

IgE in Leprosy

TO THE EDITOR:

In spite of the growing interest in immunology of leprosy, only a few reports deal with the significance of IgE in this disease. Moreover, the data available on IgE serum levels in leprosy patients are inconclusive and often discordant (^{1,2,4,5,6}).

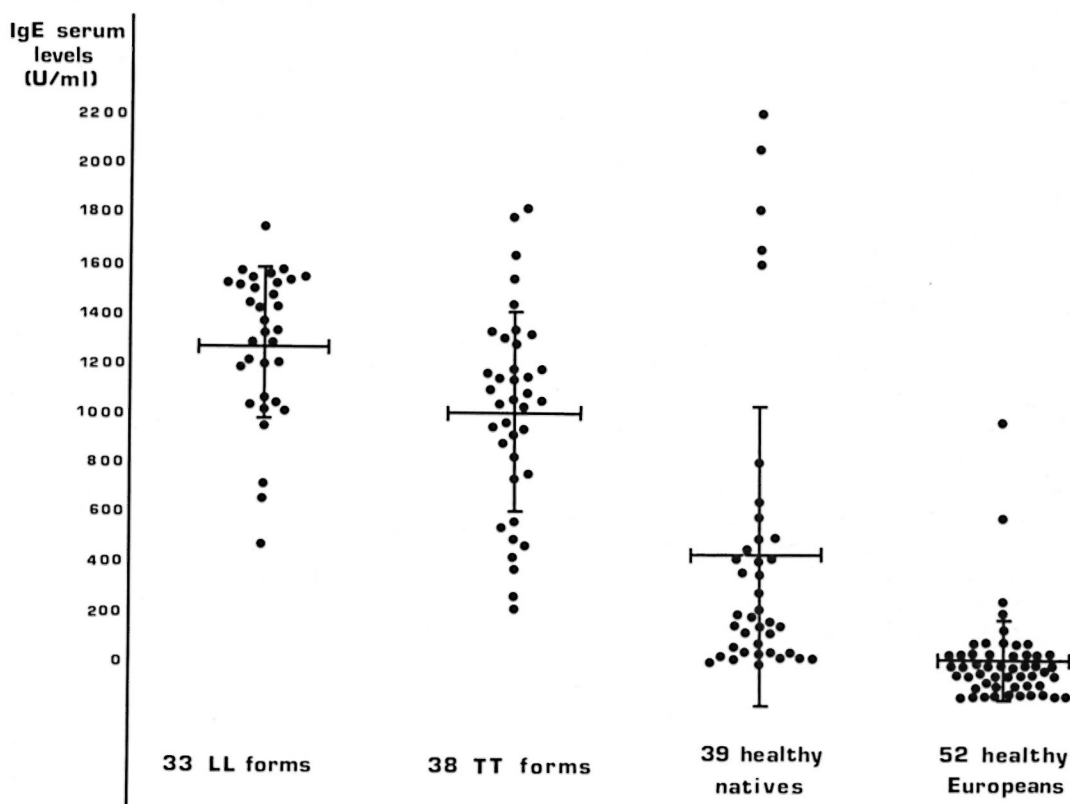
We have measured total IgE levels by PRIST (Phadebas IgE, Pharmacia Uppsala) in 71 Somalian patients selected among those admitted at the leprosy hospital of Jilib, south of Mogadishu, for having a polar form of the disease. Thirty-eight patients had tuberculoid (TT) and 33 the lepromatous (LL) form of leprosy (Ridley-Jopling classification). Thirty-nine native healthy subjects living in the same area and matched for ethnic and socioeconomic status, as well as 52 healthy Europeans, were used as controls.

The IgE values (The Figure) in the different groups were:

Patients with leprosy (total)	1128 ± 377 U/ml
Patients with LL forms	1272 ± 298 U/ml
Patients with TT forms	1003 ± 398 U/ml
Healthy natives	431 ± 601 U/ml
Healthy Europeans	111 ± 258 U/ml

The differences between the leprosy patients and both the native and European controls were statistically significant ($p < 0.001$). Patients with a LL form of leprosy had higher IgE levels than those with the TT form ($p < 0.01$).

In order to detect the eventual occurrence of specific IgE antibodies against *Mycobacterium leprae* antigens, a RAST test was performed. Cyanogen bromide activated paper discs were reacted with 10-fold



THE FIGURE. Total IgE serum levels in 71 leprosy patients, 33 lepromatous (LL) and 38 tuberculoid (TT), compared with those of native and European healthy controls.

serial dilutions (from 100 μg to 1 ng/ml) of leprosin⁽³⁾ kindly supplied by Dr. J. Bahr, Department of Bacteriology, Middlesex Hospital, London. Dr. J. Brostoff kindly provided laboratory facilities for this research in the Department of Immunology, Middlesex Hospital Medical School, London. With the technique used no specific IgE could be shown, high c.p.m. values possibly being due to non-specific binding.

Our results demonstrate that serum IgE levels are increased in leprosy, as previously reported^(2,4), even when compared with IgE values of subjects living in the same areas and matched for ethnic and socioeconomic conditions. This would imply that the increase in IgE is linked to the disease itself and does not depend only on the presence of parasitic infections, mainly ascariasis, which are very common in tropical zones.

In previous works, IgE values were not found to discriminate between the two polar forms (LL and TT) of leprosy. However, some authors^(1,6) reported an increase in IgE in the LL forms, although not significant. In our study, patients with the LL form of leprosy showed significantly higher IgE levels than those with the TT form.

It is well recognized that cell cooperation is necessary for IgE synthesis and it has been suggested⁽⁵⁾ that a T cell imbalance could be relevant to explain IgE overproduction in leprosy. Since we were not able to detect specific IgE antibodies to lepro-

sin, a defect of the cells involved in the non-specific control of IgE should be suspected. This would be more evident in the LL forms of the disease, in which a T cell deficiency is more marked.

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