Weston-super-Mare, Somerset,
16th August 1963.

DEAR SIR,

In the final paragraph of your Editorial in the April 1963 number of *Leprosy Review*, you invite comments and letters on the paper by WEDDELL and PALMER (1963).

I would particularly like to comment on the findings by these workers (pp. 58 and 59) of acid-fast dust in a degenerating nerve fasciculus, together with the presence of two viable (*sic*) organisms lying in healthy Schwann cells related to healthy nerve fibres.

These findings seem to me to be fully in keeping with an idea concerning the biology of *M. leprae* conceived several years ago. This is that the negative results of *in vivo* and *in vitro* culture experiments should be accepted, and that the cause of failure of bacillary growth should be sought not outside bacillary populations but inside each individual bacillus.

One starts with the assumption that each individual *Mycobacterium leprae* is non-viable due to a cause within itself. It is next argued that the relationship of the bacilli to the lepra cells seems to be such that the latter are overcoming this "non-viability" factor since massive multiplication of bacilli is occurring within them, and

regression of lepra cells appears to determine bacillary degeneration. (P. R. DE SOUZA and M. DE SOUZA LIMA 1952).

The next question is the teleological one—asking why lepra cells behave in this way—and the answer is to seek a positive association between bacillary non-viability and pathogenicity, instead of assuming that these two factors are necessarily in inverse relationship, as they are in the case of most other pathogens.

The observations of WEDDELL and PALMER are to my mind suggestive of such a positive association; in other words the morphologically intact bacillus is *not* damaging the nerve fibre, whereas the cloud of acid-fast dust *is* doing so.

Could it be that the Schwann cell is taking on a similar role to the lepra cell in rendering a neuropathogenic endoparasite of *M. leprae* harmless to the nerve fibre of the human host, *but* at the same time symbiotic to the bacillary host, so that the latter is able to reproduce only while under the influence of the cell, reverting to its non-viable form and being destroyed by the endoparasite growing at its expense and damaging the nerve fibre, when the influence of the cell is withdrawn?

These speculations would in no way interfere with pathological concepts assuming a direct action of tuberculoid tissue against the somatic part of *M. leprae*, but, if we think of the target organ of the latter as being the skin, might well account for very circumscribed depths at which lepra bacilli, tuberculoid tissue and lepra cells are found in man.

I have neither the specialised knowledge nor the resources to try to test the validity of this idea, but merely put it forward in the hopes that expert leprologists might be sufficiently interested to do so, and because it seems to me to fit in rather well with the observations already mentioned.

M. G. CORCOS.

References

P. R. DE SOUZA and M. DE SOUZA, Lima. The Mechanism of Action on the Sulfone Derivatives in Lepromatous Leprosy. *International Journal of Leprosy*, Vol. 20, No. 3, July-Sept. 1952.

G. WEDDELL and E. PALMER. The Pathogenesis of Leprosy. *Leprosy Rev.*, Vol. XXXIV, No. 2, April 1963.