Discrimination and Cumulative Disease Damage Among African American Women With Systemic Lupus Erythematosus

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Objectives. We examined associations between unfair treatment, attributions of unfair treatment to racial discrimination, and cumulative disease damage among African American women with systemic lupus erythematosus (SLE).

Methods. We used multivariable regression models to examine SLE damage among 578 African American women in metropolitan Atlanta, Georgia, recruited to the Georgians Organized Against Lupus cohort.

Results. When we controlled for demographic, socioeconomic, and health-related covariates, reporting any unfair treatment was associated with greater SLE damage compared with reporting no unfair treatment (b = 0.55; 95% confidence interval = 0.14, 0.97). In general, unfair treatment attributed to nonracial factors was more strongly associated with SLE damage than was unfair treatment attributed to racial discrimination, although the difference was not statistically significant.

Conclusions. Unfair treatment may contribute to worse disease outcomes among African American women with SLE. Unfair treatment attributed to nonracial causes may have a more pronounced negative effect on SLE damage. Future research may further examine possible differences in the effect of unfair treatment by attribution. (*Am J Public Health.* 2015;105:2099–2107. doi:10.2105/AJPH.2015.302727)

Systemic lupus erythematosus (SLE) is a multisystem, chronic autoimmune disorder marked by considerable racial disparities in prognosis. In addition to having a greater prevalence of SLE, African American women are more frequently affected by organ damage and comorbid conditions that emerge as a consequence of disease activity and disease-related chronic inflammation and tissue damage.[1–3] The prevalence of renal and cardiovascular damage in SLE is higher among African Americans than Whites, and African Americans with SLE suffer these complications at earlier ages.[4,5] African Americans with SLE have overall mortality rates that are up to 3 times higher than for Whites and also disproportionately suffer from premature mortality.[6,7] Although research on the causes of these divergent outcomes is in its infancy, evidence suggests that genetic and behavioral factors, including differences in access to care, detection, and treatment do not entirely account for racial disparities in SLE.[8,9]

There is increasing interest in broader stressors tied to racial minority status, including unfair treatment and discrimination experienced systematically along racial lines, as social determinants of disease susceptibility and progression among African Americans.[10,11] These experiences include everyday forms of racially motivated unfair treatment, such as instances of being treated with less courtesy or respect or of receiving poorer service.[12] These social insults are commonly reported as salient sources of psychosocial stress, particularly among African American women, who in addition to contending with the immediate psychological and physical consequences of such experiences have also expressed heightened vigilance in anticipation of being treated unfairly.[13]

Discrimination may lead to greater disease burden by undermining psychological adjustment, as well as through maladaptive behavioral coping responses such as smoking, problem alcohol consumption, and other risk-taking behaviors.[14–17] Repetitive experiences of racism-related stressors may also lead to premature physiological deterioration directly through its effects on biological systems engaged in the stress response.[18–20] Stress stimulates a cascade of biochemical reactions mediated by the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system, and it can accelerate disease progression by leading to "wear and tear" of the organism, ultimately compromising the body's ability to effectively respond to such challenges.[21,22] Repeated and more severe forms of psychosocial stress result in chronically elevated levels of proinflammatory cytokines and acute-phase proteins, and in a heightened inflammatory state.[23]

Discrimination can exacerbate disease progression via these biological channels, and it has been associated with a range of indicators of oxidative stress and inflammation.[23–28] Accordingly, discrimination may have negative consequences for SLE, which is characterized by elevated serum concentration of several biomarkers of inflammation, including interleukin-6, interleukin-1, C-reactive protein, and tumor necrosis factor, which in turn have been linked to greater disease activity.[29–32] However, findings on the association between discrimination and health outcomes have been equivocal, with some studies finding no significant association and others reporting curvilinear or inverse associations.[11,33–39] These inconsistent findings may be attributable to the ways in which researchers have operationalized discrimination. For example, whereas some studies have explicitly focused on discrimination attributed to racial causes, others have examined unfair treatment without an explicit motivational component.[40–42] There is evidence suggesting that the magnitude of the association between unfair treatment and health outcomes may differ according to

whether such experiences are perceived as being motivated by racial factors versus nonracial causes.[43–46] Some studies have found that reports of general unfair treatment and stress have stronger associations with health indicators than reports of racial discrimination specifically.[44,46,47]

