DISCUSSION: NASAL INFECTION AND TRANSMISSION OF LEPROSY

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More recent quantitative data indicates that the major excretion of *M. leprae* is in secretions from the nose, mouth and upper respiratory tract from untreated lepromatous patients (1,2 and 3). Few if any *M. leprae* are excreted from lepromatous patients with intact skin; the only untreated lepromatous patients who excrete large numbers of *M. leprae* from the skin are the relatively rare patients who present with necrotising and ulcerating lesions in the untreated state; they can excrete as many *M. leprae* from the ulcerated skin as from the nose (4). Since it is accepted that man alone of the animal kingdom is infected with *M. leprae*, then with the exception of an intermediate host for immediate transmission, the major other source of *M. leprae* for the transmission of the disease must be predominantly from the nose and to a lesser extent from the rarer ulcerating lepromatous patients. Important studies have recently shown that *M. leprae* can survive (remain infectious for mice) in dessicated nasal secretions for 1-7 days. It is reasonable to presume that dessicated *M. leprae* can survive equally well in discharges from the rarer open ulcerated lesions of lepromatous patients. Therefore these nasal secretions and the ability of *M. leprae* to survive for a period means that transmission by immediate close contact is no longer essential, and is clearly consistent with the high proportion (>50%) of new cases of leprosy that cannot be traced to a known open contact. All the similarities between nasal secretions and sputum from open cases of tuberculosis strongly indicate that infective droplets and infective dust particles containing *M. leprae* are likely to be inhaled by contacts or the population at large, as they are from similar materials containing *M. tuberculosis*. Therefore the respiratory route of infection in leprosy must be reconsidered (see paper 15 by Rees and McDougall). Alternatively, the same nasal and more rarely skin secretions in which *M. leprae* can survive for some time may be a source for indirectly entering the skin through
small abrasions or be swallowed in contaminated food or drink. The portals of entry via the respiratory tract, skin and intestinal tract will be discussed. It is of interest that close contacts of open cases of lepromatous leprosy showed a lower proportion of lymphocyte sensitisation to *M. leprae* than individuals less directly exposed (5). This observation may indicate that overwhelming exposure by mouth and by inhalation may have led to desensitisation. Finally, recent observations on the role of several genera of flies in the transmission of *M. leprae* have shown that after feeding upon nasal secretions from untreated lepromatous patients or ulcerating skin lesions, their legs, abdomens, mouth parts and intestinal contents are heavily contaminated with *M. leprae* (6). Thus the contaminated flies could indirectly deposit *M. leprae* on their next feeding site, which might be food to be consumed by man, a traumatised skin surface or, in the case of blood sucking species of flies (Stomoxys), they could inject *M. leprae* into skin or blood stream, as is the case apparently with various species of arthropods.

REFERENCES


