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Racial/Ethnic Variation in All-Cause Mortality among U.S. Medicaid Recipients with Systemic Lupus Erythematosus: An Hispanic and Asian Paradox

José A. Gómez-Puerta, MD, PhD, MPH[1,*], **Medha Barbhaiya, MD**[1,*], **Hongshu Guan, PhD**[1], **Candace Feldman, MD, MPH**[1], **Graciela S. Alarcón, MD, MPH**[2], and **Karen H. Costenbader, MD, MPH**[1]

[1]Division of Rheumatology, Immunology and Allergy, Section of Clinical Sciences, Brigham and Women's Hospital, Harvard Medical School, Boston, MA

[2]Division of Rheumatology and Clinical Immunology, University of Alabama at Birmingham, Birmingham, AL

Abstract

Objective—Incidence of systemic lupus erythematosus (SLE) is disproportionately high in non-Whites vs. Whites. However, variation in mortality according to race/ethnicity has not been well studied. We examined all-cause mortality by race/ethnicity among SLE patients in Medicaid.

Methods—Within the Medicaid Analytic eXtract 2000–2006 from 47 U.S> states and D.C., we identified individuals aged 18–65 years, enrolled for ≥3 months, with ≥ 3 claims for SLE (ICD-9 710.0), each ≥ 30 days apart. Lupus nephritis (LN) was identified by ≥ 2 additional claims for glomerulonephritis, proteinuria, or renal failure. We calculated mortality rates (MR) per 1,000 person-years with 95% confidence intervals by race/ethnicity. Multivariable Cox regression models estimated mortality risks, adjusting for age, sex, demographics and comorbidities.

Results—Among 42,221 prevalent SLE patients, 8,191 had LN. Blacks represented 40.1%, Whites 38.4%, and Hispanics 15.3%. Overall SLE MRs per 1000 person-years were highest among Native Americans (27.52), Whites (20.17) and Blacks (24.13), and lower among Hispanic (7.12) or Asian SLE patients (5.18). After multivariable adjustment, Hispanic and Asian patients had lower mortality risks [HR 0.48 (95%CI 0.40–0.59) and 0.59 (95%CI 0.40-0.86)] vs. Whites. Conversely, risks for death were significantly higher among Native American (HR 1.40, 95%CI 1.04–1.90) and Black (HR 1.21, 95%CI 1.10–1.33) compared to White patients. Among LN patients, mortality risks were lower among Hispanic and Asian patients (by 56% and 40%) than among Whites.

Conclusion—After accounting for demographic and clinical factors, Asian and Hispanic SLE Medicaid patients had lower mortality than did Blacks, Whites or Native American patients.

Corresponding author: Jose A. Gómez-Puerta, MD, PhD, MPH, Division of Rheumatology, Immunology and Allergy, PBB-B3, Section of Clinical Sciences (221 Longwood Ave, 3rd floor), Brigham and Women's Hospital, Boston, MA 02115, jgomezpuerta@mail.harvard.edu.

*Both authors contributed equally to this work.

Keywords

Systemic Lupus Erythematosus; Race; Ethnicity; Disparities; Mortality; Survival; Medicaid

Systemic lupus erythematosus (SLE) disproportionately affects non-White populations in the United States[1,2]. Black females, for example, have incidence rates three to four times higher than their White counterparts. Other racial and ethnic minorities, including Hispanics, Asians and Native Americans, are also at increased risk of developing SLE[3]. Past studies have reported poor outcomes including high rates of lupus nephritis (LN), end-stage renal disease, and SLE organ damage accrual among Blacks, Hispanics and Asians with SLE[4–9]. Academic cohort studies have suggested higher mortality among Black and Hispanic patients than among White patients with SLE[10–13]. For example, in the LUMINA study (Lupus in Minorities: Nature versus Nurture), both Blacks and Texan Hispanics with SLE had a lower five year survival than did non-Hispanic Whites in an unadjusted analysis, but after adjustment for age, socioeconomic status, disease activity and organ damage, differences in survival were no longer present[14]. Previous studies have been mainly based in academic centers with relatively few deaths. Moreover, race and socioeconomic status have historically been very hard to disentangle in their associations with poor outcomes in these SLE populations[3,15,16].

