

Ethnic difference in the prevalence of systemic lupus erythematosus

H. H. HART, R. R. GRIGOR, AND D. E. CAUGHEY

From the Department of Rheumatology, Auckland Public Hospital, Park Road, Auckland 1, New Zealand

SUMMARY A retrospective study of systemic lupus erythematosus (SLE) was carried out in Auckland, New Zealand, for the years 1975 to 1980 inclusive. One hundred and fifty-one patients were found of which 106 fulfilled the American Rheumatism Association criteria. There were 15 deaths. Age-adjusted prevalence rates per 100 000 were estimated for all cases at the end of 1980 as follows: white 14.6, Polynesian 50.63, and other 19.11. There was a preponderance of females in each ethnic group, average 87% of all cases. Age-adjusted mortality rates per million patient years were: whites 2.5 and Polynesians 13.0. Survival curves for both ethnic groups were similar. The prevalence rate in New Zealand Polynesians is significantly higher than in white counterparts which confirms the observation that the disease is commoner in the pigmented races.

Epidemiological studies have shown or suggested ethnic differences in SLE prevalence rates. Studies in New York City,¹ San Francisco,² and Jefferson County, Alabama,³ have shown a 2.4 to 3.3 times higher prevalence of the disease in blacks than in whites. Hospital based studies in West Malaysia⁴ and Singapore⁵ have suggested a greater prevalence in orientals. A similar high prevalence rate has been claimed for blacks in Jamaica,⁶ and a recent study from Hawaii⁷ has shown rates for orientals and Polynesians 3 to 4 times that for whites.

Ethnic differences in mortality rates from systemic lupus erythematosus have also been found. In the United States⁸ from 1968 to 1972 the rates for white and black females were 5.2 and 14.8 per million person years respectively. The overall rate for blacks was 2.6 times that for whites. Similar rates have been demonstrated for the period 1972 to 1976.⁹

This study describes the results of a retrospective survey of the ethnic differences of SLE in Auckland, New Zealand. Auckland is an appropriate location to conduct such a study, because there is a large population of both whites and Polynesians available for comparison. The National Census statistical area corresponds to that serviced by the 3 main hospitals. The same area is serviced by a central histopathology and immunology laboratory, and there is close liaison between these hospitals and physicians not based on hospitals.

Patients and methods

Medical records with a discharge diagnosis of SLE were retrieved from the 3 general hospitals for the period 1 January 1975 to 1 January 1981. The records of patients with significant antinuclear antibody results and renal biopsies consistent with SLE were also traced. A questionnaire was circulated among all physicians not based on hospitals in order to collect all those patients who had not been admitted to hospital. Government death records with SLE listed as the primary cause of death were also reviewed. The final list was checked against the national death register to exclude unrecorded deaths. Patients referred for hospital treatment from outside the area were excluded (Table 1).

Ethnic group was self-declared in both the National Census and hospital record data. The Polynesian ethnic group is made up of Maori, Pacific Islander, and people of mixed blood.

The criteria suggested by Fries and Holman¹⁰ were used to identify patients. These criteria require a multisystem disease with specific manifestations involving more than one system, the presence of appropriate autoantibodies, and the specific exclusion of other diseases with similar features.

The American Rheumatism Association (ARA) preliminary criteria¹¹ were also applied to all patients. Patients with 4 or more criteria were classified as definite SLE and those with 3 or fewer as possible SLE. The data used for subsequent calculations in this study were based on those patients identified by the Fries and Holman criteria.¹⁰

Table 1 Population estimates for Auckland by age, sex, and ethnic group (1976 National Census)

Age	Sex	White	Polynesian	Other	Total
0-14	M	88 745	23 679	5275	117 699
	F	84 080	22 456	5094	111 630
15-24	M	56 537	11 146	3233	70 916
	F	55 797	11 853	2825	70 475
25-34	M	49 073	7600	2627	59 300
	F	50 632	8294	2355	61 281
35-44	M	37 304	4991	1667	43 962
	F	36 316	5098	1564	42 978
45-64	M	66 084	4059	2721	72 864
	F	67 246	4161	3264	74 671
65+	M	27 275	682	1735	29 692
	F	38 132	859	2908	41 899
Total	M	325 017	52 157	17 258	394 433
	F	332 203	52 721	18 010	402 934

