22nd May, 1963

(2) From WEDDELL, PALMER and REES commenting on SPICKETT

Dear Sir,

We accept Dr. SPICKETT's comments (in this issue) with respect to the portal of entry of *M. leprae* into the body and we welcome them.

Our observations have established that *M. leprae* can be disseminated by the blood stream. They also suggest that the organism does not enter the skin merely by contact or even by inunction.

We have no evidence to support or refute the suggestion that they enter the body via the respiratory or alimentary tracts but we felt that this was a problem which required investigation since it is well recognized that there are patients with leprosy in whom skin lesions cannot be found at all.

We may have given the impression that our observations indicate that the portal of entry is *not* via the skin. This was quite unintentional for an analysis of the cases we ourselves have examined, quite apart from the evidence in the literature, suggests that the skin is the commonest *known* portal of entry. The point which we wished to stress is that though we have examined over 2,000 slides of skin from patients exhibiting a wide range of clinical forms of leprosy including those who had been in close and continuous contact with patients having leprosy but themselves exhibiting no symptoms of leprosy, we have, so far, never seen any organism in the epidermis. Moreover,

we have been unable to find organisms in any centrifuged fractions of skin biopsy homogenates taken from close contacts.

This strongly suggests that the organisms must be inoculated *into* the skin. They would then be available for entry into macrophages, the Schwann cells associated with intact or degenerating cutaneous nerves, or blood vessels or lymphatic channels.

Clinical evidence, together with our experimental observations, suggest that once the organisms are *in* the skin, either: (1) they are destroyed and the subject does not develop leprosy, (2) they become related to the Schwann cells associated with cutaneous nerves at the site of inoculation and a single self-limiting lesion eventually develops (which always appears to involve both cutaneous nerves and the sensory nerve trunk serving the area involved), (3) they enter the blood stream and are conveyed to Schwann cells related to other sensory nerves and polyneuritic leprosy eventually develops, or (4) they enter the bloodstream and become widely disseminated and lepromatous leprosy eventually develops.

So far, nothing we have said is at variance with the clinical evidence available but the point we wished to stress to leprologists is that on the evidence available inoculation *into*, not passive contact with or inunction through, the skin is the route taken by the organism into the body via the skin.

In our view this is of the greatest importance in relation to the prevention of the spread of the disease, and there are two points which emerge and which we wished to emphasise: (1) it means that subjects in areas where leprosy is endemic should understand the importance of elementary hygiene in the prevention of leprosy, e.g. (*a*) that *M. leprae* from open cases should either not get smeared onto their skin at all or that they should not be allowed to remain there in large numbers. This means washing, the destruction of infected fomites, and the disinfection of the dwellings, particularly sleeping quarters and floors; (*b*) the importance of all available measures to reduce superficial skin injuries and insect bites. In some regions light clean clothing might possibly be advocated. (2) a continuation of the efforts to reduce the number of lepromatous cases by the setting up of networks of rural clinics for the distribution of therapeutic drugs which Dr. C. M. ROSS has demonstrated in Northern Nigeria to be highly successful.

Our reasons for going into print at this possible premature stage was to stimulate leprologists into renewed vigilance with respect to hygiene for, if as SPICKETT (1961) suggests *Demodex folliculorum* Simon is a possible vector, then it is *not* enought merely to *treat* patients suffering from leprosy.

SPICKETT'S suggestion would certainly account for the occurrence of the disease in patients who have never come into contact with

either an open or closed case of leprosy, a suggestion which it is hard to discount in view of the evidence of BADGER (1959).

We remain aware that we have too little evidence at our disposal to be categorical, but we feel strongly that the evidence so far available entitles us to the view that it would be wrong not to regard leprosy as potentially highly infectious and to point out to those living in areas where leprosy is endemic the protective value of elementary personal and public hygiene which has proved such an effective measure in protecting those trained in these subjects, who have emigrated to these areas. In other words, we think that measures modelled on the prevention of tuberculosis are likely to be highly effective. We realise how difficult this may be, but because there are still a few leprologists who do not believe that leprosy is potentially infectious like other mycobacterial diseases we felt impelled to speak.

We of course accept Dr. SPICKETT's views with regard to the importance of the genetic factors and are pleased to find that another academic research worker is as interested in leprosy as we are.

GRAHAM WEDDELL
ELISABETH PALMER
R. J. W. REES

References

BADGER, L. F. (1959), Chapter VI, *Epidemiology in Leprosy in Theory and Practice*, R. G. Cochrane, John Wright, Bristol.
SPICKETT, S. G. (1961), *Leprosy Rev.*, **32**, 263.