

CORRESPONDENCE

This department is for the publication of informal communications that are of interest because they are informative and stimulating, and for the discussion of controversial matters. The mandate of this JOURNAL is to disseminate information relating to leprosy in particular and also other mycobacterial diseases. Dissident comment or interpretation on published research is of course valid, but personality attacks on individuals would seem unnecessary. Political comments, valid or not, also are unwelcome. They might result in interference with the distribution of the JOURNAL and thus interfere with its prime purpose.

Regarding Antileprosy Vaccine— an Apprehension by Dr. Parkash

TO THE EDITOR:

Dr. Parkash should be relieved to learn that his apprehensions ("Antileprosy vaccine—an apprehension"; IJL 1995; 63:572–573) are largely unfounded. Although he states that "no approved vaccine is available to date for immunoprophylactic use in the population," in fact BCG vaccines are licensed in all nations, are very widely employed (approximately 100 million individuals vaccinated in 1995 alone), and have repeatedly been shown to provide protection against leprosy (^{1–5, 7–14}). The fact that BCG vaccines are often considered to be directed against tuberculosis (TB) is irrelevant since studies to date indicate that BCG vaccines are more effective against leprosy than against TB (^{9, 11, 15}). Dr. Parkash is also concerned that, because lepromatous cases may be selectively anergic to antigens of the leprosy bacillus, and because cell-mediated immune responses are influenced by HLA, "it is worth arguing that probably for (lepromatous leprosy-prone individuals) . . . an antileprosy vaccine may not be successful in providing protective immunity." However, several studies have indicated that BCG protects against multibacillary disease, and studies in Brazil (¹²), Venezuela (⁵), Malawi (¹⁰), and Indonesia (³) have even indicated greater protection against multibacillary than paucibacillary disease. Such evidence suggests that the anergy expressed

by lepromatous cases is determined by nurture as well as by nature, and can be influenced by appropriate antigen exposure. Because of these actions the ongoing worldwide antileprosy (BCG) vaccination program is undoubtedly playing a major role in the global decline of leprosy. That said, it is also very true that BCG's effectiveness appears to vary between populations, and that there are still plenty of problems for those with interests in the immunology of antileprosy vaccines (⁶). But immunologists would do well to observe the available human data before becoming too pessimistic on the basis of theory alone.

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M. leprae and Macrophage Secretory Products Modulate the Expression of NgCAM on Schwann Cell Surface

TO THE EDITOR:

During embryogenesis and under pathological conditions, Schwann cells (SC) of the peripheral nerve are induced to express cell-surface molecules, such as the neuroglial cell adhesion molecule (NgCAM), which aid in the initial SC-axon associations needed for axon fasciculation⁽⁵⁾. Infection with *Mycobacterium leprae* of its host cell, the macrophages, renders them defective in a number of functions, including the expression of cell-surface molecules⁽¹⁾. The possibility exists that SCs, for which *M. leprae* have a special affinity, also could be rendered defective in the expression of cell-surface molecules and, therefore, contribute to the peripheral nerve pathology in leprosy. The presence of features like aberrant myelination in the sciatic nerve of the murine animal model inocu-

lated with *M. leprae* in the foot pad⁽⁷⁾ may indicate variation in adhesion molecule expression. Besides this, macrophages which infiltrate the site of a nerve lesion in order to aid SCs in nerve regeneration⁽⁶⁾, secrete a host of cytokines which have been shown to regulate the expression of cell-adhesion molecules⁽⁴⁾.

The aim of this study was, therefore, to determine if *M. leprae* infection and macrophage secretory products modulate the expression of NgCAM on the SC surface, comparing the differences in cells derived from two strains of mice, Swiss white (SW) and C57BL/6 mice, in their response to *M. leprae* infection⁽²⁾. (The viable *M. leprae* used in our study was derived from frozen armadillo liver biopsies supplied by Dr. E. Storrs, Florida Institute of Technology, Melbourne, Florida, U.S.A.)