Given the lack of studies with large populations of low income individuals affected by SLE, we investigated all-cause mortality and mortality rates, overall and by race and ethnicity, among SLE and LN patients enrolled in U.S. Medicaid from 2000 to 2006. We hypothesized that there would be significant variation in mortality rates and risks according to race and ethnicity among SLE and LN patients, with increased mortality among Hispanic and Black patients.

Patients and Methods

Study population

Medicaid is the U.S. health insurance program for individuals with low income and resources (low income children, pregnant women, mothers and people with disabilities) and provides coverage for medical expenses and prescription drugs. We employed data from the Medicaid Analytic eXtract (MAX) database, an administrative data system containing all billing claims for Medicaid enrollees in 47 U.S. states and Washington, D.C., from January 1, 2000 to December 31, 2006. (Arizona, Tennessee, and Maine do not contribute data to MAX.) We identified adults ages 18–65 years who were enrolled in Medicaid for at least three months between January 1, 2000 and December 31, 2006. The index date for SLE diagnosis was defined as occurring when individuals obtained ≥ 3 ICD-9 codes for SLE (710.0) at least 30 days apart, obtained from hospital discharge diagnoses or physician visit claims[1]. Among individuals with SLE, we identified those with LN, as having ≥ 2 additional ICD-9 hospital discharge diagnoses or physician billing claims for nephritis, proteinuria and/or renal failure, on or after the date of SLE diagnosis, and occurring at least 30 days apart[1,17]. This administrative definition has been found to have a 80% positive predictive value for LN in Medicaid claims data[17].

Exposures

Race and ethnicity in MAX are categorized based mainly on self-report. We used the following previously defined combined race and ethnicity categories: White, Black, Hispanic or Latino, Asian (including Native Hawaiian or other Pacific Islander), Native American (including American Indian or Alaskan Native)[18]. Patients with missing or unclassifiable information for race or ethnicity (e.g. “other/unknown” or “more than one race” categories) were excluded from these analyses.

Other variables

Demographic variables extracted from MAX included age, sex, and region of residence, which was determined by zip code and categorized by U.S. Census region (Northeast, Midwest, South or West). For area-based socioeconomic status (SES), we employed a composite index of seven SES indicators at the zip code level using 2000 U.S. Census data[19]. These include median household income, proportion with income below 200% of the federal poverty level, median home value, median monthly rent, mean education level, proportion of people age ≥25 years who were college graduates, and proportion of employed persons with a professional occupation. Area-level SES was divided into quartiles as previously described[1]. We also employed a previously described “SLE-specific risk adjustment index”, which has been validated for prediction of in-hospital mortality among SLE patients[20]. The SLE-specific risk adjustment index developed by Ward includes comorbidities specific for SLE, including autoimmune hemolytic anemia, thrombocytopenia, pericarditis, seizures and psychosis. We also included validated ICD-9 codes for hypertension (401.1), diabetes mellitus (250.0), smoking (305.1), obesity (278.0), acute myocardial infarction (410.0), angina (413.× or 411.1), old myocardial infarction (412.0), percutaneous coronary intervention (00.66, 36.0×, 37.22, 37.23, and 88.5×, except 88.59), coronary atherosclerosis (414.00 and 414.9; not including 414.1x), and coronary artery bypass graft (3610 and 3619)[21, 22, 23, 24, 25].

Outcomes

The outcome of our study was death from all causes. Subjects were followed from the index date through date of death, loss to follow-up (no further medical claims in the absence of documented death), or end of follow-up period of the study (December 31, 2006). Deaths were confirmed using the National Death Index.

