M. leprae oxidizes both L- and D-DOPA at the same rate, and acts on a variety of diphenols including epinephrine and norepinephrine.

8. M. leprae retains α-diphenoloxidase activity in the passage of the bacillus from the human to the animal (mousetail armadillo) host, indicating that it is a constitutive enzyme of the bacillus.

Precautions.
1. M. leprae suspensions used in enzyme assays have to be obtained from fresh tissues or tissues kept at 0°C or below, to avoid denaturation of α-diphenoloxidase. The entire separation procedure also has to be carried out in the cold. If the bacterial preparations have little activity to start with, both heated and unheated samples would give similar results. Denatured enzymes often retain a residual activity.

2. Many tissues contain inhibitors of α-diphenoloxidase. As such, testing the enzyme activity in crude homogenates of lepromatous tissues would yield no definitive results. Adequate amounts of purified organisms have to be used.

3. It is important to have heated samples as controls. We tested several cultivable mycobacteria as well as M. lepraemurium (separated from mouse spleen), and two unidentified mycobacteria (obtained from the liver and skin tissues of two other species of mammals). In presence of added DOPA, both heated and unheated suspensions of these bacteria gave similar results, proving that the organisms have no enzyme activity.

4. The mere demonstration of any enzyme in a host-derived organism is of little significance, especially if the enzyme is ubiquitously present in the host tissues as well. Acid-fast staining alone is a poor criterion for excluding host-tissue materials adsorbed on the bacilli. It has to be established that the enzyme is an inherent property of the organism itself.

Anyone who is interested is welcome to visit our laboratory. We would be willing to demonstrate that metabolically active preparations of M. leprae convert DOPA to quinine enzymatically and that this activity is distinct from mammalian tyrosinase.

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Nutrition and Leprosy

To the Editor:

The editorial “Effect of Malnutrition on Leprosy” (IUL, 44 [1976] 374) contains this statement: "There is, it is true, no evidence that any specific or group of dietary substance is promotive of leprous inflammation . . . ." Likewise, the following statement is quoted in an editorial appearing elsewhere (Lepr. Rev. 46 [1975] 5): "No direct link between malnutrition and leprosy has been convincingly demonstrated."

These statements are not true. Bergel, in more than 20 publications issued during the last 30 years, has demonstrated that a prooxidant diet, i.e., a diet with low content of vitamin E and high content of fatty acids, is promotive of leprous inflammation. The work of Bergel was confirmed by Mason and Dju (Symposium on Research in Leprosy, Leonard Wood Memorial-Johns Hopkins University, Baltimore, 8 May 1961, p 264).

We feel that the relationship, leprosy autooxidation of lipids, is the most important known factor in the pathogenesis of leprosy and it can be used as a starting point for experimental work dealing with prevention and treatment of leprosy. Unfortunately, leprologists are not very familiar with the chemistry of autooxidation, namely, with antioxidants, prooxidants, metal deactivators, free radicals, chain reactions, hydroperoxides, polymerization, copolymerization, tocopherols, endoperoxides, superoxides, etc.

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