

Letter to the Editor

Does the suppression of the immune response in pre-clinical lepromatous leprosy affect both cell-mediated and humoral responses to *Mycobacterium leprae* antigens?

Sir,

My article on 'Importance of the neural predilection of *Mycobacterium leprae* in leprosy' was summarized superbly by Dr Jopling in your September 1980 number (p. 269). I only wish to emphasize here, as I did not do originally, that little, if any, information is available on the question of the detailed cellular mechanisms of suppression in leprosy. It is, therefore, not yet possible to predict whether antibody to *M. leprae* antigens is consistently produced during the postulated suppression phase which accompanies very early (pre-clinical) lepromatous leprosy, i.e. before skin smears become positive for AFB.

One possibility, as was suggested, is that the suppression of specific cell-mediated immunity (CMI) to *M. leprae* is accompanied by a humoral immune response to antigens of *M. leprae*. However, the alternative possibility cannot be excluded, i.e. that the suppression at that early stage is mediated by suppressor cells which suppress both cell-mediated and antibody responses to *M. leprae*.

This question is important because the possibility of very early serodiagnosis of lepromatous leprosy would seem to depend on the answer. A serological test would not only be of value in leprosy control programmes, but it might also find application in the exclusion of infected individuals from vaccine trials and/or in the early identification of lepromatous leprosy in the trial group.

If suppression of CMI to *M. leprae* in very early lepromatous leprosy is accompanied by formation of significant amounts of antibody, then efforts at very early serodiagnosis are well founded. If, on the other hand, the suppression extends over the antibody response as well, then early detection of lepromatous leprosy by serological methods may be difficult. In that case the level of various antibody specificities in these individuals may not differ from those of healthy individuals in the community who were subclinically infected. The antibody levels would presumably follow the bacterial load and might become 'positive' no earlier than skin smears stained for AFB.

In a study of the immune response to *M. leprae* in lepromin-negative

indeterminate leprosy¹ there was evidence for a failure of both CMI and the antibody response. Sera were assayed for precipitins to mycobacterial antigens. This would suggest that early suppression may involve both CMI and the antibody response, and bodes ill for early serodiagnosis of lepromatous leprosy. However, the question of antibody responses in lepromin-negative indeterminate leprosy must be re-examined with the more sensitive and/or specific assays recently developed in several laboratories.^{2,3,4,5,6} With the highly sensitive fluorescent antibody test, in which 36 out of 44 tuberculoid (TT) sera (82%) were positive, 2 out of 4 indeterminate sera were positive.² With the ELISA technique, 3 out of 7 indeterminate sera were positive.⁶

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References

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