Ethnic Variation in Disease Patterns and Health Outcomes in Systemic Lupus Erythematosus

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ABSTRACT. ***Objective.*** In a single-center multiethnic lupus cohort, to investigate the influence of ethnicity on the prevalence of cumulative renal and central nervous system (CNS) lupus disease and damage, overall end-organ damage, and mortality.

Methods. Clinical features, end-organ damage, and mortality were compared by ethnic origin among patients at a lupus clinic followed prospectively in a longitudinal design over a 32-year period. Statistical analysis to compare demographic features, cumulative disease manifestations, and damage included chi-square test as well as linear, logistic, and Poisson regressions adjusting for disease duration, age at diagnosis, and presence of dialysis and hypertension. Kaplan-Meier and proportional hazard analyses were performed to compare survival.

Results. There were a total of 1017 patients: 853 Caucasian, 88 African-Canadian, and 76 Chinese-Canadian. Age at diagnosis was younger and disease duration was shorter for Chinese-Canadians compared to Caucasians, but similar between African-Canadians and Caucasians. There was no significant difference in CNS disease, comparing Caucasians to Chinese-Canadians. However, CNS disease was greater in African-Canadians than Chinese-Canadians. There was no significant difference between ethnic groups in CNS damage. Renal disease was more common in African-Canadians than Caucasians, with no significant difference between Caucasian and Chinese-Canadian patients. Renal damage was more common in African-Canadians and Chinese-Canadians than Caucasians. There was no significant difference in mortality among the 3 ethnic groups.

Conclusion. In this single referral center cohort study, there was no significant difference in CNS damage or mortality among the 3 ethnic groups. African-Canadians had a higher prevalence of renal disease and damage. Further investigation into other determinants such as genetic predisposition, treatment, and cultural perceptions is needed. (First Release Aug 15 2006; J Rheumatol 2006;33:1990–5)

Key Indexing Terms:
SYSTEMIC LUPUS ERYTHEMATOSUS | LUPUS | ETHNICITY
CENTRAL NERVOUS SYSTEM | RENAL DISEASE

Identification of variations in disease manifestations, course of illness, and prognosis among patients of differing ethnicity is increasingly common in the medical literature[1-6]. Ethnicity is thought to be a marker for genetic, environmental, behavioral, and other variables that may affect disease outcomes. There is considerable literature suggesting systemic lupus erythematosus (SLE) disease variations among African-Americans[7,8], Hispanics[7,8], Chinese[9], Arabs[10], Koreans[11], and indigenous peoples[12] compared to Caucasians. In particular, organ-specific disease prevalence, severity, and mortality have been reported to be greater in lupus patients of African and Chinese origin.

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Two studies suggest that in the United States there is an increased age-adjusted mortality rate for Chinese patients compared to Caucasians[13,14]. Thumboo, *et al* found a trend toward increased risk of developing proteinuria and central nervous system (CNS) disease over time among Chinese patients compared to Caucasians[15]. Others have reported a higher prevalence of renal disease and damage among Asians compared to Caucasians[9,16].

With regard to SLE patients of African ancestry, renal involvement is the subject of some controversy. There have been a number of studies reporting an increased prevalence of renal disease and renal damage among African-Americans compared to Caucasians[17-19]. Data from Petri, *et al*, however, failed to support an independent association of African-American ethnicity with morbidity in SLE[20]. They also reported that other classic measures of socioeconomic status such as education, income, and occupation, did not confound the relationship of ethnicity to morbidity, but suggested that medical compliance and type of medical insurance are important factors related to morbidity.

We investigated these issues in the University of Toronto Lupus Clinic. Established in 1970, it is a multiethnic, longitudinal, observational cohort study of over 1100 patients who

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are assessed and treated according to a standard protocol. The city of Toronto is a multicultural urban center with Chinese-Canadians and African-Canadians making up 10.6% (n = 259,710) and 8.3% (n = 204,075) of the population, respectively[21]. Ninety percent of Chinese-Canadians trace their origins to Hong Kong (45.6%), mainland China (27.7%), Taiwan (11.8%), and Vietnam (5.2%)[22]. African-Canadians are largely of Caribbean origin (over one-third are of Jamaican origin) and to a lesser extent of African origin (particularly Somalia)[21]. In Canada, all citizens and landed immigrants have access to universal healthcare coverage, which allows for consultations with primary care physicians and rheumatologists, free of cost to the patient. Thus our analysis would not be affected by variations in access to care based on type of medical coverage.

