tance becomes highly prevalent." ([4]). In fact, the MDT regimens, i.e., two drugs [DDS and rifampin (RMP)] for paucibacillary (PB) leprosy and three drugs (DDS, clofazimine and RMP) for multibacillary (MB) leprosy, were designed on the principle that they would be effective against all the strains of *M. leprae* regardless of their susceptibility to DDS ([6]). Hence, whether the global prevalence of DDS resistance is increasing or declining is virtually irrelevant to the therapeutic effect of MDT ([5]), and there is no need to closely monitor trends of resistance to DDS.

Roche and his co-authors expressed their concern regarding the gradual disappearance of mouse foot pad laboratories ([4]). Unfortunately, for various reasons, this is probably an irreversible trend; sooner or later, the susceptibility of *M. leprae* to drugs will be tested by molecular genetic methods ([1,2]) rather than by the mouse foot pad technique. Moreover, instead of DDS-resistance, one should pay attention especially to resistance to RMP, by far the most powerful bactericidal drug against *M. leprae* ([3]), and an irreplaceable component of the MDT regimens.

—Baohong Ji, M.D.

Bactériologie et Hygiène
Faculté de Médecine Pitié-Salpêtrière
Paris, France

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"Metabolic Theory of Leprosy"

To the Editor:

I have read the commentary of the book *Metabolic Theory of Leprosy* (IJL 68;333, 2000). Almost the entire article deals with matters unrelated to the metabolic theory. Only three paragraphs refer to it, and in a wrong way:

a) the paragraph "the treatment should consist in antioxidant diets" is false;

b) the paragraph ". . . and that should not be killed (*M. leprae*)" was extracted from: "the treatment of leprosy should be centered on the autooxidative disease and not on *M. leprae*. The use of rifampicin as monotherapy demonstrates that it is harmful to act solely on *M. leprae*." As a matter of fact, dapsone does not act on *M. leprae*, but on autooxidative disease.

The comment of Dr. Hastings is very poor and in part is false. Also, it does not give to the readers any idea about the mentioned theory.

—Prof.Dr. Meny Bergel

Director
Leprosy Research Institute
Buenos Aires, Argentina