In summary, although there is some evidence that unfair treatment may have negative consequences for SLE progression, there are no studies to our knowledge that have explicitly examined this relationship. Furthermore, studies examining other health outcomes have found mixed evidence for the health consequences of unfair treatment in general versus racial discrimination. We examined whether self-reports of unfair treatment are associated with cumulative disease damage among African American women with SLE. Additionally, we explored potential differences between unfair treatment attributed to nonracial factors versus racial discrimination.

METHODS

Participants in this study were from the Georgians Organized Against Lupus (GOAL) cohort. Recruitment and data collection methods, as well as the sociodemographic characteristics of participants, have been previously described.[48] Briefly, GOAL encompasses a large sample of adult SLE patients from metropolitan Atlanta, Georgia, primarily recruited from the Georgia Lupus Registry, a population-based registry designed to more accurately estimate the incidence and prevalence of SLE in Atlanta.[49] The GOAL cohort was supplemented through recruitment from lupus clinics at Emory University and Grady Memorial Hospital, a large indigent care hospital in Atlanta, and from community rheumatologists. All participants had a validated diagnosis of SLE, fulfilling either 4 or more American College of Rheumatology (ACR) classification criteria for SLE or 3 ACR criteria with a final diagnosis of SLE by a board-certified rheumatologist.[49,50]

Flexible administration modes (self- or interviewer-administered) and delivery methods (mail, telephone, and in person) were available. Between August 2011 and July 2012, we collected data on 751 participants with a documented diagnosis of SLE. As previously described, 95% of respondents completed the questionnaire by mail; 5% were interviewed via telephone.[48] In this study, we focused on 578 African American women.

Measures

SLE disease damage. Cumulative SLE damage is defined as an irreversible change in an organ or system occurring since the onset of SLE and present for at least 6 months.[51] SLE-related disease damage can result from disease activity, drug toxicity, comorbidity, or a combination of these factors.[51–53] Cumulative SLE damage has been found to predict mortality and a wide range of other outcomes, such as physical function, health care utilization, and disability.[51,54–56] For studies on community-based cohorts when physician assessment is not feasible, SLE disease damage can be assessed with patient-reported instruments. The Brief Index of Lupus Damage (BILD) is a widely used, validated, patient-reported measure of major irreversible organ damage in 12 organ systems, including stroke, loss of extremity, malignancy, and premature gonadal failure.[57–59] The current study used the self-administered version (SA-BILD), which was recently validated among participants in the GOAL cohort. The SA-BILD consists of 28 items, each categorized as present or absent. In a previous study, the SA-BILD was demonstrated to be reliable and to have very good criterion validity compared with physician assessment of damage among GOAL participants, and SA-BILD scores showed significant associations in the expected directions with sociodemographic and disease outcomes.[60]

Unfair treatment and racial discrimination. We assessed unfair treatment using the 5-item version of the Everyday Discrimination Scale, which measures the following interpersonal forms of routine unfair treatment: being treated with less courtesy or respect, receiving poorer service, being perceived as less smart, being feared, and being threatened or harassed.[61] We scored the frequency of these experiences from 0 (never) to 5 (almost every day; $\alpha = 0.79$). We classified participants with an unfair treatment score of 1 to 5 (out of a possible 25) as reporting low levels of unfair treatment (i.e., they reported that each of the 5 types of unfair treatment occurred on average less than once a year). Participants with a score of 6 or greater were classified as reporting high levels of unfair treatment. The full 10-item version of the Everyday Discrimination Scale has been shown to be valid and reliable and is one of the most widely used measures of discrimination in epidemiological studies.[12,41,62,63]

The Everyday Discrimination Scale includes a follow-up item assessing a single primary attribution among those reporting any unfair treatment. Participants who reported experiencing any unfair treatment were asked to provide the main reason for these experiences. Response options were as follows: ancestry or national origin, gender, race, age, height, weight, some other aspect of participant's physical appearance, sexual orientation, and "other" (participant asked to specify). We classified participants who reported "race" or "ancestry or national origin" as making an attribution to racial discrimination; we classified all others as making a nonracial attribution.

