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Original Contribution

Racial Discrimination, Disease Activity, and Organ Damage: The Black Women's Experiences Living With Lupus (BeWELL) Study

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Black women are disproportionately affected by systemic lupus erythematosus (SLE), a chronic, potentially debilitating autoimmune disease, and they also experience more rapid progression and worse outcomes compared with other groups. We examined if racial discrimination is associated with disease outcomes among 427 black women with a validated diagnosis of SLE, who live in the Atlanta, Georgia, metropolitan area, and were recruited to the Black Women's Experiences Living with Lupus Study (2015–2017). Frequency of self-reported experiences of racial discrimination in domains such as employment, housing, and medical settings was assessed using the Experiences of Discrimination measure. SLE activity in the previous 3 months, including symptoms of fatigue, fever, skin rashes, and ulcers, was measured using the Systemic Lupus Activity Questionnaire; irreversible damage to an organ or system was measured using the Brief Index of Lupus Damage. Results of multivariable linear regression analyses examining the Systemic Lupus Activity Questionnaire and log-transformed Brief Index of Lupus Damage scores indicated that increasing frequency of racial discrimination was associated with greater SLE activity (b = 2.00, 95% confidence interval: 1.32, 2.68) and organ damage (b = 0.08, 95% confidence interval: 0.02, 0.13). Comprehensive efforts to address disparities in SLE severity should include policies that address issues of racial discrimination.

black women; racial discrimination; systemic lupus erythematosus

Abbreviations: BeWELL, Black Women's Experiences Living With Lupus; BILD, Brief Index of Lupus Damage; CI, confidence interval; GOAL, Georgians Organized Against Lupus; SD, standard deviation; SLE, systemic lupus erythematosus.

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with potentially debilitating health consequences (1). It is characterized by periods of disease activity that include a vast array of clinical manifestations, such as skin rashes, oral ulcers, fever, vasculitis, myositis, and inflammatory arthritis (2, 3). Organ damage and comorbid conditions may emerge as consequences of uncontrolled disease activity and chronic inflammation (4). The number of people living with SLE in the United States is estimated at between 161,000 and 322,000 (5); still, the epidemiology of SLE is marked by significant disparities along racial and sex lines (6–9). In the state of Georgia, the prevalence of SLE among women is nearly 9 times greater than among men, and it is more than 3 times greater among blacks compared with whites (6). Moreover, there are wide variations in severity and progression between blacks and whites with SLE. For example, the prevalence of renal and cardiovascular damage in SLE is 2 to 4 times greater among blacks compared with whites (10, 11), and blacks suffer these complications 3 to 9 years earlier, on average (12, 13). Blacks with SLE also have overall death rates that are up to 3 times higher than those of whites, and blacks with SLE die earlier (14). Moreover, according to US death trend data between 1968 and 2013, there was a relatively smaller decrease in SLE-related death among blacks (13.3%) than in whites (33.3%), suggesting that racial disparities in SLE outcomes have been increasing over time (15).

The reasons for racial disparities in SLE outcomes are multifactorial. However, genetic evidence for these differences is lacking; in fact, psychosocial factors have been

more clearly identified as having a role in disease progression (16, 17). Socioeconomic stressors are associated with SLE severity and death (18–20). Geographic clusters with higher SLE death rates are concentrated in areas with higher poverty and numbers of racial minorities, implicating the role of environmental factors in SLE outcomes (17, 21–23). Compared with their white counterparts, black women are more likely to experience psychosocial stressors shown to exacerbate SLE, including those associated with poverty, unemployment, exposure to violence, and physical victimization (24–28). Black women also disproportionately experience environmental stressors and area-level deprivation associated with racial residential segregation and poverty concentration (29–31).

