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High Disease Severity Among Asians in a US Multiethnic Cohort of Individuals with Systemic Lupus Erythematosus

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Abstract

Objective.—Knowledge about systemic lupus erythematosus (SLE) outcomes among US Asians is lacking. We examined SLE disease activity, severity, and damage among Asians of primarily Chinese and Filipino descent in a multiethnic cohort.

Methods.—California Lupus Epidemiology Study (CLUES, n=328) data were analyzed. Data were collected in English, Cantonese, Mandarin or Spanish, using validated instruments for disease activity (Systemic Lupus Erythematosus Disease Activity Index), disease severity (Lupus Severity Index [LSI]) and disease damage (Systemic Lupus International Collaborating Clinics

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Damage Index). We assessed differences in SLE outcomes among racial/ethnic groups using multivariable linear regression including interaction terms for age at diagnosis and race/ethnicity.

Results.—Asians were the largest racial/ethnic group (38%; [Chinese=22%; Filipino=9%; Other=7%]). Average age at diagnosis (years) was younger among Asians (27.9), particularly Filipinos (22.2), compared with Whites (29.4) and Blacks (34.0). After adjustment, disease activity and damage were not significantly different across groups. Disease severity among Asians was significantly higher than Whites (LSI 7.1 vs 6.5; $p<0.05$) but similar to Blacks and Hispanics. Early age at diagnosis was associated with greater organ damage among Asians, Blacks, and Hispanics, but not Whites.

Conclusions.—SLE was more severe among US Asians compared to Whites. Filipinos were affected at strikingly young ages. Asians and non-White groups with younger age at diagnosis had greater organ damage than Whites. Such racial/ethnic distinctions suggest the need for heightened clinical awareness to improve health outcomes among Asians with SLE. Further study of SLE outcomes across a range of US Asian subgroups is important.

Introduction

Systemic lupus erythematosus (SLE) is a complex chronic condition that disproportionately affects racial/ethnic minorities. A growing body of literature has demonstrated that Black and Hispanic patients with SLE experience worse disease outcomes (1–4). Understanding patterns of disease among Blacks and Hispanics has prioritized controlling flares and mitigating damage in caring for these diverse SLE patients.

International multiethnic cohorts have found that SLE patients with Asian ancestry tend to have younger age at diagnosis, more frequent autoantibody positivity as well as worse disease activity, organ-specific and overall damage and SLE severity (5–7) as compared with Whites. There are outcomes for which there is no evidence that Asians differ from Whites at follow up, such as with overall damage (5,6). These findings are largely consistent with Asia-specific studies that investigate differences in genetic expression and other outcomes among Asian patients with SLE (8,9). Importantly, new large-scale prospective registries are evaluating the impact of SLE across the Asian subgroups and in comparison to non-Asian counterparts (10,11).

There is a gap in knowledge about how SLE affects US Asians. We studied disease outcomes in Asian patients with SLE to identify which aspects might require more focused attention. The Centers for Disease Control and Prevention (CDC) has supported longitudinal, population-based cohort studies to increase systematic investigation of SLE in racial/ethnic minorities. Spanning five distinct geographic regions of the US, these cohort studies provide population-based estimates of SLE disease incidence and prevalence (12–19). Across these studies, the cohort based in San Francisco County (the California Lupus Surveillance Project, CSLP), includes the largest proportion of Asians.

Patients and Methods

Data derive from CLUES baseline study visits. Some participants for CLUES were recruited from the CLSP cohort (n=134), which used outpatient, hospital, and laboratory records to identify all SLE patients residing in San Francisco City and County from 2007–2009 (12). Additional participants (n=198) in the nine counties of the San Francisco Bay Area were identified from 2015 to 2018 through academic and community rheumatology clinics and through existing local research cohorts. This study was approved by the University of California-San Francisco Institutional Review Board and all participants provided written informed consent.

