NEW LIGHTS IN LEPROSY

TO THE EDITOR:

In order to demonstrate the possible relationship between leprosy and autooxidation of lipids, I have made several experimental studies which are summarized in this communication.

- 1. Inoculation of the Hansen bacillus to rats fed with prooxidant diets.—During a period of 26 months I made three serial inoculations of the Hansen bacillus through rats kept on a prooxidant diet (low in vitamin E, with 15% linseed oil). At the end of the last inoculation the testes of the rats had considerably large numbers of acid-fast bacilli. With these bacilli two biological tests were made:
- (a) These bacilli were seeded on Loewenstein-Jensen medium, and up to 120 days there were no development of colonies, at various different temperatures. In other words, these bacilli do not grow on an artificial medium designed for acid-fast bacilli.

(b) A lepromin was made with these bacilli, and was tested in comparison with integral and bacillary lepromins made from human lepromas. The rat-tissue lepromin behaved in exactly the same way as human lepromin: tuberculoid cases reacted but lepromatous cases did not. [Semana méd. 111 (1957) 479, 1148, 1313; 112 (1958) in press.]

These two biological tests prove that the bacilli found at 26 months, after three serial inoculations, are Hansen bacilli. The great number of these bacilli proves that the Hansen bacillus grew in these rats.

- 2. Anticeroid activity of antimycobacterial compounds.—Rats kept on a prooxidant diet for periods up to five months, after which antileprosy drugs are added (isoniazid, 0.2%; or DDS, 0.2%; or TB-1, 0.1%), do not form ceroid pigment in the subcutaneous, perigonadal and perirenal fat. Controls form this pigment in large amount. This proved that these antimycobacterial compounds have a partial antioxidant activity. [Ibid. 110 (1957) 855.]
- 3. Isoniazid as a biological antioxidant.—The biological activity of isoniazid was studied in rats fed the prooxidant diets. It was found that isoniazid at the level of 0.2% in the diet protects against the "yellow fat," testicular degeneration, and pigmentation of the uterus. These symptoms appeared in all the controls. [Ibid. 110 (1957) 192.]
- 4. Histochemical similarity between lepromatous tissues and yellow fat.—The yellow fat of a rat fed a high prooxidant diet for several months, when the animal is injected with methylene blue, stains very strongly and keeps the color for a long time. The normal white fat does not stain. The sampling happens in vitro with both types of fats. To summarize, the yellow fat has the same property as the lepromatous tissues that stain strongly, in vivo, with methylene blue. This fact is related to the redox potential of these tissues. [Leprologia 2 (1957) in press.]
- 5. Mechanism of antileprotic activity of sulfones.—Take into account the following facts: (a) antioxidant activity of DDS, demonstrated by Lips; (b) anticeroid activity of DDS, demonstrated by Bergel; (c) antioxidant activity of many amine compounds in industrial use (Goodrite Chemicals, Dupont, etc.); (d) antioxidant activity of compounds related to DDS, such as 4,4'-diaminodiphenyleters, 4,4'-diaminodiphenylmethane, etc.; (e) antimycobacterial activity (in tuberculosis) of many amine compounds; (f) biological antioxidant activity of a secondary aromatic amine, n,n'-diphenyl-paraphenylendiamine (DPPD), demonstrated by Singsen and Matterson, In view of these facts I postulate that the sulfones are amine compounds and for the amine groups act as biologic antioxidants in the stabilization of the fats of the organism. Their antileprosy activity is due to their antioxidant activity. [Semana med. 111 (1957) 164.]
- 6. Treatment of lepromatous leprosy with an antioxidant system.—In order to prove the antileprosy activity of the antioxidants I have started to give to a small group of lepromatous patients (L2 and L3) the following mix-

ture: vitamin E, 100 mgm.; isoniazid, 20 mgm.; vitamin C, 50 mgm. These three compounds form an antioxidant system: vitamin E is the primary antioxidant, isoniazid is the metal deactivator, and the ascorbic acid is the synergist. I give 8 of these capsules daily, as the only treatment. Up to the present (seventh month) this antioxidant system has shown a very remarkable antilepromatous activity, and it is entirely nontoxic.

Comment.—From the facts related it is concluded that there is a very clear relationship between leprosy and the autooxidation of lipids. I can say that the pathogenesis and chemotherapy of leprosy are just problems of autooxidation of lipids.

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