Original Communications.

SLEEPING SICKNESS IN THE LADO OF THE ANGLO-EGYPTIAN SUDAN.

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Introductory.—This paper is the first of what it is hoped will be a series of short notes on sleeping sickness in various parts of the Anglo-Egyptian Sudan, giving a condensed account of the known history and distribution of the disease associated with an attempt to define the parasite.

The experimental work has been performed in Khartoum, which is far removed from tsetse-flies and sleeping sickness. The material has been obtained by means of animals inoculated from sleeping sickness patients at Yei in the Lado Enclave of the Mongalla Province by Captain Ranken, R.A.M.C., and will be called the Yei strain or Yei trypanosome until the end of the present paper.

The only means available at the present time of differentiating trypanosomes is by comparative experiments performed as nearly as possible under the same circumstances of climate, place and time, and this is the reason why the work was not performed in a sleeping sickness area, because it was considered unjustifiable to introduce *Trypanosoma rhodesiense* into such area, as with all precautions an accident might happen the results of which might have been very terrible.

The strain of *T. rhodesiense* used was a lineal descendant of the original strain discovered by Stephens and Fantham, of the University of Liverpool, to both of whom we are deeply indebted for so kindly giving us the living trypanosomes.

Therefore the two strains to be compared in this present paper have the following origins:—

T. rhodesiense.—Lineal descendant of the original strain and brought alive from Liverpool in animals.

Yei strain.—Brought alive in an animal from Yei, the full history of which will be detailed later.

Patients were not brought to Khartoum for two reasons:—

(*a*) Danger of spreading the disease by bringing individuals with trypanosomes in their peripheral blood through as yet uninfected fly regions.

(*b*) Desire not to hinder the treatment, which drives the trypanosomes from the peripheral blood and at all events temporarily benefits the patients.

The methods adopted for fixing and staining the trypanosome were as follows:—

All films were fixed wet with osmic acid vapour for about four seconds and then plunged at once into absolute alcohol, in which they were kept for two to five minutes. They were then quickly washed with distilled water and transferred into the Giemsa's solution without allowing the films to dry.

Two Giemsa's solutions were used, viz.:—

(*A*) A solution made up of 1 c.c. of the ordinary stock stain with 10 c.c. of distilled water and two drops of a 1 in 1,000 solution of potassium carbonate in distilled water.

The films were stained in this solution for one hour or longer and were then rapidly washed in distilled water and dried.

(*B*) A solution made up of 2·5 c.c. of the ordinary stock stain with 100 c.c. of distilled water and five drops of a 1 in 1,000 solution of potassium carbonate.

The films were stained from five to twenty-four hours in this solution and then washed in distilled water and dried.

Historical.—In order to make some of our remarks intelligible to any one who may read this note, it is necessary to review the history of the discovery of the trypanosomes of sleeping sickness as we understand it, and then to pass on to a brief review of the history of sleeping sickness in the countries adjoining the Lado.

Human Trypanosomes.—The trypanosomes known to exist in man may, for our present purposes, be divided into those which cause—

(*a*) *South American trypanosomiasis* (molestia de Carlos Chagas) caused by *Trypanosoma cruzi* Chagas 1909. With this disease and its causal organism we are not at present concerned.

(*b*) *The African Trypanosomiases*, more commonly called sleeping sickness, with which we are concerned at present.

In 1901 Forde and Dutton found a trypanosome which, subsequently, received the name *Trypanosoma gambiense* Dutton 1902, in the blood of a man suffering from a peculiar type of fever on the Gambia. This trypanosome was, we believe, brought alive to Europe; but, as after many inquiries we have failed to trace its present existence, we are forced to the conclusion that "the original strain" of human trypanosomes is lost. This trypanosome was also named *T. fordii* Maxwell-Adams 28 March, 1903, and *T. gambiæ* Maxwell-Adams 28 March, 1903; other synonyms are *T. hominis* Manson 1903, and *T. nepveui* Sambon 1 July, 1903.

In 1902 Castellani found a trypanosome in the cerebro-spinal fluid of persons suffering from sleeping sickness in Uganda.