Study details were previously described (12). Briefly, SLE diagnoses were confirmed at the 2015–2018 baseline by rheumatologists according to any of the following definitions: (a) meeting ≥4 of the 11 American College of Rheumatology (ACR) revised criteria for the classification of SLE as defined in 1982 and updated in 1997 (20,21), (b) meeting 3 of the 11 ACR criteria plus a documented treating rheumatologist's diagnosis of SLE, or (c) a confirmed diagnosis of SLE nephritis (12). Study procedures included an in-person study clinic visit that was separate from the regularly scheduled clinic visit. Because the recruitment was from a large catchment area and individuals could participate if they were under the care of academic tertiary, community or safety net hospital rheumatology clinics, visits were completed in a study setting at our institution. Study clinic visits included a review of medical records, a history and physical examination conducted by a rheumatologist, collection of clinical labs and stored biospecimens, and a structured interview administered by a research assistant. Study clinic visits and interviews were conducted in English, Spanish, Cantonese, or Mandarin. A total of 332 SLE patients completed the baseline in-person CLUES study visit. Four individuals were excluded from this analysis because of missing data, resulting in a final sample size of 328.

SLE Disease Outcome Measures.

Three measures of SLE disease outcomes were used in the analysis, all ascertained by the rheumatologist at the baseline study visit. The Safety of Estrogens in Lupus Erythematosus, National Assessment (SELENA) Systemic Lupus Erythematosus Disease Activity Index (SELENA-SLEDAI) is a frequently used measure of disease activity that incorporates current symptoms as well as clinical laboratory values (range 0 – 105) (22). The Lupus Severity Index (LSI) creates a weighted score of ACR criteria and sub-criteria for SLE (range 0–100) and has been shown to predict morbidity and mortality (23). The Systemic Lupus Collaborating Clinics/College of Rheumatology Damage Index (SDI) is a measure of cumulative organ damage by system (range 0 – 47) in SLE (24). For each instrument, higher scores represent poorer SLE outcomes.

Covariates.

We included self-reported, mutually exclusive categories of Hispanic of any race and the non-Hispanic categories of White, Black, Asian, and Mixed/Other and subgroups of Asian origin. For ease of readability, throughout the text/tables we refer to non-Hispanic categories of White, Black, Asian, and Mixed/Other as White, Black, Asian, and Mixed/Other. These

categories frequently encompass multiple subgroups. In particular “Asian” is a diverse categorization that includes a “person having origins in any of the original peoples of the Far East, Southeast Asia, or the Indian subcontinent” (25). When possible, we examined two subgroups of Asians, Chinese and Filipino, the two largest US Asian populations. The rest of Asian study participants, along with those identifying as Pacific Islander, were included in the “other Asian” category as their numbers were too small to be analyzed separately.

We considered the following additional baseline characteristics as covariates: sex, age at diagnosis, age at baseline, educational attainment (high school completion or less, associate degree/some college, college graduate or above), household income (below 125% or equal to/greater than 125% the Federal poverty level), and provider practice setting (academic tertiary care center, community provider, safety-net hospital clinic).

Statistical analysis.

We compared mean age at baseline, disease duration, and age at diagnosis across racial/ethnic groups and across Asian subgroups using analysis of variance (ANOVA) F-tests. Differences in the three SLE disease outcome measures between racial/ethnic groups and among subgroups of Asian origin were examined using multivariable linear regression models controlling for age at baseline, sex, educational attainment and age at diagnosis. Individuals categorized as Mixed/Other were omitted from the multivariable analyses because of the small number (n=5). We added interaction terms for age at diagnosis and race/ethnicity to the multivariable models to evaluate whether the association of age at diagnosis with the three disease outcome measures varied across racial/ethnic groups or among Asian subgroups.

We examined racial/ethnic differences in the prevalence of organ system damage as defined by the SDI by ranking the individual organ systems from highest to lowest prevalence across the whole sample and for each racial/ethnic group. We also calculated the age-adjusted prevalence of specific organ system damage across race/ethnic groups to account for the differential age distribution by race/ethnicity and compared prevalences using ANOVA F-tests.

