Prevalence of leprosy and lepromatous leprosy in the two populations

	Areas with onchocerciasis (n = 26)	Areas without onchocerciasis (n = 17)	Significance (Fisher's <i>t</i> test) DF = 41
Total population (census)	456 177	2 366 034	
Leprosy			
No (%) of cases in total population	5191 (11.4)	31 080 (13.1)	
" Of cases in each district:			
Mean $\pm$ SD	17.13 $\pm$ 6.99	13.74 $\pm$ 4.94	1.73 (NS)
Range	8.3-38.0	7.0-22.0	
Lepromatous leprosy			
No (%) of cases in total population	576 (1.26)	1356 (0.57)	
" Of cases in each district:			
Mean $\pm$ SD	1.32 $\pm$ 0.47	0.65 $\pm$ 0.33	5.1 (P = 0.001)
Range	0.64-2.9	0.22-1.42	

differed greatly in size we tested the difference in the prevalence of leprosy and lepromatous leprosy between the 27 southern districts and several random samples of the same size from the larger central and northern areas. In all cases results remained the same.

Comment

Our results indicate that in the two populations, in which the overall prevalence of leprosy is similar, the prevalence of lepromatous leprosy is about twice as high in the areas where onchocerciasis is hyper-endemic.

This finding seems to agree with observations of a reduced level of immunity because of onchocerciasis and with the hypothesis that a highly infected patient with onchocerciasis is more likely to develop the lepromatous form of leprosy and any other infection.

¹ Buck, A A, *Health and Disease in Chad*. Baltimore, The Johns Hopkins Press, 1970.

² Rougemont, A, *et al*, *Lancet*, 1977, **1**, 309.

³ Bartlett, A, *et al*, *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1978, **72**, 372.

⁴ Prost, A, *Distribution Cantonale des Aveugles en Haute-Volta—I map*. Geneva, WHO, 1976.

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Comparison of retinopathy in insulin-dependent diabetics with and without thyroid disease

Bottazzo *et al* recently described four "autoimmune families" containing 11 insulin-dependent diabetics (IDDs), of whom seven, aged 22-42 years and whose duration of diabetes was 12-30 years, had severe (extensive background or proliferative) diabetic retinopathy.¹ Despite the few cases studied, the relatively long durations of diabetes, and the lack of control observations they concluded that an autoimmune factor might have contributed to the retinopathy. Conversely, an earlier study suggested that coincident cretinism or juvenile myxoedema protects against microvascular diabetic complications.² We have compared the prevalence and severity of retinopathy in IDDs with and without associated biochemical evidence of thyrotoxicosis or thyroid failure to establish whether concomitant autoimmune endocrinopathy contributes to the risk of diabetic retinopathy.

Patients, methods, and results

The patients were divided into two groups according to the presence or absence of coincident thyroid disease. Group 1 comprised 60 IDDs (9 male,

51 female) with Graves's disease (12 secondary sulphonylurea failures requiring insulin more than three months from diagnosis, and 48 primary IDDs requiring insulin within three months of diagnosis) and 105 IDDs (21 male, 84 female) with thyroid failure (32 secondary sulphonylurea failures, and 73 primary IDDs). Of the patients with thyroid failure, 69 had confirmed primary hypothyroidism and 36 normal serum thyroxine concentrations but raised serum thyrotrophin concentrations. Group 2 comprised 164 IDDs (37 male, 127 female) who had normal serum thyrotrophin concentrations and no personal history of thyroid disease. Forty-six were secondary sulphonylurea failures, and 118 primary IDDs. There was no significant difference between the two groups in age at time of study or duration of diabetes (table). Statistical analysis was by χ^2 analysis.

The ocular fundi of each patient were carefully examined through dilated pupils under clinic conditions by one of us and the retinopathy state recorded as: 0, no retinopathy; 1, mild background retinopathy (small number of microaneurysms and occasional blot haemorrhage); 2, moderate to extensive background retinopathy (large numbers of microaneurysms, blot haemorrhages, hard or soft exudates or both); 3, new vessel formation; and 4, blindness due to vitreous haemorrhage or retinal disorganisation, or both, after haemorrhage or fibrosis.

Age, duration of diabetes, and retinopathy state of IDDs in groups 1 and 2 (figures are numbers of patients)

Age (years):	<20	20-29	30-39	40-49	50-59	60-69	70-79	80-89
Group 1	..	..	..	..	..	..	..	..
Group 2	..	..	..	..	..	..	..	..
Durations of diabetes (years):	5-9	10-14	15-19	20-24	25-29	30-34	35-39	>39
Group 1	..	..	..	..	..	..	..	..
Group 2	..	..	..	..	..	..	..	..
Retinopathy state:	0	1	2	3	4			
Group 1	..	..	..	..	..	..	..	..
Group 2	..	..	..	..	..	..	..	..

The table gives the retinopathy states of patients in the two groups and shows no significant difference between them. Similarly, there was no significant difference between primary IDDs and secondary sulphonylurea failures in respect of retinopathy state when groups 1 and 2 were considered independently and combined.

Comment

There is increasing evidence for the existence of a primary autoimmune type of diabetes, whose characteristics include female preponderance, a later age of onset, over-representation of HLA-B8, and a high frequency of persistent islet-cell antibody.³ Close scrutiny of such a group of diabetics is clearly required, especially in respect of susceptibility to microangiopathy but the necessary inclusion criteria for primary autoimmune diabetes remain poorly defined. The presence of thyroid autoantibodies or a first-degree family history of thyroid disease or thyroid antibodies may not be sufficiently exacting as evidence of autoimmunity. For our study we have regarded only biochemical evidence of thyroid overactivity or underactivity as an index of primary autoimmune diabetes, and using this criterion have compared autoimmune (group I) and non-autoimmune (group II) IDDs of similar ages and durations of diabetes. No significant difference in the prevalence or severity of retinopathy was observed in the two groups, suggesting that coincident autoimmune disease of a degree sufficient to produce thyroid failure or thyrotoxicosis neither protects against nor contributes to retinopathy. The latter is presumably the consequence of the cumulative metabolic disturbance found in insulin-dependent diabetes, although it may also depend on genetic influences.⁴

¹ Bottazzo, G F, *et al*, *British Medical Journal*, 1978, **2**, 165.

² Glick, S M, *Metabolism*, 1961, **10**, 78.

³ Bottazzo, G F, *et al*, *British Medical Journal*, 1978, **2**, 1253.

⁴ Pyke, D A, and Tattersall, R B, *Diabetes*, 1973, **22**, 613.

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