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Letters to the Editor

Dr Pedley's conscientious studies leave no doubt that the nose is far the most important site of release of *M. leprae.* In fact, I wrote: "The importance of the nasal mucosa . . . is not challenged, neither the likelihood that *M. leprae* found on the skin may frequently have originated from the nasal mucosa". So far there is no essential disagreement.

I quantified the number of bacilli which were selected from the skin as "relatively low", Dr Pedley as "practically nil". I believe that the latter is an understatement, at least with respect to reactive lepromatous patients with ulcerating, vesicular or bullous lesions, because I found that such vesicles often are filled up with bacilli. A single vesicle or bulla may contain hundreds to thousands of bacilli, a number which certainly is sufficient for infecting many individuals. The crucial question is not how many bacilli are released, but how many are needed for infection. If we agree that only few bacilli are needed, and that small numbers of bacilli are released from skin defects, sweatducts and hairfollicles, that in addition much larger numbers of bacilli may reach the skin via nasal discharge, then I believe that it is correct to say that "infection via the skin remains a definite possibility".

The main point in my paper was that there is clinical and epidemiological evidence pointing against the hypothesis that the primary lesion, as a result of droplet infection, is located in the respiratory tract and that the modes of spreading of leprosy and of tuberculosis are similar. What I miss in Pedley's comments is either a challenge of the validity of this evidence or an explanation as to how the evidence can be made compatible with his hypothesis. There are no case reports of visitors of leprosy centres who have contracted leprosy. Few expatriate general medical and paramedical workers in endemic countries develop leprosy. The incidence of leprosy in others who work in highly endemic communities is low. Most of these people must have, on one, or more probably on several occasions, inhaled *M. leprae.*

In the Netherlands small outbreaks of tuberculosis are still frequently seen, but in this densely populated country, with a high incidence of sneezing due to common cold, although countless people must have inhaled *M. leprae* originating from untreated, relapsed or even drug-resistant lepromatous patients, only exceptionally an autochthonous case of leprosy was seen. In 1959 I diagnosed highly bacilliferous lepromatous leprosy in a girl who had lived for more than 2 years in a boarding school, undiagnozed and untreated. None of the hundreds of pupils has so far developed leprosy. My only explanation is that although countless people are exposed to dispersal of droplets containing *M. leprae*, the bacilli which are inhaled do not find in the respiratory tract a suitable environment for survival. Therefore I believe that it

is unlikely that the modes of spreading of leprosy and tuberculosis are identical.

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Vitiligo and Leprosy

I would like to put on record the observation that, during my work as a leprologist in London, I have encountered a significant incidence of vitiligo in patients under treatment for lepromatous leprosy. By "vitiligo" I am not referring to the well known hypopigmentation of leprosy but to the classical circumscribed depigmentation that can affect healthy persons of all races but occasionally is found in association with organ-specific auto-immune disorders such as diabetes, pernicious anaemia, thyroid disease, Addison's disease, and alopecia areata. I have found 8 cases of vitiligo among 114 lepromatous patients, an incidence of 7%, but no cases of vitiligo among a larger number of non-lepromatous patients, and I would be interested to know if this association has been noted elsewhere or if it has been reported in the literature.

This observation, when fully investigated by my successor at the Hospital for Tropical Diseases, may lend support to the hypothesis that vitiligo is an auto-immune disorder, having regard to the wide variety of circulating auto-antibodies which have been described in lepromatous leprosy, such as antinuclear, antithyroid and antisperm antibodies, and rheumatoid factor.

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