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Trajectory of Damage Accrual in Systemic Lupus Erythematosus Based on Ethnicity and Socioeconomic Factors

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ABSTRACT. ***Objective.*** The Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index (SDI) is associated with increased healthcare costs and mortality. We compared the trajectory of total and individual damage items of the SDI in African American vs White ethnicities in a large prospective systemic lupus erythematosus (SLE) cohort. We also estimated the association between ethnicity and individual damage items after adjusting for several socioeconomic factors.

Methods. Poisson regression was used to calculate the rate of damage per year for each organ. Cox regression modeling was used to determine the association between time to the individual damage item and ethnicity.

Results. We included 2436 patients: 42.9% African American, 57.1% White, and 92% female. There was a linear relationship between time since diagnosis and mean SDI score, with no plateau. Compared to White patients, African American patients had a faster total, renal, pulmonary, and skin damage accrual rate even after adjustment for differences in socioeconomic variables.

Conclusion. The linear increase in damage in both ethnicities over time is of particular concern. African American patients accrued more damage at a faster rate compared to White patients. For a few organs, higher rates of damage in African American patients was partially explained by socioeconomic differences, whereas for most organs, the difference persisted after adjustment for these factors.

Key Indexing Terms: hypertension, outcomes, systemic lupus erythematosus

In systemic lupus erythematosus (SLE), a higher Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index (SDI) is associated with increased mortality,[1-4] poorer health-related quality of life (HRQOL),[5] and increased healthcare costs.[6] Looking at 263 patients from the Toronto Lupus Clinic inception cohort, organ damage acquired within the first year of diagnosis increased 10-year mortality by 3- to 4-fold.[1] In a multicenter study evaluating the economics of damage accrual in 1687 patients who were part of the Systemic Lupus International Collaborating Clinics (SLICC) inception cohort, an SDI ≥ 5 was found to accrue annual costs 12-fold higher than those with an SDI of 0.[6] Mok et al examined the effect of damage on HRQOL in patients with SLE.[5] Compared with age- and gender-matched controls, patients with SLE had lower 36-item Short Form Health Survey scores, which inversely correlated with SDI scores.[5]

Many studies have determined ethnicity as a predictor of higher SDI. In the Hopkins Lupus Cohort, higher rates of damage were found in African American compared to White patients.[7] In the Carolina Lupus cohort, African American patients were more likely than White patients to experience damage in almost all subitems of the SDI.[8] In the LUMINA (Lupus in Minorities: Nature Versus Nurture) cohort, Hispanics had higher SDI scores compared to White and African American patients.[9,10] In a retrospective analysis of medical records and SDI scores of 300 patients in a multiethnic British cohort, African Caribbean ethnicity was associated with increased damage.[2] Different ethnicities have different damage patterns.[8,10-12] Renal and skin damage were more pronounced in African American[10] and African Canadian[8] patients than in White patients. Mortality studies showed that mortality is higher in Black patients.[13,14] Data from the Georgia Lupus Registry suggest that Black women die earlier than their White counterparts, by an average of 13 years.[14]

Only a limited number of studies have evaluated the trajectory of damage accrual in patients with SLE in different ethnicities. In a retrospective study of 300 patients with SLE at the University College Hospital London, the divergence in the damage curves started early in the disease in African Caribbean compared to White and Asian patients with SLE.[2] In addition, a single-center, longitudinal, inception cohort study looking retrospectively at patients with juvenile SLE diagnosed at the pediatric lupus clinic at SickKids Hospital (Toronto, Canada), also determined Afro-Caribbean ethnicity as a predictor of higher

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initial and persistently greater damage accrual compared to Asian and White patients.[15]

Discrepancies between different ethnicities may be because of the role of socioeconomic measures. Few studies have evaluated the effect of individual measures of socioeconomic status (SES), such as educational level and poverty, on damage accrual in patients with SLE.[16-18] A previous study by Falasinnu et al, specifically looking at SLE-related mortality in the Eight Americas, reported that race played a more important role than socioeconomic and geographical variables.[19] To our knowledge, there was no study in the literature that looked at 3 measures of SES simultaneously in 1 study population.

The aim of our study was to calculate and compare the trajectory of both total and individual organ damage accrual in the largest longitudinal prospective SLE cohort in which African American and White ethnicities were well represented. We evaluated the role of ethnicity in damage accrual and the degree to which this was explained by socioeconomic factors.