The prevalence of SLE was estimated by counting all patients found and subtracting deaths occurring during the period. These figures were related to the population estimates derived from the 1976 National Census. Age-specific mortality rates for SLE were calculated from the white population as standard. The significance of the difference between groups was determined.¹² Survival curves were constructed for both ethnic groups.¹³

Results

One hundred and fifty-one patients with SLE were identified by the criteria of Fries and Holman.¹⁰ There were 15 deaths, leaving 136, including 96 patients with 4 or more ARA criteria. Females predominated, making up 123 of the remaining 136 patients. Although whites constitute 83% of the Auckland population, only 71% of the definite cases of SLE were from this group. The incidence of lupus manifestations for Polynesians, whites, and all Auckland cases was compared with those in a series of mixed race compiled by rheumatologists¹¹ (Table 2). Symptoms which may not be volunteered by the patient and which may require specific questions to discover, such as photosensitivity, alopecia, and pleuritis, were less frequently recorded, but this is to be expected in a retrospective study. Similarly manifestations requiring special skills for their detection, such as discoid lupus, arthritis, and urinary casts, were also less frequent than in the reference series. Notable ethnic differences were the lower incidence of Raynaud's phenomenon, photosensitivity, and arthritis in the Polynesian group and a higher incidence of chronically false positive VDRL (Venereal Disease Reference Laboratories test), haemolytic anaemia and thrombocytopenia. Psychosis was lower

Table 2 Percentage frequency of ARA criteria in whites, Polynesians, and the patient group used to construct the ARA criteria*

Criteria	ARA group (245)	Polynesian group (34)	White group (96)	All patients (136)
Facial erythema	63.7	50	58.3	58.3
Discoid	17.1	5.8	2.1	2.9
Raynaud's disease	20.0	14.7	36.5	30.1
Alopecia	43.3	26.5	21.9	23.5
Photosensitivity	36.7	20.6	31.3	27.9
Oral ulceration	15.1	23.5	12.5	15.4
Arthritis	88.9	58.8	69.8	68.3
LE cells	91.8	44.1	41.7	44.9
False positive				
VDRL	11.8	23.5	11.5	14.0
Proteinuria	19.6	26.5	16.7	19.9
Casts	47.8	29.4	11.5	17.6
Pleuritis	60.4	41.2	40.6	41.9
Pericarditis	18.8	17.6	10.4	15.4
Psychosis	19.2	2.9	5.2	7.4
Convulsions	12.7	8.8	2.8	4.4
Haemolytic				
anaemia	16.3	17.6	3.1	6.6
Leucopenia	39.6	47.1	35.4	36.0
Platelets	11.4	35.3	11.5	16.2

*Number if cases shown in parentheses.

in all groups, probably because the retrospective differentiation of cerebral lupus from steroid psychosis and reactive depression was extremely difficult. None of these differences was statistically significant (Table 3).

The prevalence rates estimated for the end of 1980 are shown in Table 4. The overall rate for Auckland was 18 per 100 000, with rates for women aged 15 to

Table 3 Patients with the diagnosis of SLE resident in Auckland on 1 January 1981*

Age	White		Polynesian		Other		Total	
	M	F	M	F	M	F	M	F
0-14	(0)	(0)	(0)	(0)	(0)	(0)	(0)	(0)
	0	0	0	0	0	0	0	0
15-24	(1)	(7)	(1)	(11)	(0)	(0)	(2)	(18)
	0	0	1	1	0	3	1	4
25-34	(3)	(32)	(1)	(9)	(1)	(3)	(5)	(44)
	0	11	0	3	0	0	0	14
35-44	(3)	(12)	(1)	(3)	(0)	(2)	(4)	(17)
	2	2	0	1	0	0	2	3
45-64	(2)	(28)	(0)	(6)	(0)	(0)	(2)	(34)
	2	9	0	2	0	0	2	11
65+	(0)	(8)	(0)	(2)	(0)	(0)	(0)	(10)
	0	3	0	0	0	0	0	3
Total	(9)	(87)	(3)	(31)	(1)	(5)	(13)	(123)
	4	25	1	7	0	3	5	35

*The figure in parentheses represents the total number of patients and the figure below the number fulfilling fewer than 4 ARA criteria.

