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Artikel: Discussion of the relevance of the trypanosome survey to the biology of large mammals in the Serengeti

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Sleeping Sickness Survey in the Serengeti Area (Tanzania) 1971

III. Discussion of the relevance of the trypanosome survey to the biology of large mammals in the Serengeti

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Abstract

Trypanosoma brucei infection rates in large mammals in the Serengeti National Park are considered in relation to the abundance of these species. On the scanty evidence available, about 7.5% of mammals are infected. It appears that lions and hyaenas, despite high infection rates, are insignificant as potential reservoirs compared with the vastly more abundant, less infected, and less-studied species. The high infection rate in lions is to be expected from a social carnivore. It is unlikely that infection rates are very different from one area of the Park to another. Possible effects of parasite on host, and vice versa, and the barrier to infection in both host mammal and in the *Glossina* vector are discussed, and the main points requiring further investigation are outlined.

A. Introduction

The recent spasmodic occurrence of sleeping sickness in the Ikoma-Serengeti region has been described by ONYANGO & WOO (1971). Investigating the implication of wild mammals as a possible reservoir of the disease, three main surveys have been carried out. SACHS and BAKER (summarized by BAKER, 1968) examined the incidence of various trypanosome species in a variety of large mammal species in 1966–67. A larger scale survey in 1970 examined trypanosome infection rates in another sample of wild mammals (GEIGY et al., 1971), in the human population (ONYANGO & WOO, 1971), in cattle (MWAMBU & MAYENDE, 1971), and examined the feeding and infection of *Glossina* (MOLOO et al., 1971), all in the same general region at the same time. A further survey (reported in parts I and II) in 1971 investigated in more detail the role of *Glossina,* and examined a larger sample of the 4 mammal species from which possible *T. rhodesiense* strains had been isolated during the previous survey. Some of us at the Serengeti Research Institute who assisted in this survey felt that a discussion of its results from the standpoint of an ecologist working within the Park would help to put these results into perspective in relation to the biology of the large mammals of the region: hence this paper.

It should be noted that in the following discussion only *T. brucei* and *T. congolense* have been considered; little information was gathered on *T. vivax*. Further, the term *"T. brucei"* has been used throughout to indicate *"T. brucei* subgroup", and is therefore inclusive of any strains which may subsequently be identified as *T. rhodesiense*.

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Table 9. Incidence of *T. brucei* in game animals in Serengeti

Host species	Proportion of individuals infected reported by: a	b	c	total	%	Approx. host population in thousands	Number of infected animals
Lion							
Panthera leo	7/11	5/9	24/43	36/63	57%	1.6 d	900
Spotted hyaena							
Crocuta crocuta	0/3	2/5	13/31	15/39	38%	3.0 e	1,200
Waterbuck							
Kobus defassa	3/6	1/10	0/1	4/17	24%	1.1 f	300
Coke's hartebeest							
Alcelaphus buselaphus	1/10	3/11	3/20	7/41	17%	8.9 f	1,500
Wildebeest							
Connochaetes taurinus	4/22	0/10		4/32	12%	800 g	100,000
Topi							
Damaliscus korrigum	2/11	0/11		2/22	9%	33 f	3,000
Warthog							
Phacochoerus aethiopicus	1/14	1/13		2/27	7%	17 h	1,300
Zebra							
Equus burchelli	2/22	0/10		2/32	6%	300 g	18,800
Impala							
Aepyceros melampus	1/6	0/11		1/17	6%	55 f	3,200
Thomson's gazelle							
Gazella thomsoni	0/7	0/11		0/18	0%	400 g	0
Buffalo							
Syncerus caffer	0/3			0/3	0%	61 i	0
Jackal							
Canis mesomelas	0/2			0/2	0%	19 h	0
Grant's gazelle							
Gazella granti	0/3	0/2		0/5	0%	18 g	0
Eland							
Taurotragus oryx	0/5			0/5	0%	11 f	0
Giraffe							
Giraffa camelopardalis	0/1			0/1	0%	6.9 f	0
Reedbuck							
Redunca redunca		0/10		0/10	0%	1.7 h	0
Bushbuck							
Tragelaphus scriptus		0/2		0/2	0%	0.8 h	0
Leopard							
Panthera pardus	0/1			0/1	0%	0.5 h	0
Hunting dog							
Lycaon pictus	0/2			0/2	0%	0.2 d	0

a BAKER (1968).
b GEIGY et al. (1971).
c GEIGY & KAUFFMANN (1973).
d SCHALLER (1972).
e KRUUK (1970).
f SINCLAIR (in press).
g NORTON-GRIFFITHS (pers. comm.).
h HENDRICHS (1970).
i SINCLAIR (pers. comm.).

