. The MIDUS Refresher 1 Project was conducted to recruit a new sample of participants to supplement the original core sample of MIDUS participants. Additionally, both MIDUS 2 and the Refresher I samples were augmented by the MIDUS Milwaukee projects (i.e., Milwaukee MIDUS 2 and Milwaukee Refresher 1), which specifically recruited Black Americans in Milwaukee, WI, in order to boost the racial diversity of the original MIDUS and Refresher samples.

Self-Report Measures Procedure

As a part of the MIDUS 2, Refresher 1, and Milwaukee projects, participants completed self-report measures assessing daily life experiences and psychosocial characteristics, including measures of discrimination and perceived control.

Biological Assessment Procedure

A subsample of these participants also completed a comprehensive biological assessment as a part of MIDUS 2 Project 4 and Refresher 1 Project 4 (i.e., Biomarker Projects), in which participants were invited to an overnight stay at one of three clinical research centers at either UCLA, University of Wisconsin, Madison, or Georgetown University. During this overnight stay, participants provided comprehensive health history information, including chronic disease/conditions diagnoses. Additionally, on day 2 of the visit, fasting blood samples were collected using standardized protocols and resulting blood plasma was assayed for inflammatory markers.

MIDUS projects were approved by the institutional review boards at the participating sites, and informed consent was obtained from all participants. All data included in this study are publicly available through the MIDUS website (https://midus.colectica.org/). A complete list of all variables with codenames included in this specific study is provided in Table S1, Supplemental Digital Content, http://links.lww.com/PSYMED/B12.

Participants

Eligibility criteria for inclusion in the current study required participants to identify their primary race as Black/African American (non-Hispanic/Latinx), have data for all psychosocial variables, and have data for at least one of the inflammatory outcomes of interest. Among all participants who completed the MIDUS 2, Refresher 1, and Milwaukee Projects, 1475 participants identified as Black/African American. Of these participants, 538 were missing data for the psychosocial variables of interest. Among this sample, 347 had relevant data from the Biomarker Project study visit. All 347 participants had IL-6 and TNF-α data. Five participants were missing CRP and fibrinogen data. An additional 33 participants were excluded due to having levels of CRP greater than 10 µg/ml, as CRP levels above this value are likely indicative of acute infection (58,59). As such, the current study included a final sample of 347 participants for analyses involving IL-6 and TNF-α, 342 participants for fibrinogen analyses, and 309 participants for analyses including CRP (see Figure S1, Supplemental Digital Content, http://links.lww.com/PSYMED/B12, for flowchart of inclusion/exclusion process). Thirty-three percent of the sample identified as female, and the average age of participants was 51.64 (SD = 11.19) years.[1] Descriptive statistics for sample characteristics are summarized in Table 1.

Measures

Perceived Discrimination

Perceived discrimination was measured using the Everyday Discrimination Scale (60), which is a nine-item self-report scale asking participants how frequently they experience unfair treatment on a daily basis (e.g., "receiving poorer service than other people at restaurants or stores,") from 1 (often) to 4 (never). Total scores were calculated by summing all items; thus, possible scores ranged from 9 to 36. Responses were reverse-coded such that higher scores reflect greater frequency of everyday discrimination (α = .93). This measure also included a follow-up question assessing discrimination attributions, in which participants were asked "What do you think is the main reason for these experiences?" Participants could then select from a checklist of various identity characteristics (e.g., race, gender, age, etc.). A summary table of participants' discrimination attributions are reported in Table S2, http://links.lww.com/PSYMED/B12.

Perceived Control

Perceived control was assessed using a 12-item self-report questionnaire that assesses the extent to which respondents agree with statements from two subscales: personal mastery and perceived constraints (48). Personal mastery refers to one's sense of efficacy and effectiveness in achieving one's goals and was assessed with four items from the measure (e.g., "when I really want to do something, I usually find a way to succeed at it."). Perceived constraints refer to individuals' beliefs about the extent to which obstacles or factors that are beyond their control interfere with reaching their goals and was assessed with eight items (e.g., "there are many things that interfere with what I want to do."). For each item,

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[1]We conducted *t* tests and chi-square tests to examine whether there were any significant demographic differences between our analytic sample (*n* = 347) and the rest of the Black participants in MIDUS who were not included in our analyses because of missing data (*n* = 1128). Our analyses revealed that there were no significant differences in age, income, and levels of discrimination. However, there were some differences in sex and education. Specifically, our analytic sample included a greater proportion of females and individuals with advanced degrees relative to the larger sample of Black participants. Our sample also had a relatively lower proportion of individuals with only a high school degree. See Supplemental Digital Content, Tables S5–S7, http://links.lww.com/PSYMED/B12 for these results.

