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Incident racial discrimination predicts elevated C-Reactive protein in the Black Women's experiences Living with Lupus (BeWELL) study

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Abstract

Introduction: Racial discrimination is a distinct health threat that increases disease risk among Black Americans. Psychosocial stress may compromise health through inflammatory mechanisms. This study examines incident experiences of racial discrimination and changes in the inflammatory biomarker C-reactive protein (CRP) over a two-year period among Black women with systemic lupus erythematosus (SLE)—an inflammatory autoimmune disease sensitive to psychosocial stress and characterized by stark racial inequities in outcomes.

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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.2023.06.004.

Methods: Data are from the Black Women's Experiences Living with Lupus (BeWELL) Study. Participants (n = 380) from metropolitan Atlanta, Georgia were enrolled from April 2015 to May 2017. Incident racial discrimination was assessed bi-annually via self-report using the Experiences of Discrimination measure. CRP was assessed annually over a two-year period. Latent change score analyses modeled longitudinal within-person associations between incident racial discrimination and change in log-transformed CRP from baseline to Year 2.

Results: Incident experiences of racial discrimination were associated with elevated log-CRP across the two-year study period (b = 0.039, SE = 0.017, 95% CI: 0.006, 0.071). For each domain of incident racial discrimination experienced, CRP increased 3.98%.

Conclusion: This study contributes to growing evidence on the biological consequences of racism and is the first to document an association between incident racial discrimination and changes in inflammation among Black women with SLE. Racial inequities in SLE outcomes and other diseases driven by inflammatory pathways may be explained in part through experiences of racial discrimination.

Keywords

Racial discrimination; Inflammation; C-reactive protein; Systemic lupus erythematosus

1. Introduction

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease characterized by fluctuations in disease activity (e.g., flares) that, if uncontrolled, can lead to organ failure and death (Fava and Petri, 2019). The epidemiology of SLE is patterned along racial and gender lines. Black women have among the highest prevalence of SLE in the United States; nearly three times as high as white women (Grabich et al., 2022; Izmirly et al., 2021). Black women also experience greater disease severity and increased risk of mortality overall and at earlier ages compared to their white counterparts (Drenkard and Lim, 2019; Pons-Estel et al., 2017). Emerging research suggests that racial inequities in disease progression and outcomes are rooted in social inequities rather than genetic or behavioral factors (Hasan et al., 2022; Ramos, 2021). For example, several studies among Black women with SLE have found that disease activity is susceptible to interpersonal experiences of racism-related psychosocial stressors, such as anticipating and experiencing racial discrimination, vicarious racism, and racial microaggressions, as well as systematic differences in residential conditions, such as exposure to neighborhood disorder (e.g., crime, abandoned buildings) associated with racial segregation (Chae et al., 2019; Hunter et al., 2020; Martz et al., 2021, 2019; Spears et al., 2021).

Inflammation is fundamental in the pathophysiology of SLE, much like numerous other chronic conditions such as heart disease, diabetes, and cancer (Aringer, 2020; Furman et al., 2019). Elevated levels of inflammation mediate comorbidities as well as irreversible damage to organs or organ systems and death (Aringer, 2020). Inflammatory processes are engaged in the embodiment of psychosocial stress and represent a biological mechanism through which stress "gets under the skin" (McEwen, 2012). Chronic and severe experiences of psychosocial stress throughout the life course can lead to negative health consequences

through repeated activation of biological systems engaged in the stress response (McEwen, 1998; Pearlin et al., 2005). Subsequent dysregulation of the parasympathetic stress-response precipitates chronic low grade and eventual high levels of systemic inflammation—a catalyst of biological "weathering" and risk factor for accelerated SLE progression (Geronimus et al., 2006; Goosby et al., 2018).