On page 9 of the First Report of the Sleeping Sickness Commission of the Royal Society Castellani says:—

> "The trypanosome found in the cerebro-spinal fluid of sleeping sickness does not, as far as I have been able to make out, differ materially in size and shape from the species one finds in the blood of trypanosome fever, *T. gambiense* (Dutton), but possibly it is to be differentiated from this one because in it, as a rule, the micro-nucleus lies nearer the extremity and the vacuole is apparently larger. Besides, its movements are not apparently so active, but this fact might be due to the effects of the centrifuge. In case it should prove to be a new species, the trypanosoma I have described might be called from the country where I have found it first, *T. ugandense*."

This name suggested by Castellani, though the paper was written in April, 1903, would bear the date

of publication in the *Proceedings of the Royal Society*, vol. lxxi, 1903, p. 501.

In the meanwhile Kruse, as Castellani points out, had suggested the name *Trypanosoma castellanii* in the "Sitzungsberichte der Niederrheinischen Gesellschaft für Natur und Heilkunde zu Bonn," dated May 18, 1903, because this is the first name to appear in print, while the name *T. ugandense* was only read before the Royal Society on May 14, 1903, and did not appear in public print till later. Therefore if, by any chance, the trypanosome found by Castellani in Uganda should prove to be different from *T. gambiense* Dutton 1902, its name would be either *T. ugandense* Castellani 1903, or, as it appeared in public print slightly earlier

T. castellanii Kruse 1903.

The differences between these two trypanosomes is not recognized at the present time because morphologically they seem to be identical.

Matters remained in this position until the year 1910 when Stephens and Fantham advanced the view that the trypanosomes found in cases of sleeping sickness in the Loangwa Valley in Rhodesia belonged to a new species which they called—

Trypanosoma rhodesiense Stephens and Fantham 1910.

Whatever views may be held concerning this species, no one has ever doubted that it was different from *T. gambiense*, in the broadest sense of the word, and some of the experiments which will be described below show how very different it is from the trypanosomes of the Anglo-Egyptian Sudan, Mongalla Province (Old Lado Enclave), and which, from epidemiological and other reasons, is thought, by the present writers, to be probably the same trypanosome as that found in the Congo and in Uganda.

In 1913 Scott Macfie described a new trypanosome in cases of sleeping sickness in Southern Nigeria, separating it from *T. gambiense* and *T. rhodesiense* by:—

(*a*) Its morphological features.

(*b*) The peculiar symptoms of the disease produced by it.

(*c*) The slight mortality it causes in animals.

This trypanosome he names:—

Trypanosoma nigeriense Scott-Macfie 1913.

Thus in differentiating a human trypanosome, it has to be compared with:—

(1) *T. gambiense* Dutton 1902,
(2) *T. castellanii* Kruse 1903, } if these are dissimilar.
(3) *T. rhodesiense* Stephens and Fantham 1910.
(4) *T. nigeriense* Scott-Macfie 1913.

To this point we shall return in the discussion of our observations.

Sleeping Sickness in Countries adjoining the Lado Enclave.—The countries which adjoin the Lado Enclave and which are known to be infected with sleeping sickness are:—

(1) Belgian Congo,
(2) Uganda,

and it is necessary, for the purposes of this paper, to review the known conditions of the disease in the parts of these countries which lie in proximity to the frontiers of the Lado.

(1) *The Belgian Congo.*—In order to understand the conditions under which sleeping sickness has arrived in the Belgian Congo and Uganda it is necessary to review briefly a few of the known salient points with regard to the general history of the disease in Africa.

> The reader of this note is asked to observe carefully that any dates merely signify that those are the periods during which the disease was definitely recognized at a given place and do not mean that the disease had just arrived in that locality.
>
> If it is realized how difficult the diagnosis of sleeping sickness may be and how necessary it is to confirm its presence, in the early stages, by gland puncture and the microscopical recognition of the trypanosome it will be obvious that it could be easily overlooked for years in a place in which it was present.
>
> Lastly it may be remembered that, as a rule, a native, out of politeness or fear, will say anything and agree to anything he thinks is required, and hence misleading evidence may be received as to absence from or the duration of sleeping sickness in a place.

With these preliminary remarks we will review what is known of the principal points of the history of the disease.

