Apparent predisposition to systemic lupus erythematosus in Chinese patients in West Malaysia

A. O. FRANK*

From the Department of Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia

SUMMARY In the first 9 years following the opening of the University Hospital in Kuala Lumpur nearly 130 000 patients have been admitted (excluding obstetric patients), and, of these, 175 fulfilled the American Rheumatism Association criteria for the diagnosis of systemic lupus erythematosus. This diagnosis was made significantly more frequently in Chinese patients than in other races. SLE is more often reported from Chinese communities in Asia than from India and tropical Africa. There may be a lower susceptibility to autoimmune disease in black Africans than the suspected increased susceptibility in their American Negro and West Indian descendants. A careful study of racial and geographical factors in autoimmune disease should throw further light on the interaction between the host and his environment which results in autoimmune disease.

Many advances in our understanding of the pathogenesis of systemic lupus erythematosus (SLE) have been made over the last few years, particularly in our understanding of the abnormal immunological responses seen in patients with SLE. Many people, however, believe that these immunological abnormalities are found in predisposed individuals, and this belief has been supported by animal models of SLE in dogs and mice (Talal, 1975), family and twin studies of patients with SLE (Block *et al.*, 1975; Arnett and Shulman, 1976) and the finding that various immunological abnormalities were more frequently found in relatives of patients with SLE than in controls (Larsen, 1972; Chantler *et al.*, 1973; Siegel and Lee, 1973; Lowenstein and Rothfield, 1977).

Three studies have demonstrated that SLE does not occur in the same frequency in patients of different races. Siegel and Lee (1973) and Fessel (1974) both found a higher prevalence of SLE in the American Negro population than in the white population, and the study of 'inbred American tribes' (Morton *et al.*, 1976) found a higher incidence of the disease in some tribes than in others. It seemed reasonable, therefore, to conduct a study of SLE in West Malaysia, where peoples of Malay,

Chinese, and Indian races coexist, to investigate whether SLE occurred with equal frequency in all ethnic groups in the University Hospital, Kuala Lumpur.

Patients and methods

This study was performed at the University Hospital, Kuala Lumpur, which opened in 1967, and which had had nearly 130 000 admissions (excluding obstetric admissions) up to 1 January 1976. At the time of the study it was the only teaching hospital in West Malaysia, and all patients admitted to the hospital were available for study.

In June 1974 the SLE Study Group was formed to continue to study this illness as seen in the University Hospital, as reported previously by Lau and White (1969) and Toh *et al.* (in press).

An analysis was performed of the number of patients admitted each year for each race, and all records coded with the international classification of disease (World Health Organisation, 1967) no. 734.1 (Lupus erythematosus) were analysed by year of admission. Those fulfilling the ARA criteria (Cohen *et al.*, 1971) were studied.

Results

Table 1 compares the ethnic composition of the population of patients in University Hospital with the population at large determined by the census of 1970 (Malaysia, 1972). It can be seen that the

*In conjunction with the SLE Study Group: B. A. Adam, C. G. Beng, S. M. Chu, T. P. Goh, K. S. Lau, C. T. Lee, K. Prathap, S. K. Wan, F. Wang, and J. C. White.

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Correspondence to Dr Frank, Salisbury General Infirmary, Salisbury, Wiltshire, England.

Apparent predisposition to systemic lupus erythematosus in Chinese patients in West Malaysia 267**Table 1** Comparison between ethnic groups of the population of West Malaysia (Census, 1970) and SLE patients 1967–October 1976

	Malay	Chinese	Indian	Other
% of population	53.1	35.5	10.6	0.8
% of SLE patients	10.4	80.7	8.4	0.5

 $\chi^2=187$; $P<0.001$.**Table 2** Comparison of Chinese and Indian and Malay admissions 1967–75 with and without SLE

Race	With SLE	Without SLE	Total
Chinese	142	66 357	66 499
Indian and Malay	33	59 547	59 580
Total	175	125 904	126 079

 χ^2 (Yates's correction), 55.569; $P<0.001$.**Table 3** Comparison of Indian and Malay admissions 1967–75 with and without SLE

Race	With SLE	Without SLE	Total
Indian	16	36 754	36 770
Malay	17	22 793	22 810
Total	33	59 547	59 580

 χ^2 (Yates's correction), 1.918; $P>0.1$.

hospital population differs greatly from the population at large, and consequently the conclusions from this hospital study cannot reflect the incidence of SLE in the community.