Systemic social and environmental disadvantage proliferates into other general sources of psychosocial stress among Black Americans (Goosby et al., 2018; Williams and Mohammed, 2009). Additionally, Black Americans experience qualitatively unique health threats, such as racial discrimination, which are rooted in white supremacy and racist notions of Black inferiority (Goosby et al., 2018). As a social evaluative threat, racial discrimination is distinct from other more general forms of psychosocial stress (Harrell, 2000). According to Social Identity Theory, racial discrimination is a direct and personal threat based largely on immutable characteristics foundational to one's identity, and has potential to impact self-worth (Kite and Whitle, 2016; Tajfel and Turner, 1979). Black women have also been found to respond to unique forms of gendered racism using high effort coping strategies (e.g., feeling the need to "work harder" to prove themselves and an obligation to overcome adversity and suppress emotions to appear strong), which while psychologically adaptive, may result in harmful physiologic health outcomes (Allen et al., 2019; Nuru-Jeter et al., 2013; 2009).

Experiencing racial discrimination has been shown to elicit a cascade of psychobiological responses, including negative emotions, depleted cognitive resources, and heightened physiologic reactivity, all of which contribute to health risk for Black Americans (Goosby et al., 2018). Inflammation is a common underlying risk factor for poor outcomes across multiple diseases and a mechanism through which racial discrimination becomes embodied (Cuevas et al., 2020). Systemic inflammation is patterned along racial lines with Black Americans having greater levels, on average, than white Americans (Boen, 2020; Stepanikova et al., 2017). Research has shown that discrimination is associated with elevated inflammation, including inflammatory cytokines and circulating levels of C-reactive protein (CRP), an acute-phase protein synthesized by the liver in response to inflammation (Cuevas et al., 2020; Lawrence et al., 2022a). Although not directly involved in disease flares, elevated levels of CRP can worsen tissue damage in SLE by triggering complement activation, thereby exacerbating inflammatory cytokines and accelerating disease pathogenesis (Enocsson et al., 2021; Walport, 2002).

Despite an emerging body of evidence on associations between discrimination and inflammation, studies have been predominantly cross-sectional or prospective and therefore unable to assess directionality of effects or within-person change (Beatty Moody et al., 2014; Boen, 2020; Brody et al., 2015, 2014; Lawrence et al., 2022a, 2022b; Nuru-Jeter et al., 2013; Simons et al., 2021; Zahodne et al., 2019). Moreover, most research has assessed discrimination at a single point in time with few studies capturing incident experiences specifically attributed to a person's race (Van Dyke et al., 2021). No studies, to our knowledge, have examined new experiences of racial discrimination over time in relation to changes in inflammation with the exception of laboratory studies where exposure to racial discrimination was experimentally manipulated and examined in relation to more

acute changes in cytokines (Cuevas et al., 2020; Lucas et al., 2017; Saban et al., 2018). Longitudinal study designs with repeated measurement of both predictor and outcome variables over time are important for causal inference regarding changes in CRP by minimizing the potential for reverse causal interpretation and unmeasured confounding in observational data (Raymaekers et al., 2020). In the current study, we examine whether incident racial discrimination predicts changes in CRP assessed at three occasions over a two-year period among 380 Black women with SLE.

2. Materials and methods

2.1. Sample

Data were from the Black Women's Experiences Living with Lupus (BeWELL) Study (Chae et al., 2019). The BeWELL Study enrolled 438 Black women with validated SLE living in metropolitan Atlanta, GA from April 2015 to May 2017. Eligibility for the BeWELL Study was based on five inclusion requirements: (1) validated SLE diagnosis based on American College of Rheumatology classification criteria (>4 criteria) or 3 criteria with a diagnosis of SLE by a board-certified rheumatologist; (2) self-identification as a Black or African American woman; (3) between 18 and 79 years of age; (4) living in metropolitan Atlanta, GA; (5) and ability to read, write, and understand English (Chae et al., 2019). Recruited participants were not enrolled if any of the five requirements were not met. Participants were primarily recruited from the Georgians Organized Against Lupus (GOAL) cohort, which drew primarily from the CDC-funded population-based Georgia Lupus Registry (Lim et al., 2014). Data collection was completed in February 2019.

Main data collection occurred at baseline, 12-months, and 24-months; two interim short surveys were administered between these study waves, at 6-months and 18-months. Participant involvement for the main study waves included self- and interviewer-administered questionnaires of psychosocial and health-related measures, and collection of dried blood spots and anthropometric measures. Interviewers were trained to collect data through standardized study procedures primarily on-site at the Emory University School of Medicine. Interim surveys involved a short, self-administered web-based questionnaire, including assessments of new experiences of racial discrimination. Attrition was minimal with >95% of participants (excluding those deceased, n = 19) completing the two-year study.