Statistical analysis

We calculated crude annual mortality rate (MR) per 1,000 person-years with 95% confidence intervals for SLE patients by racial/ethnic group. We fit three Cox regression models to examine the association of race/ethnicity with mortality risk for both SLE and LN. Model A included age (continuous) and sex. Model B added potential confounding variables to model A, including residential region, calendar year, area-SES, baseline comorbidities collected from ICD-9 diagnoses from January 1, 2000 though the study index date (including history of angina, coronary artery bypass graft, coronary atherosclerosis, percutaneous coronary intervention, hypertension, smoking, obesity) and SLE specific risk-adjustment index. Finally, model C included model A, residential region, calendar year,

area-SES, but comorbidities and the SLE-specific risk-adjustment index were included as time-varying covariates throughout the entire follow-up period. In addition, in model C, acute myocardial infarction at any time during follow-up was included as a comorbidity. We tested the proportional hazards assumption using Kaplan Meier curves, as well as time-varying covariates and observed no significant deviations in our models. In sensitivity analyses, we repeated models A, B and C using multivariable subdistribution proportional hazards models, with loss to follow-up from Medicaid as a competing event to investigate the hypothesis that loss to follow-up could account for some of the observed variation in mortality risks[26].

All statistical analysis were conducted using SAS, version 9.3. Data were obtained from the Centers for Medicare and Medicaid Services through a data use agreement and are presented in accordance with CMS policies. The Partners Healthcare Institutional Review Board waived human subjects approval for this study.

Results

We identified 42,221 patients with prevalent SLE and, among them, 8,191 patients with prevalent LN. Baseline characteristics are summarized in Table 1. The mean age among SLE patients was 38.1 ± 12.3 years, and 93% were women, and the majority of the cohort resided in the South (38%). Black SLE patients resided predominantly in the South, had a higher prevalence of hypertension and a higher SLE-specific baseline risk-adjustment index compared to White patients. Hispanic patients resided predominantly in the West, and their prevalence of diabetes mellitus was similar to that in the Black population. The baseline prevalence of hypertension, heart failure, smoking, obesity were lower, as was the SLE-specific risk adjustment index, among Hispanics than among Blacks. Asian patients lived predominantly in the West, and had a higher prevalence of LN, but a lower prevalence of heart failure, diabetes mellitus, obesity and smoking and a lower SLE-specific risk adjustment index than Whites. Native Americans had a higher prevalence of diabetes mellitus, obesity and smoking than all other races/ethnicities, and had an SLE risk adjustment index higher than that of Whites, Asians or Hispanics. Finally, White patients had a higher prevalence of previous angina, coronary atherosclerosis, but lower prevalence of LN compared to other races/ethnicities.

Baseline demographic and clinical characteristics at index date of patients with and without LN are summarized in Supplementary Tables 1A and 1B. Compared to the prevalent SLE cohort, LN patients were younger, with a mean age of 34.5 ± 12.6 years, and had a higher prevalence of coronary heart disease (including angina, coronary atherosclerosis and previous MI), hypertension and diabetes. Furthermore, the SLE-specific risk adjustment index was almost two-fold higher in the LN patients compared to the SLE cohort.

The mean duration of follow-up for all SLE patients was 2.56 ± 1.99 years and in LN patients was 2.12 ± 1.77 years. During the follow up period, there were 2,058 deaths among all SLE patients and 774 deaths among LN patients. The overall unadjusted annual MR among SLE patients was 19.07 per 1,000 person-years (95%CI 18.36–19.91), while the MR among LN patients was more than two-fold higher 44.64 (95%CI 41.60–47.90) per 1,000

person-years (Table 2). Among SLE and LN patients, all-cause MRs were significantly lower and approximately one-third as high among Hispanic patients and one-quarter as high among Asian patients compared to Whites. Unadjusted MRs were highest among Native American and Black patients among SLE patients compared to all other race/ethnicities (MR ratios 1.36 and 1.19 respectively compared to Whites) (Table 2).

In multivariable-adjusted Cox regression models, racial/ethnic variation in survival persisted. The HRs for all-cause mortality by race/ethnicity for SLE and LN patients are presented in Table 3. In age and sex-adjusted models (**model A**), compared to White SLE patients, both Hispanic and Asian SLE patients had less than half the risk of death (HRs 0.41 (95%CI 0.34–0.50) and 0.30 (95%CI 0.21–43). This was also true among Hispanic and Asian LN patients [HRs 0.39 (95CI% 0.29–0.52) and 0.31 (95%CI 0.19–0.50)] compared to Whites. Conversely, Black SLE patients had a significantly increased mortality risk compared to White patients (HR 1.36 (95%CI 1.24–1.49), as did Native Americans (HR 1.43 95%CI 1.06–1.92). Black and Native American LN patients did not have statistically elevated HRs for all-cause mortality compared to Whites however, possibly due to smaller sample size of the LN patients.

