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A New Bisalbuminaemic Family

THIS communication reports the first *bisalbuminaemic* family found in France. As the family tree shows (Fig. 1), the anomaly seems to be due to a co-dominant, autosomal

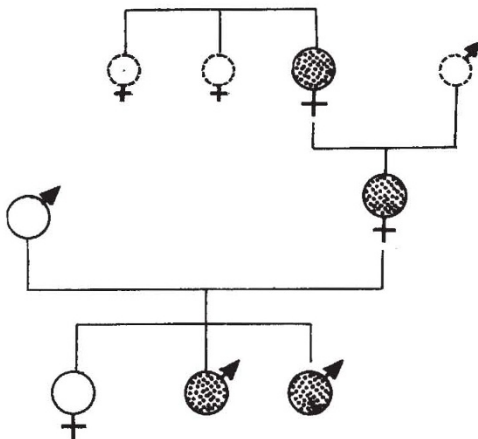


Fig. 1. The family tree. Positive cases, black spotted circles; negative cases, blank circles; cases not examined, broken circles



Fig. 2. Cellulose-acetate strip electrophoretogram of a positive case

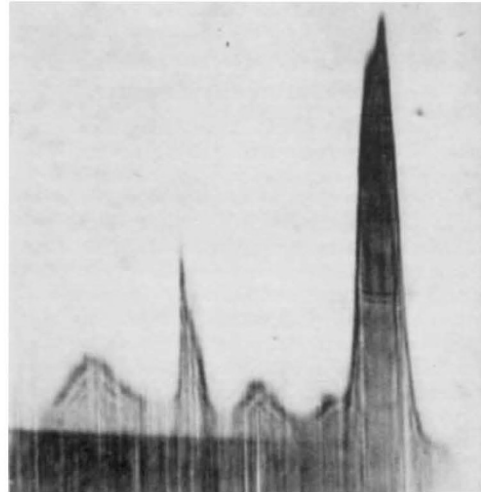


Fig. 3. Free electrophoresis of one positive case



Fig. 4. Immunoelectrophoretic analysis of a positive case. Development with equine anti-human serum. The serum of the subject is diluted to 1/30th; so the α_1 -globulin traits are distinctly separated from the albumin trait

allele at the albumin locus; we have confirmed this general conclusion. The separation of the two albumins was excellent on a cellulose acetate strip (Fig. 2) and shows, without any doubt, that the anomalous albumin is the slower one. But free electrophoresis using a Perkin-Elmer apparatus disclosed only a weak 'shoulder' on the cathodic side of the albumin band (Fig. 3). Immunoelectrophoresis and the Ouchterlony test using anti-human serum, or specific anti-human albumin serum, did not show any difference between the two albumins (Fig. 4). The anomalous albumin, evaluated after dye elution on the cellulose acetate strip, represents in all cases about 40 per cent of the total albumin. The subjects presenting the anomalous trait were normal in all other respects.

A recent check disclosed that about fifteen *bisalbuminaemic* families have been reported in the literature¹. Nearly always, the anomalous albumin demonstrated the same characters. Only Tarnoky *et al.*¹ and Wieme² conclude that the anomalous albumin was the faster-moving one. But in these cases, due perhaps to technical imperfections, the separation of the two bands was very poor and so any comparison with normal sera is not likely to lead to any clear-cut conclusion. We agree with Harris³ that *bisalbuminaemia* could be a unique hereditary trait. Taking into account the chemical results obtained by Gitlin *et al.*⁴, it may be attributed to a mutation involving a specific point in the albumin polypeptide chain.

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