TABLE 1. Sample Demographic, Health, and Psychosocial Characteristics

Variable	Count (N)	Percentage (%)
Sex (female)	109	31.40
Education level		
Less than HS	53	15.30
HS degree	181	52.20
Associate's degree/some college	28	8.10
Bachelor's degree	46	13.30
Advanced degree	39	11.20
Current smoker	88	25.40
Cardiovascular conditions		
Heart disease	43	12.40
Hypertension	181	52.20
Stroke	18	5.20
Current medications		
Antihypertensive	110	31.70
Cholesterol lowering	72	20.70
Hormone modifiers (steroids)	36	10.40
Antidepressants	35	10.10

	Mean (SD)	Range
Age	51.64 (11.24)	26.00 to 82.00
Waist-hip ratio	0.89 (0.09)	0.57 to 1.40
Number of health conditions	0.70 (0.73)	0.00 to 3.00
IL-6 (pg/ml)	3.90 (3.40)	0.46 to 22.42
IL-6 (natural log)	1.08 (0.74)	−0.78 to 3.10
CRP (μg/ml)[a]	2.90 (2.45)	0.01 to 10.00
CRP (natural log)[a]	0.60 (1.10)	−4.00 to 2.00
Fibrinogen[b]	384.63 (3.40)	167.00 to 857.00
Fibrinogen (natural log)[b]	5.92 (0.24)	5.12 to 6.75
TNF-α	2.14 (1.17)	0.69 to 16.53
TNF-α (natural log)	0.68 (0.36)	−0.37 to 2.81
Everyday discrimination	14.85 (6.27)	9.00 to 33.75
Perceived control	5.43 (1.06)	1.92 to 7.00
Personal mastery	5.73 (1.16)	1.00 to 7.00
Perceived constraints	2.67 (1.29)	1.00 to 6.63

HS = high school; SD = standard deviation; IL-6 = interleukin-6; CRP = C-reactive protein; TNF-α = tumor necrosis factor α.

Due to missing/excluded data for CRP and fibrinogen, [a] n = 309 and [b] n = 342.

participants rated how strongly they agree with each statement on a scale of 1 (strongly agree) to 7 (strongly disagree). A total measure of perceived control was computed by taking the average score across all 12 items of the measure; thus, scores ranged from 1 to 7. Items were reverse coded such that higher scores reflect higher perceived control ($\alpha = .82$). Scores for the personal mastery and perceived control subscales also ranged from 1 to 7. For the perceived constraints subscale scores, items were not reverse coded; thus, higher scores reflect higher perceptions of constraints (personal mastery: $\alpha = .65$; perceived constraints: $\alpha = .84$).

Cardiovascular Conditions

During the biological assessment visit of the MIDUS Biomarker Project, participants provided their health history, including which (if any) chronic conditions they had, including those that had been diagnosed by a physician. We used a common method for indexing multimorbidity of cardiovascular conditions (11) by summing participants' reports of whether a physician had ever diagnosed them with any of the following (no = 0, yes = 1): cardiovascular disease, hypertension, and stroke/temporary ischemic attack. As such, cardiovascular health scores ranged from 0 to 3. These particular conditions were selected because they are among the leading contributors to cardiovascular-related deaths in the U.S. (1), and Black Americans disproportionately suffer from these conditions relative to White counterparts (61). Additionally, research suggests that inflammation is a contributor to their pathophysiology (62,63).

