EDITORIAL.

We regret that the prolonged illness of the Editor during 1950 has prevented the issue of the July and October numbers of the *Leprosy Review*. It has therefore been decided that subscriptions paid for Vol. XXI (1950) will be extended to December 31st, 1951.

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More accounts have been coming to hand of the danger of the promiscuous use of diaminodiphenylsulphone. It has been argued in certain places that if there are a number of deaths from sulphonamide without its use being condemned by the profession, the same risk is justifiable with sulphone. Such an argument cannot be justified in any possible way. The diseases in which sulphonamide is used are often both deadly and desperate. They can have no possible link and connection with a disease like leprosy which, in the vast majority of cases, is not mortal. There is also a common and growing impression that diaminodiphenylsulphone was first used in Africa, is the drug of choice in sulphone therapy, and that the other sulphone derivatives have, as their only merit, a breakdown into diaminodiphenylsulphone. None of these statements and arguments can be accepted as correct at the present day. Diaminodiphenylsulphone was first used in the treatment of leprosy by Dr. Robert Cochrane in India. There is no proof which can be accepted that the other sulphones in common use derive their effect from a breakdown into diamindodiphenylsulphone. Diaminodiphenylsulphone is not the drug of choice in leprosy treatment, and this cannot be reiterated too often.

A bold and very interesting experiment is being made in Nigeria, where hydnocarpus oil is being totally discarded and replaced by diaminodiphenylsulphone. The courage and wide scope of this experiment merits both praise and criticism. It is difficult, for example, to understand why hydnocarpus oil is being totally discarded; equally why, if sulphones are being chosen, the most dangerous of this group is being selected. Presumably the selection of diaminodiphenylsulphone is influenced by considerations of cost. It is necessary, however, to point out that, in the long run, the cheapest sulphone is, and must be, the safest sulphone. But whatever our doubts of the choice of diaminodiphenylsulphone, we wish the authorities in Nigeria every success in the experiment they have undertaken.

Two letters appear in this issue which are of marked interest to readers everywhere. One from Dr. A. B. Macdonald claims that the neural and tuberculoid leprosy (we combine the terms for convenience) represent an infectious form of the disease, and secondly, that this form is in effect more intractable than lepromatous leprosy. This view is in direct contradiction to the accepted and orthodox views on the subject. That does not mean that Dr. Macdonald's view is wrong. The whole subject of leprosy has long suffered from the orthodoxy of the moment, which does not always face all the facts. No one with any wide experience of leprosy can deny that the neural and tuberculoid forms of the disease may cause very grave complications indeed. We are indebted to Dr. Macdonald for the challenge which he has thrown out, a challenge which every thinking and experienced leprologist will read with considerable interest.

We print in this issue a very interesting letter from Dr. H. W. Wade on what is already, in our opinion, prematurely designated the " Michigan inoculation " cases. It is regretted that in our issue of October, 1949, we were unaware that Dr. Wade, whose international standing and reputation is of the highest, had examined one of these cases personally. The criticisms, however, which we made in this issue of the *Leprosy Review* must still stand. Dr. Wade refers to the apparent shortness of the incubation period in these cases. We feel, however, that the term ' incubation period ' is already outmoded. Let us take, for example, an analogy of the sister disease, tuberculosis. A coal miner in Scotland returns home on an April evening feeling chilled and out of sorts. He feels unable to get up the next day, and the doctor finds that he has a chronic cough and a persistent evening temperature. The X-Ray findings show that his lungs are considerably damaged by tuberculosis. Now various claims may be made for the causation of the disease. It may be held that the pneumoconiosis from which he is suffering has helped to bring about the tuberculosis condition. It may equally be held that he has undergone a long winter during which he has never seen the sun. It may be held that his food has been lacking in adequate vitamin supply, but whatever the auxiliary causes may have been, there is one thing that we know for certain, the mycobacteria of tuberculosis were in his lungs, and had been there for a very considerable time. By some means or other, which are not clear to us, his resistance had been sufficiently lowered to allow the growth and spread of these mycobacteria of tuberculosis. It will be seen here that any talk of an incubation period is useless. The

infection was most probably acquired in childhood, and had lain dormant until the conditions for its spread had become favourable.

Now let us see where this analogy leads in leprosy. In Singapore at least 2 per cent of school children show early and sometimes transient lesions of leprosy. In the adult population of Singapore, however, the existence of leprosy is not 1 in 50, as might be argued from the childhood figures, but something less than 1 in 300. We know little of what causes leprosy to attack any given individual; we do know, however, that any severe trauma or debilitating illness may cause pre-existent and dormant mycobacterium leprae to show themselves in definite clinical lesions. The analogy of the miner, weakened from lack of sunlight, and pneumoconiosis, is self evident. Everyone has seen cases of children where tuberculoid lesions appear on the elbows and knees. Here obviously the site of the early lesion has come on the area of trauma. We have seen very frequently a tuberculoid lesion on the forehead where the patient has fallen and received injury. In these cases the term 'incubation period' becomes obviously meaningless.

The two cases quoted by Dr. Wade do not constitute evidence of infection by inoculation. In Marchoux's case a man develops leprosy after receiving a needle prick 8 or 10 years before. How many of us can, we wonder, remember a needle prick at this very considerable distance of time? From a psychological point of view, how much better it is for the patient to 'remember' a needle prick which automatically changes a shameful illness into one that is both interesting and honourable. We cannot consider that Prof. de Langen's case is any real evidence of inoculation. Those of us who knew Prof. de Langen will have to admit that he would never allow any muleish adherence to literalism to spoil the drama of a good story.

Now let us return to the "Michigan inoculation" cases, and see how much has got to be invented to make this somewhat tenuous story hang together. A man suffering from open leprosy may or may not be free to walk the streets of Michigan. He would most certainly be very indiscreet to show himself in Melbourne. This entirely supposititious case, however, not only goes in at Melbourne, but also has the temerity to visit a tattooist and display a bacteriologically positive area of his skin. The tattooist is one of these broad-minded people, who do not bother to clean needles between his clients. It will be noted that the whole story is pure invention, and unnecessary invention at that. It would seem much more likely that these Marines, in their

sojourn in the Far East, had been in an endemic area, where they acquired the seeds of leprosy. The trauma resultant on the tattooing at Melbourne would easily account for the later appearance of lesions which, as we have said, is a more or less commonplace feature in the history of leprosy.