With the unique advantages of (1) comparing different ethnic groups within the same center, and (2) reduction in confounding by differential access to care, we undertook a study assessing the relationship of ethnicity to variations in disease manifestations and patient outcomes. Specifically, we investigated the association of renal disease and damage, CNS disease and damage, overall end-organ damage, and mortality to ethnicity in the 3 largest ethnic groups in our cohort, Caucasian, African-Canadian, and Chinese-Canadian patients.

MATERIALS AND METHODS

Patient population. Patients with SLE were identified from patients followed in the University of Toronto Lupus Clinic, University Health Network. They were followed longitudinally every 2 to 6 months according to a standard protocol that included demographic data (sex, more than high school education), clinical manifestations (including age at diagnosis, comorbidities, and treatments) and laboratory features. Disease duration was calculated from date of diagnosis. Half the patients in the cohort presented within 1 year of diagnosis. Ethnicity was self-declared by the patient at first visit. African-Canadian patients predominantly originated from the Caribbean, whereas most Chinese-Canadian patients were Han Chinese originating from Hong Kong.

Outcome measures. The outcome measures of interest included renal disease, CNS disease, overall end-organ damage, renal damage, CNS damage, and mortality. Renal disease was defined as any of sterile hematuria, sterile pyuria (both in the absence of other causes)[23], casts, proteinuria, elevated serum creatinine, reduction in glomerular filtration rate, and renal biopsy showing lupus nephritis. CNS disease was defined as any of organic brain syndrome, psychosis, stroke, seizures, visual disturbance, cranial nerve disorder, and lupus headache. Renal damage, CNS damage, and overall end-organ damage were defined by the Systemic Lupus International Collaborating Clinics (SLICC) damage index (SDI) criteria[24]. Studies assessing the validity and reliability of the SDI have been published[16,25,26]. Approval for the study was obtained from the University Health Network Research Ethics Board.

Statistical analysis. Demographic values are presented as percentages or mean ± standard deviation. Chi-square tests were used to compare demographic features between ethnic groups. Both linear and logistic regressions were used to compare demographic features and disease manifestations between ethnicity while adjusting for disease duration, age at diagnosis, presence of diabetes, and hypertension. Damage was measured using the SDI and had non-negative whole-numbers scores. All patients started with a SDI score of 0 at the time of SLE diagnosis and it increased as damage accumulated. Most patients had no to little damage, consequently the SDI followed a Poisson distribution. Poisson regression is designed to model such outcomes and was used to compare damage (CNS, renal, and overall end-organ) between ethnic groups. In each of the regression models, ethnicity was an independent variable and dummy variables were created to compare African-Canadians and Chinese-Canadians to Caucasians. Kaplan-Meier curves and proportional hazard analysis adjusting for age at diagnosis were performed to compare survival in the 3 ethnic groups. All analyses were carried out using SAS 8.0 (SAS Institute, Cary, NC, USA).

RESULTS

There were 1017 patients included in the analysis. The 3 largest ethnic groups were Caucasians, African-Canadians, and Chinese-Canadians, with 853, 88, and 76 patients, respectively. There were also 25 Filipinos and 33 patients with ethnicity described as "other"; however, they were excluded from the analysis as their sample sizes were small.