Inflammatory Markers

On day 2 of their stay at the biomedical clinic site, participants provided fasting blood samples to be assayed for various biomarkers. Blood samples were stored in a −60°C to −80°C freezer until shipped to the lab where the assays were performed. Assays for CRP were conducted at the Laboratory for Clinical Biochemistry Research (University of Vermont, Burlington, VT) using BNII nephelometer with particle-enhanced immunonephelometric assay. The assay range for CRP was 0.175–1100 μg/ml with an interassay coefficient of variation (CV) of 2.1%–5.7% and an intra-assay CV of 2.3%–4.4%. Samples that fell below the lower limit of this assay range were reassayed by immunoelectrochemiluminescence using a high-sensitivity assay kit (Meso Scale Diagnostics #K151STG). Fibrinogen assays were also conducted at the Laboratory for Clinical Biochemistry Research (University of Vermont, Burlington, VT) using the BNII nephelometer. The assay range for fibrinogen was 60–1200 mg/dl, with an interassay CV of 2.6% and intra-assay CV of 2.7%. Assays for IL-6 and TNF-α were conducted at the MIDUS Biocore Laboratory (University of Wisconsin, Madison, WI) using Quantikine High Sensitivity ELISAs (R&D Systems, Minneapolis, MN). The IL-6 assay range was 0.156–10 pg/ml with an interassay CV of 12.31% and an intra-assay CV of 3.25%. The TNF-α assay range was 0.69–248 pg/ml with an interassay CV of 7.00% and an intra-assay CV of 3.19%. To adjust for positive skew in the data, raw inflammatory marker values were natural log transformed.

Covariates

Due to the complex interplay between various sociodemographic factors, inflammation, and health (64), the following covariates were included in analyses: age (in years), sex, waist-to-hip ratio, education, current smoking status, use of antihypertensive, cholesterol-lowering, steroid, or antidepressant medications. Sex was defined as participants' self-identification as either male or female. Education level was defined categorically based on participants' report of highest level of education completed (i.e., less than high school, high school degree, some college/associate's degree, bachelor's degree, or advanced degree). Selection of these specific covariates was based on recommendations of controls to use when examining inflammatory markers (28,64,65). Further, in models with inflammatory markers as outcomes (i.e., moderation analyses), analyses were run with and without controlling for the presence of the cardiovascular conditions examined in this study. Because no differences emerged as a function of including/excluding these conditions as covariates, results are reported without them in the main text (see the Supplemental Digital Content for results with

cardiovascular conditions included as covariates, http://links.lww.com/PSYMED/B12).

Data Analysis

Multiple regression analyses were first employed to assess whether discrimination and levels of inflammation were significant predictors (while controlling for covariates) of cardiovascular conditions within the current sample. Next, regression analysis using PROCESS macro model 4 (Hayes, 2013) was used to test whether levels of inflammation mediated the relationship between discrimination and cardiovascular conditions. Bootstrapping (10,000 repetitions) was used to derive 95% confidence intervals (CIs) for all indirect effects. Significant indirect effects were evidenced by the absence of zero within CIs.

Regression analysis via PROCESS macro model 1 (Hayes, 2013) was then used to test whether perceived control moderated the relationship between discrimination and inflammation. Simple slopes analyses, controlling for covariates, were then conducted to interpret significant interactions, which were defined as $p < .05$. For the simple slopes analyses, high and low values of the control measure and subscales were defined as one standard deviation above and below the mean, respectively. Additionally, although not a primary aim of this study, in an effort to replicate findings from previous studies exploring perceived control as a moderator of chronic health conditions, we also ran a regression analysis with perceived control as a moderator of discrimination and cardiovascular health conditions. The results are reported in the Supplemental Digital Content, http://links.lww.com/PSYMED/B12.

Finally, a moderated mediation was examined in a single model using PROCESS macro model 7 (Hayes, 2013). This approach tested the conditional indirect effect of perceived control on the relationship between discrimination and cardiovascular conditions via the mediating role of inflammation. As such, number of cardiovascular conditions was the outcome with discrimination entered as the predictor, inflammation as the mediator, and perceived control as the moderator. This model specifically tests the moderating effect on the predictor—mediator path (i.e., path a). An index of moderated mediation was used to ascertain whether the overall model was significant (i.e., CI with absence of zero). Conditional indirect effects with CIs without zero were indicative of significant interactions.

For all mediation, moderation, and moderated mediation analyses, discrimination, perceived control, the interaction terms, and covariates were entered into the model simultaneously. The variables comprising interaction terms were mean centered to avoid multicollinearity between the predictor variables and the interaction terms. Regressions were run with and without outliers, which were defined as any values more than three standard deviations above or below the mean. There were no differences in significance tests as a function of excluding outliers for all primary analyses, though some differences emerged in our exploratory analyses when outliers were included. Results reported within main text are with outliers excluded. See the Supplemental Digital Content for a report of results with outliers included, http://links.lww.com/PSYMED/B12.

