

CORRESPONDENCE

[In reply to the editorial request in the last issue for evidence for or against drug resistance to sulphones in leprosy, the following letter was received.]

Dear Sir,

Out of 20,000 odd patients treated since April, 1950, we have not seen a single case which shows the normal signs of drug resistance—a steady improvement, followed by lack of response, followed by a recrudescence of the disease while still under treatment, and unable to be controlled by the drug in question. The patient mentioned under “ Drug resistance ” in my article (*Lep. Rev.*, Vol. 27, p. 58) has not shown any further deterioration, and in fact his last smear was improved.

I find several patients of this nature starting with a large number of bacilli. The number is fairly rapidly reduced, but there seem to be occasional pockets of acid-fast dust, often in the form of globi. These are commonly in the ear lobes, but can be elsewhere. Being small pockets, and often showing no clinical evidence of activity, I presume that it is rather a matter of chance whether the pockets are found on bacteriological examination—hence the variable smear results.

The pockets are very persistent and I have found globi of acid-fast dust nearly two years after smears have become negative in other parts of the body. Dr. Davey is of the opinion that they are probably dead bacilli, not cleared away. We have discussed the significance, from the points of view of both infectivity and fitness to stop treatment.

I fear that the proof can only await the successful culturing of bacilli or a lab. animal which can reliably be infected, or clinical experience of at least two decades.

Some evidence in favour of these bacilli being dead is the fact that defaulters have, after leaving our care with acid-fast dust, returned after a period of time, with none. Unfortunately we cannot take as certainly true, their assertion that they have had no private treatment.

Compared with this we have many reports of drug-resistance after periods of two years or less of treatment with Thiacetazone and Isoniazid.

Another question that may be asked is whether discharged patients whose leprosy has recurred show drug-resistance. In my experience, and that of my colleagues, these patients improve about as rapidly after being placed again on treatment as would be expected of a similar group of patients with untreated leprosy.

There are a few patients who have had fairly severe reactions at a time when it appeared that all signs of the disease were past. They have not, however, shown signs of progressive worsening of the disease, as would be expected if drug-resistance were present.

To sum up, I can see no evidence at present to believe drug-resistance has occurred in leprosy under dapsone treatment, though the period of treatment of very large numbers has already been nearly three times that required for drug resistance to be produced under other drugs. On the other hand, it seems too good to be true that dapsone is the one exception in drugs, and leprosy in diseases, to a rule which is so distressingly wide in its application.

Yours sincerely,

ARTHUR GARRETT.

Oji River,
E. Nigeria.

Dear Sir,

Your editorial remarks and reference to Dr. J. A. K. Brown's article in *Lep. Rev.* of April 1956 raise, as you say, an important

problem. Lepromatous, Borderline and Tuberculoid cases in reaction are all "open" forms of leprosy. You point out that all these may show large numbers of bacilli to routine methods of examination and therefore, presumably, may pass on the infection. I suggest the inability to trace all cases of leprosy to patients falling into these categories is due to the long incubation period and, particularly in parts of Africa, to the changing population. Where there are large areas of country with no obvious lepromatous cases some of us may overlook the fact that many of the cases of leprosy found may AT ONE TIME have been Borderline or Tuberculoid cases in reaction even if no longer exhibiting such features? Further, as you point out, "concealed" (or undiagnosed) cases may spread the infection. I believe many, if not most, cases of diffuse lepromatous leprosy are contagious, perhaps for a long time, before the disease declares itself sufficiently to be recognised by a casual observer or even by the patient himself.

Yours truly,

J. T. WORSFOLD.

Chitokoloki Leprosy Settlement,
Balovale,
Northern Rhodesia.
1st August, 1956.

VINOBA BHAVE AND REHABILITATION OF LEPROSY PATIENTS

On 25th February, 1956, the Dy. Minister for Revenue, Mr. Hanmanth Rao, accompanied by the District Health Officer and Dr. Bhagwan Rao Tandade, Medical Officer, Leprosy Control Pilot Project Scheme at Narayanpet, called on Vinobaji and discussed with him a scheme for the rehabilitation of leprosy patients. Dr. Bhagwan Rao explained the purpose of the scheme and indicated that at an average of 5 acres per patient, at least 250 to 300 acres of land would be required under the scheme. After acquainting himself with every aspect of the plan, Vinobaji agreed to allocate the required acreage under the Bhoodan scheme. This plan when it gets into operation, would considerably alleviate the miserable plight and distress of those unfortunate victims of leprosy to whom society attaches a "stigma" even to-day.