The proportion of female patients was similar across all 3 ethnic groups, ranging from 85.5% to 94.3%. The age at diagnosis for Caucasians and African-Canadians was similar, with mean ages of 33.1 and 31.4 years, respectively. Chinese-Canadian patients were diagnosed at an earlier age, with a mean age of 27.7 years. Disease duration at the time of the study was similar for Caucasians and African-Canadians, with means of 12.5 and 11.0 years, but was slightly less for Chinese-Canadians, with a mean of 10.2 years. Duration of followup in the clinic was statistically longer for Caucasians (8.9 yrs) than either African-Canadians (7.7 yrs) or Chinese-Canadians (6.0 yrs) ($p = 0.005$). There was no statistically significant difference in the use of steroids, antimalarials, or immunosuppressive agents, or specific immunosuppressives, across the 3 ethnic groups. Diabetes and hypertension occurred most frequently among African-Canadians (Table 1).

Renal disease occurred more commonly among African-Canadians (83.7%) compared to Caucasian patients (70.9%). The frequency of renal disease was similar between Caucasians and Chinese-Canadian patients, at 70.9% and 69.7%. CNS disease was most common among African-Canadians (59.3%) followed by Caucasians (48.9%). CNS disease occurred in 38.2% of Chinese-Canadians. When the analysis was adjusted for age at diagnosis and disease duration, the observed differences were significant for CNS (odds ratio 1.69, 95% CI 1.10, 2.70, $p = 0.03$) and renal disease in African-Canadians compared to Caucasians only (OR 2.17, 95% CI 1.20, 4.00, $p = 0.01$]. When the analysis was adjusted further by the presence of diabetes and hypertension, these differences were no longer significant (Table 2). Mortality rates were similar across all 3 ethnic groups, ranging from 14.5% to 19.3% (Table 2). No significant differences were found by survival analysis using Kaplan-Meier curves (log-rank test $p = 0.76$; Figure 1). Similarly, there were no significant differences in survival adjusted for age at diagnosis, diabetes, and hypertension, using proportional hazard modeling, between African-Canadians ($p = 0.35$) and Chinese-Canadians ($p = 0.32$) when compared to Caucasians (Table 2).

The overall damage measured by SDI score was statistically higher ($p < 0.001$) among African-Canadian (1.85 ± 1.99) than among Caucasian (1.62 ± 2.06) or Chinese-

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Table 1. Patient demographics.

	Caucasian	African-Canadian	Chinese-Canadian	p
Sample size, n	853	88	76	
Female sex, n (%)	739 (86.6)	83 (94.3)	65 (85.5)	0.11*
More than high school education, n (%)	290 (57.2)	32 (53.3)	37 (64.9)	0.42*
Age at diagnosis, yrs	33.1 ± 14.1	31.4 ± 12.2	27.7 ± 12.7	0.004†
Disease duration, yrs (at last visit or at time of death)	12.5 ± 9.4	11.0 ± 7.9	10.2 ± 8.5	0.03†
Duration of followup in clinic, yrs	8.9 ± 8.1	7.7 ± 6.6	6.0 ± 6.0	0.005†
Treatment — ever				
Steroids, n (%)	676 (79.3)	73 (84.9)	65 (85.5)	0.22*
Antimalarials, n (%)	531 (62.3)	57 (66.3)	41 (54.0)	0.25*
Immunosuppressives, n (%)	368 (43.2)	43 (50.0)	32 (42.1)	0.46*
Imuran, %	67.9	81.4	81.3	
Cyclophosphamide, %	12.0	25.6	18.8	0.07*
Methotrexate, %	26.4	20.9	9.4	
Comorbidities				
Diabetes, n (%)	58 (6.8)	19 (22.1)	5 (6.7)	< 0.0001*
Hypertension††, n (%)	491 (57.6)	60 (69.8)	37 (48.7)	0.022*

* Chi-square test; † linear regression comparing ethnicity. †† Hypertension defined as "on antihypertensive medication" or diastolic blood pressure > 90 mm Hg or systolic blood pressure > 140 mm Hg on at least one occasion.

Table 2. Disease manifestation by ethnic group.