Adjustment for baseline cardiovascular and SLE–related comorbidities (**model B**) and for these comorbidities throughout follow-up (**model C**), did attenuate racial/ethnic variation somewhat. In particular, the risk of death among Blacks with SLE decreased to 21% above that in Whites (HR 1.21 95%CI 1.01–1.33), and, among Blacks with LN, it was no longer statistically elevated when compared to that among Whites with LN (HR 1.04, 95% CI 0.88–1.23). We did not observe a large difference in the HRs when only baseline comorbidities were included compared to when covariates were updated throughout the follow-up period. The SLE-specific risk adjustment index increased in all groups from baseline through follow-up and a one unit increase in the risk index was itself associated with increased mortality risk in all racial/ethnic groups (**model C** HR 1.15, 95%CI 1.14–1.16 among all SLE patients). Increasing area-based SES (as a continuous variable) was inversely associated overall mortality HR 0.92 (95%CI 0.90, 0.96) among all SLE patients. In multivariable-adjusted subdistribution hazards models taking the competing risk of loss to follow-up into account, racial/ethnic variation in survival persisted and the results were extremely similar (data not shown).

Discussion

In the present study with more than 40,000 adult SLE patients enrolled in Medicaid between 2000–2006, we found marked variation in all-cause mortality rates by race/ethnicity. Not unexpectedly, mortality rates were higher among Black patients, although adjustment for comorbidities and sociodemographic factors did attenuate this risk to 21% higher than that of Whites. Strikingly, Native Americans had 40% higher adjusted mortality risks than did Whites with SLE, and adjustment for comorbidities and sociodemographic factors did not substantially affect the risk estimates. The relationships of race/ethnicity to socioeconomic status, lifestyle factors, and comorbidities, such as obesity, smoking, and diabetes, all of which are related to SLE outcomes as well, are extremely complex. These factors are highly correlated and are likely mediators of health outcomes, as well as confounders of observed

relationships between race/ethnicity and outcomes. Mortality rates among Hispanic and Asian patients with SLE were lower by 52% and 41% respectively compared to White adults, even after adjusting multiple demographic and clinical factors. To the best of our knowledge, this is the first large-population study demonstrating lower mortality rates among Hispanic and Asian patients with SLE than among Black and White patients.

Previous studies in North American academic centers have reported worse prognoses, including higher mortality rates, among Black and Hispanic patients compared to Whites[4,12,14,27]. Our finding of decreased mortality rates and adjusted risks among Hispanic adults with SLE was thus surprising[14,28]. There may be several potential explanations. First, as Medicaid provides health care coverage for low-income populations in the U.S., the SES divide between Hispanic and non-Hispanic patients was likely smaller than in past academic center-based cohort studies. Second, this cohort included all Medicaid patients meeting our administrative definition of SLE, whereas academic medical centers, in particular referral centers, may tend to capture the most severe SLE cases with the worst outcomes. Third, our finding is not unique to SLE. Several past epidemiologic studies of the U.S. Hispanic population have demonstrated that, after adjusting for age and annual family income, Hispanics have a lower all-cause mortality and lower mortality due to cardiovascular disease compared with non-Hispanics, despite having a higher burden of cardiovascular risk factors[29]. This epidemiologic observation, first termed the “Hispanic paradox” two decades ago, has since been demonstrated in vital statistics, nationally representative surveys, systematic reviews and a recent meta-analysis[30,31]. To our knowledge this is the first epidemiologic study to suggest the existence of the “Hispanic paradox” among SLE patients. This seeming paradox has recently been reported for survival among rheumatoid arthritis patients in Texas as well[32]. Environmental, cultural, and social factors likely play roles. It is possible that higher neighborhood social cohesion and family and social support may improve health outcomes in Asian and Hispanic communities[33,34]. The Hispanic paradox has also been attributed in part to the “salmon effect”—the phenomenon that Hispanics may return to their home countries at the end of life, becoming statistically “immortal” and leading to an artificially low denominator[35]. For this reason, we performed competing risk analyses, accounting for the competing risk of loss to follow-up from Medicaid (no further claims or encounters of any kind without documented death). Despite accounting for potential differential follow-up, we still observed a lower mortality risk among Hispanics. Moreover, the follow-up time in Medicaid was very similar in Hispanics, Asians and other racial/ethnic groups in this study. Lastly, we acknowledge that racial and ethnic categorization by self-report is a very imperfect measure of genetic ancestry and, with time and growing genetic admixture in our society, classifying individuals in these limited categories is increasingly challenging[36].