RESULTS

The regression model predicting cardiovascular conditions by discrimination, CRP, and covariates was significant, $F(11, 295) = 16.58$,

$p < .001$. CRP was a significant predictor of cardiovascular conditions ($\beta = 0.176$, $p < .001$), whereas discrimination was not ($\beta = -0.019$, $p = .69$). The regression model predicting cardiovascular conditions by discrimination, IL-6, and covariates was also significant, $F(11, 324) = 17.94$, $p < .001$. Again, IL-6, but not discrimination, significantly predicted greater number of conditions (IL-6: $\beta = 0.169$, $p < .001$; discrimination: $\beta = -0.028$, $p = .53$). The model including fibrinogen revealed a similar pattern of results, with the inflammatory marker but not discrimination predicting cardiovascular conditions, $F(11, 327) = 17.97$, $p < .001$ (fibrinogen: $\beta = 0.128$, $p = .007$; discrimination: $\beta = -0.021$, $p = .63$). In the model with TNF-α, neither TNF-α nor discrimination predicted cardiovascular outcomes, $F(11, 322) = 17.57$, $p < .001$ (TNF-α: $\beta = 0.056$, $p = .22$; discrimination: $\beta = -0.011$, $p = .93$).

Despite the nonsignificant relationship between discrimination and cardiovascular conditions, we preceded with testing mediation models in light of both statistical and theoretical propositions that indirect effects of a predictor on an outcome can be possible and meaningful even without a significant direct relationship between the predictor and outcome (66,67). As such, we conducted separate mediation models for each inflammatory marker that was a significant predictor of cardiovascular outcomes (i.e., CRP, IL-6, and fibrinogen). The mediation model testing whether CRP mediated the relationship between discrimination and cardiovascular conditions showed that, although there was not a significant direct effect between discrimination and cardiovascular conditions ($b = -0.002$, 95% CI = −0.013 to 0.009), there was a significant indirect effect via CRP ($b = 0.004$, 95% CI = 0.001–0.007; see Figure 2A). These results were mirrored in the mediation model exploring fibrinogen as a mediator (indirect effect via fibrinogen: $b = 0.002$, 95% CI = 0.0003–0.005). The mediation model testing whether IL-6 mediated the relationship between discrimination and cardiovascular conditions did not reveal significant indirect effect of IL-6 ($b = 0.002$, 95% CI = −0.0003 to 0.004).

Next, regression analyses examined if perceived control moderated the relationship between discrimination and each inflammatory marker. These analyses revealed a significant interaction between perceived control and discrimination in predicting levels of CRP, $F(1, 293) = 4.58$, $\Delta R^2 = 0.013$, $b = -0.02$, SE = 0.01, $p = .033$. Specifically, results of simple slope analysis indicated that among individuals with higher levels of perceived control (+1 SD above mean), discrimination was not associated with levels of CRP ($p = .33$), yet for individuals with average (mean) and low (−1 SD below mean) levels of perceived control, discrimination significantly and positively predicted CRP levels (average control: $b = 0.03$, SE = 0.01, $p < .001$; low control: $b = 0.05$, SE = 0.01, $p < .001$; see Figure 2B). Perceived control did not moderate the relationship between discrimination and other the other inflammatory markers: IL-6 ($F(1, 321) = 0.140$, $\Delta R^2 = 0.0004$, $b = 0.002$, SE = 0.006, $p = .71$), fibrinogen ($F(1, 324) = 3.43$, $\Delta R^2 = 0.009$, $b = -0.004$, SE = 0.002, $p = .065$), and TNF-α ($F(1, 319) = 0.246$, $\Delta R^2 = 0.0007$, $b = 0.001$, SE = 0.003, $p = .62$). Due to these nonsignificant results, only CRP was explored in subsequent analyses.