The constellation of psychosocial risk factors experienced by black women with SLE compound those more generally associated with the management of a chronic disease, leading to worse disease trajectories in this population (32). Among these stressors is the interpersonal experience of racial discrimination, a distinct, qualitatively unique, and salient form of psychosocial stress that also may increase the risk of poor SLE outcomes (33–35). Racial discrimination can be experienced in multiple societal domains, such as employment, housing, education, health care, and legal contexts; these experiences may proliferate stress by diminishing socioeconomic attainment (33, 36–38). Experiences of racial discrimination in housing markets can also undermine health through segregation into worse neighborhood conditions (36, 38). Disparate treatment in health care can directly affect health. Patient-reported racial discrimination by physicians has been associated with heightened SLE activity and depression (39, 40). Chronic stress associated with racial discrimination, particularly when it is viewed as being outside of personal control, may compromise psychological adjustment and result in maladaptive coping responses, such as smoking and problem drinking, which negatively affect the progression of chronic diseases (33, 38, 41). Depression resulting from racial discrimination may lead to accelerated declines in health among women with SLE (39, 40). Indeed, studies have most consistently found associations for adverse mental health consequences of racial discrimination (42, 43). Accordingly, racial discrimination may increase disease severity through these mental health and behavioral channels.

As a source of psychosocial stress, racial discrimination can also elicit a cascade of biological responses that damage stress-response systems over time and, over one's life, can contribute to "weathering" or accelerated physiologic deterioration (44, 45). Discrimination is associated with a range of inflammatory markers (46–49). Repeated experiences of racial discrimination may lead to chronically elevated levels of proinflammatory cytokines and acute-phase proteins, contributing to a heightened inflammatory state (50, 51). These biological conditions may increase the risk of diseases characterized by inflammatory processes. For example, racial discrimination has been associated with increased cardiovascular risk, as well as biological processes and other health conditions that are sensitive to inflammation (52–56). Several indicators of inflammation are involved in the etiopathogenesis of SLE activity and organ damage, and the maintenance of inflammation (57–62). Accordingly, racial discrimination may have consequences for more acute SLE outcomes, such as disease activity. In addition, the tolls of racial discrimination on biological systems critical for regulating the stress response may accumulate and place black women at greater risk for earlier onset of SLE complications and disease damage. For example, experiences of contemporaneous unfair treatment attributed to racial as well as nonracial causes were associated with greater irreversible organ damage among black women with SLE (63). The purpose of the current study was to examine the association among racial discrimination, cumulative organ damage, and disease activity among black women with SLE from a large, population-based cohort.

METHODS

Sample and procedures

The cross-sectional, observational data used in this study are from the Black Women's Experiences Living with Lupus (BeWELL) Study. Participants were recruited from the Georgians Organized Against Lupus (GOAL) cohort, which drew primarily from the Georgia Lupus Registry (64). The Georgia Lupus Registry is a population-based registry funded by the Centers for Disease Control and Prevention and designed to estimate the prevalence and incidence of SLE in metropolitan Atlanta, Georgia (6). The Georgia Lupus Registry includes a full spectrum of patients, from mild to severe cases of SLE, and from all levels of socioeconomic strata. To maximize ascertainment of potential cases, a broad range of case-finding sources was used, including hospitals, health care providers (i.e., rheumatologists, dermatologists, nephrologists), commercial laboratories, and population databases. Hospital-based laboratories and regional pathology laboratories were also queried for results to identify patients with potential SLE. Data from larger commercial laboratories and the Centers for Medicare and Medicaid Services End-Stage Renal Disease database were also screened. Other unique databases, such as from the Veterans Administration, Medicaid claims, other state databases (e.g., hospital discharge), and electronic medical record systems, were analyzed. The result was one of the largest, population-based lupus epidemiology registries ever in the United States, with more than 1,500 people with validated lupus diagnoses meeting the American College of Rheumatology classification criteria for SLE (≥4 criteria) or 3 criteria with a diagnosis of SLE by a board-certified rheumatologist (65). GOAL is further enhanced through recruitment of participants from the Lupus Clinic of Grady Memorial Hospital, a large public hospital in Atlanta, as well as from diverse community rheumatologist practices.