As a sensitivity analysis, we re-estimated the multivariable models containing women only because there were few men. All analyses were conducted in SAS 9.4 (Cary, NC) and Stata (StataCorp. 2017. Stata Statistical Software: Release 15. College Station, TX: StataCorp LLC).

Results

Descriptive characteristics of the study sample (n=328) are shown in Table 1. Nearly 90% of the participants were women, the mean age at baseline of the group was 45.0 ± 14.0 years, with an average disease duration of 16.3 ± 10.5 years. Reflecting the demographics of the San Francisco Bay Area, Asians made up the largest portion of the sample (38%), including 71 participants of Chinese, 31 of Filipino and 23 of Other Asian descent. Approximately 22% reported high school education or less, 17% had household incomes below 125% of the Federal poverty level, and 18% received SLE care from a safety-net hospital clinic.

Nearly half of the sample had a diagnosis of SLE nephritis (47%). Additional data on sociodemographic variables by racial/ethnic background can be found in the Supplemental Table.

The racial/ethnic groups in the study had statistically significant differences in mean age at baseline, disease duration and age at diagnosis (Table 2). Of the main race/ethnicity categories, Asians and Hispanics were the youngest (42.1 and 40.3 years old, respectively), had similarly young ages at diagnosis (27.9 and 27.0 years old) and the shortest disease duration (14.2 and 13.3 years). Within the Asian subgroups, there were significant differences in age at baseline and at diagnosis. Filipino patients were the youngest among Asian subgroups overall (36.5 years old) and had an earlier age at diagnosis compared with Chinese or other Asian participants (22.2, 30.1, and 28.8 years old, respectively, p=0.02). Disease duration did not differ across Asian subgroups.

Lupus disease activity as measured by SLEDAI was low across all racial/ethnic groups, with a mean of 2.9 (range 0–16; Table 3) and not significantly different among racial/ethnic groups after adjustment for age at baseline, age at diagnosis, sex and educational attainment. Lupus severity as measured by the LSI was significantly higher in Asians as compared to Whites (7.1 vs 6.5; $p<0.05$) after adjustment, but Asian LSI scores were not significantly different from those of Hispanics or Blacks. Lupus organ damage as measured by SDI was lower among Asians than Blacks, but after adjustment this difference was not statistically significant (1.9 vs 2.5, respectively, p=0.12). Whites and Hispanics had similar SDI scores to those of Asians. All SLE disease outcome measures were the same across Asian subgroups. In the adjusted model, SDI scores were highest among those with childhood onset SLE and declined with increasing age at diagnosis.

The differences in age at diagnosis by race/ethnicity and the differences in disease outcomes by both race/ethnicity and age at diagnosis led us to investigate interactions between age at diagnosis and race/ethnicity. We found a statistically significant interaction (alpha=0.05) between age at diagnosis and race/ethnicity for SDI but not SLEDAI or LSI. Younger age at diagnosis was associated with higher SDI scores among Asians, after controlling for sex and age at baseline (Figure 1). A similar pattern was seen among Blacks and Hispanics, but not among Whites, among whom SDI scores were the same across age at diagnosis.

Differences in patterns of organ damage by race/ethnicity can be found in Table 4. Asians experienced ocular and musculoskeletal damage most frequently (23% and 21%), followed by neuropsychiatric (16%) and renal long-term sequelae (16%). The prevalence of involvement of the various organ systems did not differ significantly across racial/ethnic groups, except for skin manifestations which were more prevalent among Blacks.

Because of the small number of men in this cohort, we conducted a sensitivity analysis excluding men from the multivariable analyses. The results are consistent with our main analysis and are not presented here.