2.2. Measures

2.1.1. Racial discrimination—The Experiences of Discrimination (EOD) measure assesses instances of racial discrimination across nine domains: at school; getting hired or getting a job; at work; getting housing; getting medical care; getting service at a store or restaurant; getting credit, bank loans, or a mortgage; on the street or in public settings; or from the police or in the courts (Krieger et al., 2005). At baseline, participants were asked whether they had "ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior" across the nine domains "because of your race, ethnicity, or color." We utilized the situation scoring method, in which each item was coded as 0 = no or 1 = yes (Krieger et al., 2005). Scores were summed to create a baseline measure of the number of situations where a participant experienced racial discrimination

in their lifetime (range 0–9). We modified the stem of the EOD scale for subsequent assessments at interim surveys and annual study time points to capture incident experiences of racial discrimination. Participants were asked specifically about their experiences of racial discrimination "since the last time we saw you, about 6 months ago". The main exposure variable was new annual experiences of racial discrimination across each of the nine domains. Year 1 (Y1) incident discrimination was calculated as the number of domains in which racial discrimination was reported as being experienced, as indicated at either 6-months or 12-months (range 0–9). Year 2 (Y2) incident discrimination was calculated in a similar manner, as the number of domains in which racial discrimination was reported as being experienced at either 18-months or 24-months (range 0–9).

2.2.2. C-Reactive protein—C-reactive protein (CRP) was assayed from dried blood spots (DBS) collected at baseline, 12-months, and 24-months. DBS collection involved collecting five 50 μL blood droplets on filter paper via finger prick that were stored at −80 °C (McDade et al., 2007) until processing. CRP concentrations (mg/L) were quantified using a high-sensitivity, enzyme-linked, multiplex sandwich immunoassay method (McDade et al., 2004). DBS samples from all study waves were assayed simultaneously on the same plate to minimize potential batch effects. CRP was log-transformed to account for the right-skewed distribution of residuals.

2.2.3. Covariates—Potential confounding was addressed by adjusting for several sociodemographic, socioeconomic, and health-related covariates assessed at baseline, consistent with previous studies on psychosocial determinants of SLE in the BeWELL Study (Bridges et al., 2020; Chae et al., 2019; Hunter et al., 2020; Martz et al., 2019; Spears et al., 2021). Final models controlled for the following: age and disease duration (both in years), relationship status (single and never married, divorced/widowed/separated, in a romantic relationship, or married or in a marriage-like relationship), level of educational attainment (<high school, high school, some college, or college graduate/advanced degree), continuous ratio of household income to the poverty threshold (based on household composition), work status (full-time, part-time, out of labor force, and unable to work), insurance status (public, private, or none), body mass index (BMI) measured continuously based on height and weight measured at baseline, smoking (current vs not current), and self-reported use of each of the following (yes or no): steroids, hydroxychloroquine, and other immunosuppressant medications. We also adjusted for SLE severity with two validated measures: the Systemic Lupus Activity Questionnaire to assess disease activity in the past three months (range: 0–44); and the Brief Index of Lupus Damage to assess irreversible organ damage (range: 0–30) (Karlson et al., 2003; Yazdany et al., 2011).

2.3. Statistical analyses

Descriptive statistics were examined prior to conducting cross-sectional and longitudinal analyses to inspect the distribution of main study variables and identify potential outliers. We excluded participants with missing data on CRP at any of the three annual assessments (n = 54) and those with CRP values (n = 4) indicative of severe acute infection in SLE (>60 mg/L), resulting in a final analytic sample of 380 Black women with SLE (Aringer, 2020). We did not restrict the analytic sample based on levels of disease activity because of mixed

evidence on the role of CRP in SLE activity, with most studies indicating that CRP is more reflective of cardiovascular disease risk than disease activity and flares (Barnes et al., 2005; Enocsson et al., 2021).