The earliest recorded case of sleeping sickness is the death from lethargy of the King Mansa Djata in 1373-74; at that time, it is stated, the disease was *very common* in his country, which was situate in the bend of the Niger.

In the year 1721 John Atkins, Surgeon in the Royal Navy, made a journey to the Guinea Coast, touching at Sierra Leone, places on the Gold Coast, Dahomey and Cape Lopez.

As a result of his observations he says:—

> "Whydah slaves are more subject to smallpox and sore eyes; other parts to a Sleepy Distemper, and to Windward Exomphalos's."

He also mentions "the Sleepy Distemper" in his other book entitled "The Navy Surgeon."

In 1803 Winterbottom recognized the disease as being common in the natives about Sierra Leone and gave an account of the disease, especially emphasizing the importance of the presence of enlarged glands in the neck for early diagnostic purposes.

Sleeping sickness was known to exist on the Congo when Bordier wrote in 1884.

When Corre wrote his justly celebrated book in 1887 it was recognized to extend from the Senegal River in the north to the Loango river in the south. If it is realized that, at that time, hardly anything was commonly known about West Africa Congo, it will be apparent that this only indicates the fringe of the distribution of the disease.

A curious point is to be noted in these old writings, and that is, the persistency with which the authors dwell upon the endemicity of the disease.

Thus Corre says:—

> "Endémie très limitée, et ne prenant jamais la forme épidémique."

Another feature of the disease which appears to have been missed is its duration.

Again quoting from Corre:—

> "On aurait vu des individus atteints deux, trois, cinq ans après avoir quitté les centres endémiques."

Bordier says :—

"On a vu la maladie se déclarer chez les nègres depuis longtemps (7 ans) débarqués aux Antilles."

That is to say, the disease can last longer than seven years after removal from any chance of infection, but how much longer? This question we are still unable to answer.

From this history it is quite clear that sleeping sickness was of old standing in the country enclosed in the bend of the Niger and along the West Coast of Africa from the Senegal to the Loango. It is, therefore, not surprising when we find that it was recognized at Stanley Falls on the Upper Congo in 1898, but that by no means indicated that it had just arrived there, and there is no reason to doubt that, at that period, the greater part of the Congo valley was infected, but when the original infection took place we do not know.

From this source of infection it most probably spread into the western part of the Lado Enclave, perhaps by the agency of Belgian troops or perhaps still earlier.

(2) *Uganda.*—Towards the end of the eighties of last century Stanley led a large force from the Congo to relieve Emin Pasha who was at that time at Wadelai on the Nile, where it bounds the south-eastern part of the old Lado Enclave. Christy and Hodges consider it probable that some of Stanley's people were infected with sleeping sickness and thus introduced the disease into that district and infected Emin's men.

There are several points to support this, viz.: firstly, at the present time Wadelai is known to be infected as was shown by Captain Drew, R.A.M.C.; secondly, Captain Archibald, R.A.M.C., pathologist to these laboratories, travelling northwards from Uganda to the Sudan in the early part of 1908, met with sleeping sickness north of Lake Albert and found *G. palpalis* on the road from Murchison Falls to Wadelai and on the Nile in that region.

Emin's people subsequently travelled with Stanley to Kavali on Lake Albert, which is known to be infected at the present time. Later they were moved to Busoga, known now to be heavily infected, probably since 1896. Still later they passed to Uganda where Mengo, the Sese Islands and the western shores of Victoria Nyanza became infected.

This seems to be the probable history of the source of the infection of Uganda, and seeing that it started from the southern part of the Lado now belonging to Uganda or from Lake Albert it is not surprising to find that there is a heavy infection in the south-eastern part of the Mongalla Province on the Kiyu River near Kajo-Kaji and that this infection wipes out villages and in general behaves just like the Uganda and the Congo epidemics. It is, however, but just to state that it is thought that the Kiyu epidemic was introduced from Uganda years ago by Baganda traders.

The Mongalla Province.—The present Mongalla Province was acquired for Egypt by Sir Samuel Baker and was later administered by General Gordon, and still later by Emin Pasha (Dr. Edward Schnitzler). At this time the inhabitants were being decimated by Arab slave traders to an extent that is now incredible.