METHODS

The Hopkins Lupus Cohort is a prospective longitudinal single-center prevalent cohort of patients with SLE in Baltimore, Maryland, and surrounding areas. The cohort was established in 1987 and has been approved by the Johns Hopkins University School of Medicine Institutional Review Board on a yearly basis. All patients gave written informed consent to participate. This analysis was based on cohort data from its inception until January 2020.

Patients were followed up by protocol quarterly, or more often as clinically indicated. Information about SLE features since diagnosis was gathered at cohort entry by obtaining a thorough historical and medical record review of the patient. This information was updated at each cohort visit. Ethnicity and the 3 socioeconomic measures were self-reported by the patient upon initial cohort entry by means of a questionnaire. Ethnicity was recorded as African American or White. Education was reported as ≤ 12 years or >12 years. Household income was reported as < $30,000, ≥ $30,000 to < $65,000, or ≥ $65,000. Health insurance was reported as none, medical assistance, or private.

The SDI is a validated tool that was developed to measure damage, defined as irreversible organ dysfunction, present for ≥ 6 months regardless of etiology. The index evaluates 12 organ systems (ocular, neuropsychiatric, renal, pulmonary, cardiovascular [CV], peripheral vascular, gastrointestinal, musculoskeletal [MSK], skin, premature ovarian failure, diabetes, and malignancies), with 1 to 5 items per system.[20,21] The SDI was calculated based on medical record review at cohort entry and updated at every cohort clinic visit. In our analysis, the SDI was calculated based on organ damage that occurred after diagnosis with SLE until the last visit.[20,21] Patients whose damage occurred prior to the date of SLE diagnosis were excluded.

We plotted the mean damage score for each organ by year since SLE diagnosis, stratified by ethnicity. We also plotted the percent of patients with any damage in each organ by year since SLE diagnosis and by ethnicity. To compare African American patients to White patients with respect to damage rates, Poisson regression was used, allowing overdispersion for each organ system with an offset term accounting for their time followed since diagnosis. The outcome variable was the damage score of each organ system at the last available cohort visit. The associations were adjusted for sex, age, income, education, and health insurance.

We used Cox regression models approach to determine the association between time to individual damage item and ethnicity. These associations were also adjusted for sex, age, income, education, and health insurance. Associations between race and rates of damage were quantified using rate ratios (RR).

All analyses were performed using SAS software, version 9.4 (SAS Institute).

RESULTS

A total of 2674 patients with SLE classified according to the SLICC classification criteria[22] or the revised American College of Rheumatology (ACR) classification criteria[23,24] were in the cohort. Of these patients, 1049 (39.2%) were African American, 1399 (52.3%) were White, and 226 (8.5%) were of other ethnicities. In this analysis, we included only African American and White patients. We excluded 226 patients of other ethnicities and 12 patients with a missing diagnosis date.

We included 2436 patients: 1045 (42.9%) African American, 1391 (57.1%) White, and 2242 (92%) female (Table 1). The mean age at SLE diagnosis was 32.7 years (SD 13.0). Forty-nine percent of these patients were classified with SLE at an age < 30 years, and 33.6% were diagnosed at between age ≥ 30 and < 45 years. The cumulative classification criteria were 48% malar rash, 19% discoid rash, 52% photosensitivity, 53% oral ulcer, 72% arthritis, 49% serositis, 45% renal disorder, 12% neurological disorder, 67% hematological disorder, 79% immunological disorder, and 97% antinuclear antibody (ANA) positivity, based on revised ACR classification criteria. Additional SLICC classification criteria included 21% direct Coombs test, 53% low C3, 47% low C4, and 16% low CH50. The mean follow-up time from diagnosis to the last clinic visit was 13.4 years (SD 10.0).

Table 1 shows a comparison of African American and White patients with respect to demographic characteristics of the study population. They were similar in age at diagnosis, follow-up time since diagnosis, and sex. Compared to White patients, African American patients were less likely to have > 12 years of education (59.2% in African American vs 71.3%

Table 1. Characteristics of the patients included in the study.

