4. HARTSKEERL, R. A., DEWIT, M. Y. L. and KLATSER, P. R. Polymerase chain reaction for the detection of *Mycobacterium leprae*. J. Gen. Microbiol. **135** (1989) 2357–2364.
5. KARANTH, S. S., SPRINGALL, D. K., LUCAS, S., LEVY, D., ASHBY, P., LEVENE, M. M. and POLAK, J. M. Changes in nerves and neuropeptides in skin from 100 leprosy patients investigated by immunocytochemistry. J. Pathol. **157** (1989) 15–26.
6. PRAKASH, K., AGGARWAL, R. and SEHGAL, V. N. Significance of antibodies to phenolic glycolipid-I (PGL-I) in leprosy diagnosis. J. Dermatol. (Tokyo) **19** (1993) 953–958.
7. PRAKASH, K., SEHGAL, V. N. and AGGARWAL, R. Evaluation of phenolic glycolipid-I (PGL-I) antibody as a multidrug (MDT) monitor. J. Dermatol. (Tokyo) **20** (1993) 16–20.
8. SEHGAL, V. N. *Clinical Leprosy*. New Delhi: Japee Bros., 1993, pp. 27–41.
9. SEHGAL, V. N., GUPTA, R. P., KARMAKAR, S., LOGANI, K. B. and JAIN, S. *In situ* characterization of lymphocytic immunophenotypy and interleukin-2 (IL-2R) in cutaneous tuberculosis and leprosy; a comparative evaluation. Clin. Exp. Dermatol. **19** (1994) 312–316.
10. SEHGAL, V. N. and JOGINDER. Leprosy in children: correlation of clinical, histopathological, bacteriological and immunological parameters. Lepr. Rev. **60** (1989) 202–205.
11. SEHGAL, V. N., KORANNE, R. V. and SEHGAL, S. Correlation of morphological, bacteriological, histopathological and immunological feature of leprosy; a double blind study. J. Dermatol. (Tokyo) **12** (1985) 243–250.
12. SEHGAL, V. N., REGE, V. L. and REY, M. Correlation between clinical and histopathologic classification in leprosy. Int. J. Lepr. **40** (1977) 278–280.
13. SEHGAL, V. N. and SRIVASTAVA, G. Indeterminate leprosy: a passing phase in the evolution of leprosy. Lepr. Rev. **58** (1987) 291–299.

Bhatia and Katoch Respond

TO THE EDITOR:

Drs. Sehgal and Jain have highlighted some issues about our publication. By and large, their comments support our interpretations and conclusions. As detailed in the Materials and Methods section of our paper, the criteria for clinical, histopathological and reactional status were well defined (references nos. 4, 5, 8 of our paper). These criteria have some limitations regarding the differentiation of some leprosy types, such as TT/BT, borderline types, and indeterminate cases. There are always some limitations of any retrospective analysis and, as also highlighted in our Discussion (page 437), these might have affected the results to some extent. However, even after allowing some margin for these factors, there appears to be a need for the reassessment of the weight given to different signs and/or histopathological parameters for classifying leprosy cases (especially TT, BB, I). Further, as highlighted in our paper and in the comments of Drs. Sehgal and Jain, such studies are not likely to be of much therapeutic relevance. We entirely agree about the need to carry out prospective studies using fluorescence, immunological, biochemical and molecular/gene amplification techniques to gain a better understanding of these problematic areas. We have emphasized these aspects in our Discussion (page 437).

Drs. Sehgal and Jain have very nicely focused on the research needs as well as some possible methods to study these aspects further. We entirely agree with their logic and thank them for their valuable suggestions.

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