Discussion

Our study is among the first to describe SLE disease outcomes across racial/ethnic groups with a focus on Asian patients in the US. In this multiethnic population-based cohort, we found greater SLE severity in Asian as compared with White participants. Although Asians are generally thought to be a healthy group compared to other races/ethnicities (26), this work demonstrates that SLE is another chronic health condition that disproportionately affects Asians, such as tuberculosis, hepatitis-B, and certain malignancies (27).

Asians and Hispanics had the youngest age at diagnosis across the major racial/ethnic groups in the CLUES sample. Filipinos experienced the earliest average age at diagnosis of all racial/ethnic groups. Accounting for delays from SLE symptom onset and diagnosis, our findings are consistent among studies reporting a younger age at disease onset among non-White patients and Asians as compared with White patients (5,6,28–30). Similarly, meta-analyses of Asian SLE patients outside the US demonstrated the range in age at onset across Asian subgroups (31), with Filipinos having the youngest age at diagnosis (32). Differences in age at onset or diagnosis in Asians versus non-Asians may stem from biological and environmental factors. For example, differences in genetic risk among Asians may contribute to earlier onset disease via increased susceptibility to severe aspects of SLE such as SLE-associated nephritis (33–36). The higher severity of SLE found in cohorts from Australia and Southeast Asia suggest that genetic susceptibility may play a role, and some studies have started to uncover differences in immunological responses among Asian subgroups. For example, emerging literature suggests higher levels of the proinflammatory cytokine macrophage migration inhibitory factor (MIF) among Asian individuals with SLE. MIF has been linked to worse SLE disease damage (37). Among Asians in particular, higher MIF levels were independently associated with persistently active SLE (38). In contrast, the Type 1 Interferon (IFN) signature, which has been implicated in SLE pathogenesis (39) and is a target for novel therapy (40,41), was not found to be meaningfully different among Asians as compared to Whites, at least based on chemokine profile. Ongoing study of unique variations in genetic susceptibility has the potential to inform treatment pathways for Asians with SLE.

Environmental factors such as social determinants of health may negatively impact access to care by Asians in some geographic regions more than others (42). Certain factors like adverse childhood experiences, which commonly are associated with poor reported health status in SLE patients, may be less problematic for Asians, since Asians reported significantly less exposure to adverse childhood experiences compared with all other racial/ethnic groups (43). Regardless of the causes of the differences in SLE between Asians and non-Asians, our results underscore the complexity of patterns of early onset disease in Asian patients and support the need for closer study to discern underlying drivers.

There were no significant differences in disease activity by race/ethnicity, nor by age at diagnosis, age at baseline, sex, or educational attainment. This may be because this measure was collected during a study visit rather than routine or acute clinical care. Other studies have reported conflicting results regarding racial/ethnic differences in disease activity: some identified no differences (6), while another US study in Texas and Alabama determined

that disease activity was higher among Blacks and Hispanics than White patients (1). Clinically meaningful higher disease activity occurred in Australian Asians compared with non-Asian patients with SLE. Australian Asians experienced more persistently active SLE than non-Asians (odds ratio 2.14; 95% confidence interval (1.05–4.38), $p<0.04$) (7,38). Patterns of disease activity and "flares" among Asians and Asian subgroups in clinical or hospital settings in the US need further elucidation.

There was greater SLE severity among Asian as compared with White SLE patients by the LSI, a newer measure of severity that projects morbidity and mortality based on ACR diagnostic criteria alone. Asians have increased rates of renal manifestations as well as antibody abnormalities (6,44,45), elements that garner higher weights in the LSI (23). In the parent surveillance project to CLUES, Asians not only have high SLE severity as predicted by the LSI but also greater risk of developing SLE nephritis compared with other races/ethnicities (44). The worse severity among Asians in this study, which may be influenced by HLA susceptibilities or genetic risk alleles (46,47), aligns with clinical data from other multiethnic cohorts in Canada and Australia (6,7). Our work has permitted a more granular study of outcomes in US Asians and emphasizes the important findings of 2 studies with US Asians in the last 2 decades. The Manhattan Lupus Surveillance Project found US Asians had significantly higher prevalence of renal involvement based on ACR criteria as compared with Whites (13). A study on Medicare claims data demonstrated that US Asians and especially US Asian females had the highest estimated prevalence of SLE nephritis of all ethnic groups (48). Although studies have defined lupus severity differently—by organ involvement, exposure to immunosuppressive therapies and their side effects, or persistently active disease—they consistently support the notion that individuals of Asian descent tend to have severe forms of SLE.