	All, n = 2436	African American, n = 1045	White, n = 1391
Age at SLE diagnosis, yrs, mean (SD)	32.7 (13.0)	31.3 (12.1)	33.6 (13.5)
Follow-up time since SLE diagnosis, yrs, mean (SD)	13.4 (10.0)	13.6 (10.2)	13.3 (9.8)
Sex, n (%)			
Female	2242 (92)	976 (93.4)	1266 (91)
Male	194 (8)	69 (6.6)	125 (9)
Education, n (%)			
≤ 12 yrs	787 (34)	406 (40.8)	381 (28.7)
> 12 yrs	1532 (66)	588 (59.2)	944 (71.3)
Family income, n (%)			
< $30,000	706 (33)	456 (49.2)	250 (20.3)
$30,000 to < $65,000	709 (33)	268 (28.9)	441 (35.8)
≥ $65,000	743 (34)	202 (21.8)	541 (43.9)
Insurance, n (%)			
None	57 (2)	40 (4)	17 (1.3)
Medical assistance	451 (19)	306 (30.2)	145 (10.9)
Private	1835 (78)	667 (65.8)	1168 (87.8)

Values expressed as n (%) unless stated otherwise. SLE: systemic lupus erythematosus.

in White patients), a combined family income of ≥ $65,000 (21.8% in African American vs 43.9% in White patients), and private health insurance (65.8% in African American vs 87.8% in White patients).

Figure 1 shows the mean total SDI by time since SLE diagnosis, stratified by ethnicity. An approximately linear relationship between time since diagnosis and mean SDI, with no plateau, was found. The divergence of the curve started early in the disease course.

Table 2 shows the adjusted and unadjusted associations between cumulative and individual organ system damage accrual and ethnicity. Values were adjusted for sex, age at diagnosis, years of education, health insurance, and household income. The rate of cumulative damage accrual was significantly higher in African American compared to White patients after adjustment for socioeconomic measures (RR 1.14, 95% CI 1.03-1.27). Renal (RR 1.68, 95% CI 1.30-2.16), pulmonary (RR 1.38, 95% CI 1.09-1.73), and skin (RR 2.98, 95% CI 1.83-4.85) damage in African American patients accumulated at a significantly higher rate compared to White patients after adjustments. MSK and CV damage accumulation rate was higher in African American compared to White patients in the univariate analysis but lost its significance after adjustment for sex, age at diagnosis, years of education, health insurance, and household income.

Table 3 examines the association between race and individual damage items in each organ system represented in the SDI using a time-to-event approach. African American patients had a higher risk of renal insufficiency, proteinuria (3.5 g/24 h), endstage renal disease (ESRD), pulmonary hypertension, pulmonary fibrosis, cardiomyopathy, pericarditis, deforming or erosive arthritis, avascular necrosis, and scarring chronic alopecia, than White patients, at any time during follow-up. On the other hand, White patients were at higher risk for osteoporosis with fracture or vertebral collapse, bowel infarction, venous thrombosis, and malignancy. African American patients had a 40% higher risk of having renal insufficiency defined as an eGFR of < 50% (RR 1.44, 95% CI 1.16-1.79). In addition, African American patients were more than twice as likely to have nephrotic proteinuria (RR 2.14, 95% CI 1.47-3.12) and ESRD (RR 2.09, 95% CI 1.34-3.28) compared to White patients. African American patients had almost twice the risk of White patients for pulmonary hypertension (RR 1.81, 95% CI 1.30-2.52). The likelihood of cardiomyopathy (RR 2.52, 95% CI 1.48-4.29) and chronic pericarditis (> 6 months; RR 2.48, 95% CI 1.08-5.71) to develop