130,200 animals out of 1,738,700 (i.e. 7.5 %) are infected.

B. Numbers of host species

In considering the importance of a mammal species as a possible reservoir, attention must clearly be paid to the abundance of that species, as well as to the infection rate found within it. In Table 9 are set down *T. brucei* infection rates (as discovered during 3 recent Serengeti surveys), and approximate numbers of each species in the Serengeti National Park.

From Table 9 the following points should be made:

1. The overall *T. brucei* infection rate in large mammals in the Serengeti National Park, from the data available, is about 7.5 %; i.e. some 130,000 animals are infected.

2. The great majority of these are wildebeest, due to the enormous abundance of this species. Yet, regrettably, relatively little information is available on the infection rate in wildebeest. The results of examination of mice inoculated in the course of recent larger collections of wildebeest by the Serengeti Research Institute are still awaited.

3. Although the *T. brucei* infection rate found in lions and hyaenas is high, the scarcity of these species makes them insignificant as reservoirs when compared with the vastly more abundant wildebeest, even if only a very small proportion of this species is infected.

4. On the above evidence, few of the differences in infection rates are statistically significant (i.e p's less than 0.05 two-tailed on a χ^2 Two-sample Test or Fisher Exact Probability Test as appropriate: SIEGEL, 1956). Lions have a significantly higher infection rate than Coke's hartebeest, zebra, wildebeest, and the other ungulates sufficiently sampled (but not waterbuck). They are not significantly more highly infected than hyaenas. Hyaenas in turn are significantly more highly infected than the ungulates, with the exception of hartebeest and waterbuck. There are no significant differences in infection rate among any of the ungulates.

5. It can be seen that for most species the sample collected is extremely small. If one is to generalise from this sample to consider infection rates in the species as a whole, it is most important to be able to obtain a random sample from the population of that species. This is extremely difficult to achieve and was understandably not possible during the 1971 survey. Indications of the non-randomness of the samples are:

a) only 4 out of 20 kongoni were female, since only territorial males could easily be approached;

b) only 7 out of 33 hyaenas were female, probably because the culvert-dwelling habit (see under "Methods" in part I) is more characteristic of nomadic animals;

c) the 43 lions collected were from a limited number (14) of social groups, as indicated in Table 10.

To point out this non-randomness is not in any way to detract from the value of the survey, but it must be borne in mind when considering infection rates in the species as a whole, infection rates which are derived from such samples.

6. As reported by GEIGY & KAUFFMANN (in part I), one hyaena when re-darted showed a different parasitaemia. It should be understood that the "infection rate" of 38 % for hyaenas could indicate either that 38 % of hyaenas are infected, or that all hyaenas are infected with a parasitaemia which is detectable for 38 % of the time, or anything in between. In considering the probability of a tsetse's hyaena-blood meal resulting in infection, this is not important; if one is considering the possible spread of the trypanosome through migration of individuals, it is. No animals of other species were re-darted; they too may show changes in parasitaemia with time.

C. Lion infection rates

Virtually all adult lions were found to carry either *T. brucei* or *T. congolense,* an infection rate considerably higher than in other species sampled. This would be expected for the several different reasons below, most resulting from the fact that lions are carnivores and that they live in close-knit permanent social groups wherein if one member becomes infected the probability is high that others in that group will also become infected.