Identification of groups at higher risk for organ damage in SLE is vital since existing organ damage predicts future damage accrual and mortality (49). Compared with Whites, non-White racial/ethnic groups experience more frequent and greater organ damage (50). In CLUES, damage among Asian patients was not significantly different from Hispanics or Whites, whereas Blacks experienced the worst organ damage overall. Despite high disease severity, accumulation of damage in Asians is inconsistent in other studies (5–7,49). Variations in damage do not sufficiently explain disability or mortality among Asian patients with SLE and need to be further explored.

We discerned a significant interaction between race and age at diagnosis on organ damage as non-Whites experienced more damage with earlier disease onset than the Whites. Previous studies found earlier age at diagnosis predicts worse organ damage, but our study suggests that this relationship is not uniform across racial/ethnic groups (49).

There are limitations to our study. We relied on self-report for age at diagnosis, thus age could be over- or underestimated in the sample. Another limitation is the overall low disease activity scores, which is likely because patients were unable, or chose not, to attend study visits when they had clinically active disease. Since all persons in our study resided in the San Francisco Bay area, these results may not be generalizable to the population at large. Furthermore, sample sizes among Asians, Hispanics and Whites were robust for

comparisons, yet the low numbers of Black participants limited comparisons to this group. Asians represent a large portion of the world population; our subgroup was primarily Chinese. The ability to compare among Asian subgroups was limited by small numbers. Since our study suggests that Filipinos had the earliest age at diagnosis, further investigation into subgroup differences among Asians is essential.

The major strength of this work was that it permitted close study of an available US Asian population with well-defined SLE. Concerted efforts to employ study personnel who spoke patients' native languages and to provide materials available in Chinese and Spanish were integral to successfully recruit and follow up Asian and Hispanic individuals with SLE in CLUES.

Conclusion

We found that US Asians, primarily Chinese but especially Filipinos, demonstrated earlier age at diagnosis and higher disease severity as measured by LSI than Whites. Increased vigilance among clinicians caring for Asians with SLE may detect more severe disease earlier in this group of patients, which could improve outcomes. Further defining unique disease patterns, genetic influences and treatment responses among Asian subgroups may help to identify patients at greatest risk for disability and death.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Significance and Innovation

- This is the first US multiethnic population-based cohort where Asian patients with SLE represent the most prevalent racial/ethnic group. Because prior reports indicate that the impact of SLE is higher for Asians than other races/ethnicities, examining outcomes of disease activity, severity, and damage were of chief interest.
- US Asians experience early age at diagnosis with higher disease severity as measured by the Lupus Severity Index as compared with Whites (7.1 vs. 6.5 $p<0.05$). Awareness of these greater risks should factor into diagnosis, monitoring and treatment efforts for US patients of Asian descent.

