TRYPANOSOMIASIS IN NORTHERN UGANDA.

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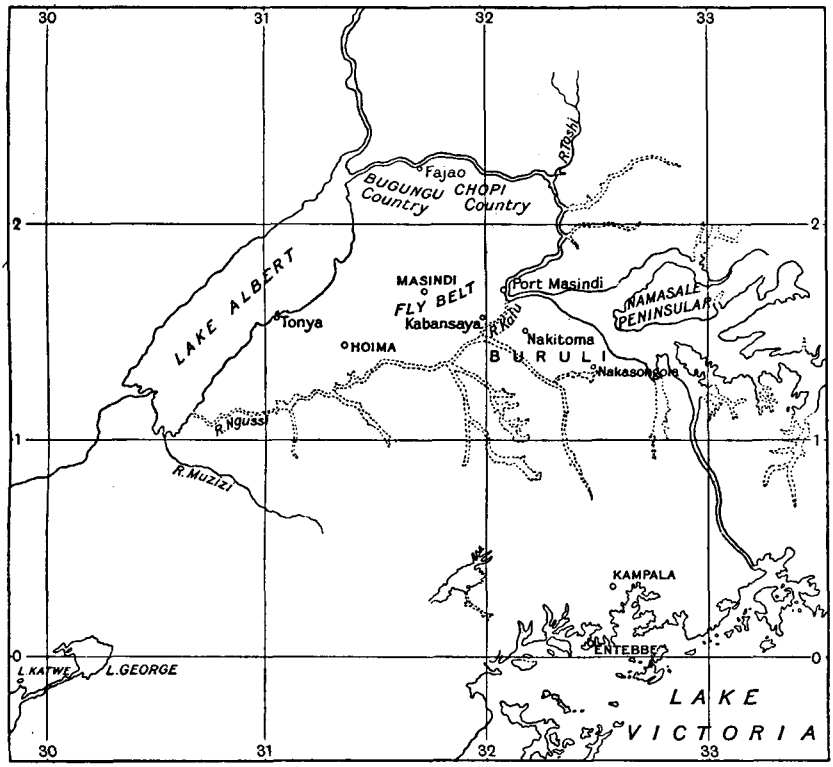
(With Map.)

In January, 1914, Dr J. H. Reford, Uganda Medical Staff, reported the discovery, in the blood of a dog belonging to a European missionary, of a trypanosome showing marked morphological resemblance to the human parasite, the so-called *Trypanosome rhodesiense*. The dog had made a single journey through the *morsitans* belt south of Masindi. It died after a short and severe illness characterised by marked emaciation and keratitis. Dr Reford recognised a certain number of trypanosomes in which the nucleus was displaced posteriorly, and also sundry irregular, more or less rounded-off forms. Miss Robertson, who had just completed a tour in this same fly belt, examined some of Dr Reford's slides and expressed her agreement with his view as to the affinities of the organism to the Rhodesian trypanosome. As she had not met with any trypanosomes of the *gambiense-brucei* group during her experiments in the same district, Miss Robertson, in a report to the Principal Medical Officer dated 20th January, 1914, laid considerable stress on the importance of the find and discussed at some length the pros and cons of the arrival of the dreaded South African organism in this already overburdened Protectorate.

I was accordingly instructed to proceed to the Northern Province and thoroughly test the tentative hypothesis put forward by Miss Robertson. I arrived at Masindi in May, 1914, and continued in the district until September, 1914, when I was recalled for military service. The writing up of the experiments performed was unavoidably delayed until March, 1915, when I was enabled to prepare this report on the results obtained, while on temporary duty at Entebbe. There remains much to be done in this northern fly area, and I hope to renew my investigations at some future date.

During the latter part of 1913 Miss Robertson was engaged in the Northern Province on a series of investigations in connection with cattle trypanosomiasis in the Masindi *morsitans* region. A brief epitome of this work is here necessary, before considering the experiments which I subsequently carried out in the same district and which led me to somewhat different conclusions.

In June, July, and August, 1913, Miss Robertson visited and



examined the various scattered herds of cattle still remaining in the plains north and south of the Kafu river, and interrogated the natives regarding the past history of the Buruli-Kafu grazing grounds. At a spot on the Nakasongola-Kibangaya-Masindi Road (cf. Map), which traverses the fly belt, experiments were carried out. On no single occasion was any evidence obtained either in fly, experimental animals, or cattle, of the existence of a trypanosome of the *brucei* group. The fly experiments were carried out on the road where the mission dog

acquired its infection some three months later. Miss Robertson says however, "...but a survey of herds and the examination of a relatively small number of *Glossina morsitans*, such as I have made, cannot be considered a really exhaustive investigation of the country, and no subinoculations have been made of game."

During the reign of Kabalega, King of Bunyoro, the Buruli country, which at that time was a part of the Bunyoro kingdom, and the plains to the north of the Kafu supported large numbers of cattle. After the final defeat of the rebel king in 1895 he retreated to Bukedi across the Nile, and drove all his cattle away with him, practically clearing the country. About 1900–1901 cattle began to arrive from Buganda kingdom, and the old grazing grounds were restocked. In 1902–1903 disease began to show itself among the cattle in the Nakasongola district to the south, and slowly spread northwards to Nakitoma some two or three miles south of the Kafu river. "The disease was carried north of the Kafu by the moving of a single infected herd from Nakitoma in Buruli to Kibangaya in the Northern Province[1]." After this event "the sick animals in this herd died of their disease in about six months, and there was no further illness until about June, 1908, when all the herds in the Kafu district showed the disease one after the other. Now there is no reasonable doubt that Kabosolita's herd pasturing in country full of game, with tsetses in small numbers in the open grass plains and in tremendous numbers in the Kakora bush country only four or five miles away, must have been the source of the trouble. The fact that there was an interval of nearly two years between the arrival of Kabosolita's herd and the general infection of the district shows that the country at Kibangaya was clean when Kabosolita's cattle were taken there; also the general history of all other herds confirms this." "...It is important to note that it took nearly two years to produce an efficient endemic reservoir in the Kafu district" (north of Kafu)[2]; "moreover, owing to mutual relations of the game and the *morsitans*, the short period of six years has brought about a 10 % infection of the fly throughout the Masindi fly belt, even in places 10–12 miles from domestic herds."

From the above, which, as already stated, is Miss Robertson's account, the words in quotation marks being extracted from her reports, it is quite evident that she considers the cattle trypanosomes of the

[1] *Tropical Diseases Bulletin*, III. 419. Section 3, Miss Robertson's Report, September, 1913.

[2] My brackets.

Buruli and Kafu country, as well as those of the *morsitans* there found, were originally derived from the south.

As regards the trypanosome found by Dr Reford, Miss Robertson adopted a similar explanation of its presence in the *morsitans* of the Masindi belt. Her reasons were briefly as follows: during June, July and August, 1913, she was touring the district examining cattle and endeavouring to trace the aetiology and sources of the trypanosomiasis which had worked such havoc among the stock. She performed a series of experiments with *morsitans* caught on the very road where the mission dog became infected with the *brucei*-like organism in December, 1913.

In the course of these experiments 450 flies were fed on a monkey and a goat. The monkey developed *T. pecorum* "but never showed any infection with a member of the *gambiense-rhodesiense* group." The goat died of a mixed infection of *T. pecorum*, *T. vivax* and *T. uniforme*. Of these flies, 445 were dissected and 9·4 % found to contain developmental stages of various trypanosomes. "Proboscis only," "proboscis and gut" and "gut only" infections were noted; *i.e.* as will be seen later, none of these 445 flies harboured the full developmental cycle of the *brucei*-like organism described by Dr Reford.

Further 718 cattle were examined in various parts of the grazing area north of the Kafu without any sign of this latter organism being found, although 12 % were infected with trypanosomes.

The mission dog acquired its infection in December, 1913, *i.e.* some three months after the completion of Miss Robertson's experiments. In her report on the blood slide from the dog, sent from Masindi by Dr Reford, she says "it seems probable that the trypanosome which has appeared in the dog was not present in a sufficiently high percentage of the flies in the *morsitans* belt in July, 1913, to be detected by the methods adopted in the investigation. From the fact, however, that the dog passed apparently only once through the belt one is inclined to conclude, in spite of the element of chance which enters to a certain extent, that the trypanosome in question is now present in a greater proportion of the flies than in July, 1913. The deduction being that the trypanosome has been recently introduced into the *morsitans* belt" (Miss Robertson's report, 20th January, 1914).

It will be seen therefore that, according to Miss Robertson, both the trypanosomes responsible for the disease in cattle and this free-flagellated *brucei*-like organism have been imported from outside, and have secondarily established themselves in the *morsitans* belt near

Masindi; the reservoir being the wild game of the district. In other words the fly and game of this belt were presumably 'clean' say 20 years ago, and have reached their present degree of infectivity entirely as a result of a relatively recent introduction of trypanosomes from outlying parts of the Protectorate, in the case of the cattle parasites probably from the south.

I will now put forward some criticisms of this view, based partly on an exhaustive enquiry among the natives, with and without interpreters, supplemented by a series of questions put at my request to the Lukiko or native parliament of Bunyoro kingdom by the District Commissioners of Masindi and Hoima Stations; partly on my own experiments and observations.

First and foremost, as a general principle, the idea of a large area of very sparsely populated bush country, thick with game and *morsitans*, being free from trypanosomes is, I consider, untenable.

I hold the opinion that the pathogenic trypanosomes of cattle and domestic animals were originally derived from the apparently harmless parasites of wild game, the ultimate origin of these game trypanosomes being, of course, outside the range of the present discussion. It is curious to note, in various parts of Africa, that native owners often avoid grazing their cattle in the proximity of waterbuck and other scrub-feeding game because these animals are supposed to infect the grass with their saliva and other excretions. These same natives have no fear of the tsetse as such, although the results are the same as regards minimising the exposure of the cattle to disease.

In reviewing Miss Robertson's conclusions two criticisms at once suggest themselves.

(1) Disease or death in cattle in the area visited is assumed to be invariably due to trypanosomes, whereas in reality several other diseases are known to have occurred among these herds. The acting C.V.O., Mr V. F. Richardson, writes "with regard to native names and descriptions of disease, I should like to point out that these are very misleading. I have not found Bahima herdsmen able to distinguish rinderpest from trypanosomiasis, whilst the name *Nsotoka* appears originally to have referred to pleuro-pneumonia and is now used for rinderpest or acute trypanosomiasis. Even *Amakebbe*, the best known native name for a disease of cattle, is of doubtful significance, and I have known it applied to rinderpest and to undoubted cases of trypanosomiasis."

Nsotoka was one of the diseases described by the herdsmen in connection with the recent epidemic.

Again, Mr Hutchins, the C.V.O., Uganda Protectorate, on being consulted as to the probable diagnosis of the symptoms shown by the diseased cattle during the epidemic as supplied by various herdsmen around Masindi, expresses the opinion that some of these accounts point to *Babesia* as the causative agent.

(2) Miss Robertson is inclined to assume that once an animal is infected with trypanosomes it is doomed to a more or less speedy death. In the case of such trypanosomes as *T. nanum*, *T. vivax* and *T. uniforme* the result is not necessarily a fatal one, or, at any rate, not rapidly fatal. Both Kleine and myself have drawn attention to the extreme chronicity which often characterises infections with these parasites. Dr Marshall, early in 1914, found two of the animals in which Miss Robertson found trypanosomes in June–July, 1913, still alive and in excellent condition. Thus the conclusion that an animal has been recently infected because the only sign of disease is the occurrence of *T. vivax* or *T. nanum* in the peripheral blood is, I consider, not justifiable. Such an animal might have been infected for 12 months or more. With the more pathogenic trypanosomes the case is of course different, but the distinction is of importance in considering the history of an epidemic in relation to recent events.

As regards the native names for cattle diseases, the C.V.O. confirms my opinion that when the herdsmen refer to the disease *Lwakipumpuru* they mean genuine trypanosomiasis. Now it is interesting to note that, according to native testimony, there have been at least two epidemics of *Lwakipumpuru* in the Buruli cattle country before the one under discussion. Both these epidemics caused a very serious mortality among the cattle, and occurred respectively in the reign of Kabalega's father Kamrasi, say about 1860, and when the present King Anderea was a young boy, *i.e.* about 1890. *Lwakipumpuru* also played an important part in the recent epidemic. After each epidemic the Buruli country has been restocked with cattle, at longer or shorter intervals, from different sources, Buganda, Busoga and especially Bukedi. When Kabalega and his Banyoro were driven out of Buruli in 1895, they drove off all their cattle to Bukedi, to the neighbourhood of the Toshi river, a district thickly infested with *G. morsitans*. The majority of these cattle were brought back to Buruli by the victorious Buganda and re-established in their old grazing grounds. From what is now known of the Toshi river country, it is certain that a large percentage of these animals were infected with trypanosomiasis. Now *G. morsitans*, though found in the country south of the Kafu, is very

sparsely distributed. Nothing like the numbers found in the Masindi fly belt, north of the Kafu, occur in northern Buruli. The great tsetse reservoir is separated from these grazing grounds by the Kafu river and a belt of short grass plains some 8–10 miles wide—under ordinary circumstances an almost insuperable barrier to the fly. The Bukedi trypanosomes, under the fly conditions normally existing in Buruli, might be expected to die out with their original hosts. But given some particularly favourable year or season in which *morsitans* for some reason or other multiplied and spread, then we have all the requirements for an epidemic in Buruli. It is highly probable that such favourable seasons do occur from time to time, and *morsitans* become much more numerous in Buruli than they are at present. In the face of such an epidemic the cattle would be driven northwards across the Kafu, and the probability of their encroaching on the great Masindi fly belt would be increased. Again, with an increased number of cattle on the narrow strip of plain between the river and the fly country, there would be an increased likelihood of 'following fly,' accompanying traffic along the roads through the fly belt, meeting herds.

The part played by 'following fly' in the spread of disease is a very important one, as the following experiment arranged by Mr Fiske and myself shows. Two cyclists started from the plain near the Kafu at about 7.30 a.m. in bright sunshine, and rode some miles into the Masindi fly belt along two of the main roads. Turning round they then rode rapidly back to the ford where both roads cross the Kafu. Here, as each arrived, a canoe took him across to the Buruli side, and he walked his bicycle some 200 yards to where fly boys were awaiting him. In this way 16 *G. morsitans* were conducted across the river to a point some five or six miles from their home, across absolutely bare plains—and, curiously enough, on dissection five of these flies contained developmental stages of trypanosomes. No one who has cycled through *morsitans* country can have failed to be struck by the number of fly which follow the machine, and the distance they will travel. Mr Fiske tells me that one accompanied him on his bicycle as far as Masindi Station, which is some 12 miles from the northern edge of the fly belt. A slow pedestrian will soon shake off 'following fly,' but the pace of the cyclist seems to exert an irresistible attraction, and large numbers will follow for miles. Such agencies doubtless play their part in the spread of trypanosomiasis, and I consider it possible that the introduction of bicycles among the natives of Busindi, dating from 1908, had a definite influence on the recent epidemic.

The great fly belt which forms the northern limit of the Buruli-Kafu cattle country I regard as the principal source of all the trypanosome species found in the cattle, particularly those grazing north of the river. It is a permanent source, merely awaiting any condition which facilitates frequent exposure of the animal to fly bite. From this northern belt trypanosomes may be taken into central Buruli by

(1) 'Following fly' either with elephant, buffalo or other game which can easily cross the Kafu, especially in the dry season, or by cyclists and other traffic.

(2) By the passage of infected animals, game or cattle.

Other sources of trypanosomes in southern Buruli are afforded by the small *morsitans* foci which undoubtedly exist in that country, probably especially in association with the buffalo in the eastern part of the country; and also by the intermittent introduction of infected Bukedi or Busoga cattle.

It must be remembered that a native owner moves his herds directly he detects the presence of serious disease, and, as such movements are indiscriminate, he may well make matters worse by increasing their exposure to fly. The introduction of an infected herd into another region where the stock is apparently healthy, will expose these latter animals to the chance of direct transmission by biting flies other than *Glossinae*.

FLY EXPERIMENTS IN THE MASINDI MORSITANS BELT.

1857 wild *G. morsitans*, caught at various places in the fly belt south of Masindi, were fed upon suitable animals, more especially monkeys, and the trypanosome which, for convenience I will refer to as "*T. brucei*," appeared on eleven occasions. This gives a percentage of 0·59 % wild *morsitans* infected with this trypanosome. The figure is of little value, however, because the numbers of flies used for each experiment were unnecessarily large In all, 13 experiments were performed, and only two of these failed to produce the long trypanosome; these two experiments involved 48 and 44 flies respectively. The above feedings were carried out from May 14th, 1914, to June 14th, 1914, *i.e.* approximately one year after Miss Robertson's visit.

Trypanosoma pecorum, nanum, vivax and *uniforme* were recovered on numerous occasions during these experiments, double and treble infections in the experimental animal often resulting.

During my stay in the Masindi belt, I came across two 'natural' infections in domestic animals which had been exposed to fly:

(*a*) A 'clean' dog, bought at Masindi and kept and examined at the laboratory there, sent to me along the road where the original mission dog became infected. This animal emerged from the fly belt at about 8 a.m. having commenced the journey during the night. It only made one passage, and five days later developed "*T. brucei.*"

(*b*) A dog kept by Mr Fiske at his camp in the fly belt, which died of a pure *T. pecorum* infection.

The following figures show the results obtained by fly dissection performed in connection with the above feeding experiments. In interpreting the figures it must be borne in mind that (*a*) infections of proboscis only is ascribable to *T. vivax* or *T. uniforme*; (*b*) those of the proboscis and gut to *T. nanum* or *T. pecorum*; (*c*) those of the gut and salivary glands to the *brucei*-like organisms; those of the gut only to the immature infections with (*b*) or (*c*). There is no reason to suppose that any other pathogenic trypanosome than those mentioned occurs in this district.

A. Total of 1117 flies caught during experiments in fly belt, May and June, 1914:

13·4 % infected with trypanosomes,
8·5 % proboscis only,
3·1 % proboscis and gut,
1·8 % gut not proboscis (three of these flies showed infected salivary glands).

B. With 264 of these flies caught within possible range of Miss Robertson's fly boys (*i.e.* around the place where her experiments were performed):

9·7 % infected with trypanosomes,
6·8 % proboscis only,
2·2 % proboscis and gut,
0·7 % gut not proboscis (no infected salivary glands).

C. With 715 flies caught at places where waterbuck are not found or are very rare visitors:

9·6 % infected with trypanosomes,
6·3 % proboscis only,
1·8 % proboscis and gut,
1·5 % gut not proboscis (1 fly showed infected salivary glands).

D. With 198 flies caught in a waterbuck locality:

25·2 % infected with trypanosomes,
20·2 % proboscis only,
4·0 % proboscis and gut,
1·0 % gut not proboscis (1 fly had infected salivary glands).

E. With 206 flies caught in a second waterbuck locality:

15·8 % infected with trypanosomes,
4·8 % proboscis only,
6·7 % proboscis and gut,
4·3 % gut not proboscis (1 fly showed infected salivary glands).

Miss Robertson's figures for July and August, 1913, given for comparison, are

445 flies dissected 9·4 % infected with trypanosomes,
3·8 % proboscis only,
2·9 % proboscis and gut,
2·7 % gut, not proboscis

(no flies found with infected salivary glands).

These figures are very significant in reference to Miss Robertson's opinion that the *T. brucei* is a new arrival in these Masindi *morsitans*. If this *T. brucei* were a new introduction it would be reasonable to expect an increase in the 'gut not proboscis' figures in one or both of Sections A and B of my figures.

It is interesting also, as pointing to the presence of this trypanosome in the *morsitans* of the Masindi belt before Miss Robertson's arrival, that the native dogs were dying in 1912–13 with symptoms of keratitis. This symptom is almost always due to *T. brucei*, and rarely if ever to the *T. pecorum* present in these fly.

In the following fly-feeding experiments the results were controlled by dissection of all the flies employed as they died, either during the experiment or at its conclusion.

It will be seen that on every occasion where a positive salivary gland was seen, the animal fed upon developed "*T. brucei*"; also that *T. nanum* appears to be more common in these wild *morsitans* than *T. pecorum*.

Animal fed upon	Number of flies fed	Positive flies: Number found during experiment	Site of trypanosomes: Proboscis only	Proboscis and gut	Gut and salivary glands	Gut only	Trypanosome species appearing in animal fed upon	Remarks
Goat D	28	5	3	1	0	1*	"*T. brucei*," *vivax* and *pecorum* or *nanum*	Only a small piece of the salivary gland of * fly seen
„ C	48	5	0	2	1	2	"*brucei*" and *nanum*	Subinoculation from goat into dog excluded *pecorum*
Sheep A	48	11	9	2	0	0	*vivax* and *nanum*	Subinoculation from sheep into dog excluded *pecorum*
Goat E	44	4	1	1	0	2	*vivax*	
Monkey K	32	9	7	1	1	0	"*brucei*"	Injection of salivary gland into monkey produced "*brucei*"
„ P	64	9	5	3	1	0	"*brucei*"	
„ Q	46	9	8	1	0	0	nil	
„ H	27	9	8	1	0	0	nil	

The following inoculations were made of the blood of game shot in and around the fly belt into clean experimental animals:

Game species inoculated	Number injected	Number + on microscopic examination	Nature of infection in game; single or mixed	Species of organism appearing in sub-inoculated animals
Hartebeest	7	1	single	"*brucei*"
Ugand cob	7	0	—	0
Reedbuck	1	0	—	0
Bushbuck	5	2	single	*uniforme*; *pecorum*
Warthog	1	0	—	0
Buffalo	4	0	—	0
Duiker	2	0	—	0
Waterbuck	3	2	mixed; simple	*nanum*; *vivax*; "*brucei*"
Totals	30	5	—	0

This gives a total infected with trypanosomes of 16·6 %: 6·6 % carried "*T. brucei.*"

Experiments in other tsetse districts of the Northern Province to determine the distribution of the *brucei*-like organism:

1. Ngussi river, 3–4 miles from the falls (*cf.* Map).

In this neighbourhood three species of *Glossina* were obtained, *G. pallidipes*, *G. palpalis*, and *G. fusca*.

Fly dissections:

G. pallidipes. 65 flies dissected.
18·4 % infected with trypanosomes,
1·5 % proboscis only,

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7·6 % gut only,
6·3 % proboscis and gut,
3·0 % gut and salivary glands.

G. palpalis. 95 flies dissected.
8·4 % infected with trypanosomes,
2·2 % proboscis only,
1·0 % gut only,
5·2 % proboscis and gut.

No salivary gland infections seen.

G. fusca. 5 dissected—all negative.

Fly feeding experiments:

Dog X, fed upon by 110 flies, a mixture of *pallidipes* and *palpalis,* developed an uncontaminated "*T. brucei*" infection. Dissection of these flies revealed two with the salivary glands swarming with trypanosomes, and several flies with 'proboscis' and 'proboscis and gut' infections.

Dog Y, fed upon by a mixture of 48 *G. palpalis* and *G. pallidipes,* the majority *palpalis,* remained clean. Dissection of the flies revealed several 'proboscis' and 'proboscis and gut' infections, but no sign of any infected salivary glands.

Here again a positive salivary gland leads to infection with "*T. brucei*"; while the fact that the majority of the 'proboscis and gut' infected flies had obviously fed on the dogs without producing infection indicates that, here also, *T. nanum* is commoner than *T. pecorum.*

I have already pointed out (*Sleeping Sickness Report,* XII, 7, and XIII, 5, and *Proc. Roy. Soc.* B. vol. LXXXV, 1912) that *T. nanum* is better adapted to development in and transmission by *G. palpalis* than is *T. pecorum,* and these figures point to the same conclusion.

No game inoculations were made in this district.

2. REGION OF THE TONYA PENINSULA AND SHORE OF LAKE ALBERT.

Tsetse species found, *G. palpalis.*

407 flies dissected: 9·9 % infected with trypanosomes,
2·7 % proboscis only,
1·4 % proboscis and gut,
5·8 % gut only.

A considerable proportion of these 'gut only' flies contained *T. grayi.* No salivary gland infections were found.

Game inoculations:

Game injected	Number infected on microscopic examination	Number injected	Infection in game; single or mixed	Trypanosome recovered
Buffalo	0	4		
Bushbuck	2	9	single	*nanum*
Waterbuck	0	2		
Duiker	0	3		
Cob	0	1		
Warthog	0	1		
Lion	0	2		
Totals	2	22		

i.e. 9 % infected with *T. nanum* which is here carried by *G. palpalis*.

Fly feeding experiments:

Goat F, fed upon by 393 *palpalis*, developed *T. nanum* or *T. uniforme*.
Goat G, fed upon by 570 *palpalis*, developed *T. nanum* or *T. uniforme*.
Goat H, fed upon by 370 *palpalis*, developed *T. nanum* or *T. uniforme*.

3. Chopi, Victoria Nile and Bugungu region.

Fly dissections: *G. palpalis* caught around Foweira on Victoria Nile.

154 flies dissected: 6·4 % infected with trypanosomes,
3·2 % proboscis only,
0·00 % proboscis and gut,
3·2 % gut only.

A large proportion of these 'gut only' flies carried *T. grayi*.
No game was obtained here.

G. palpalis caught on Bugungu plain.

57 flies dissected: 8·7 % infected with trypanosomes,
7·0 % proboscis only,
0·00 % proboscis and gut,
1·7 % gut only.

A proportion of these 'gut only' flies carried *T. grayi*.

G. morsitans caught in Bugungu and around Fajao.

606 dissected: 9·6 % infected with trypanosomes,
7·0 % proboscis only,
1·8 % proboscis and gut,
0·8 % gut only.

No *T. grayi* seen.

Fly breeding experiments with fly caught in Bugungu country and around Fajao.

Dog M, 218 *G. palpalis*, no trypanosomes recovered.

Dog N, 520 *G. morsitans*, developed *T. pecorum*.

Goat X, 760 *G. morsitans*, developed a mixed infection with *T. vivax*, "*T. brucei*," and a *pecorum-nanum* type of organism.

Monkey L, 440 *G. morsitans*, died on seventh day after last batch of flies had fed, having no trypanosomes in its blood.

Injection of blood of game shot around Fajao and in Bugungu country.

Game injected	Number injected	Number + on microscopic examination	Infection in game	Trypanosome
Waterbuck	9	2	—	*uniforme* or *nanum*
Bushbuck	4	2	mixed	*nanum* and *vivax*
Elephant	1	0		
Buffalo	1	0		
Cob	3	0		
Hartebeest	1	0		
Warthog	1	0		
Totals	20	4		

Total infected with trypanosomes, 19 %.

4. NAMASALE PENINSULA.

No tsetse seen.

Blood of following game was injected and examined: hartebeest 3, cob 3, warthog 1, reedbuck 1, rhino 1, elephant 1 = total of 10 animals. No trypanosomes were seen.

5. NORTHERN BURULI.

No tsetse seen.

Blood of following game was inoculated and examined without any trypanosomes being seen: hartebeest 6, eland 2, cob 5, bushbuck 2, waterbuck 2 = 17 animals.

