EDITORIAL

Mycobacterial Enigmata

Much has been written in recent years on the effect of tuberculosis infection, and particularly BCG, in converting a negative to a positive lepra reaction. Presumably, though we still lack positive proof, resistance to leprosy may also be raised at the same time.

Dr. McFadzean's paper in this issue raises the question as to whether there is any action in the reverse direction—does leprosy infection in the community have any effect in causing the low-grade tuberculin positives common in some countries but not in others.

In a report on a BCG campaign under WHO in Pakistan, abstracted on p. 122 of the last issue, mention is made of the frequency of low-grade tuberculin reactions in Eastern Pakistan, as compared with its comparative infrequency in Western Pakistan. Could this be due, at least in part, to infection of the community with leprosy, which is much more common in the former region?

On the other hand, is there not a possibility that some sub-pathological, or at least sub-clinical, mycobacterial agent may be at work in the body causing a low-grade sensitivity and/or resistance to the pathogenic mycobacteria. Repeatedly cultures of acid-fast or facultative acid-fast organisms have been cultured from biopsies of leprosy patients, the material having been taken from well under the surface of the skin, and with all precautions to avoid surface infection. Not infrequently it has been claimed, though without general support, that these cultures represent *Myco. leprae*. It has even been claimed that injection of some of them can convert the negative lepromin reaction to positive. Are similar organisms to be found in non-leprous subjects? If so, are they more frequent in some countries than in others? Can their presence be correlated with low-grade positive tuberculin or with positive lepromin reactions? Can they spread from the soil or from one person to another, and if so through what channels? These are matters worthy of investigation.

If BCG administered orally is able, without causing any recognisable symptoms, to convert a negative lepromin or tuberculin reaction, why should not other unrecognised organisms, belonging to or allied to the mycobacterial group, have similar effects?

Dr. Brown's paper approaches the question from the genetic angle. First a case is propounded in which a mother and her child are infected, the child having lepromatous leprosy, and the mother

a single tuberculoid lesion. Of the three methods of infection mentioned, the first seems the most likely. If both were not infected by an unknown person with concealed lepromatous leprosy, then it seems likely that the child was infected in that way and then, when his lesions had become infectious but were still concealed, he infected his mother, but only mildly, as she had comparatively high resistance.

The question of susceptibility varying according to genetic rules as set forth in the paper is of considerable interest. It might be useful to test its application in circumstances such as those in Culion, where children brought up under chances of repeated infection either escaped the disease or recovered without treatment (p. 164).