The Mahdist rising in the Northern Sudan isolated this Equatorial Province, and Emin Pasha and his people were left there until rescued by Stanley's expedition in 1888. This is a memorable date, as it is believed that sleeping sickness was introduced into this part of Africa by Stanley's followers.

After the departure of Emin Pasha and his people the local inhabitants were left to war with one another at their own free wills, until during the closing years of the last century they were controlled by the Belgians who came from the West, and to whom the Lado Enclave, a territory extending along the left bank of the Nile from Albert Nyanza to 5 deg. 30 min. N. latitude, was leased in order to afford an outlet for the trade of the eastern parts of the Belgian Congo via the Nile.

This traffic, which was probably of importance in the spread of sleeping sickness, was maintained by carriers drawn from the neighbouring regions and from around the lakes.

After the death of King Leopold the Lado reverted to the Anglo-Egyptian Sudan in June, 1910.

As now constituted, the Mongalla Province is bounded—

On the North by the Upper Nile Province and a horizontal east and west line running from the Nile at about 7 deg. 40 min. N. latitude to the Abyssinian frontier.

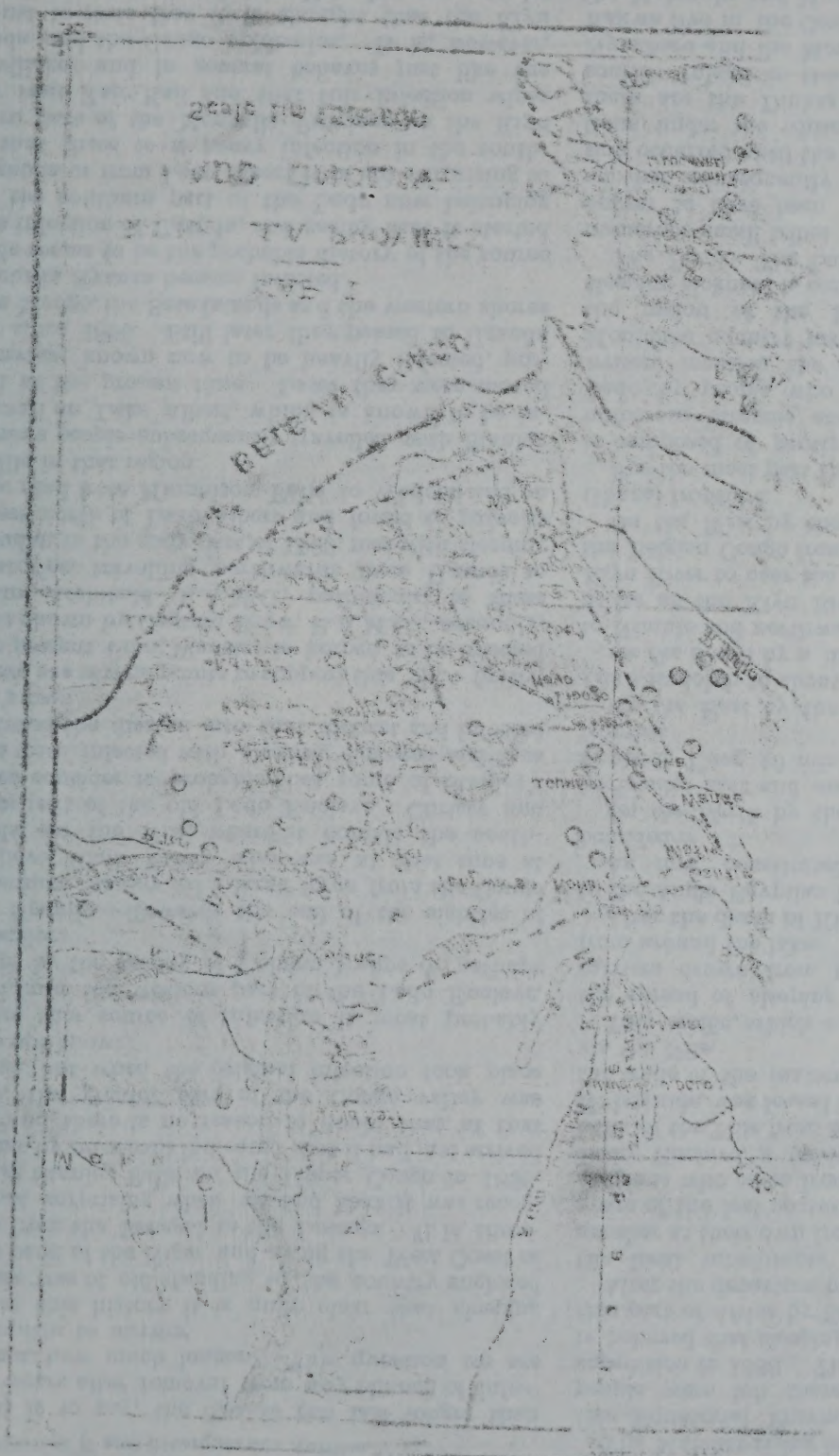
On the East by the Abyssinian boundary and by Lake Rudolph to about 3 deg. 30 min. N. latitude.