The animal reactions and morphology of all the strains of the "*T. brucei*" which I recovered during the above investigations were such as to warrant the conclusion that they belong to the same species. The disease is acute in dogs and is characterised by keratitis in the great

majority of cases. Oedema of the face is often present in sheep and goats. One striking exception to the rapid course usually pursued by infections with this trypanosome was afforded by a young monkey which was infected by wild fly on May 30, 1914, and was still alive and active on Sept. 11, 1914, though intermittently showing trypanosomes in its blood.

The average duration of the disease in eight experimental dogs was 26 days, and in five monkeys 40 days; the young monkey mentioned above was not included in estimating this average. In seven out of the eight dogs, keratitis intervened. It is difficult to estimate the course of the disease in sheep and goats as no sub-inoculations were made into these animals; and in every instance where "*T. brucei*" developed as a result of feeding wild fly upon them, mixed infection resulted.

The above facts show that a trypanosome of the *brucei* group is widely distributed throughout the southern part of the Northern Province, probably, indeed, wherever *G. morsitans* and *pallidipes* are found. Thus it has been recovered from the Ngussi river to the south-west, from Bugungu and Chopi to the north, in addition to being found throughout the main belt south of Masindi station. Further, Dr Bagshawe, in his report for December, 1906, alludes to the occurrence of a similar organism on the Muzizi river at the southern end of Lake Albert, and also in the neighbourhood of Lake George. Dr Hodges, Principal Medical Officer, Uganda Protectorate, described a similar trypanosomiasis in the neighbourhood of Gondokoro in 1904–5, in which oedema of the face and keratitis were prominent symptoms, and which strongly suggests the presence of the same or a similar organism. In *Sleeping Sickness Report*, XIV, 2, I described a trypanosome from fly and game recovered from the Katwe region of Toro Province, and Lake George.

Speaking generally, wherever cattle are exposed to the bites of tsetse, especially of *morsitans* and *pallidipes*, they sooner or later sicken and die out. No one, so far as I am aware, has yet reported the existence of a locality where cattle are tolerant to the game tsetses.

It would appear then that this, at first sight, alarming discovery in the Masindi fly belt of a trypanosome showing close affinity to the organism recently isolated from man in South Africa need not cause any undue alarm. A similar or identical trypanosome will probably be found in every *morsitans* or *pallidipes* area in Africa. Here and there, for what reasons we know not, it may, as in Rhodesia, develop

the faculty of more or less permanent survival in man, a host normally immune. As such it must be viewed as a potential source of danger to human beings[1]. As regards the local aspect of the question, I visited nearly all of the few scattered villages in the fly belt and examined as many of the natives as possible. In this way 288 natives were seen and examined by gland palpation with negative result. There was no sign of any form of human trypanosomiasis, acute or chronic. The actual figures were

Adult males	131
Adult females	135
Children	22
Total	288

These figures, though small, none the less represent a considerable proportion of the inhabitants of the actual fly belt.

As regards the nomenclature of this organism and its full significance to the Protectorate, I cannot at present pronounce a final opinion. Experiments had been devised for further research on this interesting subject, but they had to be suspended owing to the outbreak of war.

Meantime, from an administrative point of view, I do not consider it to be a human parasite. The natives examined were selected from villages within the fly belt, and *morsitans* were to be seen actually in their villages. These fly have been shown to be infected with the *brucei* organism in the proportion of 0·5 %. The percentage varies in different parts of the belt, the difference apparently having some relation to the distribution of game. These people must be bitten many times a day. Domestic animals cannot survive; dogs die with keratitis and emaciation, and an occasional goat, all one finds in these scrub villages.

From a theoretical and scientific point of view, we must admit that the presence of a trypanosome so nearly allied to the human parasite of Rhodesia constitutes a potential source of danger. Practically, however, provided that steps are taken against the introduction of a large number into the at present sparsely populated fly area, there is no reason to expect any untoward developments.

[1] Cf. *Journ. Trop. Med. and Hyg.* June 15, 1915.