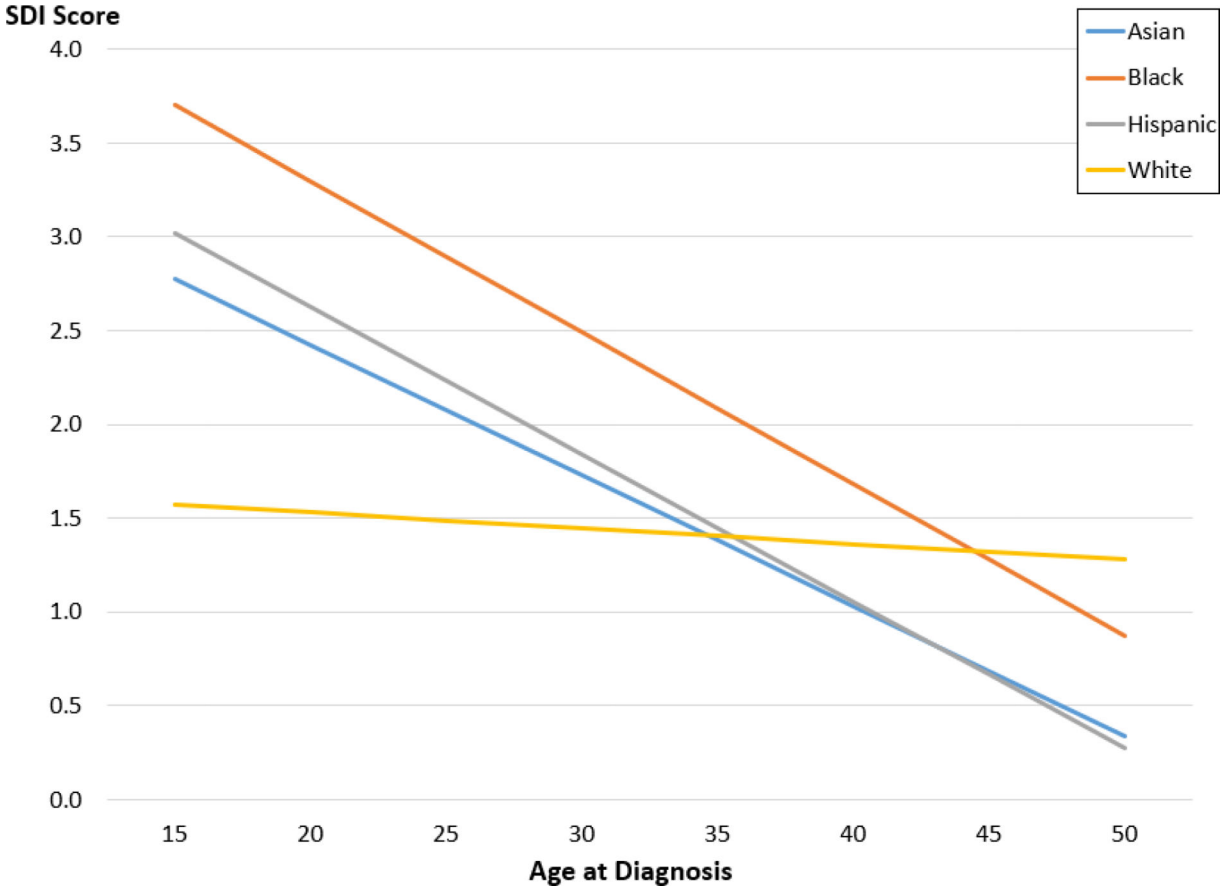

Figure 1.
Race/ethnicity categories include Hispanic and non-Hispanic White, Asian and Black

Table 1.

Baseline demographic and SLE characteristics of 328 California Lupus Epidemiology Study participants, 2015–2018

Demographic characteristics	
Age, mean ± sd (range)	45.0 ± 14.0 (19–83)
Women	291 (89%)
Race/Ethnicity	
Asian	125 (38%)
Chinese	71 (22%)
Filipino	31 (9%)
Other Asian *	23 (7%)
White	95 (29%)
Hispanic	69 (21%)
Black	34 (10%)
Mixed/Other	5 (2%)
Interview Language	
English	283 (86%)
Chinese (Mandarin or Cantonese)	25 (8%)
Spanish	20 (6%)
Educational Attainment	
High school completion or less	72 (22%)
Associate degree/some College	10 (30%)
College graduate	156 (48%)
Below poverty or near poor +	57 (17%)
SLE Characteristics	
Disease Duration, mean ± sd (range)	16.3±10.5 (0–58)
Age at Diagnosis, years	
< 18	57 (17%)
18–25	95 (28%)
26–35	93 (28%)
36–67 (max)	83 (25%)
Lupus Nephritis	153 (47%)
Provider Practice Setting	
Academic Tertiary Care	136 (42%)
Community	131 (40%)
Safety-Net hospital clinics	60 (18%)

* Southeast Asian (n=9), Korean (n=5), Japanese (n=3), Indian (n=3), Taiwanese (n=2), and Hawaiian (n=1).