The risks of death among Native Americans with SLE were the highest of any racial/ethnic group in the US Medicaid population during the years of study. Prior data about mortality among Native American SLE patients have been limited. In a past study of Canadian Native Americans with SLE by Peschken and colleagues, SLE prevalence, severity and mortality were all reported to be elevated compared to non-native patients[37]. Fifty-nine Native Americans were included in that study of patients seen in a regional arthritis center, and were found to have more vasculitis, proteinuria, cellular casts, receive more

immunosuppressants and prednisone and have higher damage scores and fatality rates. Our study allows an examination of a much larger Native American population and highlights that after adjustment for multiple comorbidities and socioeconomic factors, mortality risks were 20% higher among Native Americans than among African Americans and 40% higher than among White patients. Clearly the reasons for these striking disparities deserve further study. Among individuals of Asian origin, SLE has been reported to be increased in both incidence and severity[38]. Past population-based studies of SLE mortality from the 1970s and 1980s reported that Asians had three to six times higher mortality rates than Whites[39,40]. However, in more recent studies in other countries, comparable mortality has been seen among Asian and White patients with SLE[11,41]. In the current study, U.S. Asian SLE patients enrolled in Medicaid had lower mortality rates and adjusted risks compared to White patients. Variation in mortality across racial and ethnic groups likely depends on both genetic and environmental factors, such as poverty, education, health care access, cultural and health behaviors, and it is interesting to see that, in the U.S. Medicaid system, Asians with SLE have increased survival compared to Whites.

The main strength of this study is the use of nationwide data including over 40,000 SLE patients, providing robust information concerning all-cause mortality over a seven-year period. We fit several models to adjust for potential confounders that might contribute to a high risk of mortality in SLE patients and performed sensitivity analyses accounting for competing risks of loss to follow-up. Additionally, although the SLE-specific risk adjustment index was developed as a specific risk-adjustment index for in-hospital mortality, we successfully applied the SLE-specific risk adjustment index to the Medicaid population and found that it captured SLE-related comorbidity and was itself a predictor for mortality.

A limitation of our study is that it is a prevalent cohort that by definition includes both incident cases, and prevalent cases who have survived. Our estimates cannot address variation by race/ethnicity in mortality anchored at diagnosis and the follow-up period was relatively short at less than four years on average. We did not have access to specific causes of death. Moreover, while we did adjust for many demographic and clinical factors, we were unable to account for SLE disease duration or activity, manifestations or SLE organ damage. Based on work by others, we have developed and used this definition of SLE in past studies, and it is very similar to administrative definitions used in other cohorts, and stringent in that it requires three diagnosis each 30 days apart to eliminate “rule out SLE” and subsequent follow-up visits[1,42]. While these data have not been directly validated in Medicaid, our estimates of SLE prevalence, overall and in demographic strata, are very close to those published in the CDC-funded epidemiology projects in Michigan and Georgia, providing external validation of our methods[2,43]. (For example, among Black women in Medicaid we found the prevalence of SLE to be 286/100,000[1]. Among Black women unrestricted by medical insurance type, in Georgia it was reported to be 241/100,000 and in Michigan it was 181/100,000)[2,43]. Again, we also acknowledge that self-reported race/ethnicity is imperfect and we were bound by the system of reporting race/ethnicity category used by the Medicaid system, which does not correspond to that of the U.S. Census. Finally, results from this U.S. Medicaid population may not be generalizable to populations abroad or higher SES groups.