Parametric analyses were conducted because of the normal distribution of log-transformed CRP values at each time point. We first examined regression diagnostics for bivariate regression models to assess the normality of residuals and other model assumptions (e.g., homoscedasticity, linearity), as well as potential influential observations and leverage points. Next, we examined cross-sectional associations between CRP and racial discrimination at each time point using multivariable linear regression. We then used change score analyses within a structural equation modeling framework to examine longitudinal associations between new experiences of racial discrimination and annual change in CRP (McArdle, 2009). Latent change score analyses emphasize within-person change by modeling time-dependent variation as the outcome of interest (Grimm et al., 2016; McArdle, 2009). This method to examine developmental processes minimizes issues of measurement error, such as regression to the mean and variance inflation (McArdle, 2009). Latent change scores representing proportional change in CRP from baseline to Y1, and from Y1 to Y2 were created by constraining the paths between observed variables to be equal across annual time points and regressed on previous assessments. We focus on a hybrid model of change because our repeated assessment of racial discrimination inherently captured new experiences (i.e., change) across time points in tandem with annual CRP assessments.

Iterative univariate models were fit to determine the best fitting type of change, starting with models of no change and progressing to models of proportional change (i.e., predicted changes are proportional to state of prior true scores), constant change (i.e., within-person predicted changes are constant but allowed to vary between individuals), and dual change (i.e., proportional and constant change) (Grimm et al., 2016). Fit criteria for determining the best-fitting model of change were BIC (lower represents better fit), RMSEA (<0.08 = satisfactory fit), and CFI (>0.90 = satisfactory fit) (Grimm et al., 2016). Latent CRP change scores were examined in relation to annual incident racial discrimination (Model 1), adding previous discrimination (Model 2), sociodemographic (Model 3), socioeconomic (Model 4) and health-related (Model 5) covariates in block groups to assess potential confounding. Continuous covariates were grand mean centered (age, disease duration, income-poverty ratio, BMI, disease activity, organ damage). Fitted models constrained estimates of CRP change scores regressed on incident racial discrimination to be equal across annual time points to examine an overall association between incident racial discrimination and change in CRP over the two-year period. All variables were allowed to freely covary except latent change scores which encountered convergence issues (estimates allowing change scores to covary did not substantially differ from estimates restricting change score covariance despite a non-positive definite covariance matrix).

Annual incident discrimination scores were considered missing for participants with incomplete data for either of the bi-annual assessments within a study year (e.g., missing at 6-month or 12-month, missing at 18-month or 24-month). Full information maximum likelihood accounted for data missing at random on racial discrimination as well as covariates (n = 5 participants) (Graham, 2009). Latent change score analyses were

conducted using Mplus version 8.6. Syntax for the final model is available on GitHub (https://github.com/connordmartz/rd_crp.git). Data is available upon request to the BeWELL Study investigators.

3. Results

The mean age of the sample was 46.81 years (SD = 12.07). The average participant-reported household income was approximately two times the federal poverty threshold, the near poor range (Mean = 2.03, SD = 1.67). Our sample spanned a broad spectrum of SLE severity. The mean disease duration was 15.92 years (SD = 10.23), mean disease activity was 15.21 (SD = 8.05; possible range 0–44), and mean organ damage was 2.47 (SD = 2.55; possible range 0–30). Baseline descriptive information is presented in Table 1.

Mean CRP (non-transformed) at all three time points ranged from 4.26 to 5.01 mg/L, with a median range of 1.94 to 2.62 mg/L. Descriptive statistics and correlations for main study variables are shown in Table 2. Most participants (81.48%) reported at least one lifetime experience of racial discrimination in any of nine domains at baseline (Table 3). Approximately 54.74% of the sample reported experiencing a new instance of racial discrimination in at least one of the nine domains at one-year follow-up; between one-year and two-year follow-up, 52.37% reported a new instance of racial discrimination in at least one domain. Over two-thirds of participants reported incident experiences of racial discrimination in multiple domains in Y1 (39.74%) and Y2 (37.63%). Relative prevalence of incident racial discrimination over the two-year period were generally consistent with those of lifetime experiences reported at baseline. Incident racial discrimination was most common in domains that reflect routine experiences of slights and bias (e.g., getting service in a store or restaurant, on the street or in public settings). Relatively less common were experiences of acute forms of racial discrimination occurring in specific institutional domains (e.g., at school, getting hired, getting housing, from the police or in the courts).