+ Household income below 125% of the Federal poverty level.

Race/Ethnicity categories include Hispanic and Non-Hispanic White, Asian, Black and Mixed/Other.

All n (%) except where noted.

sd, standard deviation.

Table 2.

Mean Age, Disease Duration, and Age at Diagnosis at baseline, by Major Race/Ethnicity Category and by Asian Subgroups

By major race/ethnicity category	N	Age at baseline (years)	Disease duration at baseline (years)	Age at diagnosis (years)
Total mean ± sd (range)	328	45.0 ± 14.0	16.3 ± 10.5	28.7 ± 12.2
			mean (95% CI)	
Asian	125	42.1 (39.6, 44.7)	14.2 (12.4, 16.0)	27.9 (25.6, 30.3)
White	95	50.3 (47.6, 53.0)	21.0 (18.9, 23.0)	29.4 (26.9, 31.8)
Hispanic	69	40.3 (37.1, 43.5)	13.3 (11.0, 15.7)	27.0 (24.1, 29.9)
Black	34	49.9 (45.4, 54.4)	15.9 (12.5, 19.3)	34.0 (29.9, 38.1)
Other	5	44.2 (32.4, 56.0)	21.6 (12.8, 30.4)	22.6 (12.0, 33.2)
p-value		<0.001	<0.001	0.04
By Asian Subgroups				
Asian mean ± sd (range)	125	42.1 ± 14.3	14.2 ± 9.9	27.9 ± 13.4
Subgroup			mean (95% CI)	
Chinese	71	44.2 (40.9, 47.5)	14.1 (11.7, 16.4)	30.1 (27.1, 33.2)
Filipino	31	36.5 (31.5, 41.5)	14.3 (10.7, 17.8)	22.2 (17.6, 26.9)
Other Asian *	23	43.3 (37.5, 49.1)	14.5 (10.3, 18.6)	28.8 (23.4, 34.2)
p-value		0.04	0.99	0.02

* Southeast Asian (n=9), Korean (n=5), Japanese (n=3), Indian (n=3), Taiwanese (n=2), and Hawaiian (n=1).

Race/Ethnicity categories include Hispanic and Non-Hispanic White, Asian, Black, and Mixed/Other.

sd, standard deviation.

Table 3.

SLE disease activity, severity, and damage[^] at baseline by race/ethnicity and age at diagnosis, with and without adjustment[**]

