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Letters to the Editor

Dr. Brubaker's letter in *Leprosy Review* of April, 1970 (**41**, 2, 128) concerning the use of dapsone in Northern Nigeria raises some very important issues. I do think, however, it was the Government's policy to provide treatment only for those showing definite evidence of leprosy. There is, as is well known, a great desire on the part of the public to obtain dapsone for reasons I have not understood, and in the clinics where I began working there were many people who had no signs of leprosy. Some of these could be immediately discharged, but there was a considerable number who presented a dilemma. The description of the initial lesions at the time of the patient's first attendance was often scanty or absent and it was impossible to decide whether the person had ever had leprosy or whether genuine skin patches had disappeared with treatment. If these patients were discharged there was then a possibility of relapse because of inadequate therapy. I therefore adopted a very cautious approach and kept on treatment a large number of people, but obviously many of them probably had not the disease. This then was the reason for the large numbers of attenders reported in my paper, rather than a deliberate policy of chemoprophylaxis.

Similarly, in other parts of Northern Nigeria there were many people obtaining dapsone because leprosy attendants were too ready to accept them without adequate examination, and so Dr. Brubaker is quite right to argue that the decline in the disease could in part be due to the chemoprophylactic effect of the drug. However, I feel that the direct effect of dapsone in the leprosy patient is more important, particularly in those with multibacillary disease in which bacteria are rendered non-viable after a few months of therapy, thus preventing new cases from arising. The well-documented articles published in recent numbers of *Leprosy Review,* describing control projects where patients were closely supervised, have shown how successful this approach has been, and it was my intention also to advocate this in my paper. I too am strongly opposed to the haphazard distribution of dapsone tablets.

Faculty of Medicine,
The University
Dar es Salaam, Tanzania

C. L. CRAWFORD

16 November, 1970

For many years the history of leprosy has suffered from the contradictory opinions expressed regarding the method of transmission of the disease. In India it used to be thought that there was no need to take special precautions in working with leprosy patients; I had to work cheek by jowl with an infective patient, looking down the same microscope. The wind of change then began to blow, and masks and gloves were worn.

A paper has recently been published (*Leprosy Review* (1970) **41**, 31 and 167) whose main contention is that skin-to-skin contact is a minor source of infection, and that the principal method of spread is *via* the nasal secretions. The skin-to-skin route, with the skin unbroken, is ignored. While it is true that a positive nasal smear indicates a greater possibility that *Myco. leprae* may be transferred from an infected person to a healthy one, yet it cannot therefore be inferred that the nasal route is the sole method by which a person can become infected. In the first place, how often, except in a case of diffuse lepromatous leprosy, is the skin intact? In the second place, bacilli can be detected in the sweat of a patient perspiring profusely. Further, bacilli can be found in the epithelial cells of the skin.

Therefore, while it is true that the nasal mucosa may, in many instances, be the chief route of infection, it is unwise to consider this as the sole source; by so doing, other possible routes are ignored, and an unwise policy may be advocated. I have been long enough in leprosy work, approaching half a century, to be very sceptical of statements that emphasize only one aspect of the transmission of the disease. I would emphatically stress that, while we give due importance to the nasal route of spread, we do not thereby ignore other possibilities.

Let us keep our feet firmly on the ground, and neither be unduly scared in regard to hypothetical routes of infection, nor nonchalant about methods of dissemination whose importance in the transmission of leprosy from the infective person to the susceptible individual cannot at present be readily determined.

20 February, 1971 R. G. COCHRANE

I should like to comment on two points in Dr. Cochrane's letter, which queries certain statements in my recent papers (Pedley, 1970*a, b*), first on the statement: "bacilli can be detected in the sweat of patients perspiring profusely".

If bacilli are indeed excreted in the sweat on to the surface of the skin, I should have expected, in a systematic search of an area of 813 sq cm of maximum infiltrated lepromatous skin in 24 patients, to have found a large number of *Myco. leprae,* since partly dried sweat would have been present in every case, leaving its solid contents adhering to the skin. As it was, only 28 bacilli were found by Composite Skin Contact Smears (C.S.C.S.), of which 25 were present on the face of 4 patients, in each of whom the nose-blowings were heavily infected (Pedley, 1970*b*). Two of these patients had been sweating profusely, for the weather was humid, and their faces were still clammy with perspiration when I made the examinations. Had I not first examined the nose-blowings, I might have concluded (wrongly) that the bacilli had been excreted in the sweat. The patients here dispose of their nasal discharge and sputum most unhygienically; one patient, whose nose-blowings were heavily infected, was seen to wipe the nasal discharge on to hand, forearm, face and thigh. From any of these contaminated areas, bacilli on the skin surface could doubtless have been demonstrated by the technique described.

In the 24 patients in my series (Pedley, 1970*b*) with untreated lepromatous leprosy, 18 showed large numbers of solid-staining *Myco. leprae,* together with many globi, in their nasal discharge. Bacilli would probably have been found in the nasal mucus of the remaining 6 patients had they been examined more than once, or had the nasal mucosa itself been examined. Thus, I conclude that in the

majority of patients with untreated lepromatous leprosy, enormous numbers of *Myco. leprae* may be present in the nasal discharge.

I admit that bacilli may be present in the sweat of patients with lepromatous leprosy, but before it is justifiable to conclude that bacilli found on the skin of the face had been deposited in the sweat, the nasal discharge must be examined (more than once, if necessary) for the presence of acid-fast organisms.

Second, Dr. Cochrane asserts that ". . . bacilli can be found in the epithelial cells of the skin". This is undoubtedly true, but it does not necessarily follow that these bacilli will emerge on to the skin surface. The likelihood of this happening may be doubted for the following reasons: if this were true, more than 28 bacilli on the area of skin examined would have been found; and the sections I have seen show that when intracellular organisms are present in an epidermal cell, the cell is situated deeply. In this layer, the cells are considerably flattened and the nucleus and cytoplasm appear to be disintegrating before being transformed into the horny layer.

It is, of course, possible that by the time the cell containing the bacilli reaches the surface of the epidermis, the acid-fast organisms will have disintegrated along with the nucleus and cytoplasm of the cell.

Leprosy Mission,
Tan Sen, Nepal

J. C. PEDLEY

11 March, 1971

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