Eligibility criteria for the BeWELL Study were as follows: consent given to participate in the GOAL cohort, self-identification as black or African American; between 18 and 79 years of age; living in metropolitan Atlanta; and able to read, write, and understand English and respond to questions on a computer. We attempted to contact a total of 710 potentially eligible women who were enlisted in GOAL during the recruitment period, from April 2015 to May 2017. Attempted

contact occurred initially through mail, which included study information and a request to contact study staff by telephone or by returning an interest reply form in a prepaid envelope. For those who did not respond in 2 weeks, study staff followed up through telephone calls. We were unable to reach 102 women. Of the remaining 608 participants, 12 did not meet eligibility criteria and 55 refused to participate (refusal rate = 9.2%); 103 women who were contacted could not be scheduled despite repeated attempts. This left a total sample size of 438 participants.

We compared the 260 black women in GOAL who were believed to be eligible but did not participate with the women who participated in BeWELL. Examining responses provided in the GOAL survey, we found that BeWELL participants were younger at the time of recruitment in the GOAL cohort (mean = 46.1 (standard deviation (SD), 12.3) years vs. 47.9 (SD, 12.9) years; $P < 0.001$), were diagnosed with SLE at a younger age (mean = 31.3 (SD, 11.0) years vs. 34.6 (12.0) years; $P < 0.001$), and had higher levels of disease activity (mean Systemic Lupus Activity Questionnaire score = 17.6 (SD, 9.1) vs. 15.8 (9.1); $P < 0.001$). Furthermore, examining the GOAL survey, BeWELL participants were more likely to be poor (50.1% vs. 40.7%; $P = 0.03$). There were no significant differences between the groups in terms of other SLE characteristics (i.e., disease duration, organ damage) or sociodemographic variables (i.e., marital status, education, employment, insurance).

Respondents were assessed primarily on-site at the Division of Rheumatology of the Emory University School of Medicine; 20 respondents participated through home visits. Trained lay interviewers assessed demographic characteristics and measures of organ damage and disease activity. More sensitive questions, including those assessing racial discrimination, were self-administered via computer-assisted software. Signed informed consent was obtained from all study participants. The median duration of study visit was 2.2 hours. All protocols and procedures were approved by the Institutional Review Board of Emory University.

Measures

SLE outcomes. SLE activity was measured using the Systemic Lupus Activity Questionnaire, a validated, patient-reported measure developed to track disease activity (66, 67). The questionnaire includes 24 items related to disease activity in the past 3 months, such as fatigue, fever, oral ulcers, rashes, vasculitis, myalgias, and joint swelling. Items were grouped and weighted, with possible scores ranging from 0 to 44. Higher scores indicate greater disease activity.

SLE organ damage was measured using the Brief Index of Lupus Damage (BILD), a validated, patient-reported measure of damage due to SLE in 12 organ systems; the index is used in clinical research studies (68–70). Cumulative organ damage is an important outcome and predicts death, physical function, quality of life, and disability. The BILD enables researchers to assess major irreversible damage to an organ or system since the onset of SLE and present for at least 6 months. Items are endorsed as present or absent, with possible scores ranging from 0 to 30. Higher scores indicate greater organ damage.

Racial discrimination. Racial discrimination was measured using the Experiences of Discrimination measure, which asks participants, "Have you ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior in any of the following situations because of your race, ethnicity or color," followed by 9 specific domains: at school; getting a job; at work; getting housing; medical care; service at a store or restaurant; obtaining credit or a loan; on the street or in a public setting; and from the police or in the courts (71–73). Response choices were "no," "once," "two or three times," and "four or more times." We examined 2 scoring methods: 1) the situation version, which is a count of the number of items endorsed at least once, and which ranges from 0 to 9; and 2) the frequency version, which is calculated as the mean score of items with the following values assigned to response choices: 0 = no; 1 = once; 2.5 = 2 or 3 times; and 5 = 4 or more times.