1. A very probable means of becoming infected is through eating infected prey, if newly dead. This has been suggested by BAKER (1968), quoting experimental cases. MOLOO (pers. comm.) has also infected cats and dogs by feeding them on infected goats. The trypanosomes may enter the new host blood stream through small wounds in the buccal mucosa; additionally, they could enter through the many external wounds on the nose and muzzle, which are frequently covered in blood when lions are feeding. If 7.5 % of prey animals are infected with *T. brucei,* the number of occasions when lions are open to infection by these means is large; it is probably increased if (as discussed in section E) a high parasitaemia is a cause of, or a symptom of, even a slight

deterioration in condition or alertness of the prey animals. Lions take a greater proportion of sick animals than is found in the wild population (SCHALLER, 1972) and thereby eliminate an appreciable number of sources of infection.

2. If direct transmission by these means is possible, it is also presumably possible from one lion to another when they lick one another's wounds in the course of extensive social grooming.

3. Cyclical transmission by tsetse flies is unlikely to account for the fact that there is a much higher infection rate in lions than in most other species. Lions are not a preferred host of *Glossina swynnertoni* in this region, i.e they are not fed on more often than would be expected if the flies were feeding on different host species at random according to their abundance. From the figures of MOLOO et al. (1971), "cat" comprised 0.7 % of tsetse blood meals and 1 % of the potential host population in the area sampled. But double feeds, as reported by MOLOO et al. and by ROGERS & BOREHAM (in part II), are frequent. They are only detectable when the two hosts are of different species. It must occur much more frequently that the fly feeds on more than one individual of the same species, and the closer together the host individuals are to one another, the greater (pesumably) is the likelihood of a disturbed fly resuming its feeding from a different individual; and in doing so it may transfer trypanosomes directly from one to the other. Few animals are as close together as lions at rest during the day, so the chances of direct, non cyclical transmission must be great. This applies both for tsetse and for other biting flies.

4. In addition, almost all lions carry hippoboscid flies, providing yet another possible means of direct transmission between individuals (BAKER, 1967). ROGERS & BOREHAM (in part II) found no *T. brucei* salivary gland infection in 200 hippoboscid examined, flies which were taken off lions which were later proved to be infected. This renders cyclical transmission by hippoboscids unlikely, although no *T. brucei* salivary gland infections were found either in over 10,000 tsetse flies MOLOO et al., 1971; ROGERS & BOREHAM, cf. part II), yet cyclical transmission is known to occur in this species.

5. In contrast to a sick ungulate, the chances of survival of a sick lion are high, since it can feed on kills made by others of its social group. If poor condition and high parasitaemia go together (as discussed in section E), the presence of a sick lion increases the likelihood of infection for other members of its group.

6. There are of course other unknown factors which could cause the high infection rate found in lions; weaker barriers to infection, response to infection, and level of parasitaemia are obvious possibilities, as yet untested.

The possible causes listed above are not exclusive; all probably act, although the relative importance of each is unknown. That there are factors operating through the social group is indicated by Table 10, in which infection type is related to social group. The lions collected belonged to 14 social groups. (Adult males of over 5 years are treated as belonging to a group different from that of any females they may have been with at the time of darting, since males do not joint a lion pride until they are at least 5 years old, by which time they are already infected.) The infection types, *T. brucei, T. congolense,* and mixed *brucei* and *congolense,* are listed for the individuals in each social group.

Table 10. Lion social group and trypanosome infection type

	Trypanosome infection type			
Social group	*T. brucei*	*T. congolense*	Mixed	Not infected
1.	1	3	3	–
2.	–	–	3	–
3.	–	–	–	2
4.	1	2	1	1
5.	–	4	–	–
6.	1	–	1	–
7.	–	2	–	–
8.	2	1	–	–
9.	–	1	–	–
10.	–	1	–	–
11.	1	–	–	–
12.	2	–	–	–
13.	2	–	1	–
14.	5	1	–	1
Total	15	15	9	4

Of the 39 lions infected, a total of 29 have that infection type which is commonest in their social group. To test whether this is a higher proportion than would be expected by chance, the same number (39) of lions, in social groups of the same sizes, were distributed at random among the 3 possible infection types, with probabilities of infection types B, C, and B + C occurring being 15/39, 15/39, and 9/39, respectively. The process was repeated 19 times. For each of these 20 random distributions, the number of lions with that infection type which was the commonest in their social group was determined; this figure ranged from 21 to 27; thus all were smaller than the observed figure of 29. The probability of a distribution as extreme as that observed being due to chance is thus less than 0.05. Thus lions within the same social group tend to have the same infection type as one another.

