DAPSONE-RESISTANT LEPROSY

Sir,

Leprosy Review must be congratulated for the timely tribute to Dr R J W Rees, in the special issue of June 1983. Dr Rees' dedication to the scientific battle against *Mycobacterium leprae* is an example to researchers all over the world.

The same special issue of June 1983 carried an article by J M H Pearson entitled 'Dapsone-resistant leprosy', on pages 85S–89S. Before making a few comments, may we indicate some important corrections. In the references cited, the title of the eleventh reference (Almeida *et al.*) is 'Studies on DDS-resistant *M. leprae* in leprosy patients of Gudiyatham Taluk . . .' and not 'Prevalence of secondary dapsone resistance in Gudiyatham Taluk . . .'[1] Secondly, Balraj *et al* (from Gudiyatham Taluk) are represented as having reported a '5-10% prevalence of dapsone-resistant leprosy'. The exact figure reported by them was 2·3%.[2]

Dr Pearson quite rightly points out that the findings of his study in Ethiopia 'rather quickly overshadowed' other reports of dapsone resistance. The reported prevalence of '10–20%', from Ethiopia stands in marked contrast to reports of 2·3 to 6·8% from Malaysia,[3] Costa Rica,[4] Israel,[5] and Gudiyatham Taluk, South India.[2] Since the findings in Ethiopia were 'not the result of formal surveys', and depended on 'cases with clinical suspicion of dapsone-resistant leprosy' being 'referred', some questions arise.

Patients with predominantly dapsone-sensitive *M. leprae* who fail to take dapsone can easily be included among those diagnosed to have 'dapsone-resistant leprosy'. Dr Pearson could perhaps explain how he avoided such misclassification. If investigations continued in Ethiopia after Dr Pearson's departure, the findings would be of interest.

Patients tested for 'primary dapsone-resistant leprosy' are said by Dr Pearson, to have been reported for the first time in 1977. Some papers by Rees were overlooked. In 1967, Rees[6] published data showing that *M. leprae* from previously untreated patients grew in the foot-pads of mice fed 0·1% DDS: roughly equivalent to a daily DDS dose of 1000 mg (1g) in an adult patient. In 1965, Rees[7] reported on the results of feeding DDS, to mice inoculated with *M. leprae* from previously untreated patients: 'The overall result on nearly a hundred foot-pads has been complete inhibition in only 82%.'[7] *M. leprae* from untreated patients were, therefore, found to grow in the foot-pads of mice fed high doses of DDS in some of the earliest tests performed. It is no surprise that such DDS-resistant mutants of *M. leprae* which were detected by Rees[6, 7] in 1965 should again be reported by Pearson[8] in 1977, and should continue to be found wherever they are sought. The spread of DDS-resistant *M. leprae* from treated patients is not the only explanation for DDS-resistant *M. leprae* in untreated patients.

However, Dr Pearson's interesting hypothesis of an 'epidemic of primary dapsone-resistant leprosy' can be evaluated. In areas where suitable records are available, the response to dapsone monotherapy can be compared in recently infected patients and those infected relatively long ago. If those infected recently show relatively diminished response to DDS, Dr Pearson's hypothesis would be strengthened.

We look forward to hearing the comments of Dr Pearson and any workers in Ethiopia who can shed more light on his findings in that country.

The discussion of drug resistance in leprosy must in no way be construed as criticism of multiple drug therapy. On the contrary, only a full discussion will allow crucial questions on drug resistance in leprosy to be answered.

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