

CORRESPONDENCE

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THE KAHN UNIVERSAL REACTION IN LEPROSY

[In an article by Pineiro, Bishop and Kahn, noted in the abstract section of this issue (p. 255), there was a statement about the exceptional occurrence of weak reactions in forms of leprosy in which the disease is widely disseminated. The authors were asked about that point, and Doctor Kahn responded with the following letter.—EDITOR.]

TO THE EDITOR:

Your interest in our studies of the universal reaction in leprosy is greatly appreciated. These studies have been very limited in our laboratory because of lack of opportunity to get blood specimens from leprosy patients.

So far as our studies indicate, the universal reaction behaves like other antibody reactions in immunity. In tuberculosis, for example, the universal reaction will increase in early tuberculosis and, as the patient gets well, the reaction drops to the normal level of that person, but in miliary or highly advanced tuberculosis, instead of an increase in the strength of the universal reaction, there is a reduction. The explanation is that these patients do not have the capacity to produce antibodies; hence, the universal reaction becomes weak.

In lepromatous leprosy the pattern is closely similar. Let me quote from page 299 of my book "Serology with Lipid Antigen." Although I am not showing the graphs, the text is understandable:

An examination of the graphs of the universal reactions of patients DN and JG with lepromatous leprosy reveals extraordinarily marked precipitation. These patients were reported as having moderate lesions. On the other hand, the graphs of the universal reactions of patient JL with "burnt out" lepromatous leprosy, and patient CL with advanced lepromatous leprosy, reveal far less precipitation.

It would seem reasonable to interpret these results by assuming that the patients with moderate lesions possess a high degree of immunity. Having a high degree of immunity, they possess a marked capability for the production of antibodies to tissue lipids, which explains the graphs of DN and JG. In "burnt out" leprosy the capability for antibody production may be marked, but the quantity of liberated antigenic lipids is probably low since tissue destruction is practically at a standstill. In the advanced form, on the other hand, the capability for antibody production is low. The result is that precipitation in the graphs of JL and CL is not as marked as in the graphs of DN and JG.

We were interested in the report by Olitzki in the *Journal of Investigative Dermatology* [27 (1956) 35] in which he shows the marked

reduction in universal reactions of lepromatous patients following therapy. But the outstanding feature of Olitzki's work was that he and his associate, Sagher, extended their observation period to two to three years. Then, the differences in the reactions were quite outstanding. This observation is of special interest to me because, as is well known, the pathologic processes in leprosy go on at a slow rate, and it would therefore seem reasonable to expect that the immunologic processes would similarly go on at a slow rate. Time is apparently an important factor in the study of serologic changes in leprosy. We must keep this in mind because we are so accustomed in the serology of syphilis to expect a change in the serologic picture in the course of a few months.

In summary, I would add that I believe the universal reaction could prove of value in the therapy of leprosy, provided the results are interpreted in relation to the clinical condition of the patient.

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