3.1. Multivariable linear regression analyses

In multivariable linear regression models, baseline lifetime racial discrimination was not associated with baseline CRP (b = −0.025, SE = 0.023, 95% Confidence Interval (CI): −0.070, 0.020; Table A1). Controlling for baseline lifetime racial discrimination, baseline CRP, and other covariates, incident racial discrimination experienced between baseline and Y1 did not have a statistically significant association with Y1 CRP, albeit in the expected direction (b = 0.019, SE = 0.023, 95% CI:−0.026, 0.064; Table A2); it was also not significantly associated with Y2 CRP (b = 0.007, SE = 0.026, 95%: −0.043, 0.058; Table A3). Incident racial discrimination experienced between Y1 and Y2 was significantly associated CRP at Y2 after adjusting for baseline discrimination, Y1 incident discrimination, Y1 CRP, and covariates (b = 0.070, SE = 0.030, 95% CI: 0.012, 0.1290; Table A3).

3.2. Latent change score analyses

Fit statistics indicated that a proportional change model (versus constant change or dual change) best fit the data (Table A4). Fully-adjusted latent change score models fit the data well: BIC = 29023.87, CFI = 0.972, RMSEA = 0.080, SRMR = 0.014 (values for acceptable

model fit are CFI > 0.900, RMSEA < 0.080, SRMR < 0.080) (Kline, 2011). Stability path estimates for repeated assessments of racial discrimination indicated that greater lifetime experiences at baseline were associated with more reports of discrimination in Y1 (b = 0.332, SE = 0.040, 95% CI: 0.254, 0.410), which predicted discrimination in Y2 (b = 0.391, SE = 0.040, 95% CI: 0.313, 0.470). CRP change scores were not associated with CRP levels at prior time points (b_{Y1} = −0.043, SE = 0.060, 95% CI: −0.159, 0.074; b_{Y2} = 0.072, SE = 0.067, 95% CI: −0.058, 0.203).

Incident experiences of racial discrimination were associated with annual change in log-transformed CRP over the two-year period (b = 0.039, SE = 0.017, 95% CI: 0.006, 0.071) (Fig. 1 and Table A5). This corresponds to a 3.98% increase in non-transformed CRP ((exp(0.039)−1)*100) for each domain in which incident racial discrimination was experienced. Results were consistent with cross-sectional regression estimates in Y2 but not Y1 (Table A5). Additional models examined freely-estimated associations between incident racial discrimination and annual change in CRP from baseline to Y1, and from Y1 to Y2. These models provided a satisfactory but relatively worse fit to the data (BIC = 29029.48, CFI = 0.972, RMSEA = 0.014) compared to overall models that constrained estimates across Y1 and Y2. Estimates were consistent with those from cross-sectional linear regression models.

4. Discussion

Racial discrimination is a distinct source of psychosocial stress that may become biologically embedded through inflammatory channels (Cuevas et al., 2020; Goosby et al., 2018). Our study advances research in this area by being the first, to our knowledge, to longitudinally examine whether reports of new, incident experiences of racial discrimination are associated with changes in CRP levels, thus providing more robust evidence to support causal inference (Raymaekers et al., 2020). SLE operates through inflammatory mechanisms that, when exacerbated, can hasten disease progression via heightened disease activity and organ damage (Aringer, 2020). We found that incident racial discrimination predicted annual change in CRP over two-years among Black women with SLE, who experience more rapid disease progression and worse outcomes and at earlier ages than white women (Drenkard and Lim, 2019). The findings of our study suggest racial inequities in SLE progression and outcomes are due in part to experiences of racial discrimination and subsequent elevated levels of inflammation.

