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## **Further studies on the *Trypanosoma brucei* group trypanosome, isolated from a patient infected in Anger-Didessa Valley, West Ethiopia, using the blood incubation infectivity test (BIIT)**

### **Short communication**

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An autochthonous sleeping sickness case was recently diagnosed from the Anger-Didessa Valley, West Ethiopia (Feseha Gebre-Selassie, 1987). A preliminary blood incubation infectivity test (BIIT) (Rickman, 1984) carried out on the *Trypanosoma brucei* group trypanosome isolated from the patient, showed it to be sensitive to pooled human sera.

Since human isolates that gave negative BIIT results have previously been reported (Mehlitz et al., 1982), it was decided to test the present isolate against the serum of the patient from whom it was isolated. Sera were therefore collected from the patient and from the three individuals used in the preliminary BIIT study. Serum was collected six months after the patient had been treated and cured. The four sera were used separately in a second set of two BIIT experiments using the first passage of the trypanosome isolate. Results from both experiments showed the trypanosome to be resistant to the patient's serum and sensitive to the other three human sera, despite repeated administration of cyclophosphamide (1 mg/20 g/dose) to the respective test mice.

No confirmed sleeping sickness case has ever been diagnosed from the Anger-Didessa Valley, which is endemic for *T. brucei* group trypanosomes (Langridge, 1976). The BIIT results suggested that either antitrypanosomal antibodies are present in the three human sera, or that the patient might be deficient in high density lipoprotein (HDL) which is considered as one of the non-immunological barriers that *T. brucei* group trypanosomes should overcome in order to establish themselves in man (Rifkin, 1978; Mims, 1982).

However, in spite of the fact that the patient's liver was in normal condition, determination of HDL and low density lipoprotein (LDL) using polyanion precipitation technique (Friedwald et al., 1972), revealed his HDL to be much lower than his LDL (HDL = 30.04 mg%, LDL = 120.04 mg%), while that of the other three sera was more or less equal (HDL = 52.73 mg, LDL = 68.05 mg%, average values). The indirect fluorescent antibody test (IFAT), using freeze dried *T. rhodesiense* antigen (Etat 1/18 LOT 83B28) (from Antwerp Institute of Tropical Medicine) and the pretreatment serum of the patient as positive control, revealed no antitrypanosomal antibodies demonstrable in all the test sera except for some in that of the patient.

Therefore, the results obtained infer that the decreased production of HDL of the patient, which could be due to malnutrition or under genetic control, might have played a pivotal role in the establishment of a *T. brucei* strain that is unlikely to infect people under normal circumstances. Although HDL is the trypanolytic factor in human serum, its activity is modulated in the presence of excessive LDL (Rifkin, 1978): i.e. a strain of *T. brucei* group trypanosome that is lysed by pure HDL

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survives this lytic effect in the presence of excessive LDL. The mechanism(s) involved is not clearly understood.

The features of the trypanosome necessary to confer infectivity to man are poorly understood. In human volunteer inoculation studies (Onyango et al., 1966; Geigy et al., 1975), it has been shown that *T. brucei* group trypanosomes which were isolated from animals and were infective to volunteers, were also resistant to volunteers' sera in laboratory mice. In the present study, the resistance of the trypanosome isolate to the serum of the previously infected individual corroborates with the volunteer inoculation experiments.

The present investigation demonstrates the existence of individuals that could play a significant role in the epidemiology of *T. brucei* group trypanosomes under conditions whereby transmission is essentially from animal to man.

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