calculating the incidence of disability and deformity. Second, the study is based on data collected by paramedical workers, the authors of the paper having examined only about 20 per cent of the patients reported on. For reaching such a serious conclusion, one that has far reaching implications, a more careful study carried out by medical personnel is required.

One of the advantages of sulfone treatment of early cases of leprosy is generally believed to be the prevention of deformity. This general belief is based not entirely on impressions. There is some reported evidence on the matter, although the reports are few. Dr. R. V. Wardekar, Director, Gandhi Memorial Leprosy Foundation reported such findings at the VIIIth International Congress of Leprology at Tokyo in 1958. In a general survey of 2,340 cases he found the incidence of deformity to be 24 per cent, a figure that is in agreement with those of other workers. In a separate investigation on a follow-up study for two to six years of 2,327 patients without deformity and treated with sulfones, he reported the following findings. Among the 2,327 patients without involvement of the large nerve trunks at the time of starting treatment, deformity developed later in only 25 (1.0%) cases; among the 692 patients with involvement of large nerve trunks, deformity developed in 36 (6.0%) cases. The figure of 6 per cent may well be compared with the figure of 24 per cent in the general patient population. From these findings Wardekar concluded that practically no deformity developed in the patients detected and put on sulfone treatment in early stages.

In view of the above report, I would like to emphasize again that before cognizance is taken of the possibility that sulfone therapy in the field may induce deformity, there is need for a well planned long term study on the subject. The paper under comment should be considered only as providing a stimulus for a further study of the matter, and the views stated therein should not be seriously taken into account before more solid and convincing evidence is forthcoming to support them. In the meantime mass sulfone therapy, as a means to control the spread of leprosy, should be continued, with unmitigated efforts, on an increasingly wide scale.

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TO THE EDITOR:

With regard to present experience in the inoculation of M. leprae in animals I wish to make the following comments:

1. Most of the inoculation reports deal only with M. leprae, ignoring the disease, i.e., leprosy. In fact, most workers do not pay attention to the biologic, immunologic, biochemical and nutritional condition of the animals. It is well known that growth of M. leprae does not necessarily mean leprosy infection.

2. Much of the experimental work in leprologic centers in the world is based on Shepard's method of inoculation of M. leprae. It is well known that with this type of inoculation it is not possible to obtain a true leprosy infection in mice, with formation of globi, vacuolization of histiocytes, neural involvement, etc. Besides this, the growth of M. leprae is not progressive. As we can see, we are dealing only with a very limited multiplication of M. leprae and not with a true leprosy infection.
A limited multiplication of *M. leprae* during a short period of time is very different from progressive growth, with the histopathologic alterations mentioned above. Because of this, probably this type of bacterial growth is unrelated to the biologic ground necessary for the establishment of a true leprosy infection. Therefore it seems to me that it represents a risk, because of questionable interpretation, to do so much work on experimental leprosy on the basis of one very poor and doubtful leprosy infection, such as the one that follows inoculation of *M. leprae* into the foot pad of normal mice, which Rees compares, from the immunologic viewpoint, with human tuberculous leprosy.

For these reasons we feel that this type of inoculation is not a good example of leprosy infection, furnishing a basis for experimental work relative to some aspects of human leprosy, such as vaccination, chemotherapy, etc.

3. It is easy to observe among the researches on this problem, viz., those of Bergel, Binford, Convit, Chatterjee, Hibson, Kirchheimer, Nishimura, Pattyn, Rees, Sato, Shepard, Waters and others, that the results and interpretations of their works are very different. As examples: On inoculation of *M. leprae* into hamsters Convit induced tremendous lepromas in the ears, while Binford, Waters and Wiersma produced microscopic growth only of *M. leprae*; Shepard, Rees, Bergel and Pattyn induced growth of *M. leprae* when it was inoculated in the foot pad of mice; Nishimura and Kirchheimer failed to confirm this finding; Chatterjee induced a massive infection with the inoculation of *M. leprae* in mice, while Sato obtained only a few microscopic granulomas; Shepard explained the growth of *M. leprae* on the basis of a low temperature of some parts of the organism, and Rees opposes this interpretation, taking into account the growth of *M. leprae* in the liver of lepromatous patients and in the muscular tissue of the extremities of mice. This is in agreement with the concept that a very severe and persistent infection, such as leprosy, cannot depend on slight modifications of the organic temperature of an organism.

4. A complete experimental leprosy infection was obtained by Bergel (*Derma
tologica Tropical 3* (1964) 115-121). In this work account is taken of the biologic ground for the growth of *M. leprae*. Twenty months after inoculation of *M. leprae* in the foot pad of rats given a prooxidant diet, the formation of globi and necrotic alterations were observed.

It would be highly desirable that other qualified investigators with experience in nutritional work try to repeat Bergel's work with the use of prooxidant diets, as was recommended by the Committee on Pathology and Experimental Transmission of the VIIIth International Congress of Leprology, Rio de Janeiro, Brazil, 1963.

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