To examine unfair treatment attributed to racial as well as nonracial causes, we constructed a 5-level categorical variable, as follows: 1 = no unfair treatment; 2 = low unfair treatment with a nonracial attribution; 3 = high unfair treatment with a nonracial attribution; 4 = low unfair treatment attributed to racial discrimination; 5 = high unfair treatment attributed to racial discrimination.

Covariates. We calculated years since diagnosis using self-reported month and year of diagnosis. Health-related covariates were as follows: insurance status, categorized as private versus public, other, or none; smoking status (never, former, or current), defined by Centers for Disease Control and Prevention criteria based on self-reported current smoking and lifetime smoking of at least 100 cigarettes[64]; body mass index (BMI; defined as weight in kilograms divided by the square of height in meters), based on self-reported weight and height; and exercise in the past month (yes vs no). We also adjusted for current SLE medication use of the following: steroids (e.g., prednisone, methylprednisolone) or antimalarials (e.g., hydroxychloroquine), which are drugs frequently used to treat a broad range of SLE manifestations, and other less commonly used immunosuppressive drugs (e.g., cyclophosphamide, azathioprine, mycophenolate mofetil, methotrexate, cyclosporine).

TABLE 1—Descriptive Characteristics of African American Women With Systemic Lupus Erythematosus (SLE): Georgians Organized Against Lupus (GOAL) Study, Atlanta, 2011–2012

Characteristic	No. (%)	SA-BILD Score, Mean (SD)
Self-reported unfair treatment*		
None	159 (27.6)	1.99 (2.00)
Any	418 (72.4)	2.47 (2.54)
Attribution for unfair treatment[a]		
Nonracial attribution, low	138 (24.5)	2.49 (2.25)
Nonracial attribution, high	120 (21.3)	2.63 (3.10)
Racial attribution, low	76 (13.5)	2.37 (2.58)
Racial attribution, high	70 (12.4)	2.37 (2.22)
Age,*** y		
< 35	131 (22.7)	1.76 (1.99)
35–49	211 (36.6)	1.92 (2.34)
50–64	193 (33.4)	2.98 (2.61)
≥ 65	42 (7.3)	3.19 (2.22)
Years since diagnosis***		
< 5	103 (18.1)	1.53 (1.97)
5–9	124 (21.8)	1.99 (2.15)
10–14	131 (23.0)	2.21 (2.53)
15–19	90 (15.8)	2.83 (2.33)
≥ 20	122 (21.4)	3.18 (2.69)
Marital status		
Married or living with a partner	163 (28.2)	2.50 (2.67)
Never married	223 (38.6)	2.04 (2.23)
Widowed, separated, or divorced	192 (33.2)	2.54 (2.36)
Household income, $		
< 10 000	204 (36.4)	2.59 (2.44)
10 000–19 999	103 (18.4)	2.44 (2.33)
20 000–29 999	71 (12.7)	2.34 (2.45)
≥ 30 000	183 (32.6)	2.01 (2.43)
Education, y		
≤ 12	225 (38.9)	2.46 (2.39)
13–15	194 (33.6)	2.43 (2.53)
≥ 16	159 (27.5)	2.04 (2.30)
Work status***		
Working	192 (33.3)	1.41 (1.58)
Retired, homemaker, or student	106 (18.4)	3.07 (2.62)
Unemployed	279 (48.4)	2.70 (2.62)
Health insurance		
Private	189 (33.9)	2.12 (2.50)
Public	368 (66.1)	2.50 (2.40)
Smoking status***		
Never	402 (70.2)	2.15 (2.41)
Former	95 (16.6)	3.19 (2.69)
Current	76 (13.3)	2.29 (1.84)

Continued

Sociodemographic variables were age, relationship status (married or living with a partner, never married, separated, widowed, divorced), education (≤ 12 years, 13–15 years, ≥ 16 years), and employment status (working; retired, homemaker, or student; unemployed). We measured annual household income in $10 000 increments (ranging from 1 ≤ $10 000 to 8 ≥ $70 000), examined continuously in multivariable analyses.

