Investigation of the incidence of sleeping sickness in the human population

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Sleeping Sickness Survey in Musoma District, Tanzania

Part I. Onyango, R. J. & Woo, P. Investigation of the Incidence of Sleeping Sickness in the Human Population ................................. 181
Part III. Mwambu, P. M. & Mayende, J. S. P. Survey of Cattle for the Evidence of T. rhodesiense Infections ......................................... 206
Part IV. Geigy, R., Mwambu, P. M. & Kauffmann, M. Examination of Wild Mammals as a Potential Reservoir for T. rhodesiense ............. 211
Part V. Onyango, R. J., Geigy, R., Mwambu, P. M. & Moloo, S. K. The Endemicity of Rhodesian Sleeping Sickness in Ikoma-Serengeti Area – Final Discussion ................................. 221

I. Investigation of the Incidence of Sleeping Sickness
in the Human Population

R. J. Onyango and P. T. K. Woo

Abstract

In a survey of sleeping sickness in the Ikoma-Serengeti area, carried out in October and November 1970, about 3,000 people living in the area were examined and none was found infected.

Introduction

Rhodesian sleeping sickness was introduced into the Musoma District mainly in the Ikoma area in the 1920’s. It is believed to have been an extension of an epidemic in Maswa, Mwanza District, which lies to the south. The outbreak of Rhodesian sleeping sickness in Maswa probably began in 1919-1921 during a period of famine but early patients were first diagnosed in 1922 (Davey 1924). Glossina swynnertoni was incriminated as the main vector of the outbreak. The spread of the disease was thought to be due to infected persons and the maintenance of intensive man-fly contact as the game population was quite small and scattered (Swynnerton 1923, 1925). Davey and McClean, however, having travelled in the affected area during an investigation of the same outbreak, found ample evidence that game was quite abundant in the area (Davey 1924). From 1925 the yearly incidence of sleeping sickness in Ikoma ranged between 12 and 265 (Fairbairn 1948). The endemic situation continued until 1954 when the last 3 cases of sleeping sickness were notified from the area. It is reported that a large number of cases of sleeping sickness was diagnosed during the endemic period

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largely among goldmine workers in the Kilimafedha and associated mines in the district. One possible reason for the disappearance of the disease in the mid-fifties is that a number of mines had been closed because of their unprofitability; consequently the population moved away (Young 1968).

In 1964, 2 patients suffering from sleeping sickness were notified from the Musoma District, one of them a resident of Seronera, the administrative centre of Serengeti National Park. From 1964 to date the following cases have been notified: 1 case in 1965; 4 in 1966; 6 in 1967; 14 in 1968 and 6 in 1969. Of these, 2 were tourists. The reappearance of sleeping sickness into this heavily tsetse-infested area caused some concern particularly as this area had been developed as a tourist centre. For this reason a survey of human trypanosomiasis (sleeping sickness) in the Musoma District was undertaken mainly in the Serengeti National Park, Ikoma Game Reserve and populated areas surrounding the game sanctuary particularly those to the north and north west about 10 miles from the borders of the Park. The aims of the survey were to find out the mechanism and the extent of transmission of sleeping sickness in this part of Tanzania. To achieve this objective it was necessary to examine people living in the area for evidence of parasitaemia; to examine the tsetse species so as to ascertain the rates and types of their infections and also their feeding habits; and to determine the infection rates in a limited number of game species and cattle with pathogenic trypanosomes particularly those of the *Trypanosoma brucei* subgroup. Full accounts of the investigations and their results are reported in this paper in four parts. In the first part an account is given of the investigations undertaken among the human population living in the survey area.

**Subjects**

A preliminary census of human population was not taken before the survey. It was assumed that because an excellent co-operation between the administration and the people existed, the majority of the population would respond to written and verbal notices and come willingly for examination at the preselected centres. In fact with regard to the people living in the Serengeti National Park, nearly a 100% attendance was achieved. Although a good number of people in the Serengeti National Park are immigrant labourers, many of them had lived and worked within the park for over 2 years. The period of residence for the female population and children may have been shorter, temporary and renewed frequently after visits to the original homeland miles away from the Park. Contact of this latter group with the vector and the infective agent was therefore irregular. Population sampled in the homesteads and in the villages surrounding the Park represented the resident local population and therefore were more permanent. The number of young adult males in the villages tended to be fewer than expected because members of this group left home in search of lucrative employment elsewhere. An attempt was made to examine people of all ages and both sexes. During the survey, inquiries were made about ill persons who could not come to the centres. Those reported to be ill were either
visited at their homes or brought in a vehicle for examination at the relevant centres.

All the resident population in the various establishments within the Serengeti National Park and villages in the Ikoma area and a few tourists at Serengeti Lodge were examined. Examinations of the people were carried out at 29 centres (see maps Fig. 1 and 2). The histogram (Fig. 3) shows the age and sex distribution of all the people examined at these centres. In all there were a total of 2,941 people.
Methods

Four methods of examinations were used during the investigation:
1. Blood film (thin and thick on glass slides) examination.
2. The haematocrit centrifuge technique (HCT).
3. The serum IgM (immunoglobulin M₂) level determination.
4. The immunofluorescent antibody test (IFT).