In conclusion, even within a relatively short time window of less than three years of average follow-up, we have found marked variation in mortality rates and adjusted hazards ratios among Medicaid patients with SLE according to patient race/ethnicity. Documenting and understanding this variation is important for determining prognoses for individual patients, as well as for further investigation into the root causes of such variation in mortality, including genetic and environmental factors. Further research is needed to identify the mechanisms mediating observed variation among SLE patients. Ultimately, the goal is understand the factors contributing to increased mortality in SLE, in order to modify risk factors and provide tailored therapies to enhance survival.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Characteristics at Index Date of 42,221 Medicaid Recipients with SLE (2000–2006)

	All patients n (%)	White n (%)	Black n (%)	Hispanic n (%)	Asian n (%)	Native American n (%)
Total number of patients	42,221	16,219	16,956	6,489	1,880	677
Female	39,320 (93.13)	15,040 (92.73)	15,859 (93.53)	6,058 (93.36)	1,740 (92.55)	623 (92.02)
Age, years (mean, SD)	38.13 (12.29)	40.23 (12.26)	36.91 (11.86)	36.32 (12.40)	36.99 (13.23)	38.66 (12.27)
Residential region						
Midwest	8,324 (19.72)	4,011 (24.73)	3,582 (21.13)	466 (7.18)	97 (5.16)	168 (24.82)
Northeast	8,296 (19.65)	3,149 (19.42)	2,937 (17.32)	1,903(29.33)	235 (12.50)	72 (10.64)
South	16,028 (37.96)	5,363 (33.07)	8,621 (50.84)	1,715 (26.43)	154 (8.19)	175 (25.85)
West	9,573 (22.67)	3,696 (22.79)	1,816 (10.71)	2,405 (37.06)	1,394 (74.15)	262 (38.70)
Comorbidities*						
Previous Angina	5,116 (12.12)	2,174 (13.40)	1,965 (11.59)	714 (11.00)	197 (10.48)	66 (9.75)
Previous CABG	441 (1.04)	142 (0.88)	185 (1.09)	77 (1.19)	30 (1.60)	–
Previous CVA	1,568 (3.71)	547 (3.37)	758 (4.47)	182 (2.80)	56 (2.98)	25 (3.69)
Coronary atherosclerosis	6,412 (15.19)	2,737 (16.88)	2,483 (14.64)	886 (13.65)	233 (12.39)	73 (10.78)
Previous MI	1327 (3.14)	423 (2.61)	505 (2.98)	118 (1.82)	24 (1.28)	20 (2.95)
Previous PCI	94 (0.22)	660 (4.07)	18 (0.11)	16 (0.25)	–	–
Diabetes mellitus	10,264 (24.31)	3,792 (23.38)	4,237 (24.99)	1,623 (25.01)	414 (22.02)	198 (29.25)
Heart failure	3,827 (9.06)	1,299 (8.01)	1,990 (11.74)	386 (5.95)	96 (5.11)	56 (8.27)
Hypertension	13,081 (30.98)	4,644 (28.63)	6,215 (36.65)	1,616 (24.90)	476 (25.32)	130 (19.20)
Obesity	5,857 (13.87)	2,346 (14.46)	2,467 (14.55)	803 (12.37)	138 (7.34)	103 (15.21)
Smoking	6,579 (15.58)	3,646 (22.48)	2,226 (13.13)	440 (6.78)	88 (4.68)	179 (26.44)
Lupus nephritis	8,191 (19.40)	1,988 (4.71)	4,099 (9.71)	1,418 (3.36)	545 (1.29)	141 (0.33)
SLE risk adjustment index¥ (mean, SD)	3.65 (4.19)	3.52 (4.28)	3.93 (4.22)	3.31 (3.92)	3.32 (3.86)	3.81 (4.19)

CABG: coronary artery bypass graft, CVA: cerebrovascular accident, MI: myocardial infarction, PCI: percutaneous coronary intervention

*Comorbidities collected at any time during the follow-up, cell sizes under 11 suppressed per Centers for Medicare and Medicaid policy

¥SLE specific index ranges from 0–46.