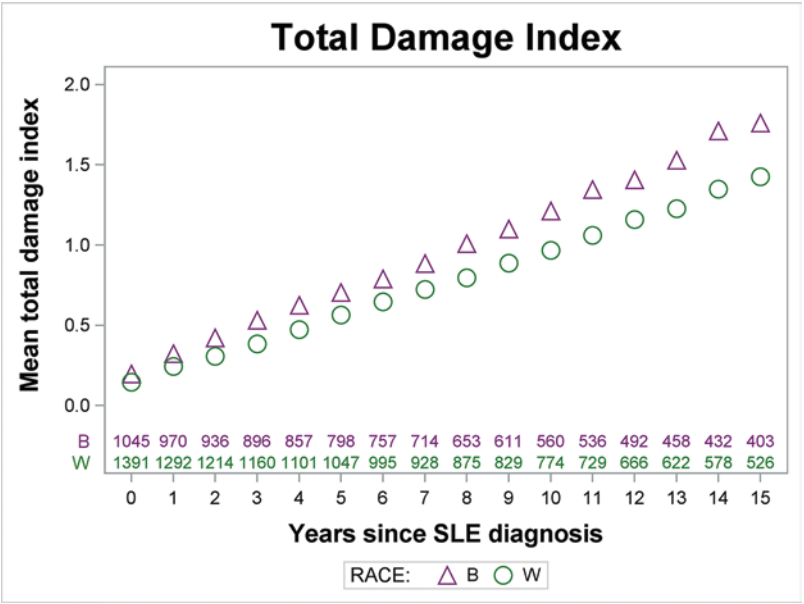

Figure 1. Mean total damage index by years since diagnosis, stratified by ethnicity. B: Black; SLE: systemic lupus erythematosus; W: White.

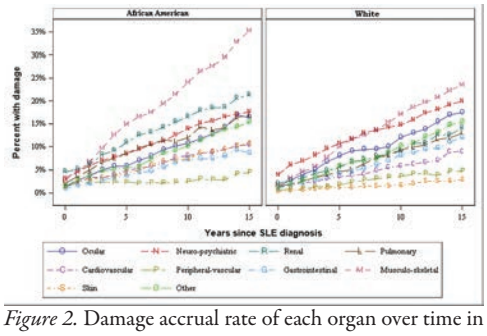

Figure 2. Damage accrual rate of each organ over time in African American and White patients.

in African American patients was approximately 3 times that of White patients. With MSK damage, African American patients had double the risk of having erosive arthritis (RR 2.02, 95% CI 1.48-2.75) and avascular necrosis (RR 1.84, 95% CI 1.34-2.52), while White patients had double the risk of having osteoporosis with fracture (RR 0.46, 95% CI 0.35-0.60). Scarring alopecia was 6.3 times higher (RR 6.25, 95% CI 3.16-12.36) in African American compared to White patients.

Figure 2 shows the linear trends in specific organ systems. Mean pulmonary, renal, MSK, and skin damage scores were higher in African American patients compared to White patients,

Table 2. Adjusted and unadjusted rates of damage accrual in total and individual organs after SLE diagnosis by ethnicity.

Type of Damage	Rate of SDI, per yr		RR (95% CI)	*P*	Adjusted RR[a] (95% CI)	*P*
	African American	White				
Total damage	0.16	0.12	1.30 (1.18-1.43)	< 0.001	1.14 (1.03-1.27)	0.01
Ocular	0.015	0.015	0.99 (0.83-1.19)	0.95	1.06 (0.88-1.28)	0.55
Neuropsychiatric	0.017	0.018	0.96 (0.78-1.18)	0.69	0.82 (0.65-1.03)	0.09
Renal	0.03	0.016	1.99 (1.58-2.49)	< 0.001	1.68 (1.30-2.16)	< 0.001
Pulmonary	0.016	0.011	1.47 (1.18-1.82)	< 0.001	1.38 (1.09-1.73)	0.01
Cardiovascular	0.012	0.009	1.32 (1.02-1.71)	0.04	1.26 (0.95-1.68)	0.10
Peripheral-vascular	0.004	0.004	1.04 (0.72-1.52)	0.82	0.75 (0.49-1.16)	0.20
Gastrointestinal	0.007	0.009	0.82 (0.59-1.14)	0.24	0.76 (0.52-1.10)	0.15
Musculoskeletal	0.03	0.02	1.31 (1.14-1.51)	< 0.001	1.11 (0.94-1.30)	0.21
Skin	0.007	0.002	4.15 (2.63-6.55)	< 0.001	2.98 (1.83-4.85)	< 0.001
Other	0.013	0.012	1.10 (0.90-1.35)	0.34	0.97 (0.79-1.20)	0.81

[a] Values adjusted for sex, age at diagnosis, years of education, insurance, and combined family income. RR: rate ratio; SDI: Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index; SLE: systemic lupus erythematosus.

at any point after SLE diagnosis. A small difference between the 2 ethnicities was also noted in CV damage. By 5 years after SLE diagnosis, 11% of African American patients compared to 6% of White patients had renal damage. By 15 years after SLE diagnosis, compared to White patients, African American patients had more renal damage (21% vs 14%), pulmonary damage (17% vs 14%), skin damage (11% vs 3%), and MSK damage (35% vs 25%).