Our findings are consistent with previous cross-sectional and prospective studies that suggest racial discrimination is associated with higher levels of CRP and other circulating inflammatory biomarkers (Cuevas et al., 2020; Lawrence et al., 2022a; b). Estimates from final models can be interpreted as a 3.98% increase in CRP for each incident experience of racial discrimination, which, in context of baseline mean CRP values for all participants (4.26 mg/L), corresponds to an approximately 0.17 mg/L increase in CRP for each domain of racial discrimination experienced over the two-year period. Inconsistent associations between racial discrimination and CRP from baseline to Y1, and from Y1 to Y2 in cross-sectional regression and freely-estimated latent change score models may be due to measurement error in self-reported racial discrimination. Estimates from Y1

to Y2 accounted for previous year incident racial discrimination, compared to baseline to Y1 models that adjusted for lifetime experiences, as reflected in poorer model fit relative to constrained estimates. Recall bias and underestimation of lifetime experiences is a significant concern with retrospective assessments of racial discrimination, particularly for younger Black women of lower socioeconomic position (i.e., a majority of BeWELL sample) (Van Dyke et al., 2021).

Inflammation activates the complement system in SLE which can worsen tissue damage and influence disease progression and comorbidities (Enocsson et al., 2021; Walport, 2002). Prior studies have shown that psychosocial stress, when chronic, severe, and perceived to be outside of one's control, can dysregulate biological systems engaged in the stress response (Furman et al., 2019; McEwen, 2012). The immune system is highly adaptive and allostatic states resulting from acute psychosocial stressors are generally temporary. However, the accumulation of new experiences of discrimination over a two-year study period can prolong a return to homeostasis, resulting in extended allostasis with heightened levels of inflammation (Geronimus et al., 2006; Goosby et al., 2018; McEwen, 2012). This is in line with research among Black women that found stronger associations between coronary artery calcification and chronic discrimination assessed over the past five years compared to more recent experiences in the previous 12 months (Lewis et al., 2006). Shared inflammatory pathways involved in a dysregulated stress-response and the pathogenesis of SLE can accelerate disease progression and undermine health through biological "wear and tear" (Aringer, 2020; McEwen, 2012). Chronically elevated inflammation also is a risk factor for cardiovascular disease – a leading cause of overall and race-specific mortality in SLE due in part to profound immune dysregulation (Garg et al., 2022; Liu et al., 2022; Yazdany et al., 2020). Our findings are consistent with other research that has linked experiences of racial discrimination with increased disease activity and greater organ damage among Black women with SLE (Chae et al., 2019).

Compared to more subtle "everyday" forms of racial discrimination, acute instances occurring within institutional contexts pose greater risk for elevated inflammation due to strategies used by Black women to cope with routine experiences of mistreatment, as well as potential collateral effects of rumination and stress proliferation (e.g., being denied a mortgage and living in a neighborhood with high disorder and few health-promoting resources) (Cuevas et al., 2022; Lawrence et al., 2022b; Pearlin et al., 2005; Szabo et al., 2022; Thomas et al., 2019). Experiences of racial discrimination in medical settings may directly compromise disease management and CRP levels given our clinical population that frequently engages with the healthcare system. Incident everyday experiences of racial discrimination (e.g., occurring in public settings) were more common than major forms in specific institutional domains (e.g., getting hired) for our sample of Black women living with SLE in metropolitan Atlanta, Georgia from 2015 to 2019. We did not examine whether specific forms of incident racial discrimination were associated with change in CRP, which is a direction for research using measures and scales designed to compare major and everyday sub-types of discrimination.

4.1. Strengths and limitations

We note several limitations of this study. It is possible that our associations were artifacts of unmeasured confounders, such as indicators of disease severity or acute infection that are relevant to a clinical sample. Concurrent infection was not directly assessed in the study. However, final models adjusted for SLE severity (disease activity and organ damage) and excluded participants with CRP values indicative of severe infection in SLE (>60 mg/L) at any time point, and conclusions are consistent with past research on racial discrimination and inflammation (Aringer, 2020). The potential for recall bias and underreporting of racial discrimination is another limitation, although we conducted assessments of new experiences every six months. Mean CRP values in our sample are higher than those expected for non-clinical populations but are within normal limits for SLE (Aringer, 2020; Cuevas et al., 2020; Dima et al., 2016; Lawrence et al., 2022a). Despite these limitations, our study has multiple strengths that advance research on racial discrimination and inflammation. Our use of repeated measures for both racial discrimination and CRP strengthens the case for causality and explicitly focuses on within-person effects rather than between-person differences (Grimm et al., 2016; McArdle, 2009; Raymaekers et al., 2020). We also address measurement error in the Experiences of Discrimination measure by modifying the stem to capture incident experiences of discrimination in the past six months since the previous assessment (Gaston and Jackson, 2022; Van Dyke et al., 2021). Finally, the BeWELL Study addresses the paucity of social epidemiologic research on SLE, and provides unique insight into the causes of racial inequities in disease progression by focusing specifically on psychosocial experiences relevant to Black women (Celious and Oyserman, 2001; Volpe et al., 2022).

