TUBERCULOID LEPROSY IN IDENTICAL TWINS

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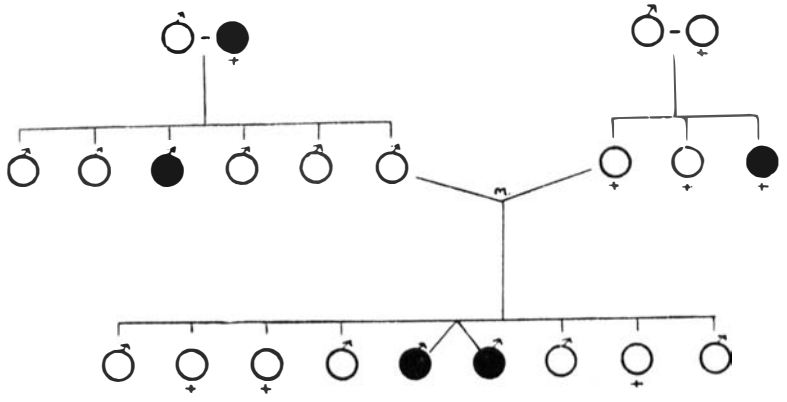
In a general paper on leprosy, Ryrie referred to a particular instance of this disease in twins. He did not say whether they were identical twins, and from his brief description it appears that one had tuberculoid leprosy and one lepromatous leprosy. Keil wrote specifically about hereditary factors in leprosy and gave several instances of twins in contact with infection, both of whom or neither of whom developed the disease. Rotberg and others have from time to time referred to a constitutional factor which determines the onset of clinical disease and which was probably hereditary.

Twins, who from all appearances were identical, aged 10, were seen by one of us (M.M.S.) at a rural treatment village in the Teso district of Uganda, and admitted to the Kumi-Ongino settlement in July, 1957. They had already had several months' sulphone treatment, and the patches were repigmented. The initial lesions had appeared about the same time in each child in 1955. They consisted of typical hypopigmented areas. In one twin they were more extensive, involving both legs anteriorly and posteriorly above and below the knee. The other twin had more limited areas affected on the limbs and one discreet patch on the back just above the right iliac crest. Bacteriological examination was negative but a biopsy in each child confirmed the diagnosis of resolving tuberculoid leprosy: the Mitsuda or late lepromin reaction was 3 mm. in each child.

A visit was paid to their home 55 miles further north, and the various members of their family seen, together with the local chiefs who had been instrumental in calling them together. Reliable details of great aunts and great uncles were difficult to ascertain, but the following information was obtained and is regarded as reasonably authentic:—

Of the grandparents, the grandmother on the father's side had leprosy, and from the description it was probably the lepromatous type. She died before the twins were born.

The father had five brothers, but no sisters. One of the brothers had tuberculoid leprosy and has now been discharged after three years' treatment. The mother had two sisters, one of whom had tuberculoid leprosy. She had no brothers. There was a monogamous marriage and neither the father nor the mother of the twins had been married before. By this marriage there were twelve children, three of whom died in infancy. Of the survivors,

