# Diabetogenic Effect of Dapsone

TO THE EDITOR:

The isoniazid (INH) acetylator phenotype of 79 Brazilian leprosy patients, mostly Caucasoids (22 Negroids), which included 45 males and 34 females, 47 of them with diabetes mellitus (26 males and 21 females, 12 of them Negroids), was assessed by means of Eidus, *et al.*'s method ([2]). All of them were under dapsone therapy for at least 5 years. The same procedure for investigating the INH-acetylator phenotype was applied to 30 Brazilian Caucasoids with diabetes mellitus but without leprosy (14 males and 16 females).

The frequency of the slow INH-acetylator phenotype among the 32 nondiabetic leprosy cases (47%) did not differ significantly from that found among the 30 diabetic individuals without leprosy (53%) or among Brazilian Caucasoids (57%; N = 119) and Negroids (50.4%; N = 115) with pulmonary tuberculosis ([1]). In contrast, the slow INH-acetylator phenotype predominated among the 47 diabetic leprosy patients (76.6%), this frequency being significantly higher than that seen among the nondiabetic leprosy patients, the diabetic persons without leprosy, or the patients with pulmonary tuberculosis.

Regression analysis applied to the data recorded on the leprosy cases has shown that the blood level of glycosylated hemoglobin does not depend upon age, sex, duration of the disease, or years of dapsone therapy, but it is correlated to both the slow INH-acetylator phenotype and the fasting plasma glucose.

The results presented here indicate that diabetes mellitus has no influence on the INH-acetylator phenotype, since the frequency of slow INH-acetylators found among the diabetic individuals without leprosy was almost identical to that observed among nondiabetic Brazilian Caucasoids or Negroids. On the other hand, since the blood level of glycosylated hemoglobin is associated with the slow INH-acetylator phenotype, while the *in vivo* acetylation of dapsone depends on the same acetyltransferase used for acetylating INH ([3–6]), one would infer that slow INH-acetylator leprosy patients when under dapsone therapy would be more exposed to an undescribed diabetogenic effect of this drug than fast INH-acetylators. This fact would explain the high frequency of slow INH-acetylators among the diabetic leprosy patients.

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