Analyses

Missing data. The highest number of missing data for any single variable was 21 (for insurance status), and the total number of participants with missing data for any variable under investigation was 79 (13.7% of participants). We generated 5 imputations for missing data, assuming an arbitrary missing data pattern. Multiple imputation is considered to be a principled technique for handling missing data by taking into account the uncertainty inherent in missing values.[65] We truncated imputed values to fit the bounds of possible values, and we did not round them for categorical variables.[66] Sensitivity analyses conducted on participants with complete data resulted in substantively similar conclusions.

Analysis plan. We conducted descriptive analyses to characterize the sample on primary study variables. We used the *t* test and analysis of variance (ANOVA) to examine bivariate associations between independent variables and SA-BILD score. We constructed categories of age, years since diagnosis, household income, and BMI to examine the functional form of these variables in bivariate analyses, but we examined them continuously in multivariable models. We used ordinary least squares regression analyses to examine SA-BILD score, first by unfair treatment (any vs none), then by level of unfair treatment (high and low vs none) and attribution (nonracial factors and racial discrimination vs none). Final models examined SA-BILD score by the combination of level and attribution. We conducted all analyses with SAS version 9.3 (SAS Institute, Cary, NC).

RESULTS

The average SLE damage score in our sample was 2.3 (SD = 2.4; possible range = 0–30),

TABLE 1—*Continued*

Body mass index, kg/m^2		
< 25.0	166 (28.9)	2.28 (2.15)
25.0–29.9	158 (27.5)	2.13 (2.39)
≥ 30	251 (43.7)	2.50 (2.59)
Exercise in past month		
No	266 (46.3)	2.47 (2.50)
Yes	308 (53.7)	2.19 (2.32)
Taking steroid or Plaquenil		
No	113 (19.6)	2.19 (2.31)
Yes	463 (80.4)	2.36 (2.44)
Other SLE medication		
No	381 (67.7)	2.25 (2.33)
Yes	182 (32.3)	2.46 (2.55)

Note. SA-BILD = self-administered version of the Brief Index of Lupus Damage. *P* values correspond to significant bivariate associations with SLE damage measured with the SA-BILD. The number of participants for categorical variables may not sum to 578 because of missing data. The sample size was n = 578.
[a]Whether participant attributed unfair treatment to racial factors or other factors, and whether unfair treatment was perceived as high-level or low-level.
*P < .05; **P < .01; ***P < .001.

and the mean years since initial diagnosis was 13.6 years (SD = 9.3). A total of 159 participants (27.6%) reported not experiencing any unfair treatment. Participants who reported any unfair treatment had significantly higher SA-BILD scores than those reporting no unfair treatment ($t = -2.2$; $P = .03$).

Among those reporting any unfair treatment, participants were approximately split in terms of reporting high versus low levels. An examination of level of unfair treatment found no significant difference between those reporting no, low, and high unfair treatment ($F_2 = 2.4$; $P = .09$). Of those who reported any unfair treatment, most did not make an attribution of racial discrimination. The most commonly reported nonracial response choice was "some other aspect of your physical appearance" (n = 57), followed by age (n = 21) and gender (n = 19). Less frequently endorsed were weight (n = 8), height (n = 5), and sexual orientation (n = 4). Among participants who selected "other" (n = 144), a survey of open-ended responses showed that most common were attributing unfair treatment to the characteristics of others (e.g., people's attitudes, ignorance), not knowing the reason for unfair treatment (e.g., unsure, don't know), and their own personality and behavior (e.g., because I'm opinionated, because of the way I act sometimes). Less common responses were work-related circumstances (e.g., mistreatment by colleagues or supervisors), relationship dynamics (e.g., by family members and romantic partners), everyday life (e.g., human nature, people having a bad day), disease and disability (e.g., having lupus), and religion.