Covariates. Age in years was measured on the basis of date of birth. Years since diagnosis were calculated on the basis of response to 1 of the following: the number of years and months since being diagnosed; the month and year of diagnosis; or the age at diagnosis. Relationship status was categorized as married or in a marriage-like relationship; romantic relationship; divorced, separated, or widowed; or single. Socioeconomic covariates were as follows: education (less than high school, high school, some college, or college graduate or advanced degree), work status (full-time; part-time; out of labor force, including retired, homemaker, or student; or not working, including those unemployed, laid-off, or unable to work due to health or disability), insurance status (private, public, or none), and ratio of household income to the poverty threshold. Household income in the past month was reported in categories of $500 increments, from which we took the midpoint of the response category and multiplied by 12. For those who volunteered past-year household income, responses were recorded in categories of $5,000 increments, from which we took the category midpoint to represent annual household income. A follow-up question assessed whether the income reported was before or after taxes; for those reporting that it was after taxes, we calculated the pre-tax amount on the basis of Georgia income tax rates for participant interview year (74). We calculated the ratio of household income to the federal poverty threshold on the basis of the number of adults and children in the household (75).

Health-related covariates were body mass index, examined continuously from height and weight, which were measured using standardized protocols; self-reported days of exercise per week in the past year; self-reported current smoking status (yes vs. no); and information on current SLE medication use from lists brought by participants to the interview, in addition to a checklist of lupus medications that interviewers went through with each participant. In the current study, SLE medication used was coded as yes versus no for the following: steroids (e.g., prednisone, medrol, methylprednisolone), antimalarials (e.g., hydroxychloroquine sulfate), and other immunosuppressant drugs (e.g., methotrexate, cyclophosphamide, cyclosporine, mycophenolate, dapsone, azathioprine, belimumab, rituximab).

Table 1. Sociodemographic Characteristics of Participants in the Black Women's Experiences Living With Lupus Study ($n = 427$), 2015–2017

Variable	No.	%	Mean (SD)
SLE organ damage (BILD score)			
0	68	15.93	
1	95	22.25	
2	78	18.27	
≥3	186	43.56	
SLE activity (SLAQ score)			15.11 (7.94)
Racial discrimination: situations			
0	83	19.44	
1–2	86	20.14	
3–4	87	20.37	
≥5	171	40.05	
Racial discrimination: frequency			
0	83	19.44	
0.01–1.00	189	44.26	
1.01–2.00	102	23.89	
≥2.01	53	12.41	
Age, years			46.71 (12.28)
Years since diagnosis			15.88 (10.32)
Relationship status			
Married or marriage-like	194	45.43	
Romantic relationship	26	6.09	
Divorced/separated or widowed	94	22.01	
Single, never married	113	26.46	
Education			
Less than high school	36	8.43	
High school	77	18.03	
Some college	194	45.43	
Bachelor's degree or higher	120	28.10	
Income-to-poverty ratio			2.00 (1.68)
≤100% poverty income	134	31.38	
Work status			
Full-time	122	28.57	
Half-time	54	12.65	
Out of labor force	21	4.92	
Unable to work	230	53.86	
Insurance status			
Private	153	35.83	
Public	226	52.93	
None	48	11.24	
Body mass index[a]			30.91 (8.11)
Exercise			2.06 (1.71)
Smoking status			
No	365	85.48	
Yes	62	14.52	
Steroids			
No	192	44.96	
Yes	235	55.04	
Hydroxychloroquine			
No	114	26.70	
Yes	313	73.30	
Immunosuppressants			
No	239	55.97	
Yes	189	44.03	

Abbreviations: BILD, Brief Index of Lupus Damage; SD, standard deviation; SLE, systemic lupus erythematosus; SLAQ, Systemic Lupus Activity Questionnaire.

[a] Weight (kg)/height (m)^2.

Analysis plan

Eleven participants (2.5%) with missing data for any of the variables being investigated were excluded from analyses, leaving a total analytic sample size of 427 participants. We specified multivariable linear regression models examining SLE activity. We also used multivariable linear regression to examine log-transformed BILD, given its right-skewed distribution. Results were substantively similar to models examining BILD continuously. We also compared models using the situation versus frequency scoring methods for the Experiences of Discrimination measure, which did not lead to different conclusions. Here, we present results using the frequency version of the Experiences of Discrimination measure. Results from models using the situation version to score the Experiences of Discrimination measure are available upon request. Nested models were specified, controlling for demographic, socioeconomic, and health-related characteristics entered in block groups.