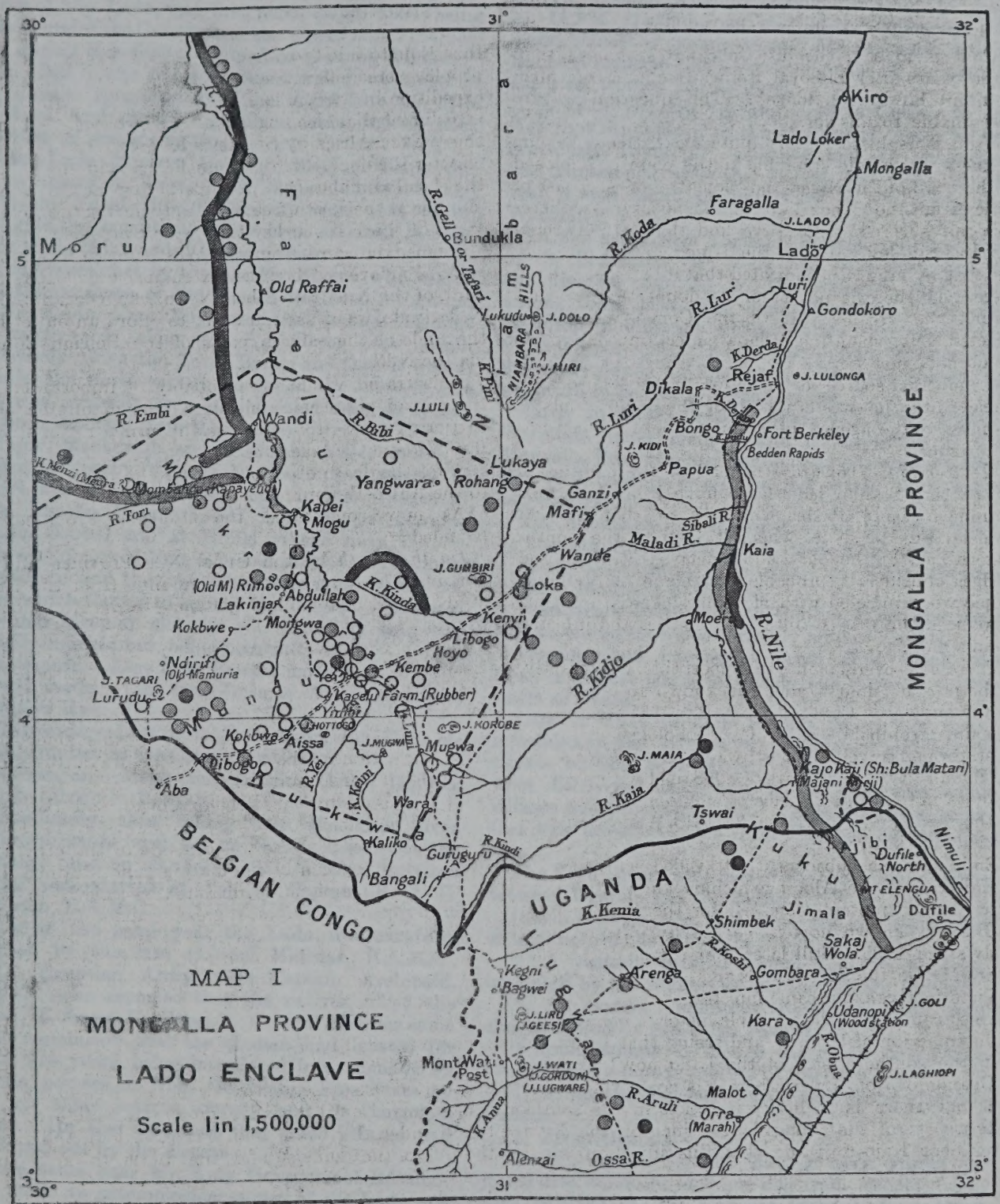
On the South by a line drawn from Lake Rudolph to Nimule and northward just to the west of the Nile as far as the Kiyu River, then westwards along the Kiyu River to near the source of the Kaya River on the Belgian Congo frontier.