Because this study sought to examine attributions specifically to racial discrimination, participants who did not make an attribution to race or to ancestry–national origin were considered to make a primary nonracial attribution. An examination of attribution found no significant difference in SA-BILD score between those reporting no unfair treatment, nonracial unfair treatment, and racial discrimination ($F_2 = 2.5$; $P = .07$). Analyses examining the combination of level of unfair treatment and attribution also showed no significant differences in SA-BILD score ($F_4 = 1.4$; $P = .24$).

The distributions of additional participant characteristics and associations with SLE damage are shown in Table 1. Age ($F_3 = 11.5$; $P < .001$) and years since diagnosis ($F_4 = 8.6$; $P < .001$) were significantly associated with SLE damage, with increasing age and years since diagnosis being associated with greater SA-BILD score. We found additional significant bivariate relationships with work status ($F_2 = 23.7$; $P < .001$) and smoking status ($F_2 = 7.3$; $P < .001$), with those currently working and having never smoked having the lowest SLE damage scores, respectively.

Results from multivariable regression analyses are presented in Table 2. When we controlled for age and years since diagnosis (model 1), those reporting any unfair treatment had significantly higher SA-BILD scores than those reporting no unfair treatment (b = 0.50; 95% confidence interval [CI] = 0.08, 0.92); these scores remained significantly higher after we controlled for sociodemographic (model 2) and health characteristics (model 3). An examination of SA-BILD by level of unfair treatment showed that reporting low (b = 0.58; 95% CI = 0.12, 1.04) as well as high (b = 0.53, 95% CI = 0.05, 1.00) levels was associated with significantly higher SA-BILD scores compared with reporting no unfair treatment.

When we controlled for age and years since diagnosis, those who reported unfair treatment attributed to nonracial factors had significantly higher SA-BILD scores than those reporting no unfair treatment (b = 0.62; 95% CI = 0.17, 1.08); however, those who attributed unfair treatment to racial discrimination did not (b = 0.28; 95% CI = −0.24, 0.79). Further adjustment for demographic and socioeconomic factors resulted in an increase in the effect size of making attributions to racial discrimination, but it remained nonsignificant. Further controlling for health-related factors did not appreciably change effect estimates.

Final models examined the combination of level of unfair treatment and attribution (Table 3). As in previous models, reporting unfair treatment (both high and low levels) attributed to nonracial causes was associated with significantly higher SA-BILD scores than reporting no unfair treatment. By contrast, reports of unfair treatment attributed to racial discrimination were not significantly associated with SA-BILD scores for either low or high levels. This was the case in all models: first adjusting for age and years since diagnosis (model 1), then for marital status and socioeconomic variables (model 2), and lastly for insurance status and other health variables (model 3). Although the magnitude of the association between low and high levels of unfair treatment attributed

TABLE 2—Multivariable Regression Analyses of Disease Damage Among African American Women With Systemic Lupus Erythematosus (SLE): Georgians Organized Against Lupus (GOAL) Study, Atlanta, 2011–2012

Variable	Model 1, b (95% CI)[a]	Model 2, b (95% CI)[b]	Model 3, b (95% CI)[c]
Any self-reported unfair treatment vs none	0.50* (0.08, 0.92)	0.59** (0.17, 1.00)	0.55** (0.14, 0.97)
Unfair treatment			
Low level	0.47 (–0.01, 0.94)	0.59* (0.13, 1.05)	0.58* (0.12, 1.04)
High level	0.55* (0.06, 1.03)	0.59* (0.12, 1.06)	0.53* (0.05, 1.00)
No unfair treatment (Ref)	0.00	0.00	0.00
Attribution			
Nonracial attribution	0.62** (0.17, 1.08)	0.65** (0.21, 1.10)	0.62** (0.17, 1.06)
Racial attribution	0.28 (–0.24, 0.79)	0.45 (–0.05, 0.96)	0.44 (–0.07, 0.94)
No unfair treatment (Ref)	0.00	0.00	0.00

Note. CI = confidence interval. Independent variables are examined in separate models. The sample size was n = 578.
[a]Controlling for age and years since diagnosis.
[b]Model 1 + marital status, household income, education, and work status.
[c]Model 2 + health insurance, smoking status, body mass index, exercise, steroid or Plaquenil use, and other SLE medication.
*$P < .05$; **$P < .01$.

to racial discrimination increased with the adjustment of covariates, they remained nonsignificant.