	Caucasian	African-Canadian	Chinese-Canadian
Renal			
n (%)	605 (70.9)	72 (83.7)	53 (69.7)
OR (95% CI)†	1.00	1.64 (0.88, 3.03)	1.02 (0.60, 1.79)
p†	NA	0.12	0.93
CNS			
n (%)	417 (48.9)	51 (59.3)	29 (38.2)
OR (95% CI)†	1.00	1.47 (0.93, 2.38)	0.71 (0.43, 1.18)
p†	NA	0.10	0.18
Mortality			
n (%)	157 (18.4)	17 (19.3)	11 (14.5)
Time to death (year)	12.4 ± 9.4	10.1 ± 7.9	10.0 ± 9.2
Hazard ratio** (95% CI*)	1.00	1.28 (0.76, 2.13)	1.37 (0.74, 2.54)
p	NA	0.35	0.32

† Logistic regression comparing ethnic group to Caucasians, after adjusting for age at diagnosis, disease duration, diabetes, and hypertension. ** Proportional hazard model of time to death comparing ethnic group to Caucasians after adjusting for age at diagnosis, diabetes, and hypertension. CNS: central nervous system; NA: not applicable.

Canadian lupus patients (1.41 ± 1.60). The renal component of the SDI was higher among African-Canadians (0.20 ± 0.64) and Chinese patients (0.26 ± 0.79) compared to Caucasians (0.06 ± 0.32). This remained true even after adjustment for diabetes and hypertension in addition to age at diagnosis. There was no statistically significant difference in the SDI-CNS score across the 3 ethnic groups, with mean scores ranging from 0.10 to 0.16 (Table 3).

DISCUSSION

Identification of variations in target organ predilection, accrual of damage, and mortality across patients of differing ethnicity has implications for treatment, monitoring, and prognostication. Although we found increased renal damage among Chinese-Canadians, we found no statistical difference in the frequency of renal disease, CNS disease, overall damage, CNS damage, and mortality in Chinese-Canadian patients with SLE compared to Caucasians. This is in contrast to previous reports[9,13,14,27]. Although Thumboo, *et al* found a trend toward more proteinuria and CNS disease in Chinese patients with SLE, they compared Chinese patients in Singapore to Caucasian patients in Rochester, Minnesota, USA. Our results reflect a direct comparison of Chinese-Canadian and Caucasian patients treated at the same clinic, with a more homogenous environment, as opposed to a comparison of 2 populations in 2 different countries.

We also found an increased frequency of renal disease, renal damage, CNS disease, and overall damage in African-Canadians compared to Caucasians, despite similar healthcare coverage and education. The increased frequency of renal dis-

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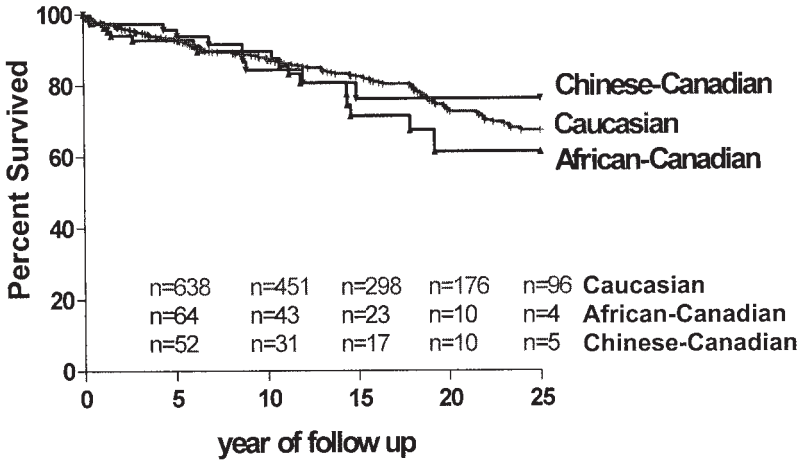

Figure 1. Kaplan-Meier curves illustrate survival by ethnic group. *Log-rank test p = 0.76.

Table 3. End-organ damage and overall damage by ethnic group.

