If the fundamental validity of the model of *M. leprae* infections in the footpads of normal mice is questioned, then the fundamental validity of many of the assumptions made in the more recent recommendations for the treatment of leprosy may be questioned. Indeed, if the view is taken that the mouse footpad model is completely lacking in validity, then many of the more recently recommended treatment regimens may also completely lack validity. If there is indeed no suitable model to test new antileprosy drugs in animals or *in vitro*, then the many drugs, the many regimens, the revival of old and ineffective drugs, etc., could be interpreted as being completely disorderly, confused and likely to be ineffective. Taken further, this line of reasoning could lead to the conclusion that leprosy control by chemotherapy is unattainable and that it is impossible to predict the future course of leprosy in the world.

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Superoxide Production in PMNs from Leprosy Patients

To the Editor:

In a recent paper (Int. J. Lepr. **46** [1978] 337–441), Dr. O. Rojas-Espinosa reported his results in determining superoxide production of polymorphonuclear leukocytes (PMNs) from patients with leprosy as compared with those from normal individuals. Levels obtained were essentially similar. In addition, he found no significant difference between superoxide production of PMNs from patients with standard lepromatous leprosy and that of PMNs from patients with reactional leprosy (RLL). The author analyzed his results and compared them with those previously reported by us (Clin. Exp. Immunol. **20** [1975] 257–264) as follows: "Goihman-Yahr, *et al.*, found that patients with any type of leprosy, except reactional (RLL) lepromatous leprosy, had normal numbers of NBT-reducing cells. In patients with RLL, the proportion of reducing cells was significantly raised. We did not find a significant increase in the O_2^- levels produced by PMN from patients with RLL when compared with lepromatous patients without reaction."

From these comments, the reader might conclude that Dr. Rojas' results are at variance with ours, at least concerning RLL. This is not the case at all. As I feel that PMN activation is a rather distinctive feature of RLL, the issue should be clarified.

In the method which we used (a modification of Matula and Paterson's, New Engl. J. Med. **285** [1971] 311–317), heparinized peripheral blood is incubated with NBT at 37°C in siliconized excavated glass slides. We found that blood from patients with active RLL had a significantly higher proportion of NBT-reducing PMNs than blood from normal individuals or from any other kind of leprosy patients. We also found that the above was not due to any intrinsic difference between PMNs from RLL patients and those from other persons. Thus, if blood was incubated *in vitro* with endotoxin and NBT, the proportion of NBT-reducing PMNs reached a similarly high level in all groups. We concluded that spontaneous activation (i.e., without incubation with an additional activator) was brought about in RLL patients by some factor, presumably of immunologic nature. Further work has been done in this direction, but it is not germane to the current discussion.

Dr. Rojas-Espinosa employed a method by which PMNs were isolated from peripheral blood and then put in the cold (thereby presumably suppressing any pre-existing metabolic burst). PMNs were then incubated at 37°C with cytochrome and latex particles. The latter are quite capable of inducing an activation comparable to that caused by endotoxin. Dr. Rojas was then

simply determining the capacity of PMNs to become activated *in vitro* and to generate superoxide. He did not find out whether activation pre-existed in patients with RLL. His results are concordant with ours except that he did not explore what was happening to the metabolism of PMNs in the patients when they had symptomatic RLL. This latter point is a key one in our concept. It must be added that it is conceivable that NBT reduction, as estimated by cytologic methods, is affected not only by true metabolic activation but also by availability of NBT to the cell. Factors such as PMN permeability may be of importance.

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Reply to Dr. Goihman-Yahr's Letter to the Editor

To the Editor:

In relation to Dr. Goihman-Yahr's comments on my paper on superoxide production (O_2^-) in leprosy (Int. J. Lepr. **46** [1978] 337–341), I accept (and regret) that the paragraph he mentions seems to point out discrepancies between my results and his (Clin. Exp. Immunol. **20** [1975] 257–264). My intention was not to show these supposed discrepancies (which are not real) but to simply indicate that lepromatous patients with an active leprosy reaction do not differ from those without it in regard to their leukocytes' ability to generate O_2^- when appropriately stimulated.

As Dr. Goihman-Yahr mentioned, I did not measure the spontaneous production of O_2^- by leukocytes of the patients under study and because of this my results cannot be at variance with his. What is happening to the metabolism (in terms of O_2^- production) of PMN leukocytes in the patient with reactional leprosy, is a point that has to be studied.

The very important point is, I believe, that PMN leukocytes from lepromatous leprosy, with or without a complicating leprosy reaction, do not seem to be defective in regard to the metabolic activities so far examined.

—Oscar Rojas-Espinosa, Ph.D.

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Teaching and Training in Leprosy

To the Editor:

I refer to Volume 47, Number 2 of your esteemed journal dated June 1979 in which there is given a summary of the present teaching and training facilities in leprosy on pages 176–196.

On page 195 there is stated as a Note: "In September 1978, political and military disturbances in Ethiopia are most likely to disrupt this center's activities, and it may have to close."

As you will know from your visit to Ethiopia when we had the pleasure of having you as our visitor in connection with the Kellersberger Memorial Lecture, ALERT is still carrying on with uninterrupted activities, and there is no reason to believe that this center will have to close. On the contrary, we are intending to give even more comprehensive teaching in leprosy in the future.

In order to correct the false picture your readers may have gotten because of the referred note, I shall be grateful if you will be kind enough to insert a correction in your next issue.