Blood samples for all the tests were obtained from a skin puncture on the tip of a finger in the case of every adult and juvenile and on the tip of the big toe of every child and infant. One skin puncture produced enough blood for samples required for all the four tests. The first drops were used to make both thin and thick blood films, a subsequent squeeze of the skin puncture produced enough blood for capillary tubes used in the HCT and additional pressure produced blood samples to be absorbed onto a filter paper and left to dry for use in both the serum IgM level estimations and the IFT.

All the thin blood films were fixed in ethanol at the time of taking the samples. Both these and the thick films were brought back to the laboratory at the Serengeti Research Institute (S.R.I.) and stained with Giemsa stain. Some were examined under the microscope at S.R.I. and others were brought back to the laboratories of the East African Try-
panosomiasis Research Organization, Tororo, for examination. Each blood film was examined under oil immersion lens and discarded after 200 microscope fields had been scanned.

Blood samples were examined immediately by the HCT described by Woo (1970). A portable Honda (1 kilowatt) generator was available to supply electric current for the centrifuge. It was carried at the back of a landrover to the examination centres at which surveys were carried out. Only at a few inaccessible places that were reached only by a four-seater aeroplane which was not big enough to carry all the equipment was the generator not taken and hence the HCT was not used to test the few samples obtained. The tests for the estimation of serum IgM level and for the immunofluorescent antibodies were carried out on blood spots on absorbent filter paper at E.A.T.R.O. The methods of the tests used are similar to those described by Cunningham et al. (1967) and Bailey et al. (1967), respectively.

Results

Blood films

Out of a total of 2,941 people sampled, blood films taken from 102 were lost. Examination of the rest showed no trypanosomes. About 47 blood films were found to be positive for malaria parasites (P. falciparum) and one for microfilaria (D. perstans).

Haematocrit centrifuge technique

A total of 2,761 freshly obtained blood specimens in capillary tubes were examined by this method; none showed the presence of trypanosomes. Thirteen specimens were positive for microfilaria.

Serum IgM levels

Of the specimens collected 47 were lost and so only 2,894 were tested. Those that showed a significant rise in IgM (immunoglobulin M) levels are shown in the histogram (Fig. 4) by their ages and sexes. They numbered 909 and formed 31.4% of the population.

Indirect immunofluorescent tests (IFT)

The first 1,691 people had the test performed on their dried blood sample irrespective of whether they had raised IgM serum levels or not. Only 11 of these were positive by the test; all except one were males. Of the remaining 1,203, only those that had raised IgM serum levels
had their samples tested for immunofluorescent antibodies. In this group those with raised serum IgM levels numbered 484; among them 25 showed evidence of immunofluorescent antibodies. Fourteen were males and 11 females. The degree of fluorescence achieved was only 3, none showed maximum fluorescence of degree 4. People positive by the IFT formed 1.2% of the whole population examined.

Lost specimens

One hundred and two blood films and only 47 dried blood samples on filter paper were lost. Many whose blood films were lost had their blood samples examined for raised IgM serum levels as well as for immunofluorescent antibodies. Some of those whose dried samples were lost had their blood slides examined for the presence of parasites. Thus the people whose both blood films and dried blood samples on filter paper were lost numbered only 15 and they came from 6 centres only. They consisted of 4 females and 11 males. The largest number came from Ikoma Robanda where 7 were lost. In other places the number lost was as follows: Mugumu Primary Court: 3 lost out of 205 samples;
Mugumu Lungahule: 2 out of 237; Nata Sibora: 1 out of 114; Lobo: 1 out of 165 and Serengeti National Park: 1 out of 526 samples.

Discussion

The results of this investigation showed that at the time of the survey (i.e. between 6th October and 16th November, 1970) no overt trypanosome infection was discovered among the human population examined. Even the HCT, which is said to be more sensitive than blood film examination, gave negative results. The screening tests used together (i.e. IgM estimation and IFT) also gave negative results. The interpretation of this is that a few months before and including the duration of the survey of that year there was no transmission of pathogenic trypanosome strains capable of producing parasitaemia in man. Nor did the survey team find an evidence of a healthy carrier among that population. Of a total of 17 cases notified between 1964 to 1968 the highest monthly incidence occurred in the month of November (i.e. 5 cases altogether). From monthly incidence of cases of sleeping sickness at Ikoma between the years 1934 and 1946 there appeared to be no increase in incidence during any particular month or season (Fairbairn 1948). The negative results together with those found by other survey teams whose reports appear in parts II, III and IV will be discussed together in part V where an attempt will then be made to explain the epidemiological factors which are at play in this part of Tanzania.

References