Table 2

Crude Incidence Rates for Death among SLE and LN patients, stratified by Race/Ethnicity

	Adult SLE Patients, n= 42,221				
	Number of Patients	**Number of Events**	**Person-years, mean (SD)**	**MR* (95% CI)**	**MR Ratio (95%CI)**
All patients	42,221	2,058	2.56 (1.99)	19.07 (18.36–19.91)	
White	16,219	824	2.41 (1.97)	20.17 (18.84–21.60)	1.0 (Ref)
Black	16,959	1,040	2.54 (1.98)	24.13 (22.71–25.64)	1.19 (1.09–1.31)
Hispanic	6,489	119	2.58 (2.00)	7.12 (5.95–8.52)	0.35 (0.29–0.43)
Asian	1,880	29	2.98 (2.15)	5.18 (3.60–7.45)	0.26 (0.18–0.37)
Native American	677	46	2.47 (1.94)	27.52 (20.61–36.74)	1.36 (1.01–1.83)

	Adult Lupus Nephritis Patients, n= 8,191				
	Number of Patients	**Number of Events**	**Person-years, mean (SD)**	**MR* (95% CI)**	**MR Ratio (95%CI)**
All patients	8,191	774	2.12(1.77)	44.64 (41.60- 47.90)	
White	1,988	230	2.16(1.80)	53.49 (47.01–60.87)	1.0 (Ref.)
Black	4,099	461	2.05(1.72)	54.95 (50.16–60.20)	1.03 (0.88–1.20)
Hispanic	1,418	52	2.12(1.76)	17.33 (13.21–22.74)	0.32 (0.24–0.44)
Asian	545	18	2.46(1.95)	13.45 (8.47–21.35)	0.25 (0.16–0.41)
Native American	141	13	2.19(1.72)	42.05 (24.42–72.42)	0.79 (0.45–1.37)

* MR = annual mortality rate per 1,000 person years, SD= Standard deviation

Table 3

Hazard Ratios (HR) for Death in Adult SLE patients and Lupus Nephritis patients

	SLE, n= 42,221			Lupus Nephritis, n=8,191		
Race/Ethnicity	**Model A (HR, 95%CI)**	**Model B* (HR, 95%CI)**	**Model C** (HR, 95%CI)**	**Model A (HR, 95%CI)**	**Model B* (HR, 95%CI)**	**Model C** (HR, 95%CI)**
White	1.0 (Ref)	1.0 (Ref)	1.0 (Ref)	1.0 (Ref)	1.0 (Ref)	1.0 (Ref)
Black	**1.36 (1.24–1.49)**	**1.25 (1.13–1.37)**	**1.21 (1.10–1.33)**	**1.18 (1.00–1.38)**	1.13 (0.95–1.34)	1.04 (0.88–1.23)
Hispanic	**0.41 (0.34–0.50)**	**0.50 (0.41–0.61)**	**0.48 (0.40–0.59)**	**0.39 (0.29–0.52)**	**0.48 (0.35–0.65)**	**0.44 (0.32–0.59)**
Asian	**0.30 (0.21–0.43)**	**0.62 (0.42–0.90)**	**0.59 (0.40–0.86)**	**0.31 (0.19–0.50)**	0.68 (0.41–1.13)	**0.60 (0.37–1.00)**
Native American	**1.43 (1.06–1.92)**	**1.51 (1.12–2.04)**	**1.40 (1.04–1.90)**	0.85 (0.49–1.49)	0.86 (0.49–1.52)	0.85 (0.48–1.50)

Model A: age (continuous) and sex

*__Model B__: Model A + region of residence, calendar year, area-level socioeconomic status, comorbidities *at study index date* including history of angina, coronary artery bypass graft, coronary atherosclerosis, percutaneous coronary intervention, hypertension, smoking and obesity and SLE-specific index at study index date

**__Model C__: Model A + region of residence, calendar year, area-level socioeconomic status, comorbidities *at any point* including previous history of angina, coronary artery bypass graft, coronary atherosclerosis, percutaneous coronary intervention, acute myocardial infarction, hypertension, smoking and obesity and SLE-specific index through last follow-up