DISCUSSION

Our study, the largest ever done to our knowledge, confirmed that cumulative damage accrual in SLE is faster in African American compared to White patients. The organ systems mostly affected by ethnicity are pulmonary, MSK, renal, and skin. The damage in these organ systems is not only more likely in African American patients but also occurs at a faster rate compared to in White patients. These differences persisted after adjusting for socioeconomic factors except for MSK damage.

First, a considerable percentage of patients acquired damage by 5 years of diagnosis and continued to do so over the course of their disease. In our cohort, 40% of patients had accrued damage by 5 years with no evidence of a plateau effect. This is in agreement with other studies that showed that approximately 50% of patients with SLE will accrue damage by 5 years of the disease regardless of their ethnicities and geographic backgrounds.[10,25] The absence of plateau effect in damage accrual in patients with SLE was also demonstrated in the SLICC inception cohort.[13] These findings highlight the importance of continuous follow-up and aggressive management of SLE, considering there was no flattening of the damage curve over the disease course.

Second, ethnicity was an important determinant of severity and rate of damage accrual. From the outset of the disease and thereafter throughout the course of the disease, African American patients accumulated more damage, and earlier on, at a faster rate compared to White patients. In the Canadian juvenile SLE cohort, African Caribbean patients accrued earlier and faster damage compared to Asian and White patients.[15] In a large international SLE inception cohort, US patients of African ancestry and Hispanic patients in Mexico had a higher risk of progressing from baseline damage to higher damage compared to White patients in Europe or Canada.[13] In contrast, Asians had lower transition rates compared to White patients in Europe or Canada.[13] There are a number of possible explanations for these findings, including differences in clinical phenotype, response to therapy, comorbidities, socioeconomic factors, differences in corticosteroid use, and access to healthcare among different ethnic groups. In our study, increased rates of overall and organ-specific damage persisted after adjustment for demographics and measures of SES. We did not adjust for corticosteroid use in comparing ethnic groups because we viewed that to be in the causal pathway from disease manifestations to damage.

Third, ethnicity plays a role in determining organ-specific damage accrual rate and pattern. Our study, along with others, demonstrated that renal[10,26] and skin damage[8,10] occurred more frequently among patients with SLE of African descent compared to Asians or White patients. Another new finding of our study is that African American patients accrued faster lung and MSK damage. Studies of other ethnicities also have demonstrated different patterns in organ damage. In the LUMINA multiethnic cohort, neuropsychiatric problems accounted for the greatest proportion of organ system damage in Hispanics and White patients.[10] Hispanics were more likely than African American and White patients to exhibit cataracts, muscle atrophy, and cognitive impairment.[10] In a Chinese cohort, the main contributors to organ damage were renal insufficiency, osteonecrosis, and gonadal failure.[25] In a cross-sectional study from Malaysia, patients of Indian origins predominantly developed diabetes while Malay patients developed osteoporosis.[12] The majority of patients who developed malignancy were Chinese.[12]

Fourth, the difference in damage accumulation between African American and White patients could not be explained by socioeconomic disparities. Despite contributing to organ damage in SLE,[7,8,10,27] SES could not explain disparities in organ damage and mortality in patients with SLE with different ethnicities in some past studies.[10,28,29] A study conducted at the

Table 3. Associations between time to SDI items and ethnicity (African American vs White).