On the West by the Belgian Congo and Bahr-el-Ghazal frontiers.

For the most part the western side of the province is composed of gently undulating land drained by numerous streams and inhabited especially in the Lado by people who are great wanderers. Early writers mention the existence of tse-tse fly in the Monbuttu country just south of the Lado, which is the region of the Mongallo Province to which sleeping sickness is confined.

The Lado.—The Lado (*vide* map) is inhabited by numerous small tribes who in their natural condition appear to have been on very poor terms with one another, consequently little or no inter-communication occurred until the advent of more stable conditions under the white man's rule. Along the river there are the Dinkas and the Baris towards the south. Inland in the Northern Lado there are the Nyanbaru and the Morru. The Fajelu, Avokaya and Kakwa live in the Central Lado. To their west are the Makaraka and Mundu tribes. The former are an offshoot of the Azande or Niam-Niams and are a comparatively recent intrusion in this part. In the southern part of the Lado there are the Kaliko towards the west and the Kuku on the plateau near Kajo-Kaji.





Sleeping sickness.
Sleeping sickness areas.
Glossina palpalis.
Glossina morsitans.
Provincial boundaries as regards Lado Enclave only.


MOMBUTTU PROVINCE

The small original Mongalla Province, *i.e.*, the part of the present province to the west of the Nile and north of the Lado, was first occupied in 1901, being placed under the Governor of the Upper Nile Province, and during this time contained no sleeping sickness.

In 1909 it was felt that there was great danger of the disease spreading from the south into the little province, and special regulations were framed with the view of preventing this.

The Lado Enclave lease to Belgium being due to terminate in June, 1910, Major Mackenzie and Yusbashi Yusef Effendi Derwish were sent to investigate the conditions of sleeping sickness therein, prior to its being added to the Sudan.

They were met by the late Dr. Errara, who stated that the presence of the disease had been recognized in the Lado since 1908, and that it had extended far northwards along the Yei River (*vide* map), westwards along the Torre River, and that it was at Kiro in the north-eastern corner of the Enclave, but this was probably only an imported case, as it has not been reported from there since.

Major Mackenzie and Yusbashi Yusef found it present in many villages on the upper waters of Yembi and Kowba Rivers, also in the villages of Wata, Lasuba, Kambora, Sei, Lua, Morgan, Baraba, Lugalla (Luba) and at Bringi village near Wande. They also observed that *Glossina palpalis* was almost everywhere and that the tribes were great wanderers, a fact which tended to spread the disease. The area in which sleeping sickness was definitely seen was carefully marked out, being bounded :—

Eastwards.—Line from Bangali to Loka.

Northwards.—Line from Loka to Wande, and from Wande to Ewe.

Westwards.—Line from Ewe along the Bahr-el-Ghazal frontier to the Congo frontier.

Southwards.—Along the Belgian Congo frontier through Libogo to Bangali.

Immediately after taking over the Lado from Belgium schemes were set on foot to cope with the epidemic ; and in January, 1911, a large isolation hospital was started at Yei, by Captain R. J. C. Thompson, R.A.M.C.

Later in the same year the Lado was carefully inspected by the late Colonel Mathias, R.A.M.C., P.M.O. Egyptian Army, with Captain Archibald, R.A.M.C., who reported that the natives called the disease "Kubeera Na Pongi." Colonel Mathias came to the conclusion that the disease had existed for four to five years, being introduced from Uganda by Baganda porters or from the Congo Free State by them on their return journey and by Congolese soldiers. He also mentions that some villages had been wiped out by the disease.

