Inoculation of *M. leprae* in Animals

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 leprology 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 tuberculoid 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, Hilson, 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 Wiersema 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 (*Dermatologica Tropica* 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 neuritic 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.

—Meny Bergel
Director
Laboratorio de Investigaciones Leprologicas
E. Zeballos 3411
Rosario, Argentina
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