SDI Items	No. of Events	African American vs White RR[a] (95% CI)	*P*[*]
Any cataract ever	414	1.07 (0.87-1.31)	0.51
Retinal change or optic atrophy	101	0.91 (0.59-1.42)	0.95
Cognitive impairment or major psychosis	130	0.83 (0.55-1.23)	0.35
Seizures requiring therapy for 6 months	57	0.58 (0.32-1.04)	0.07
Cerebral vascular accident ever or resection	180	0.84 (0.60-1.17)	0.30
Cranial or peripheral neuropathy	183	0.80 (0.57-1.12)	0.19
Transverse myelitis	15	0.79 (0.26-2.42)	0.68
Estimated or measured GFR < 50%	416	1.44 (1.16-1.79)	< 0.001
Proteinuria 3.5 g/24 h	156	2.14 (1.47-3.12)	< 0.001
Endstage renal disease	112	2.09 (1.34-3.28)	0.001
Pulmonary hypertension	184	1.81 (1.30-2.52)	< 0.001
Pulmonary fibrosis	173	1.40 (1.00-1.96)	0.05
Shrinking lung	9	0.82 (0.18-3.65)	0.79
Pleural fibrosis	64	1.36 (0.78-2.36)	0.28
Pulmonary infarction or resection	8	0.20 (0.02-1.77)	0.15
Angina or coronary artery bypass	84	0.66 (0.39-1.11)	0.12
Myocardial infarction ever	97	1.03 (0.66-1.62)	0.89
Cardiomyopathy	79	2.52 (1.48-4.29)	< 0.001
Valvular disease	58	1.00 (0.56-1.80)	0.999
Pericarditis > 6 months, or pericardiectomy	29	2.48 (1.08-5.71)	0.03
Claudication × 6 months	36	1.10 (0.51-2.39)	0.81
Minor tissue loss (pulp space)	14	1.73 (0.51-5.86)	0.38
Significant tissue loss ever	20	1.04 (0.39-2.76)	0.94
Venous thrombosis with swelling, ulceration, or venous stasis	66	0.48 (0.26-0.87)	0.02
Infarction or resection of bowel	204	0.64 (0.46-0.88)	0.007
Mesenteric insufficiency	8	0.92 (0.17-4.94)	0.93
Chronic peritonitis	7	1.34 (0.27-6.66)	0.72
Stricture or upper GI tract surgery ever	16	2.89 (0.89-9.35)	0.076
Pancreatitis	9	0.23 (0.04-1.21)	0.08
Muscle atrophy or weakness	46	0.89 (0.46-1.73)	0.74
Deforming or erosive arthritis	219	2.02 (1.48-2.75)	< 0.001
Osteoporosis with fracture or vertebral collapse	311	0.46 (0.35-0.60)	< 0.001
Avascular necrosis	200	1.84 (1.34-2.52)	< 0.001
Osteomyelitis	19	0.51 (0.18-1.44)	0.20
Ruptured tendon	70	0.84 (0.49-1.45)	0.54
Scarring chronic alopecia	65	6.25 (3.16-12.36)	< 0.001
Extensive scarring or panniculum other than scalp and pulp space	37	2.19 (0.99-4.83)	0.05
Skin ulceration (not due to thrombosis) > 6 months	26	0.60 (0.26-1.42)	0.25
Premature gonadal failure	58	0.93 (0.52-1.65)	0.80
Diabetes	130	1.36 (0.91-2.03)	0.13
Malignancy (excluding dysplasia)	248	0.74 (0.55-0.99)	0.04

[a] Adjusted for sex, age at diagnosis, years of education, income, and insurance. GFR: glomerular filtration rate; GI: gastrointestinal; RR: rate ratios; SDI: Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index.

University of California, San Francisco, on 783 participants, showed that poverty resulted in damage accumulation and that exiting poverty was associated with lower levels of accumulated damage.[17] Using the same data source, it was suggested that poverty resulted in a higher mortality in SLE by increasing organ damage.[18] In 2005, Ward found that among White patients, higher education was associated with lower mortality but this association was not found in Black patients.[16] Our study is more in line with the Eight Americas mortality study, which found that ethnicity played a major role, regardless of SES.[19]

The strength of our study is that it reviewed the trajectory of damage in 2 ethnicities in a large cohort of patients with SLE followed up for a mean of 13 years, taking into account the effects of socioeconomic measures. The 3 measures of SES were self-reported by the patients. A limitation is that socioeconomic variables were colinear and, hence, it was difficult to tease out

any individual effect of each measure of SES. A second limitation is that our analysis assumes that SES remained fixed since SLE diagnosis. While patient education is generally established after a certain age, income and health insurance could vary over time, leading to some misclassifications. Determining the effect of the change in SES on the trajectory of damage in SLE is extremely challenging and beyond the scope of this study. We did not find any study in the literature that evaluated these repercussions. Another limitation is the inability to account for potentially confounding variables that could affect the study findings, due to their multitude. These variables include, but are not limited to, disease activity, corticosteroid use, medication adherence, and obesity. A final limitation is that our study was a single-center cohort predominantly consisting of African American and White patients and cannot be generalized to other ethnicities.

In conclusion, the linear increase in damage in both African American and White patients over time is of particular concern. At any point during the disease, the SDI was higher and accrued at a faster rate in African American compared to White patients with SLE. Damage in most organ systems progressed at a faster rate in African American patients with SLE. Ethnicity, therefore, is a strong contributor to organ damage. Our study further highlights that differences between African American and White patients in SLE organ damage are not adequately explained by disparities in SES, similar to the Eight Americas mortality study.[19]

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