Posthoc analyses showed that effect estimates for unfair treatment attributed to racial discrimination appreciably increased only after adjustment for socioeconomic variables. We found that participants attributing unfair treatment to racial discrimination had higher household income (mean = 3.38; SD = 2.36) than those reporting no unfair treatment (mean = 2.96; SD = 2.22) and those making nonracial attributions (mean = 2.84; SD = 2.18), albeit not significantly ($F_2 = 2.7$; $P = .07$).

We also examined whether associations between unfair treatment and SA-BILD score varied by household income, given prior research suggesting that greater socioeconomic status (SES) may be associated with greater exposure to and reports of unfair treatment, and may also moderate associations with health outcomes.[34,67–69] Although there was no significant association between household income and unfair treatment ($F_4 = 1.80$; $P = .13$), those reporting low levels of unfair treatment had higher average household income than those reporting high levels of unfair treatment, for both those making nonracial attributions (mean = 3.00 vs 2.65) and those making attributions to racial discrimination (mean = 3.47 vs 3.28). Other studies have also found that those of lower SES report higher levels of discrimination.[42,70] The interaction between household income and unfair treatment in predicting SA-BILD score was not significant ($F_4 = 1.4$; $P = .24$). In short, we did not find evidence that the associations we found between unfair treatment and SLE damage varied by household income.

DISCUSSION

Our study highlights the role of social stressors in contributing to the progression of SLE, and it is the first to report that unfair treatment is associated with greater disease damage among African American women. Consistent with findings from previous studies, our results suggest that unfair treatment is a risk factor for worse health outcomes. As a source of psychosocial stress, unfair treatment may trigger inflammatory responses through biobehavioral channels that have been associated with heightened SLE activity.[23–25] We found that the association between unfair treatment and SLE damage was pronounced among those who attributed such experiences primarily to nonracial factors. Accordingly, it is possible that individuals may be less able to cope with or receive social support for negative experiences that are viewed to be the result of nonracial causes, which may be more likely to be perceived as deserved.[34,71,72]

Participants attributing unfair treatment to racial discrimination also had greater SLE damage than those reporting no unfair treatment; however, the associations were consistently lower in magnitude than for those attributing unfair treatment to nonracial factors, and they were not significantly different from those reporting no unfair treatment. This observation resonates with findings from other studies, which have reported that the effect of unfair treatment may differ according to whether it is perceived as being motivated by race or by other factors.[43,73] Prior research has suggested that the relationship between discrimination and health outcomes among African Americans is contingent on other relevant psychosocial factors, such as appraisal, coping, and psychological responses, as well as attributions.[18,74–76] For example, a study found that among African American women, high levels of major lifetime discrimination attributed to nonracial factors were associated with greater visceral and subcutaneous fat prior to adjustment for BMI; however, these associations were not found for major lifetime discrimination attributed to race.[46] We found that part of the reason for this observation may be higher levels of income and other socioeconomic indicators among those making attributions to racial discrimination. When we controlled only for age and years since diagnosis, effect estimates for attributions of unfair treatment based on racial discrimination were considerably lower than estimates for nonracial attributions, and they increased noticeably only after the inclusion of socioeconomic variables. Other studies, however, have found no effect of attribution, or that attributing unfair treatment to racial discrimination may in fact have more detrimental health consequences.[45,77]

Despite inconsistent findings reported in the literature, our results are consonant with some theories of racial identity and minority stress.[78,79] Because of the motivational ambiguity that characterizes contemporary forms of racial discrimination, attributing unfair treatment to race in lieu of other sources, including possible personal deficiencies, may