Given the significant moderation of composite perceived control scores on the relationship between discrimination and CRP, exploratory regressions were run after decomposing the perceived control measure into its two subscales, to assess whether there were separable moderating effects of personal mastery and/or perceived constraints. For the personal mastery subscale, regression
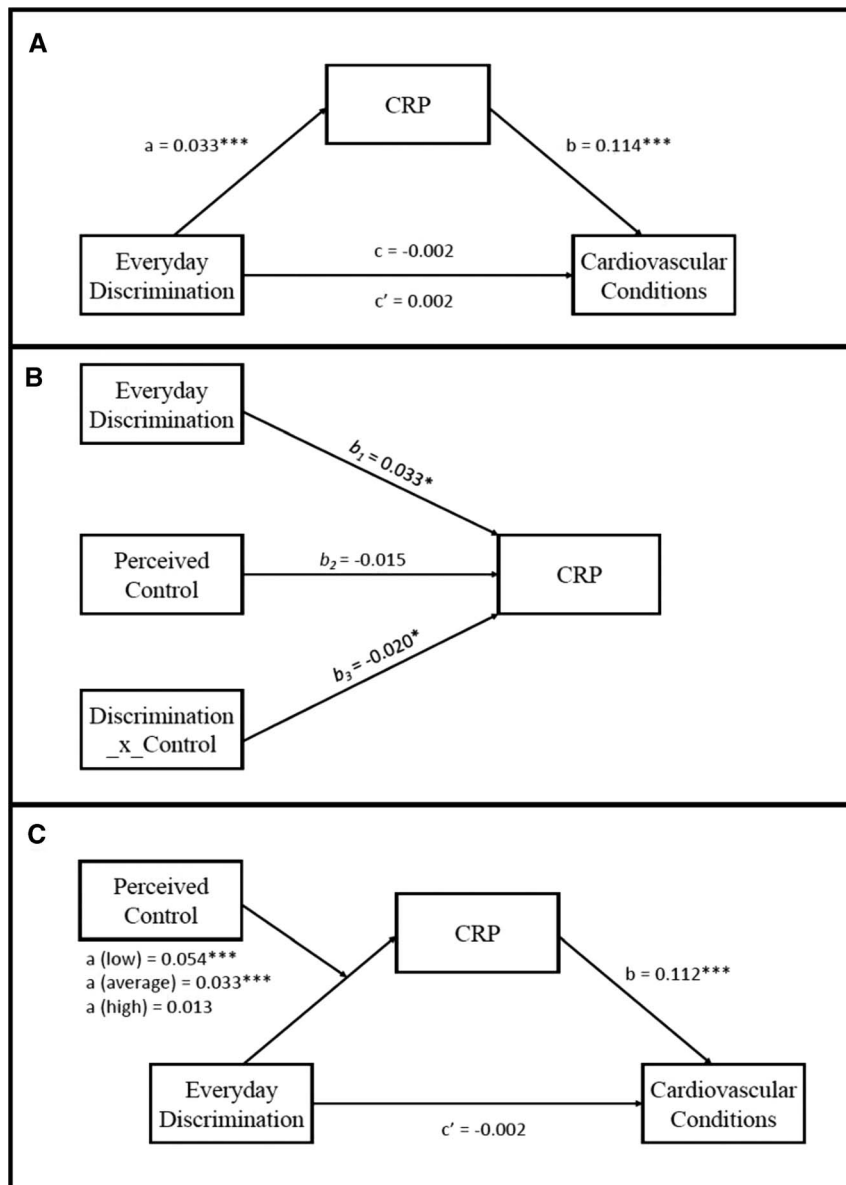
FIGURE 2. Models depicting mediation, moderation, and moderated mediation results for discrimination, perceived control (composite), CRP, and cardiovascular conditions. Note. Panel A models the indirect effect of discrimination on cardiovascular conditions through CRP. Panel B models the moderation effect of perceived control in the relationship between discrimination and CRP. Panel C models the conditional indirect effect of discrimination on cardiovascular conditions through CRP based on level of perceived control (i.e., low, average, and high). *** $p < .001$, ** $p < .01$, * $p < .05$. CRP = C-reactive protein.

analyses revealed a similar pattern to what was found with the composite perceived control measure: There was a significant interaction between personal mastery and discrimination in predicting CRP levels ($F(1, 292) = 5.30$, $\Delta R^2 = 0.015$, $b = -0.02$, SE = 0.01, $p = .022$). Among individuals with high levels of personal mastery, discrimination was not associated with levels of CRP ($p = .30$), yet for individuals with average to low levels of personal mastery, discrimination significantly and positively predicted CRP (average mastery: $b = 0.04$, SE = 0.01, $p = .002$; low mastery: $b = 0.06$, SE = 0.01, $p = .0001$). The interaction effect of discrimination and perceived constraints in predicting CRP levels was not significant ($F(1, 294) = 3.11$, $\Delta R^2 = 0.010$, $b = 0.01$, SE = 0.01, $p = .08$).