Between 1967 and October 1976, 202 patients were seen in University Hospital with SLE fulfilling the ARA criteria. Chinese patients predominated (80.7%), with Malays 10.4%, Indians 8.4%, and others 0.5%. Of these 202 patients 175 presented before 1 January 1976, the period for which control data are available.

Table 2 compares Chinese admissions with and without SLE in the University Hospital between 1967 and 1975 with Indian and Malay admissions over the same period. There was a highly significant difference ($P<0.001$). Table 3 compares Indian patients with and without SLE with Malay patients with and without SLE over the same period. In this case there was no significant difference.

Discussion

Support for the hypothesis that SLE could be influenced by racial factors was further sought by reviewing reports of SLE worldwide. Fig. 1 depicts

a map of the world with sites of some reports of SLE marked. A number denotes the size of the series. It is noted that SLE has been reported from several places in Asia. Reports from India are rare (Malaviya *et al.*, 1973; Menon *et al.*, 1976), and conflicting views are expressed as to the extent of SLE there. Few patients are actually reported, however, and this contrasts with the reports from the predominantly Chinese communities (in addition to this report) in Hong Kong (Wong, 1969) and Singapore (Tay and Khoo, 1971). A large series has also been described from Japan (Fukase and Ofuzi, 1973) of 689 patients, of whom 275 were thought to fulfil the ARA criteria (Cohen *et al.*, 1971). It may be coincidental, but the infrequent reports from India and the larger reports from Chinese communities also reflect the ethnic composition of this series.

The increased prevalence of SLE in the American Negro has been referred to (see above), and Hughes in Kingston, Jamaica, saw 81 new patients with SLE in 1 year (Hughes, 1977). This contrasts with the 2 patients seen by Greenwood (1968) in a study in Ibadan, Western Nigeria, spanning 9 years. Other reports from equatorial Africa are rare (in contrast to reports from Singapore, which is also near the equator). In South Africa, however, the general feeling that autoimmune disease is seen infrequently in black Africans (O. L. Meyers, personal communication) has been supported by 2 studies (Jessop and Meyers, 1973; Seedat and Pudifin, 1977). Although diagnostic techniques and time to report cases may be scarce in tropical Africa, it is not unreasonable to contrast the reports from Ibadan and Kingston, since both come from teaching centres in the third world. Greenwood (1968) commented that there appears to be a decreased susceptibility to autoimmune disease in West Africa compared to the increased prevalence in the American Negro descendants, and Hughes's (1977) report from Jamaica supports this. If true environmental rather than genetic factors may explain the racial differences. A comparative study of SLE in Japanese people in Japan and in America, or of West Indians in Jamaica and in London might throw further light on this. It is suggested that geographical and racial factors in SLE are worthy of further study.

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Fig. 1 Sites of some reports of SLE, each number denoting size of report

possible without the help and encouragement of my wife, who personally punched the details of 202 patients on to punch-cards and designed suitable computer analyses. I am also indebted to Mr A. Cotton from the Department of Community Medicine at Southampton University for making available the facilities of the Department, to Mr John Alexander for continuous guidance on statistical analysis, and to Dr M. I. D. Cawley and Mr John Alexander for criticism of the manuscript. I am also indebted to Mr Bob Gann and Miss Christine Taylor for providing me with the relevant literature and for help in preparing Fig. 1. Finally I thank Mrs Mary Willis for typing the manuscript.

Addendum: The recent findings by Serdula and Rhoads (1979) in an epidemiological study that the Chinese had the highest age adjusted prevalence rate for SLE among the different ethnic groups in Hawaii strongly supports our findings in a hospital population that the Chinese are more prone to SLE than the other racial groups in the study.

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