It can be seen that lions tend to have the same infection types as others within the same social group. This is further suggestive of cross-infection within the group.

For hyaenas, the points 1 to 5 above probably all apply but to a lesser extent than with lions, since hyaenas are less predatory and have less close-knit social groups.

D. Geographical distribution of *T. brucei*

The surveys of 1970 and 1971 concentrated on the Ikoma/Banagi region since it was from this region that the majority of human sleeping sickness cases have been reported. It is tempting to consider this region to be a focus of *T. brucei* infection in the wild mammal host species, but the evidence available at present does not support this. The probability of a *T. brucei* infection in a lion over 1 year old appears to be independent of locality, as can be seen from Table 10 and map Fig. 1 (p. 17). In addition, 8 of the 24 lions containing *T. brucei* (with or without *T. congolense*) were nomadic individuals travelling over a large area of the Park, and happening to be within the sampled area at the time of the survey. Many hyaenas, and probably most of those collected during the survey, travel long distances, either as nomadic individuals or commuting to dens more than 50 km away (KRUUK, 1970), and were collected only because they happened to be within the survey area. Data on hartebeest (a resident species) show no differences in probability of a *T. brucei* infection between the different areas within the Ikoma/Banagi region where they were collected; they have not been collected from other parts of the Park. It is clear that the area E, found in the 1970 survey to have the highest incidence of *T. brucei* (GEIGY et al., 1971), did so only because it was the area in which the lions and hyaenas were collected. It can also be seen from Table 9 that wildebeest, zebra, eland, and gazelles comprise some 91% of infected animals; these species move extensively over the whole area of the Park. On the present evidence, the areas where human sleeping sickness occurs are those areas where both humans and wild animals occur, rather than areas where wild animals are any more infected with *T. brucei.*

E. Effects of parasite on host, and vice versa

The pathogenicity of *T. brucei* and *T. congolense* infections to wild animals is almost unknown. BAKER (1968) summarized what information is available. A few species may be killed directly by a *T. brucei* infection; if so, infection rates in that species will be low. But death is

not the only criterion of pathogenicity; it is likely that many animals are to some extent handicapped by a trypanosome infection, even if this is not easily detectable externally. Losos & Gwamaka (1973) have examined histologically a number of the animals collected during the 1970 and 1971 surveys, and have reported lesions which were probably caused by trypanosomes in the brain and heart of several of them. The effects of such lesions on the host are very difficult to determine. In the ungulates, it may be that even a slight reduction in alertness, stamina, speed, or condition may increase significantly the chances that that animal will be killed by a predator or will succumb to stress such as malnutrition. To measure such a reduction in fitness is almost impossible at present. From the survey data, 3 attempts have been made to determine whether there is a relation between parasitaemia and condition:

1. For 22 hartebeest (in 1970 and 1971), whose condition was categorised, the incidence of infection was determined. Of the 11 in "Good" condition, 1 was infected; of the 11 in the "Medium" and "Poor" condition categories, 5 were infected. The difference is not significant.
2. For lions, only 7 were not classified as in "Good" condition. There was no higher infection rate in this group than in the remainder.
3. Response to darting can be considered as a measure of ability to deal with a stress situation. Mean recovery times were: for lions infected with *T. brucei* only 94 minutes; for lions infected with *T. congolense* 118 minutes; for lions with mixed infections 138 minutes. However, there is great variation, and the differences are not significant.

If a higher parasitaemia is found in animals whose condition is below average the problem still remains of assessing cause and effect. Young (reported by Sinclair, 1970, p. 138) found that trypanosome infection increased in a tame buffalo calf when the condition of the animal was poor due to other causes (lion injury). Sinclair found a 17% trypanosome infection rate in wild buffalo in the Serengeti; most of these were in the young age groups, and he concluded that the older animals must have become immune. (It is also possible that they simply had a lower parasitaemia.) This provides an instance of an effect of the host on the parasite. There are others: for example, Ashcroft (1959) reported that more animals were doubly infected than would be expected by chance; possibly poor condition (due either to the first infection or to other causes) allowed the second infection to establish itself. In lions, the reverse appears to be the case: there are fewer double infections than would be expected. If (as was found) all lions are infected with either *T. brucei* or *T. congolense* by the age of 1 year, one would expect that all adults would be infected with both;

yet few are, and there is no increase in the incidence of double infections with age. This may be due to interaction between parasite species, or to immunity of the host to new infections.

