Reply to Dr. Bergel's Letter to the Editor

TO THE EDITOR:

The Editor has asked me if I wish to reply to Dr. Bergel's letter. I am reluctant to do so because I believe that communications of this sort too easily degenerate into useless exchanges of personally motivated assertions. The only really suitable scientific communication is a full-scale publication with adequate description of methods, full presentation of results, cogent discussion, and conclusions; the paper should be published in a journal that requires review by knowledgeable scientists before possible revision and acceptance. The reader is thereby provided with the evidence that he needs to form his own conclusions about what has been written in the paper.

If I did not reply to the statements of Dr. Bergel, however, some readers might conclude that I have at least partially accepted his assertions. To avoid such confusion let me simply state that, with one exception, I think all of the limitations and observations listed by Dr. Bergel are either wrong (entirely or in substantial part) or trivial. The exception, of course, is his first limitation; as far as I know, no one has claimed that the experimental *M. leprae* infection in normal mice resembles human lepromatous disease histologically. The concentration of *M. leprae* in mice is usually lower, but the values in mouse-foot-pad lesions and in human lepromatous tissue overlap.

Because Letters to the Editor often are political rather than scientific, the reader is well-advised to consider their purpose. I have given the reason for my reply. It may be that Dr. Bergel feels that the infection

of normal mice with *M. leprae* provides evidence against his theories, which emphasize the harmful effects of pro-oxidant diets. If so, this is most unfortunate because the experimental infection in the mouse and the similar infection in the rat provide opportunities to explore the effect of diet on leprosy. I feel that this experimental area has been ignored much too long. In my own case, discussions with experts in the area have been discouraging because of stated difficulties in experimentally reproducing the usual types of human malnutrition in the mouse or rat. Dr. Bergel's nutritional theories are unique, however, and I believe that they are testable experimentally with these systems. He may be discouraged from proceeding by his belief that values that do not have a normal frequency distribution cannot be analyzed statistically (his limitation number 3). Fortunately, nonparametric methods are entirely suitable for such distributions ([1]). An approach through incisive experimentation would be most helpful. Many observers have been impressed by the association of endemic leprosy with inadequate nutrition.

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REFERENCE

1. GOLDSTEIN, A. *Biostatistics, an Introductory Text.* New York: Macmillan, 1964, pp. 61–63.