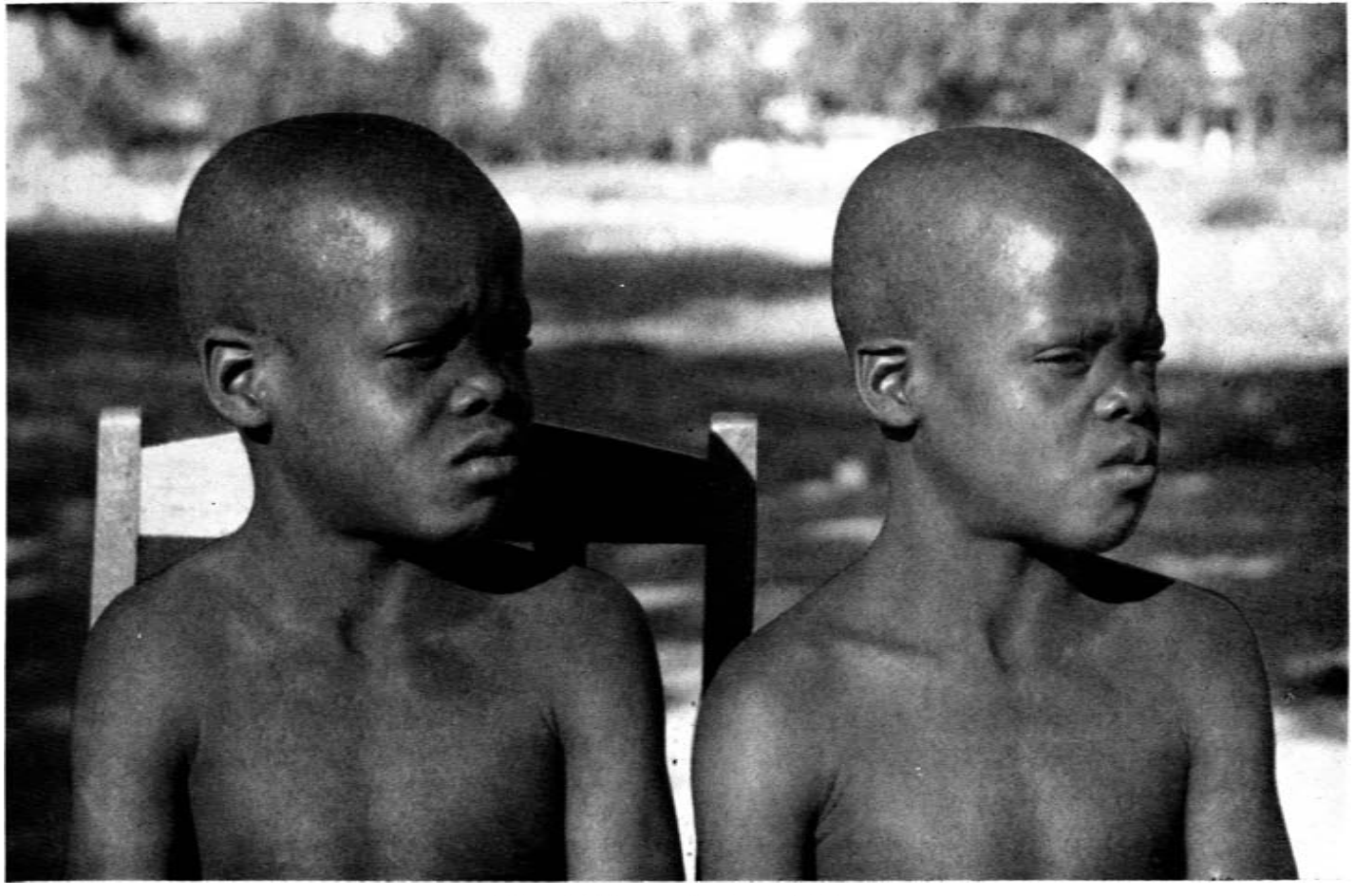


Genealogical Tree showing infected members of the family.

four were older than the twins, three younger. Only the twins developed leprosy, and it was of the same type.

Two interesting points arise. In the first place, in this part of Uganda the population density is approximately 90 to the square mile, or one individual to seven acres. There are no villages and each family homestead is centred in the middle of its farmland in comparative isolation. Thus, a family of ten or twelve would probably live 500 yards from its own boundaries, or at least half a mile from its neighbours' houses. Infection with leprosy in childhood in Uganda is therefore nearly always the result of contact within the family. The only family sources in this instance were an aunt on the mother's side, and an uncle on the father's side, both of whom had tuberculoid leprosy. To those unfamiliar with the country, ingenious explanations may suggest themselves of why and how two children out of nine should contact the disease, explanations perhaps more in line with the views that association with an open case is necessary for infection. It is difficult to believe, however, that the twins had any more exposure to infection than the other seven children, and as it is not customary for a household to include aunts and uncles, whatever contact took place must have been limited and not of the prolonged intimate type often asserted to be necessary. The prevalence of the disease in this part of Uganda is around 20 per thousand, and the lepromatous rate is less than 10%. In the average one open case can be found in every five square miles. The simplest explanation is, therefore, that the twins derived their infection within the family by contact with an aunt and uncle, both of whom had tuberculoid disease.

Secondly, and particularly, the interest of the disease occurring in identical twins is demonstrated in the genealogical



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tree. In three generations, only a minority contracted the disease although all were exposed to a similar risk of infection. In the third generation only two out of nine siblings have so far developed leprosy, and they the identical twins.

It has been suggested elsewhere by one of us (J.A.K.B.) that there are many anomalies in the epidemiology of leprosy. Conjugal infection is relatively uncommon—only a fraction of the children of infected parents develop the disease; the majority of people living in constant relationship with patients do not develop leprosy whilst others do so after brief or trifling contact. These anomalies are most easily explained on the assumption of a constitutional factor that is transmitted genetically and which determines whether successful invasion will take place. The occurrence of leprosy in these twins, particularly of the same type, is regarded as confirmatory evidence of this hypothesis. Patients with leprosy belong to a race within a race, they come to light as a result of contact; but those who are not infected because they never meet infection, continue to transmit the constitutional factor to some of their progeny. It is not suggested that this factor is a single gene, indeed it is far more probable that it is the presence or absence of components of a polygene. In a country so rural as East Africa where there is no herding of the population into artificial communities, this is the simplest explanation of the haphazard occurrence of patients who stoutly and rightly deny any memory of the disease within their families, and any but the most accidental contact with infected persons. In the epidemiology and aetiology of leprosy, and in the organization of control, as much prominence should be given to the hereditary factor in susceptibility as is usually given to the bacillus.

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