There is no reason to assume that all individuals of the host species are susceptible to infection. There were cases during the survey where 1 or 2 rats out of 5 inoculated developed no infection, despite injection of 4–5 ml of blood with a *T. brucei* or *T. congolense* parasitaemia sufficiently high for these parasites to be detected in blood slides. Clearly some rats are less susceptible than others, and it is likely that the same differential barrier to infection is present in wild animals.

F. Transmission

ROGERS & BOREHAM (in part II) and MOLOO et al. (1971) have shown that many more tsetse flies carry *T. congolense* infections than *T. brucei* infections, yet the surveys have shown in wild animals little difference in infection rate between *T. congolense* and *T. brucei.*

ROGERS & BOREHAM found mature *T. congolense* infections in about 2.5 % of *Glossina swynnertoni.* There is no data on how often a game animal is fed on by tsetse flies; a human in the same region is fed on painfully many times in the course of a day (pers. obs.). It is likely that a wild animal during its life is fed on by an enormous number of *congolense*-infected tsetse flies, yet probably does not develop an infection. There must be an extremely strong barrier to infection which is only rarely broken. The nature of the barrier and the causes of such breaks need to be determined. Possibly only a small proportion of host individuals are susceptible; or possibly only a very small proportion of mature *T. congolense* in the flies are in fact infective. ROGERS & BOREHAM also showed that only a small proportion of infected blood meals gave rise to infection in the flies. Clearly transmission takes place in only a much smaller proportion of cases than would be expected, and therefore there is surely no reason whatever to assume that transmission in these cases is random. Possibly only trypanosomes from one or two host species are capable of establishing infection in the flies, or in the next host; possibly it is only under very occasional, but precise, circumstances that the barrier to infection of fly or particularly game host is penetrated. The effects of condition and the incidence of different infection types as already discussed are evidence of non-randomness in the transmission process. And if the process is so rarely successful, and is non-random, to concentrate studies only or excessively on infection rates in different host species, and on feeding selection by the tsetse flies, may not be the most fruitful way of determining how humans occasionally become infected. As BAKER (1968) showed, the

favoured host species tend to be less infected than the less favoured hosts. (It should be borne in mind that good knowledge both of the numbers and of the spatial distribution of host species needs to be available before it is meaningful to use the term "favoured" in its useful sense of "more than would be expected by chance encounter". For example, a very scattered species will be encountered more often than an equally common one which lives in compact groups.) Transmission via any individual fly is very unlikely to occur, and whether it does occur may be determined by many non-random factors; it would appear to be these factors which particularly require attention.

G. Further investigations

The following would appear to be some of the most fruitful directions for further investigation:

1. Infection rates in the commonest wild animals need to be better ascertained; some of the material required is being examined by the Serengeti Research Institute at present.

2. It may be as important to know parasite levels in the host blood as to know infection rates. Clearly, a host species with a 100 % infection rate but a parasitaemia so low that flies feeding on it are rarely able to become infected, may be equivalent as a potential reservoir to a host species with a low infection rate and high parasitaemia. Quantitatively, parasite levels have been neglected so far, as have any possible changes in these levels.

3. The nature of the barriers to infection, both of the mammal host and of the tsetse fly, and the causes of breaks in these barriers, must be determined.

4. The effects of trypanosome infection (including *T. vivax*) on wild animals need to be known. For this, fine measures of even slight pathogenicity are required.

5. The role of cattle needs more attention. MWAMBU & MAYENDE (1971) found 12 out of the 260 cattle examined by both mouse-inoculation and blood slides to be infected with *T. brucei,* an infection rate of 4.6 %. (Their quoted figure of 3.5 % is derived including another 538 cattle examined by blood slides only, and is not therefore comparable with the figure for wild animals.) Outside the Park, Man has much greater contact with cattle than with wild mammals, and therefore there would appear to be a greater chance of becoming infected from cattle than from such mammals.