Characteristics	Disease Activity (SLEDAI) unadjusted	Disease Activity (SLEDAI) adjusted	Disease Severity (LSI) unadjusted	Disease Severity (LSI) adjusted	Disease Damage (SDI) unadjusted	Disease Damage (SDI) adjusted
Overall (mean and range)	2.9 (0–16)		6.9 (3.4–9.5)		1.8 (0–10)	
Race/Ethnicity	mean (95% CI)		mean (95% CI)		mean (95% CI)	
Asian	3.0 (2.5, 3.6)	3.0 (2.4, 3.5)	7.1 (6.8, 7.4) *	7.1 (6.8, 7.4) *	1.6 (1.3, 2.0)	1.9 (1.5, 2.2)
Chinese	2.9 (2.2, 3.5)	3.0 (2.3, 3.6)	7.1 (6.7, 7.4)	7.3 (6.9, 7.6)	1.5 (1.1, 1.9)	1.6 (1.2, 2.0)
Filipino	3.4 (2.4, 4.4)	3.6 (2.6, 4.6)	6.9 (6.4, 7.5)	6.9 (6.4, 7.5)	1.7 (1.1, 2.3)	1.7 (1.1, 2.4)
Other Asian[+]	3.1 (1.9, 4.3)	3.4 (2.2, 4.6)	7.5 (6.8, 8.1)	7.6 (7.0, 8.3)	2.1 (1.4, 2.9)	2.1 (1.4, 2.9)
White	2.5 (1.8, 3.1)	2.9 (2.2, 3.6)	6.2 (5.9, 6.5)	6.5 (6.1, 6.8)	1.9 (1.5, 2.3)	1.5 (1.1, 1.9)
Hispanic	3.7 (3.0, 4.4) *	3.4 (2.6, 4.1)	7.4 (7.1, 7.8) *	7.3 (6.9, 7.6) *	1.8 (1.3, 2.3)	2.0 (1.6, 2.5)
Black	2.4 (1.3, 3.4)	2.5 (1.4, 3.5)	6.6 (6.1, 7.2)	6.9 (6.4, 7.4)	2.4 (1.8, 3.1)	2.5 (1.8, 3.1)
Age at Diagnosis (years)						
< 18	2.9 (2.1, 3.8)	2.1 (1.2, 3.0)	7.5 (7.1, 7.8) †	7.2 (6.7, 7.6)	1.7 (1.2, 2.2)	2.6 (2.0,3.1) †
18–25	3.3 (2.5, 4.0)	2.7 (2.0, 3.4)	7.2 (6.9, 7.5) †	7.0 (6.7, 7.4)	1.8 (1.4, 2.2)	2.5 (2.1,2.9) †
26–35	3.2 (2.6, 3.9)	3.3 (2.7, 4.0)	6.6 (6.3, 7.0)	6.8 (6.5, 7.1)	2.0 (1.5,2.5)	1.9 (1.5,2.3) †
36–67 (max)	2.4 (1.9, 2.9)	3.1 (2.3, 3.9)	6.3 (5.9, 6.7)	6.6 (6.2,7.0)	1.8(1.4,2.2)	0.9 (0.4,1.4)

[^] higher value is worse outcome.

* p<0.05 for difference with Asians.

† p<0.05 for difference with oldest age at diagnosis.

[+] South East Asian (n=9), Korean (n=5), Japanese (n=3), Indian (n=3), Taiwanese (n=2), and Hawaiian (n=1).

** Adjusted models include variables for race/ethnicity, age at diagnosis and baseline, sex, and education. Mixed/Other n=5 not included in adjusted models.

Systemic Lupus Erythematosus Disease Activity Index (SLEDAI); Lupus Severity Index (LSI); Systemic Lupus Collaborating Clinics/College of Rheumatology Damage Index (SDI).

Table 4.

Age-adjusted percentage with specific baseline SDI organ system damage, by race/ethnicity

	Total (n=323)		Asian (n=125)		White (n=95)		Hispanic (n=69)		Black (n=34)		p-value^
SDI organ system	**%**	**Rank***	**%**	**Rank***	**%**	**Rank***	**%**	**Rank***	**%**	**Rank***	
Musculoskeletal	24%	1	21%	2	29%	1	24%	2	24%	2	0.648
Ocular	21%	2	23%	1	17%	3	27%	1	19%	3	0.398
Neuropsychiatric	19%	3	16%	3	25%	2	13%	6	18%	4	0.265
Skin	14%	4	11%	6	10%	5	14%	4	31%	1	0.021
Renal	14%	5	16%	4	7%	6	21%	3	17%	5	0.087
Vascular	10%	6	12%	5	6%	7	14%	5	11%	8	0.415
Malignancy	8%	7	3%	11	14%	4	7%	8	8%	9	0.060

*Organ systems ranked by prevalence in each group.

^p-value for test of any difference in proportions across racial/ethnic groups.

Mixed/Other omitted due to small sample size.

Systemic Lupus International Collaborating Clinics Damage Index (SDI).