TABLE 3—Final Multivariable Regression Analyses of Disease Damage Among African American Women With Systemic Lupus Erythematosus (SLE) by Combination of Unfair Treatment and Attribution: Georgians Organized Against Lupus (GOAL) Study, 2011–2012

Variable	Model 1, b (95% CI)[a]	Model 2, b (95% CI)[b]	Model 3, b (95% CI)[c]
Intercept	0.06 (-0.72, 0.83)	0.07 (-1.02, 1.16)	-0.44 (-1.91, 1.04)
Attribution for unfair treatment[d]			
Nonracial attribution, low	0.56* (0.03, 1.08)	0.66* (0.15, 1.17)	0.64* (0.13, 1.15)
Nonracial attribution, high	0.65* (0.11, 1.19)	0.61* (0.08, 1.14)	0.57* (0.03, 1.10)
Racial attribution, low	0.22 (-0.40, 0.85)	0.39 (-0.22, 1.00)	0.43 (-0.18, 1.04)
Racial attribution, high	0.28 (-0.37, 0.92)	0.49 (-0.14, 1.12)	0.42 (-0.22, 1.05)
No unfair treatment (Ref)	0.00	0.00	0.00
Age	0.03*** (0.01, 0.04)	0.02* (0.01, 0.04)	0.02* (0.00, 0.04)
Years since diagnosis	0.05*** (0.02, 0.07)	0.05*** (0.03, 0.07)	0.05*** (0.03, 0.07)
Marital status			
Never married		-0.45 (-0.98, 0.08)	-0.41 (-0.94, 0.13)
Widowed, separated, or divorced		-0.51 (-1.03, 0.01)	-0.46 (-0.98, 0.06)
Married (Ref)		0.00	0.00
Household income		-0.14* (-0.25, -0.02)	-0.17** (-0.29, -0.04)
Education, y			
≤ 12 (Ref)		0.00	0.00
13–15		0.16 (-0.28, 0.61)	0.13 (-0.32, 0.58)
≥ 16		0.21 (-0.32, 0.75)	0.24 (-0.30, 0.78)
Work status			
Retired, homemaker, or student		1.19*** (0.61, 1.77)	1.17*** (0.58, 1.76)
Unemployed		1.04*** (0.57, 1.52)	1.10 *** (0.60, 1.60)
Working (Ref)		0.00	0.00
Public vs private health insurance			-0.33 (-0.85, 0.19)
Smoking status			
Former			0.38 (-0.16, 0.91)
Current			-0.05 (-0.63, 0.52)
Never (Ref)			0.00
Body mass index			0.02 (-0.01, 0.04)
Any exercise: yes vs no			-0.13 (-0.51, 0.25)
Steroid or Plaquenil: yes vs no			0.36 (-0.13, 0.85)
Other SLE medication: yes vs no			0.18 (-0.23, 0.59)

Note. CI = confidence interval. The sample size was n = 578.
[a]Controlling for age and years since diagnosis.
[b]Model 1 + marital status, household income, education, and work status.
[c]Model 2 + health insurance, smoking status, body mass index, exercise, steroid or Plaquenil use, and other SLE medication.
[d]Whether participant attributed unfair treatment to racial factors or other factors, and whether unfair treatment was perceived as high-level or low-level.
*$P < .05$; **$P < .01$; ***$P < .001$.

have self-protective properties (e.g., through the preservation of self-esteem).[80,81] Researchers have hypothesized that attributing negative events to external factors, such as racism, may have less detrimental health consequences.[82] Attributions for unfair treatment may be particularly relevant given the subtleties of present racial discrimination, in contrast to traditional forms of racism that have tended to be more clearly racially motivated.[83] Studies have consistently documented the covert ways in which racial discrimination operates across various settings, including in housing, health care, and the criminal justice system.[84–87] In light of the persistent systemic social disadvantage experienced by African Americans, being able to recognize such experiences as instances of racial discrimination may be adaptive and a truer assessment of the reason for unfair treatment. Along these lines, attributing unfair treatment to racial discrimination may be somewhat less deleterious for health. Our study suggests that future research may further examine possible differential associations between unfair treatment and health outcomes by attribution.