5. Conclusions

Our study advances understandings of racial inequities in SLE and potentially other chronic diseases mediated by inflammatory mechanisms. An important direction for future research will be to examine associations between incident discrimination and changes in inflammation in non-clinical samples, as well as consider effect modifiers, such as racial identity, that have been shown to buffer the effects of racial discrimination (Brody et al., 2015). Additionally, future work should consider the role of discrimination and elevated CRP in comorbid conditions, such as cardiovascular disease, which may contribute to racial inequities in SLE outcomes (Garg et al., 2022). The results of this study contribute to a growing body of evidence indicating that racial discrimination is a toxic health threat, and that the embodiment of racial discrimination is one pathway through which racism contributes to inequitable health outcomes for Black Americans (Chae et al., 2020; Williams et al., 2019). Policies and laws aimed at eliminating contemporary and persistent forms of racial discrimination are likely to advance health equity for Black women with SLE, as well as Black Americans more broadly.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data availability

Data will be made available upon request to BeWELL Study Investigators.

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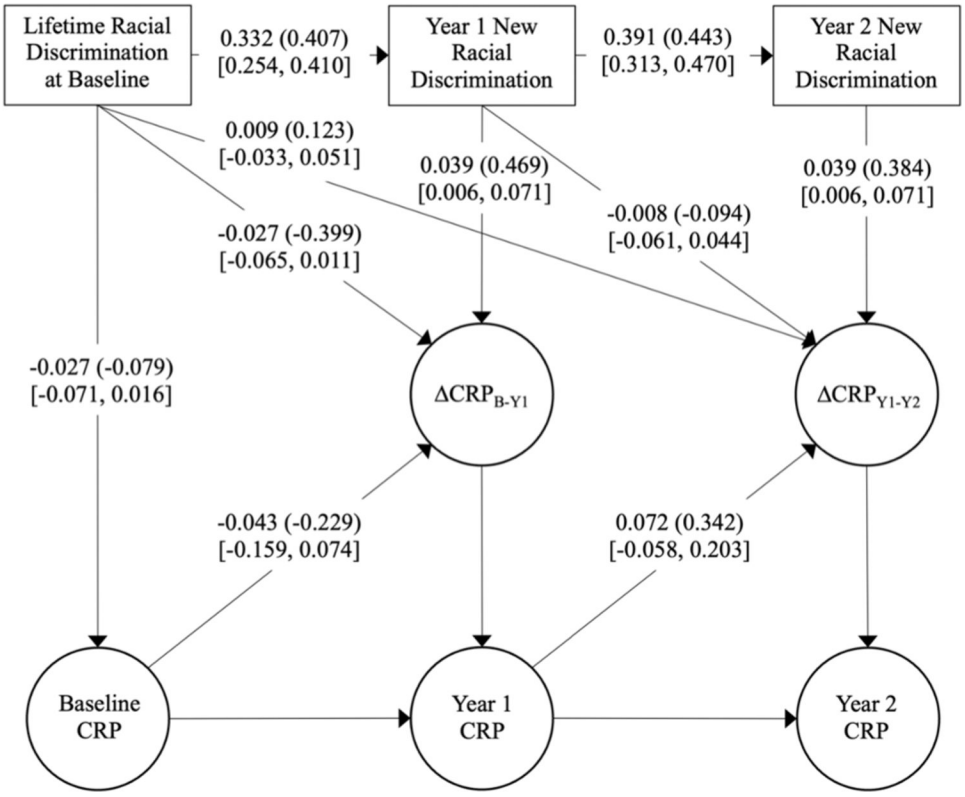

Fig. 1.