Finally, we conducted moderated mediation analyses to assess whether perceived control moderated the indirect path linking discrimination to cardiovascular conditions via CRP. The overall moderated mediation model was significant (Index = -0.003, 95% CI = -0.007 to -0.0001), indicating that perceived control moderated the indirect effect of discrimination on cardiovascular conditions through CRP. Specifically, the conditional indirect effect of discrimination on cardiovascular conditions via CRP was significant at average ($b = 0.005$, SE = 0.002, 95% CI = 0.001–0.009) and low levels of perceived control ($b = 0.01$, SE = 0.003, 95% CI = 0.002–0.015), but not at high levels (95% CI = -0.002 to 0.006; see Figure 2C). See Tables

S3a–3b for full moderated mediation results, http://links.lww.com/PSYMED/B12.

Additional moderated mediation models were run with the subscales of personal mastery and perceived constraints. As with composite perceived control, the model with personal mastery was significant (Index = −0.003, 95% CI = −0.007 to −0.001). The conditional indirect effect of discrimination on cardiovascular conditions via CRP was significant only at average (b = 0.005, SE = 0.002, 95% CI = 0.001–0.010) and low levels of personal mastery (b = 0.01, SE = 0.003, 95% CI = 0.002–0.016), but not at high levels (95% CI = 0.002–0.007). The model including perceived constraints was nonsignificant (Index = 0.002, 95% CI = −0.0002 to 0.005), suggesting that perceived constraints did not moderate the impact of discrimination on cardiovascular conditions via CRP. Taken together, these findings suggest that the mediating effect of CRP on the link between discrimination and cardiovascular conditions occurred only among individuals who reported average to low levels of perceived control, and this may be specifically driven by their low endorsement of personal mastery (versus high perceived constraints).

DISCUSSION

The current study examined inflammation as a mechanism linking discrimination and cardiovascular health and identified perceived control as a potential psychological buffer of this relationship in a sample of Black midlife adults. Although we did not find a direct link between discrimination and cardiovascular conditions in this sample, we did find that CRP and fibrinogen mediated the link between discrimination and cardiovascular conditions, such that higher levels of discrimination predicted higher levels of these biomarkers, which in turn predicted a greater number of cardiovascular conditions. Further, perceived control moderated the relationship between discrimination and CRP such that individuals with higher levels of perceived control did not show an association between discrimination and CRP, whereas those with low and average levels of control showed a positive association between discrimination and CRP. Additionally, a moderated-mediation analysis revealed that CRP mediated the link between discrimination and cardiovascular conditions only for those who reported low and average levels of control but did not for those who had high levels of control. This protective effect of perceived control may be particularly driven by greater perceptions of personal mastery (versus low perceived constraints), as personal mastery but not perceived constraints was a significant moderator of the mediation effect. Thus, these findings ultimately indicate that inflammation may be a mechanism linking discrimination to cardiovascular health, and that higher perceptions of control are health-protective for Black middle-aged adults who have experienced discrimination—in part because control "breaks the link" between discrimination, inflammation, and worse cardiovascular health.

The present study adds to emerging empirical literature demonstrating inflammation, specifically CRP and fibrinogen, as a mechanism through which experiences of discrimination may translate to poorer cardiovascular health among Black Americans. As theorized by many scholars of Black-White health disparities in the U.S., persistent exposure to social disadvantage (e.g., discrimination, racism, etc.) may cause chronic stress that leads to heightened or dysregulated activity of biological systems, which ultimately contributes to the development of chronic health conditions (10–12,24,68). The mediating role of CRP and fibrinogen observed in the present study aligns with this theory, as it demonstrates the innate immune system as a potential biological pathway connecting experiences of discrimination and to greater burden of cardiovascular conditions. The nonsignificant direct effect of discrimination on cardiovascular conditions also adds to the mix of existing work aimed at elucidating the relationship between discrimination and cardiovascular health. Although some previous studies have found significant associations between discrimination and worse cardiovascular outcomes, there are also many that have not (4,69), suggesting some inconsistency in the current literature. This may be because the impact of discrimination on cardiovascular indices is intricate and dependent on other factors such as coping strategies (70,71), health behaviors (72), internalized beliefs (37,38), or in the case of this study, inflammatory biomarkers (i.e., CRP, fibrinogen).

Further, although we did find that CRP and fibrinogen mediated the association between discrimination and cardiovascular conditions, IL-6 was not a significant mediator of this relationship (and TNF-α was not even a predictor of cardiovascular conditions). There are a few possible explanations for these differing results across inflammatory markers. First, existing literature has more consistently linked CRP and fibrinogen with cardiovascular disease risk (73–76); thus, our null results with IL-6 and TNF-α may be because these markers are not as strongly implicated in the pathophysiology of the cardiovascular conditions assessed in this study. Secondly, levels of IL-6 and TNF-α are more consistently elevated following acute stress relative to CRP and fibrinogen, and they have a more similar peak time-course in response to such stress (17,77). Additionally, increased IL-6 and TNF-α trigger the downstream production of acute-phase proteins like CRP and fibrinogen (75,78). Given that the present study assessed past experiences of discrimination (versus an acute instance), it is possible that CRP and fibrinogen may be more reliable indicators of the chronic impact of discrimination on inflammation, whereas IL-6 and TNF-α may be better indices of acute inflammatory responses to discrimination. Accordingly, our finding that CRP and fibrinogen (but not IL-6 and TNF-α) acted as a mechanism linking discrimination to cardiovascular conditions may simply reflect that this pathway is particularly driven by chronic inflammatory activity.

Differences in results across inflammatory markers also emerged in our moderation analyses. Specifically, only the relationship between discrimination and CRP (but not IL-6, TNF-α, or fibrinogen) was moderated by perceived control. The lack of significant moderation with IL-6 and TNF-α may be explained by the fact that discrimination was not significantly related to these markers in the present sample (for the same reasons discussed above). However, it is interesting that the moderation results with fibrinogen were null given that it was strongly linked with discrimination. Although the results of this analysis did not quite reach significance (p = .065), the effect size was close to that of CRP (fibrinogen: R^2 change = 0.009, CRP: R^2 change = 0.013); therefore, it is possible that the effect of perceived control on this fibrinogen may be smaller than that of CRP and we were simply underpowered to detect it. Alternatively, this result may indicate that the effects of this psychological resilience factor are specific to CRP. However, support for this hypothesis is limited given that most studies exploring the influence of perceived control on inflammation have primarily

assessed CRP (57,79,80); thus, its association with other inflammatory markers is not well established.

Still, our significant findings with CRP offer evidence that the relationship between discrimination and inflammation (and more distal cardiovascular outcomes) can be influenced by beliefs about control. Prior research on perceived control offers some insights as to why it might be protective for health in the context of experiencing discrimination. For example, it has been shown that greater feelings of control can be protective by reducing reactivity to stressors and facilitating emotion regulation processes (41). Indeed, one's perception of control is influential in the process of making subjective appraisals of threat in response to stressors (57), which may subsequently impact physiological reactivity to such stressors. Along these lines, the present findings might suggest that Black individuals who encounter discrimination but have high perceptions of control may appraise these occurrences as less distressing and may be less physiologically reactive to them. Moreover, it has been proposed that perceived control promotes adaptive coping behaviors such as seeking social support and problem solving (81,82). Thus, our findings may also suggest that Black individuals with greater perceptions of control may be more adept at engaging in healthy coping behaviors, which could also minimize the physiological impact of discrimination stress. Of course, this explanation is currently speculative; future experimental research could test this empirically.

Importantly, we must also consider that when examining the domains of personal mastery and perceived constraints separately, personal mastery reached statistical significance as a moderator, but perceived constraints did not. This suggests that having *stronger* beliefs about one's own self-efficacy rather than having *weaker* beliefs about the influence of external forces may be more influential in protecting against the negative health impacts of discrimination. We interpret this finding as cautiously hopeful. This is because discrimination is typically enacted by other individuals who are in positions of power relative to the target of the discrimination; thus, it may be unrealistic to alter individuals' perceptions of constraints in this specific context. However, beliefs about personal mastery are likely to be more modifiable; thus, interventions that foster such beliefs could critically mitigate the effects of discrimination on cardiovascular health among Black Americans. Indeed, previous research has shown that personal beliefs about control can be intervened upon in other contexts (41). This indicates that developing interventions for increasing perceptions of personal mastery among Black individuals and studying the subsequent effects on inflammation may be a fruitful avenue for future research. Such interventions could be a promising proximal solution to reducing the harmful effects of discrimination on cardiovascular health among Black Americans. However, such individual-level solutions are certainly not enough to ameliorate Black-White health disparities in the U.S. Instead, efforts to build up greater perceptions of control among Black Americans should be viewed as supplementary to broader systemic changes that address the long-standing inequities in our society.

This study, of course, has limitations. The primary limitation is the cross-sectional nature of the data, which limits our ability to make any causal interpretations. Relatedly, although this study conceptualized perceived control as an individual resilience factor that moderates the relationship between discrimination and inflammation, future research may also aim to explore perceived control as a mechanism (i.e., mediator) in the context of discrimination and cardiovascular health. Prior work has found that discrimination was associated worse psychological health through reductions in perceptions of control (55); thus, perhaps perceived control could also play a mechanistic role in linking discrimination and physical health outcomes. As such, exploring how intervening on Black individuals' perceptions of control/personal mastery may subsequently impact inflammatory processes and more distal health outcomes could be an especially worthwhile future endeavor. Moreover, although we tested mediation with our inflammatory markers, this was not a longitudinal design. Future studies should thus measure perceived control, discrimination, inflammation, and cardiovascular outcomes sequentially at different time points to establish a causal chain.

Additionally, due to the nature of the medical history collected from participants, we were only able to include heart disease, hypertension, and stroke as our index of cardiovascular health. Future work should thus explore whether these relationships are consistent across other types of cardiovascular conditions (e.g., heart failure, heart attack, peripheral arterial disease, etc.). Our study also relied on a relatively small sample given the limited availability of biomarker data for many of the Black participants in the MIDUS study. Further, the Black participants that make up this sample are predominantly from Milwaukee, WI, as MIDUS researchers intentionally recruited Black Americans from this city to enhance Black representation in the MIDUS 2 and Refresher samples. This consequently limits the generalizability of the present findings and highlights the need to replicate these findings in a larger, more nationally representative sample.

Finally, our measure of discrimination did not assess experiences of racial discrimination specifically. Instead, it involved follow-up questions that asked participants to indicate the primary reason they believed they were discriminated against (e.g., race, gender, social class, weight, etc.). Although over half of the participants in our sample indicated race as the primary reason for the discrimination they experienced, some attributed it to other identity characteristics (see Table S2, Supplemental Digital Content, http://links.lww.com/PSYMED/B12, for a summary of discrimination attributions). As such, we cannot necessarily say that our findings are specific to experiences of racial discrimination. However, prior research has shown that Black individuals are more likely to experience discrimination, both racial and nonracial (7), so the present findings still offer important insights into how this psychosocial stressor may impact cardiovascular health for this more vulnerable population. Furthermore, findings from extant meta-analyses and reviews have suggested that both race-related and non–race-related experiences of discrimination have similar consequences for health (7,83,84). Given this, it is possible that the role of discrimination in contributing to poorer cardiovascular health for Black Americans could have less to do with the specific attribution for discrimination and more to do with the relative frequency of discriminatory experiences and one's position in the racial hierarchy (i.e., experiences may be more impactful for those in nondominant racial groups). Investigating this presumption may be yet another avenue for future research.

Despite the limitations of this study and need for further research, our findings are the first to show that perceived control buffers the mediating effect of inflammation on the relationship between discrimination and cardiovascular health among a sample

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of Black adults in the U.S. Notably, we explored these relationships specifically within a sample of Black Americans, rather than using a sample of Black and White (or other race) individuals, which is often done in studies exploring the connections between discrimination and inflammation (14). We found it important to focus solely on Black Americans given that they are disproportionately affected by cardiovascular-related morbidity and mortality. This population is also more often exposed to instances of discrimination (7,85–87), and such experiences likely carry different meaning and implications relative to White Americans who experience discrimination. As such, this study contributes to current understanding of the biopsychosocial pathways linking discrimination and health, and identifies yet another characteristic of Black Americans that can foster resilience in the face of pervasive social disadvantage and marginalization.

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