It should be noted that analyses did not show evidence of a dose–response relationship, and those reporting low and high levels of unfair treatment had similarly higher SLE damage scores than those reporting no unfair treatment. A possible reason for this observation is that the SA-BILD, which measures irreversible organ or system damage, is somewhat narrow in range and relatively stable.[88,89] An SA-BILD score of 0 was the most commonly reported (n = 135, 23.4%), followed by a score of 1 (n = 131, 22.7%); more than 75% of participants had scores of 3 or less. Furthermore, the physiological impact of psychosocial stressors may require more time to accumulate and appear as SLE damage. Hence, the effect of high unfair treatment may not necessarily be differentiated from lower levels for this particular outcome.

Our results support those of previous studies that have found that lower SES is associated with SLE severity and mortality.[90,91] For example, the geographic distribution of clusters with higher SLE mortality rates has been found to be concentrated in areas with greater poverty and numbers of racial minorities.[92,93] In this study, we found that participants with lower household income had higher SLE damage scores. Greater socioeconomic deprivation experienced by African American women may also contribute to racial disparities in SLE damage and accelerated disease progression.

A strength of our study is the large population-based sample of African American women with SLE. Research on SLE has typically relied on convenience samples, often from university centers that attract and treat an unrepresentative subset of patients.[94] The relatively low prevalence of SLE, coupled with the

fact that there is no single test or clinical feature serving as a marker for SLE, has been an additional obstacle to participant recruitment. Although our findings are less generalizable to those outside the Atlanta metropolitan area, the GOAL cohort represents a significant enhancement in SLE research.

A limitation of our study is its cross-sectional and correlational nature, which presents challenges to establishing the temporal sequence of events and making causal inferences. For example, it is possible that participants with greater disease severity were more likely to perceive unfair treatment. Another limitation is that data on social desirability bias are not available, which may affect reports of the extent of unfair treatment as well as whether such experiences are attributed to racial or nonracial causes. Our reliance on self-reports of SLE damage and other health indicators, and the absence of biological markers of disease severity or progression, also present limitations to our study. For example, the lack of association between medication use variables and the SA-BILD might suggest that this measure is not valid. This lack of association, however, is not necessarily counterintuitive, as medication use is not a direct indicator of disease severity (e.g., the use of medications may deter disease progression). Furthermore, the BILD is a widely used and validated patient-reported measure of SLE damage[57–59]; the self-administered version used in the current study has also been validated in the GOAL cohort.[60]

An additional caveat to our findings is that we could not assess the impact of making multiple attributions for unfair treatment. Given that the Everyday Discrimination Scale assesses a single main attribution for unfair treatment, we could not examine differences among those perceiving a combination of reasons for unfair treatment, including both racial and nonracial factors. We also did not distinguish between nonracial attributions in this study—for example, attributions to one's personality or behavior versus attributions to other external or systemic factors. Nevertheless, we believe that there is value in examining the effect of making a primary attribution to racial discrimination as the reason for unfair treatment given its social significance.

Despite these limitations, our study advances scientific knowledge on the social determinants of SLE outcomes among African American women. Social stressors salient in the lives of African American women may contribute to SLE severity and progression. We provide a more nuanced perspective on how persistent, routine forms of unfair treatment that are more commonly experienced by African Americans than by other racial groups may adversely affect disease outcomes and contribute to racial disparities in SLE. ■

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Contributors

D. H. Chae conceptualized the study, conducted data analyses, and held primary responsibility for data interpretation and writing. C. M. Drenkard and S. S. Lim collected the data. All authors contributed to the interpretation of findings and article development.

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Human Participant Protection

This study was approved by the Emory University institutional review board, the Grady Health System Research Oversight Committee, and the Georgia Department of Public Health institutional review board.

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