Table 4 Estimated prevalence of SLE in Auckland at the end of 1980

Age	White		Polynesian		Other		Total	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate
0-14	0	0	0	0	0	0	0	0
15-24	8	7.12	12	52.2	0	0	20	14.14
25-34	35	35.1	10	62.9	4	80.28	49	40.63
35-44	15	20.4	4	39.64	2	61.9	21	24.15
45-64	30	22.5	6	72.99	0	0	36	24.4
65+	8	12.2	2	129.8	0	0	10	13.96
Total	96	14.6	34	32.41	6	17.01	136	17.05
Age adjusted		14.6		50.63		19.11		17.62

64 of 45 per 100 000 and Polynesian women in the same age group 99 per 100 000. Age-adjusted rates show that SLE is 3.5 times commoner in Polynesians than whites (50.63 versus 14.6 per 100 000), and this is statistically significant (chi-square 32.2, $p < 0.001$). The difference in rate occurs in all age groups, but there is significant inhomogeneity (Mantel-Haenszel test, chi-square 20.5, $p < 0.001$) between age groups due to sample size. The mean age at onset for whites was 32 years and Polynesians 25.3 years. This difference may be partly explained by the different age distribution in the 2 populations. The 'other races' category, which is mainly Chinese and Indian, shows an intermediate prevalence rate of 19.11 per 100 000 (Table 4).

Age-specific mortality rates for SLE were calculated from the white population as standard. This procedure showed the rate for Polynesians to be 13.0 and whites 2.5 per million person years. Survival curves were constructed which showed the Polynesian 6-year survival to be 87% and whites 96%. This difference was not significant (log rank test), suggesting that there is no difference in the severity of the disease in the 2 populations.

Discussion

A study from San Francisco² of members of a prepaid health plan that cares for 125 000 people showed the prevalence of definite SLE to be 51 per 100 000. The rate for women aged 15 to 64 years was 142 per 100 000 and in black women in the same age group 408 per 100 000. It should be noted that in this study the investigators had access to complete medical records. Similar ethnic differences have been confirmed by other studies, but the prevalence rates have been considerably lower. This is particularly true of those based on hospital records, for this method undoubtedly leads to the exclusion of many milder

cases. In a study from Hawaii,⁷ for example, the rates for whites was 5.8 per 100 000 compared with 20 per 100 000 for Chinese, Philipinos, and Polynesians.

Our study suggests that in Auckland SLE is also substantially more common among people of black (Polynesian) ancestry. The overall prevalence rates lie somewhere between the 2 studies mentioned above, which is in keeping with our method of finding patients. Inevitably some cases in the community will have been missed, particularly in a retrospective survey.

Racial differences in the severity of the disease are more difficult to assess. Taking death as the end point we have shown that the age-specific mortality rate is substantially greater in the Polynesian group. These rates are similar to those obtained for blacks in the United States.⁹ However, life table analysis failed to demonstrate a significant difference, suggesting that Polynesian survival is no different from that of whites. The overall survival rates were similar to those observed in San Francisco.²

An explanation for these differences in prevalence is lacking. In particular the evidence from HLA tissue typing studies has been unhelpful. While there is no clearly defined association of SLE and a particular tissue type, a weak association with HLA B8 has been found in two studies.^{14, 15} However, in North America, the frequency of HLA B8 has been found to be 17.1% in whites and 5.2% in blacks.¹⁶ A preliminary study from New Zealand has found much the same situation, with HLA B8 in 24% of whites and 2% of Polynesians (Woodfield D G, personal communication).

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