RESULTS

The mean (standard deviation) SLE activity score in our sample was 15.11 (7.94). More than half of participants (61.8%; $n = 264$) had a damage score of 2 or more, 22.3% ($n = 95$) had damage to 1 organ or system, and 15.9% of participants ($n = 68$) had no major organ damage Participants were an average of 46.7 (SD, 12.3) years and the average time since diagnosis with SLE was 15.9 (SD, 10.3) years. The majority of participants (80.6%; $n = 344$) reported experiencing racial discrimination in at least 1 domain, with 40.1% ($n = 171$) reporting experiencing racial discrimination in 5 or more. The most commonly reported domain of racial discrimination was "getting service at a store or restaurant" (65.6%). Participants were least likely to report racial discrimination "getting medical care," although this still represented a relatively large percentage of participants (27.6%). Additional characteristics of our sample are listed in Table 1.

Table 2. Linear Regression Analysis of Disease Activity Among Participants in the Black Women's Experiences Living With Lupus Study ($n = 427$), 2015–2017

Variable	Model 1		Model 2		Model 3	
	b	95% CI	b	95% CI	b	95% CI
Racial discrimination	1.92	1.18, 2.66	2.20	1.52, 2.89	2.00	1.32, 2.68
Age	−0.03	−0.10, 0.05	−0.02	−0.10, 0.05	−0.03	−0.11, 0.04
Years since diagnosis	−0.03	−0.11, 0.06	−0.04	−0.11, 0.04	−0.03	−0.11, 0.05
Relationship status[a]						
Romantic relationship	−0.05	−3.23, 3.13	−0.48	−3.37, 2.41	−1.18	−4.04, 1.67
Divorced/separated, widowed	1.10	−0.85, 3.05	0.07	−1.76, 1.90	−0.26	−2.08, 1.55
Single, never married	−0.28	−2.13, 1.56	−1.02	−2.74, 0.69	−1.21	−2.90, 0.48
Education[b]						
High school			−1.30	−4.14, 1.54	−0.60	−3.40, 2.21
Some college			−0.96	−3.54, 1.62	−0.54	−3.10, 2.02
Bachelor's degree or higher			−3.64	−6.54, −0.75	−2.89	−5.80, 0.02
Income-to-poverty ratio			−1.02	−1.54, −0.51	−0.96	−1.46, −0.45
Work status[c]						
Half-time			−0.31	−2.77, 2.16	−0.56	−3.00, 1.88
Out of labor force			1.41	−2.15, 4.97	1.61	−1.92, 5.14
Unable to work			3.59	1.58, 5.60	3.06	1.03, 5.09
Insurance status[d]						
Public			−0.42	−2.38, 1.54	−0.40	−2.34, 1.53
None			−1.11	−3.63, 1.40	−1.36	−3.84, 1.11
Body mass index[e]					0.07	−0.02, 0.15
Exercise					0.19	−0.20, 0.58
Smoker: yes vs. no					3.17	1.19, 5.14
Steroids: yes vs. no					2.43	0.98, 3.88
Hydroxychloroquine: yes vs. no					−1.89	−3.43, −0.35
Immunosuppressants: yes vs. no					−0.29	−1.74, 1.16

Abbreviation: CI, confidence interval.
[a] The reference category was married or a marriage-like relationship.
[b] The reference category was less than a high school education.
[c] The reference category was full-time work.
[d] The reference category was private insurance.
[e] Weight (kg)/height $(m)^2$.

According to linear regression analyses, racial discrimination had a significant bivariate relationship with SLE activity (b = 1.89, 95% confidence interval (CI): 1.16, 2.62). Results from multivariable analyses are reported in Table 2. Adjusting for potential demographic confounders (model 1: age, years since diagnosis, and relationship status), racial discrimination continued to be associated with SLE activity (b = 1.92, 95% CI: 1.18, 2.66). Although socioeconomic characteristics may be considered possible mediators, additional adjustment for these factors (model 2: model 1 plus education, poverty ratio, work status, and insurance status) somewhat increased the magnitude of the association between racial discrimination and SLE activity (b = 2.20, 95% CI: 1.52, 2.89). Adjustment for health-related variables (model 3: model 2 plus body mass index, exercise, smoking status, and use of steroids, hydroxychloroquine, and other immunosuppressants) did not substantively change this relationship (b = 2.00, 95% CI: 1.32, 2.68).