	Caucasian, n = 734 (86.0%)	African-Canadian, n = 80 (90.9%)	Chinese-Canadian, n = 70 (92.1%)
Overall SDI	1.62 ± 2.06	1.85 ± 1.99	1.41 ± 1.60
PE ± SE	1.00	0.12 ± 0.09	0.11 ± 0.11
p*	NA	0.17	0.28
SDI-Renal	0.06 ± 0.32	0.20 ± 0.64	0.26 ± 0.79
PE ± SE	1.00	0.95 ± 0.30	1.47 ± 0.29
p*	NA	0.002	< 0.0001
SDI-CNS	0.16 ± 0.49	0.11 ± 0.36	0.10 ± 0.35
PE ± SE	1.00	–0.19 ± 0.35	–0.15 ± 0.39
p*	NA	0.60	0.70

SDI: SLICC damage index; PE ± SE: parameter estimate ± standard error; CNS: central nervous system; NA: not applicable. * Poisson regression comparing ethnic group to Caucasians after adjusting for age at diagnosis, disease duration, diabetes, and hypertension.

ease and renal damage is concordant with previous reports; however, it appears to contrast with reports that suggest the disparity is related to socioeconomic factors of type of healthcare coverage and education[18,28-31]. Our study suggests there may be other confounding factors such as genetic predisposition and behavioral variables including nonadherence to medication[32,33]. Hypertension and diabetes occurred more frequently among the African-Canadian lupus patients compared to the other 2 ethnic groups. Indeed, the adjustment for the presence of these 2 factors to the analyses showed that neither renal disease nor CNS disease was found statistically more frequently among the African-Canadians than the Caucasians. However, renal damage remained higher among African-Canadians even after adjustment for diabetes and hypertension. The increased frequency of these comorbid illnesses in people of African heritage, after adjustment for age, sex, and education level, has been described previously[34].

We also found no significant difference in either unadjusted mortality or age-adjusted survival time across the 3 ethnic groups. Our findings contradict the notion that non-Caucasian ethnicity is a risk factor for death from SLE. A report from the US Centers for Disease Control found that between 1979 and 1998 the mortality rate increased by roughly 70% among African-American women[35]. It may be that in the United States, the prognosis of African-American women is heavily confounded by socioeconomic status and access to care. Our results suggest that despite differences in end-organ disease and damage across ethnic groups, access to similar care and similar therapy equalizes the risk of death.

A potential limitation of our study is that despite universal healthcare in Canada, there continue to be disparities in healthcare outcomes based on socioeconomic status[36]. For this study, education was an indicator of socioeconomic status, as it has been shown to be a more stable determinant of socioeconomic status than income, which changes with time and disease onset[37]. Although education is an excellent marker of socioeconomic status, one should bear in mind that its value diminishes if not measured accurately. In our study, level of education was similar across all 3 ethnic groups. With similar education levels and similar healthcare coverage, we

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feel our study was relatively less confounded by access to care issues than studies performed in the USA, where lack of access to private insurance or government-run programs (which include both Medicare and Medicaid) is believed to have a negative effect on health outcomes[37,38].

This is an observational study and interpretation of results should be limited to associations between variables of interest. No causal inferences should be drawn. Further, SLE disease manifestations and damage may be confounded by a multitude of other factors. The constraints of feasibility limit data collection in observational studies. Future researchers should consider collecting data on other factors that may influence disease manifestations, such as measures of medication compliance, use of alternative therapies, cultural perceptions of healthcare, or genetic/proteonomic markers of those who are more likely to respond to therapy.

Finally, our study may reflect site-confounding, given that our results describe disease manifestations and outcomes in a single tertiary care institution. The proportion of Chinese-Canadian and African-Canadian patients reflects similar proportions of these 2 ethnic groups within our city. However, the patients in our cohort may have more severe disease than patients followed in the community due to referral bias. Thus the results are more generalizable to patients in tertiary care lupus specialty clinics in urban multiethnic centers.

In the University of Toronto multiethnic cohort of 1017 patients, we found renal damage, adjusting for age at diagnosis and disease duration, and overall damage occurred more frequently among African-Canadians than Caucasian patients with SLE. Renal damage was also more frequent among Chinese-Canadians than Caucasians. CNS disease was more frequent among African-Canadians than among Caucasians. There was no significant difference in mortality among the 3 ethnic groups. Further investigation into other determinants such as genetic predisposition, comorbid illness, treatment, and cultural perceptions is needed.

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