Co-incident (Simultaneous) Dapsone Sensitivity and Dapsone-resistant Leprosy

TO THE EDITOR:

It was interesting to read the letter of Dr. McDougall and Dr. Felton Ross [IJL **50** (1982) 214–215] concerning co-incident (simultaneous) dapsone sensitivity and dapsone-resistant leprosy. They ask whether leprosy workers in other parts of the world have observed findings similar to theirs.

This occurrence was first proved by workers from the British Medical Research Council's unit in Sungei Buloh, Malaysia, in the initial series of papers which proved the existence of sulfone-resistant leprosy.

Our Indian patient No. 5075 was No. 1 in the first seven cases we studied for resistance ([2]) and also No. 1 in the more detailed report printed in 1966 ([3]). Although the sensitivity tests that were first performed found him to have a strain of dapsone-sensitive organisms, his Morphological Index did not diminish as much as might have been expected after six months on injectible DDS (600 mg weekly).

This case was reported in much greater detail in 1968 as "A Case of 'Partial' Resistance" ([1]) which describes how his Morphological Index fluctuated wildly while on sulfone treatment. In March 1963 the biopsies (we always took two from different sites) showed definite active lepromas and he was treated with 300 mg of dapsone twice weekly. About eight months later further biopsies seemed quiescent although Dr. Ridley noted "one more than the other." In April 1964 however the biopsies again showed definitely active leprosy. In the next eight months he had a severe bout of erythema nodosum leprosum which we believe is associated with successful anti-mycobacterial therapy, yet by February 1965 the biopsies still showed definite evidence of activity. At that time he was put on clofazimine therapy and from then on got steadily better by all criteria—clinical, bacteriological, and pathological.

We were treating a patient whose initial lesions showed dapsone sensitivity but who improved and then got worse while under treatment—the skin plaques got smaller but a diffuse skin infiltration appeared.

In those early days we were not yet able to recognize the clinical appearance of sulfone-resistant leprosy, but in retrospect it is obvious that this patient had some lesions containing resistant organisms and others with sensitive organisms. We said in our summary that it was likely that more cases of this type will be found. Apparently this is now the case.

We must therefore accept that resistant leprosy may be due to two things:

1) The development of a resistant clone in a patient most of whose bacilli are sulfone-sensitive. It is a moot point whether this clone develops because of inadequate therapy or whether it was always there and

only revealed when the sensitive population disappeared.

2) Any lepromatous patient under satisfactory sulfone treatment is liable to catch a resistant form of leprosy, particularly if such immunologically susceptible individuals are living in a place where other cases with resistant organisms are likely to be found.

Some time ago I was asked to write an article for the "house paper" of a famous leprosarium and I wrote to the effect that leprosaria should be closed down—it was not published. It seems to me to be the height of stupidity to send lepromatous patients for admission to a hospital where, almost by definition, they are likely to meet sulfone-resistant mycobacteria. If this continues (to repeat our warning of 15 years ago), it is likely that more cases of this type will be found.

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