TO THE EDITOR:

In a recent communication to the JOUR-NAL (²), Nuti, *et al.* reported the existence of elevated IgE levels in Somalian leprosy patients.

On the basis of these results, the authors endorse a previous hypothesis (³) of hyperproduction of IgE due to deficient T lymphocyte control activity in leprosy (particularly LL) patients.

We would, however, like to remark that we have found no significant elevation of serum IgE levels in Venezuelan leprosy patients (¹), and have emphasized the importance of both the appropriate selection of control groups (due to the augmenting effect of intestinal parasitosis on IgE synthesis) and the statistical methods employed (because of the non-normal distribution of IgE levels). Nuti, *et al.* have included the proper controls in their study (individuals of the same socio-economic level as the leprosy patients), but the statistical analysis applied should be clarified. Considering the fact that the authors do not specify the form in which their results are presented and compared, the data in the table are probably arithmetic means and standard deviations.

Since the most common statistical treatment of such parameters is Student's *t* test, we presume that this was applied; indeed the probability values reported are consistent with this assumption. However, the non-normal distribution of IgE levels (demonstrated by calculation of coefficients of the 3rd and 4th moments about the mean; skewness and Kurtosis) precludes the use of these statistical methods.

It is now widely accepted that geometric means are the most appropriate form of presentation, and that the statistical tests applied should be "non-parametric" (e.g., Wilcoxon-Mann and Whitney tests). A much-used alternative statistical treatment is normalization by logarithmic transformation and subsequent use of conventional analysis. We would be particularly interested in the recalculated significance of the relatively modest (27%) difference between LL and TT patients, because if their overall T

51, 3

Correspondence

lymphocyte reactivity is really greatly different, so should be their IgE levels, according to the proposed hypotheses (¹).

> -Neil R. Lynch, Ph.D. -Reina I. Lopez, B.S.

Panamerican Center for Research and Training in Leprosy and Tropical Diseases (PAHO-WHO) Instituto Nacional de Dermatologia Universidad Central de Venezuela Aptdo. 4043 Caracas 1010A. Venezuela

REFERENCES

- LYNCH, N. R., LÓPEZ, R. I., ULRICH, M., ARANZAZU, N. and CONVIT, J. IgE in leprosy: effect of a *M. leprae*-BCG vaccine. Int. J. Lepr. **51** (1983) 169– 173.
- NUTI, M., RASI, G., ROSA, C. and BONINI, S. IgE in leprosy. Int. J. Lepr. 50 (1982) 217–218.
- SAHA, K., DUTTA, R. N. and DASGUPTA, A. Immunological aspects of leprosy with special reference to the study of immunoglobulin E. Int. J. Lepr. 43 (1975) 314–319.