In the same year Mr. King, Government Entomologist, made an entomological survey of the Lado and reported that *G. palpalis* could be found at any suitable place the whole way from M'volo in the Bahr-el-Ghazal Province to Yei. He also visited the eastern part of the Lado, and mapped out the distribution of *G. palpalis* and *G. morsitans.*

Captain Drew, R.A.M.C., made a careful examination of the Enclave and wrote a most valuable report on the sleeping sickness therein, finding 218 cases in 14,976 examinations and after performing 742 gland puncture examinations. He estimates that in the area inspected he examined about 95 per cent. of the men, women and children.

In 1912 Captain Stigand drew attention to the fact that Kajo-Kaji, in the vicinity of which he had previously found *G. palpalis* and *G. morsitans,* was threatened with the disease and later in the year it was found to be infected as was Loka and Wadelai.

In the same year Captain Ranken reported that up to September 30, there had been 408 cases of the disease admitted to the isolation camp and among these there had been 88 deaths. The case infection of different villages varied very much from 22 to 0·3 per cent. In one set of 695 persons with enlarged glands in the neck 139 were proved by puncture to be due to trypanosomes.

Early in 1913, Colonel Bray, R.A.M.C., P.M.O. Egyptian Army, made a tour of inspection of the Mongalla Province and found that the area of infection had become larger, having spread northwards and eastwards.

The boundaries as described to us in June, 1914, by Captain Ranken, R.A.M.C. (*vide* map), are :—

Eastern.—From a little south of Wara through Loga to Mafi east of Wande.

Northern.—From Mafi to the Yei River and from this to where Ewe was formerly on the frontier.

Western and Southern.—From Ewe along the frontier to just south of Libogo and then to a little south of Wara.

Late in 1913 Captain Ranken found very heavy infections in the villages of Bulamatari and Jokwat situate on the Kiyu River, where about 100 cases were discovered and where it was said that whole villages had been wiped out. Captain Ranken thinks that this infection came from Baganda traders long ago.

The Kiyu River forms part of the proposed boundary between Uganda and the Sudan.

It will thus be seen that there are two main areas of infection in the Mongalla Province, viz. :—

(1) A western : centred around Yei (*vide* map) and inhabited by the Makaraka and the Mundu peoples.

(2) An eastern : adjoining Kajo-Kaji (*vide* map) and inhabited by the Kuku peoples.