Structural equation model of annual latent change in log-transformed C-reactive protein (CRP) and associations with new annual experiences of racial discrimination in the Black Women's Experiences Living with Lupus (BeWELL) Study (n = 380). Notes: Estimates adjust for sociodemographic, socioeconomic, and health-related covariates. Standardized coefficients are in parentheses and 95% confidence intervals are in brackets. Paths without estimates were fixed to 1 for model identification. Model fit: CFI = 0.972, RMSEA = 0.080, SRMR = 0.014.

Table 1

Descriptive statistics for baseline covariates in latent change score analyses in the Black Women's Experiences Living with Lupus (BeWELL) Study (n = 380).

Covariate	n	%	Mean	SD
Age	380	–	46.81	12.07
Years since diagnosis	380	–	15.92	10.23
Relationship Status				
Married/Marriage-Like	174	45.79	–	–
Romantic Relationship	22	5.79	–	–
Divorced/Separated, Widowed	85	22.37	–	–
Single	99	26.05	–	–
Income-Poverty Ratio	378	–	2.03	1.67
Educational Attainment				
College/Advanced Degree	112	29.55	–	–
Some College	172	45.38	–	–
High School Degree	66	17.41	–	–
<High School	29	7.65	–	–
Work Status				
Full-time	111	29.21	–	–
Part-time	49	12.89	–	–
Out of labor force	20	5.26	–	–
Not working	200	52.63	–	–
Insurance Status				
Private	141	37.11	–	–
Public	199	52.37	–	–
None	40	10.53	–	–
Body Mass Index	380	–	30.96	7.98
Current Smoker	59	15.61	–	–
Steroid Use	204	53.68	–	–
Hydroxychloroquine Use	285	75.00	–	–
Other Immunosuppressant Use	165	43.42	–	–
Disease Activity (SLAQ)	380	–	15.21	8.05
Inactive (SLAQ = 0)	6	1.58	–	–
Mild (SLAQ = 1–10)	105	27.63	–	–
Moderate (SLAQ = 11–16)	111	29.21	–	–
Severe (SLAQ = 17+)	158	41.58	–	–
Organ Damage (BILD)	380	–	2.47	2.55

Table 2

Descriptive statistics and bivariate correlations for experiences of racial discrimination (EOD) and C-reactive protein (CRP) in the Black Women's Experiences Living with Lupus (BeWELL) Study (n = 380).

Variable	n	Mean	SD	Median	IQR	BL EOD*	Y1 EOD	Y2 EOD	BL CRP*	Y2 CRP	Y3 CRP
Baseline discrimination	377	3.73	2.88	4.00	5.00	–					
Year 1 new discrimination	380	1.80	2.35	1.00	3.00	**0.42**	–				
Year 2 new discrimination	380	1.55	2.07	1.00	2.00	**0.40**	**0.49**	–			
Baseline CRP	380	4.26	6.61	1.94	3.71	−0.04	0.06	0.03	–		
Year 2 CRP	380	4.79	6.73	2.61	4.40	−0.09	0.04	−0.03	**0.57**	–	
Year 3 CRP	380	5.01	6.93	2.62	5.15	−0.05	0.08	0.09	**0.50**	**0.49**	–

Note: CRP values are not log-transformed for interpretability. Bolded correlations indicate statistical significance at $p < 0.05$.

*Baseline (BL).

Table 3

Percent of participants reporting lifetime and incident experiences of racial discrimination across nine domains of the Experiences of Discrimination (EOD) measure in the BeWELL Study (n = 380).

EOD Item	Baseline (Lifetime)	Year 1 Incident	Year 2 Incident
1. At school	30.16	8.68	6.32
2. Getting hired or getting a job	46.97	12.89	13.68
3. At work	48.28	21.32	16.58
4. Getting housing	29.02	13.68	10.53
5. Getting medical care	28.50	19.74	17.98
6. Getting service in a store or restaurant	66.75	38.95	36.84
7. Getting credit, bank loans, or a mortgage	36.24	19.74	16.32
8. On the street or in a public setting	52.51	29.74	25.53
9. From the police or in the courts	35.88	15.26	11.05

Krieger et al. (2005).