A significant bivariate relationship with racial discrimination (b = 0.09, 95% CI: 0.03, 0.15) was determined from linear regression models in which log-transformed organ damage was examined. Results from multivariable analyses are listed in Table 3. Similar to results from models examining SLE activity, greater reported racial discrimination was associated with higher organ damage scores. This association was also robust to adjustment for demographic factors (model 1: b = 0.08, 95% CI: 0.02, 0.14), socioeconomic characteristics (model 2: b = 0.08, 95% CI: 0.02, 0.14), and health-related variables (model 3: b = 0.08, 95% CI: 0.02, 0.13). The estimate from our final model indicated that each unit increase in the frequency of racial discrimination was associated with an increase of 0.08 units in log-BILD score.

Table 3. Regression Analysis of Log-Transformed Organ Damage Score Among Participants in the Black Women's Experiences Living With Lupus Study ($n = 427$), 2015–2017

Variable	Model 1		Model 2		Model 3	
	b	95% CI	b	95% CI	b	95% CI
Racial discrimination	0.08	0.02, 0.14	0.08	0.02, 0.14	0.08	0.02, 0.13
Age	0.01	0.00, 0.01	0.01	0.00, 0.01	0.01	0.00, 0.01
Years since diagnosis	0.01	0.01, 0.02	0.01	0.01, 0.02	0.01	0.00, 0.02
Relationship status[a]						
Romantic relationship	0.07	−0.19, 0.33	0.08	−0.17, 0.32	0.06	−0.18, 0.31
Divorced/separated, widowed	0.17	0.01, 0.33	0.11	−0.04, 0.27	0.01	−0.06, 0.26
Single, never married	0.14	−0.02, 0.29	0.09	−0.06, 0.23	0.01	−0.05, 0.24
Education[b]						
High school			0.08	−0.16, 0.33	0.07	−0.17, 0.32
Some college			0.13	−0.09, 0.36	0.10	−0.12, 0.32
Bachelor's degree or higher			−0.03	−0.28, 0.22	−0.07	−0.32, 0.18
Income-to-poverty ratio			0.00	−0.04, 0.05	0.01	−0.04, 0.05
Work status[c]						
Half-time			0.18	−0.03, 0.39	0.15	−0.07, 0.36
Out of labor force			0.07	−0.24, 0.38	0.05	−0.26, 0.35
Unable to work			0.40	0.23, 0.57	0.33	0.15, 0.50
Insurance status[d]						
Public			0.00	−0.17, 0.17	0.03	−0.14, 0.19
None			−0.25	−0.47, −0.04	−0.24	−0.45, −0.03
Body mass index[e]					0.00	−0.01, 0.01
Exercise					−0.01	−0.05, 0.02
Smoker: yes vs. no					−0.06	−0.23, 0.11
Steroids: yes vs. no					0.19	0.07, 0.32
Hydroxychloroquine: yes vs. no					−0.24	−0.37, −0.10
Immunosuppressants: yes vs. no					−0.01	−0.14, 0.12

Abbreviation: CI, confidence interval.
[a] The reference category was married or a marriage-like relationship.
[b] The reference category was less than a high school education.
[c] The reference category was full-time work.
[d] The reference category was private insurance.
[e] Weight (kg)/height $(m)^2$.

Alternatively, the exponential of this estimate, 1.20, indicated that each unit increase in racial discrimination was associated with a 20% increase in the geometric mean of BILD score.

DISCUSSION

Results from the present study are concordant with those of a prior study of unfair treatment attributed to race and organ damage among black women with SLE, as well as of other research on racial discrimination and health more broadly (63, 76, 77). Specifically, we found that greater frequency of racial discrimination was associated with increased SLE activity and organ damage. Our findings suggest that experiences of racial discrimination contribute to racial disparities in SLE outcomes. We leveraged a large population-based sample of black women with validated SLE, which allows us to generalize inferences about the association between racial discrimination and SLE severity to a greater diversity of patients. Our study advances knowledge in this understudied area of research.