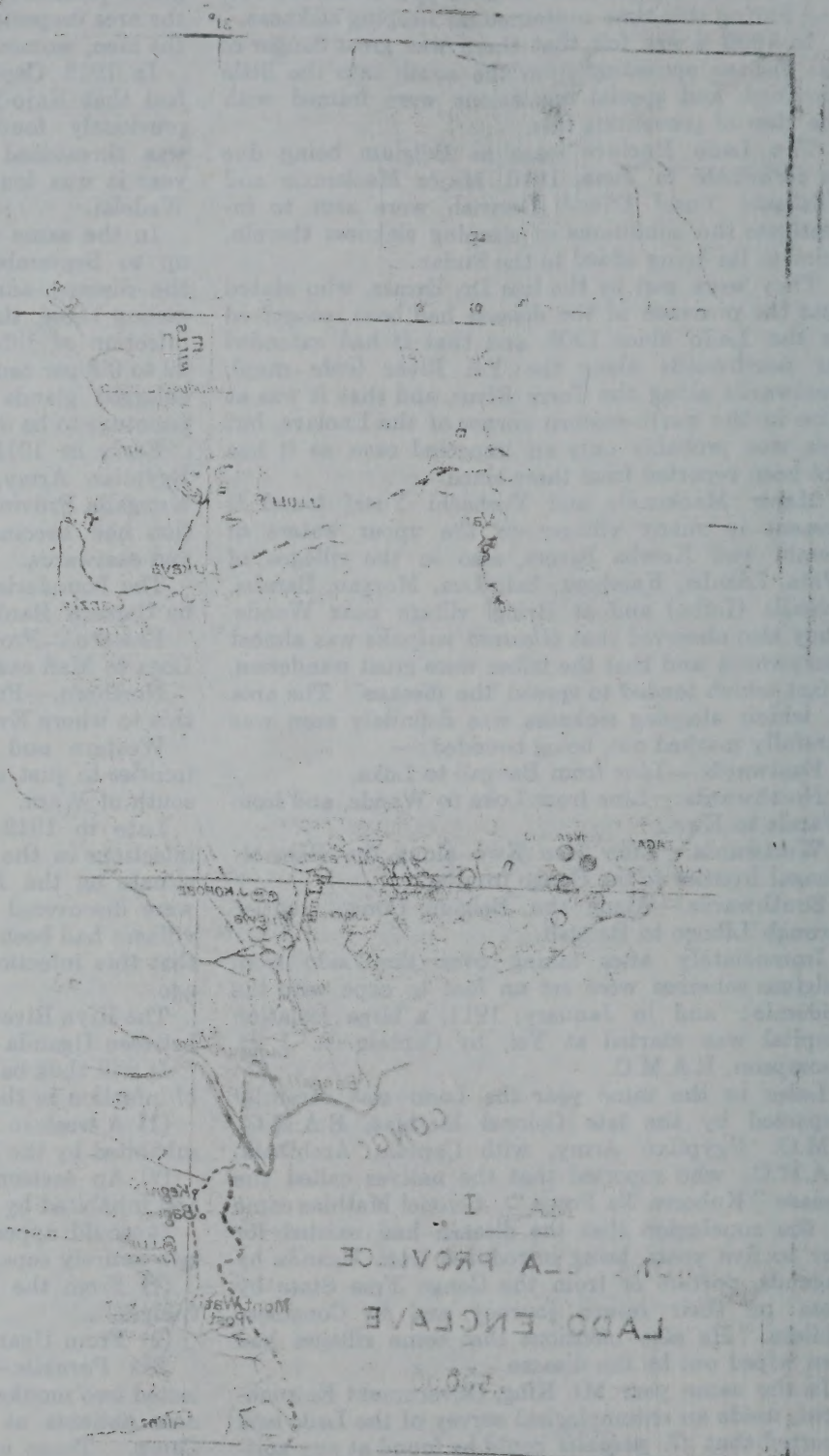
It would appear as though these had arisen from two entirely separate sources of infection, viz. :—

(1) From the Belgian Congo, assisted by Baganda traders.

(2) From Uganda.

The Parasite.—Captain Ranken very kindly injected two monkeys and one dog from sleeping sickness patients at Yei Sleeping Sickness Segregation Camp. These animals were brought to Khartoum by Captain Simpson, who left Rejaf on July 21, 1913, and who arrived in Khartoum on August 10, 1913.

On examination only one monkey was found to be infected and from this animal the strain called the trypanosome of Yei was obtained.



An attempt to forward another strain in December, 1913, was unfortunately not successful, as neither the dog nor the monkey showed any infection on arrival in Khartoum, so that this present note as regards the Mongalla Province is written solely from experiments made on the first strain.

Captain Ranken has kindly supplied the following history of the case from which the monkey was inoculated :—

"The woman was an advanced case of sleeping sickness coming from Abuddal, a Makaraka village situate originally on the banks of the Yei River some twenty miles north of Yei itself, but recently moved to a distance from the river bank. The Makaraka area used to be celebrated for its ivory, and many Baganda traders formerly visited it and may possibly have been the original source of infection. It is of importance to note this fact as it indicates the same source of infection as that which has wiped out the villages of Bulamatari and Jokwat (this should be remembered in reading the account of the action of human serum on this trypanosome given below).

"Each of the animals was inoculated subcutaneously with blood, gland juice, liver blood and cerebro-spinal fluid in order to ensure infection, as the woman was the only untreated case in the camp and the trypanosomes had disappeared from the peripheral blood on the day of inoculation."

It is possible that the strain may represent that present in Uganda or present on the Congo, but this hardly matters, believing, as we do, that the Uganda infection originally came from the Congo.

It is now proposed to give certain details concerning this parasite under the following headings :—

(1) Morphology.
(2) Animal reactions.
(3) Immunity.
(4) Cross immunity.
(5) Cytolytic sera.
(6) