Reply to Dr. Bergel's Letter to the Editor

TO THE EDITOR:

The results on pharmacological and toxicological effects of dapsone (DDS) obtained and discussed by M. Bergel in his Letter to the Editor might be of great interest; they have, however, nothing to do with the antibacterial mode of action of DDS on the enzymatic level of the bacterial cell. The action of DDS is highly specific on the 7,8-dihydropteroate synthetase, i.e., the inhibition of bacterial growth occurs via depletion of dihydrofolate. It can be totally antagonized by p-aminobenzoic acid (PABA), which competes with DDS for the binding site on 7,8-dihydropteroate synthetase.

—J. K. Seydel, Dr. rer. nat.
Professor and Head
Biochemistry Department
Forschungsinstitut Borstel
2061 Borstel, Federal Republic of Germany

The Fingers in Non-lepromatous Leprosy

TO THE EDITOR:

Dr. McDougall ([4]) comments on Dr. Pearson's finding of inflammatory cells in the skin of the fingers of patients with non-lepromatous leprosy in the absence of skin lesions. He concludes that the fingers may be a focus for leprosy bacilli similar to the observations in patients with lepromatous leprosy. However, in describing Dr. Pearson's findings, he states that although inflammatory cells were present "*no acid-fast bacilli were seen.*" Previously, I described inflammatory cells in the skin of the hand of a patient who had severe edema of the hands and feet and acute sensory loss. There was no hypopigmented skin lesion at this site, and in spite of inflammatory cells, no acid-fast bacilli were found ([1]). At the X International Leprosy Congress at Bergen in 1973, I described two further non-lepromatous leprosy patients who had edema of the hands and feet in which skin biopsies showed inflammatory cell infiltrates at these sites ([2]). Dr. Pearson's observations in patients with swollen extremities would be consistent with these findings.

The significance of these findings lies in the fact that this inflammatory cell infiltrate is associated with sensory loss which may be acute and is of glove and stocking distribution, i.e., a sensory polyneuritis probably occurring in the *absence* of leprosy bacilli. If unrecognized and untreated, sensory loss will be irreversible, leading to the inevitable sequelae: mutilation of the fingers and toes, ulcers of the feet, osteomyelitis, and Charcot's joints. Current descriptions of "reversal reactions" emphasize the pain and swelling in the hypopigmented skin lesions and also in the peripheral nerve trunks but infrequently mention the edema of the hands and feet. In my opinion, it is the edema of the hands and feet which is much more important in indicating nerve damage, and it usually occurs in the absence of visible changes in the skin lesions and pain in the nerve trunks ([3]). Furthermore, the experimental finding of foot pad swelling, mononuclear cell infiltration, and cutaneous nerve damage in thymectomized and irradiated mice which have been given injections of lymphocytes is similar to this clinical syndrome ([5]).

Dr. McDougall's suggestion of the need for further investigation of this syndrome is welcome. The dose and timing of corticosteroid therapy could be monitored by performing serial skin biopsies in the hands and feet.

—C. L. Crawford, M.B., M.R.C.P., D.T.M.&H.

Department of Anatomy
Charing Cross Hospital
Medical School
Fulham Palace Road
London W6 8RP, England

REFERENCES

1. CRAWFORD, C. L. Neurological lesions in leprosy. Lepr. Rev. **39** (1968) 9–13.
2. CRAWFORD, C. L. Hansen's classification of leprosy. Int. J. Lepr. **41** (1973) 584–585.
3. CRAWFORD, C. L. Leprosy—an alternative viewpoint. Trop. Doct. **3** (1973) 137–139.
4. MCDOUGALL, A. C. The fingers in non-lepromatous leprosy. (Letter to the Editor) Int. J. Lepr. **48** (1980) 210.
5. REES, R. J. W. and WEDDELL, A. G. M. Experimental models for studying leprosy. Ann. N.Y. Acad. Sci. **154** (1968) 214–235.

Reply to Dr. Crawford's Letter to the Editor

TO THE EDITOR:

I am grateful for the opportunity to reply to Dr. Crawford's letter on this interesting subject and hasten to apologize to him for overlooking his previous observations on the presence of inflammatory cell infiltrates in biopsies from patients with edema of the extremities. His belief that a sensory polyneuritis may occur in these patients in the absence of bacilli, with serious consequences, confirms my suspicion that the subject of edematous swelling of the extremities in patients with borderline reactions has never been fully investigated and explained. If there is indeed an inflammatory infiltrate of cells in these edematous areas, *unrelated* to the obvious skin lesions of borderline leprosy, then the subject is surely worth further investigation since it may reveal something new concerning the pathogenesis of leprosy, at least in this part of the spectrum.

—A. Colin McDougall, M.D., F.R.C.P.
The Slade Hospital
Headington
Oxford OX3 7JH England

Adherent Suppressor Cells in Polar Lepromatous Leprosy

TO THE EDITOR:

Patients with polar lepromatous leprosy exhibit tolerance to *M. leprae* specific antigens in a variety of systems to test for delayed-type hypersensitivity and cell-mediated immunity. We wish to report results of preliminary investigations into the type of cells responsible for this immunological tolerance.

Mononuclear cells from three patients with lepromatous leprosy were isolated from whole blood by conventional methodology ([1]). The patients had been treated with dapsone (DDS) for one to two years and had not experienced complications during the period of treatment. Adherent cells were removed from the mononuclear cell suspension in a two-step procedure. In brief, the mononuclear cells were cultured on plastic petri dishes in RPMI 1640 containing 20% heat inactivated fetal calf serum but without antibiotics for 2 hr at 37°C in 5% CO_2 and 98% humidity. Nonadherent cells were removed by decantation and incubated overnight under the same conditions. After overnight incubation, nonadherent cells were removed and set up in culture. The procedure was, in our hands, highly effective and resulted in a depletion of adherent cells, which are activated mac-