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CORRESPONDENCE

This department is for the publication of informal communications that are of interest because they are informative and stimulating, and for the discussion of controversial matters. The mandate of this JOURNAL is to disseminate information relating to leprosy in particular and also other mycobacterial diseases. Dissident comment or interpretation on published research is of course valid, but personality attacks on individuals would seem unnecessary. Political comments, valid or not, also are unwelcome. They might result in interference with the distribution of the JOURNAL and thus interfere with its prime purpose.

Co-incident (Simultaneous) Dapsone Sensitive and Dapsone Resistant Leprosy

TO THE EDITOR:

In considering the possible sources of infection by *Mycobacterium leprae*, it has long been recognized that any patient may have acquired the organism from a variety of different sources. Increasing knowledge of resistance to the drugs available for the treatment of leprosy now indicates that such sources may include patients with different degrees of responsiveness to drugs. It recently occurred to us that one of the ways in which an existing leprosy patient could acquire resistance to therapy might be through super-infection with already resistant organisms. In the case of lepromatous leprosy, it is a common observation that a small proportion of patients demonstrate a curious combination of skin lesions which are difficult to interpret. These lesions consist of a background of "typical" or "classical" lepromatous lesions, usually fairly widespread on the skin surface, together with other lesions of an "atypical" type, many of which are frankly histoid, with close resemblance in appearance, size, and location to histoid lesions as described in the literature ([2,6,7,8]). Many of these patients have been on treatment, frequently in low doses and irregularly, over a long period, usually more than five years and often of the order of 10 or even 15 years. Could it be that these different types of clincal skin lesions are the result of coincidental infection with strains of *M. leprae*, a) resistant and b) sensitive to dapsone? We discussed this idea over a year ago but did not pursue it since there were apparently no published data to support it and we knew of no parallel in the tuberculosis or other literature. It has now, however, come to our notice that sequential infections with drug sensitive and drug resistant bacilli have been reported in tuberculosis and reviewed by Bates ([1]). Furthermore, Mankiewicz and Liivak ([5]) found that 33 out of 233 tuberculous Eskimo patients had more than one phage type in initial isolations of sputum, 22 of whom showed variation in susceptibility to anti-tuberculous drugs. In the U.K. in a study of 120 patients with positive cultures of *M. tuberculosis* obtained from both pulmonary and non-pulmonary sites, two revealed organisms which differed in drug susceptibility (but were identical in other bacteriological tests) ([3]).

We are prompted to correspond on this matter by the recent publication of data in a journal which may not be routinely available to all those working with leprosy ([4]). This article dealt essentially with rifampin resistance in two different patients, but it is of interest that the mouse foot pad data were compatible with the possibility that one of them (the second patient) harbored ". . . two strains of bacilli, one strain resistant to both dapsone and rifampin, and the other strain resistant to rifampin but sensitive to dapsone."

It would be of interest to know if leprosy workers, particularly clinicians, in other parts of the world have observed the coincidence of lesions described above. Presumably, its significance could be investigated by taking biopsies for foot pad

examination from "histoid" and "normal" lesions simultaneously.

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Chemotaxis of Monocytes in Hanseniasis

TO THE EDITOR:

Mycobacterium leprae is an intramacrophagic parasite. The performance of the macrophage during infection with this organism constitutes the basis for the polarity doctrine ([3]); if the macrophage has the ability to lyse *M. leprae*, the tuberculoid form of hanseniasis (TH) will occur; if not, the virchowian form (VH) will develop. It is possible that this characteristic of the macrophage is hereditary. Additionally, lymphocytes play an important role in the lysis of *M. leprae*. If T cells are specifically sensitized by *M. leprae*, delayed or cellular hypersensitivity will occur with the production of specific lymphocyte mediators. These mediators, in turn, can activate specific lysis by the macrophage. If it is a B cell that is sensitized, humoral immunity will be produced without any effect on macrophagic lysis of *M. leprae*.

Recently numerous reports have centered their attention on the phenomenon of chemotaxis in infections, i.e., the directed migration of leukocytes following stimulation by substances known as chemotactic factors. In general, these factors are nonspecific, but there are a few that are selective such as the C 567 activator for neutrophils, the eosinophil chemotactic factor of anaphylaxis (ECF-A) and certain peptides derived from lysosomal granules of neutrophils with specificity for the chemotaxis of monocytes. The action of these factors explains the presence of cells, specific or nonspecific, in inflammatory processes. On the other hand, a chemotactic inactivating factor (CIF) has been detected in trace amounts in normal serum. It is probably an enzyme, possibly an aminopeptidase. The significant elevation of CIF in certain diseases has made different interpretations of the role of inflammation possible. In delayed or cellular hypersensitivity, the specific antigen causes the T cell to liberate lymphocyte mediators, among which are chemotactic factors. There seems to be a correlation between delayed hypersensitivity and the amount of CIF. Van Epps, *et al.* ([2]) and Ward and Beremberg ([5]) found an elevation of CIF in cases with depressed delayed hypersensitivity. Ward and Beremberg ([5]) found high levels of CIF in nine patients with Hodgkin's disease, thus demonstrating a decrease in the ability to mobilize inflammatory cells in this entity.

In hanseniasis, Bullock, *et al.* ([1]) and Ward, *et al.* ([6]) found marked depressions