

## GENETIC POLYMORPHIC SYSTEMS AND HANSENIASIS

EDITORIAL

Up to the present the following genetic polymorphic systems were analysed in samples of Hansen's disease patients : ABO, Rh, MNSs, P, Kell, Lewis, Duffy, Kidd and Diego blood groups; secretion of ABH substances; taste sensitivity to phenylthiourea; S hemoglobin; beta-thalassaemia; glucose - 6 - phosphate dehydrogenase; phosphoglucomutases 1, 2 and 3; glyoxalase; properdin factor B; acid phosphatase; adenosine deaminase; esterase D; adenylate kinase; glutamic pyruvic transaminase; 6-phosphogluconate dehydrogenase; haptoglobins ; transferrins; group specific protein; beta-lipoprotein Ag; alpha-1-antitrypsin ; ceruloplasmin; beta-2-glycoprotein I; third component of complement (C<sub>3</sub>) ; Inv antigens; pseudo-cholinesterase; HL-A antigens. Of course, these almost forty polymorphisms were studied with the hope of finding associations between hanseniasis and genetic markers. However, most of these investigations provided negative or controversial results, while very few have shown associations of disputable importance.

The negative results were indeed expected with greatest probability 1, 2, since most of the genetic polymorphisms were chosen for study without a logical indication that susceptibility to hanseniasis might depend upon the

polymorphic genes under investigation. Concerning the conflicting results, they may be most probably attributable to large sampling fluctuations due to small samples, to racial and geographical variations, to inappropriate controls and to variation in the composition of the hansenic samples. Thus, some of them included only Virchowians patients, others were composed of patients belonging to both polar types of hanseniasis, others included all forms of hanseniasis, and so on.

At any rate, such types of studies, in spite of being relevant for some geneticists, are useless for practical hansenologists. As a matter of fact, even if an association between a well-known polymorphic system and hanseniasis could be demonstrated beyond any doubt, this association would only serve to indicate that hanseniasis is one of the several forces that are maintaining the analysed polymorphism. However, the practical hansenologists, who are interested in the applications that Genetics may provide to Hansenology, will make no use of such information, since it has no value for diagnostic and prognostic purposes. Therefore, in our opinion, the choice by hazard of genetic polymorphic systems for investigation in hanseniasis should not be stimulated among hansenologists.

(1) BEIGUELMAN, B. An appraisal of genetic studies on leprosy. *Acta Genet. Med. Gemellol.*, 21:21-52, 1972.

(2) BEIGUELMAN, B. Leprosy and genetics. A review of past research with remarks concerning future Investigations. *Bull. WHO*, 37:461.476, 1967.

At the present status of knowledge we think that the only polymorphic systems that should deserve the attention of hansenologists are the glucose-6-phosphate dehydrogenase (G-6PD), dapsone acetylation and methemoglobin NADH reductase. Of course, such polymorphisms should not be investigated with the aim of finding

associations between them and hanseniasis, but with the main purpose of verifying the pharmacogenetic response to dapsone presented by patients with G-6PD deficiency, slow and rapid dapsone acetylators, and patients who are heterozygous for the NADH reductase deficiency gene<sup>3</sup>.

Bernardo BEIGUELMAN

---

(3) BEIGUELMAN, B. Terapêutica da lepra e Farmacogenética. *Hansen. Int.*, 1(1):61-78, 1976.

(4) Departamento de Genética Médica, Faculdade de Ciências Médicas, Universidade Estadual de Campinas, C.P. 1170, 13.100, Campinas, SP.