There is a growing body of evidence indicating psychosocial stress exacerbates the clinical symptomatology of SLE and contributes to worsening health. For example, in a recent study, general perceived stress was associated with cognitive symptoms in patients with SLE (78). Lower socioeconomic status has been associated with greater functional disability and organ damage (21, 22, 79, 80). Moreover, within socioeconomic strata, racial disparities in health consistently have been apparent in lower as well as higher ranges (20, 81, 82). The findings of these studies suggest structural inequalities related to being a racial minority, such as those linked to

racism, result in health tolls (36, 38). In addition, in carefully controlled observational research, racial disparities in SLE progression were not entirely accounted for by differences in access to health care, detection, and treatment (16, 83). Our findings indicate that racial discrimination is a unique source of stress that exerts a negative health impact even after adjustment for socioeconomic variations and differences in health-related characteristics among black women with SLE.

Our results indicate that racial discrimination is commonly reported in this population and that such experiences have negative consequences for SLE severity. For example, differential treatment in medical settings has direct implications for disease management. Supporting this finding, patient-reported racial discrimination by physicians has been associated with heightened SLE activity and depression (39, 40); this relationship may be mediated by a lack of trust in physicians, poor treatment adherence, and avoidance of care (84). The causal effect of racial discrimination on SLE outcomes is also biologically plausible. Evidence for associations between discrimination and inflammation has been found in both cross-sectional and prospective studies (46, 47); in turn, inflammation has been strongly linked to SLE severity (58, 61, 62). Furthermore, prior research suggests that black women may be particularly impacted by such experiences; they report greater distress from racial discrimination than do black men (85). For example, in a large, multiethnic sample, among women from the general population, greater experiences of general as well as racially attributed lifetime and everyday discrimination were associated with higher levels of interleukin-6 (86), an inflammatory biomarker that is significantly elevated during periods of SLE activity. These associations, however, were mixed or of lower magnitude among men. These and other findings suggest that racial discrimination is associated with biological factors shown to aggravate SLE activity, which over time accrue and lead to irreversible physiologic damage (17).

Several limitations of this study should be noted. Because these results are based on cross-sectional data, direction of causality is not definitive and third-variable explanations are more difficult to rule out. For example, it is possible that greater SLE activity or organ damage resulted in increased perceptions of racial discrimination. Although the interpretation of our findings is consistent with other research demonstrating a causal effect of racial discrimination on the progression of other diseases (49, 87–89), additional studies using more than 1 wave of data are an important forthcoming step. Also, although our self-reported measures of disease activity and organ damage are well-validated, additional insight may be gleaned through examination of objective health indicators (e.g., SLE-relevant biomarkers). Finally, only 1 racial and sex group was considered in a specific geographic area. Although this allowed for in-depth consideration of our hypotheses in a specific population known to be at particularly high risk for poor SLE outcomes, our results may not be generalizable to other groups not represented in our study.

Despite these limitations, our study is 1 of the largest investigations of the social epidemiology of SLE among black women. These findings point to the salience of racial discrimination in the lives of black women and its relevance to health outcomes. Although results from this study are specific to SLE, they may also have implications for other chronic conditions, particularly those mediated by inflammatory mechanisms. Our findings contribute to a growing body of research that suggests experiences of racial discrimination, as a source of psychosocial stress, can generate health inequities and accelerate progression of multiple diseases. Because inflammation is a central characteristic of SLE, it may be a particularly useful context in which to identify the mechanisms and health consequences of racial discrimination. Research that integrates biological markers of stress and inflammation may help further elucidate these relationships. Our study highlights the critical need to eliminate racial discrimination across multiple domains of society, through greater enforcement of existing antidiscrimination policies at institutional levels, including in health care settings, and addressing the perpetration of discriminatory acts in other social domains. These steps represent important components of comprehensive efforts aimed at reducing racial disparities in health.

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