6. Further laboratory calibration of techniques appears called for, particularly on such points as individual host susceptibility.

7. The nature of *T. rhodesiense* is not clear. On the evidence at present, a small proportion of the *T. brucei* strains isolated are infective to Man, regardless of the host species from which they were obtained. This, and the several equivocal results obtained when these strains are tested by the BIIT test, suggest the possibility that *T. rhodesiense* and *T. brucei* (sensu stricto) are not completely discrete species but that they may interchange or interact.

8. Better case records of each infected human are required, with an assessment of the degree of contact with cattle, tsetse or wild animals, of the areas in which he is likely to have contracted the disease, and of his medical history.

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Zusammenfassung von Teil I, II und III

Die Ergebnisse der Schlafkrankheits-Kampagne, welche 1970 im Serengeti-Ikoma-Gebiet (Tanzania) durchgeführt worden war, ließen es wünschenswert erscheinen, dem Problemkreis Übertragung–Reservoir weiter nachzugehen. Die diesbezüglichen neuen Felduntersuchungen wurden November und Dezember 1971 im selben Gebiet durchgeführt mit Beschränkung auf Glossinen und Wildtiere.

Wie im Vorjahr konnte bei 3500 untersuchten Tsetsefliegen *(Glossina swynnertoni)* wiederum keine Speicheldrüseninfektion festgestellt, d. h. kein sicherer Nachweis von Brucei-Gruppe Trypanosomen erbracht werden. Die gesammelten Daten betreffend Alter, Ernährungszustand, Infektionsrate und Verbreitung der Fliegen wurden analysiert, wobei es sich in 3 verschiedenen Untersuchungsgebieten zeigte, daß die gefundenen Unterschiede auf das jeweilige Futterangebot zurückzuführen sind.

Aus den 95 untersuchten Wildtieren (Löwen, Hyänen und Kuhantilopen) konnten insgesamt 40 Brucei-Stämme isoliert und für weitere Untersuchungen als

Stabilate tiefgekühlt werden. Bei den später durchgeführten Blut-Inkubations-Testen nach RICKMAN & ROBSON reagierten 4 Stämme positiv, d. h. *T. rhodesiense*-artig.

Es folgt eine Analyse der Populationsdichte und der Infektionsraten der großen Wildsäuger des Serengeti-Nationalparkes und der sich daraus ergebenden Bedeutung der einzelnen Tierarten als mögliches Reservoir für Trypanosomen der Brucei-Gruppe.

Résumé des parties I, II et III

L'enquête sur l'épidémiologie de la trypanosomiase Est-africaine, réalisée en 1970 dans la région de Serengeti–Ikoma (Tanzanie), a montré qu'il serait intéressant de réexaminer certains problèmes concernant la transmission et les réservoirs. La nouvelle enquête effectuée en novembre et décembre 1971 dans la même région s'est bornée à étudier les glossines et les animaux sauvages.

Comme l'année précédente, la dissection de 3500 mouches tsé-tsé *(Glossina swynnertoni)* n'a permis de mettre en évidence aucune infection des glandes salivaires par des trypanosomes du groupe Brucei. L'ensemble des renseignements obtenus sur l'âge, les conditions d'alimentation, le taux d'infection et la répartition des mouches a été analysé. Les variations trouvées peuvent s'expliquer par des différences de nourriture disponible dans les trois régions étudiées.

Sur 95 mammifères sauvages examinés (lions, hyènes et bubales) 40 souches de trypanosomes du groupe Brucei ont été isolées et conservées sous forme de stabilats dans de l'azote liquide en vue d'être étudiés ultérieurement. Plus tard, quatre d'entre eux ont réagi positivement au B.I.I.T. test de RICKMAN & ROBSON, c'est-à-dire de la même façon que *T. rhodesiense*.

L'analyse corrélative des densités de population et des taux d'infection des grands mammifères du Parc National de la Serengeti a montré l'importance relative de certaines espèces animales comme réservoir possible de trypanosomes du groupe Brucei.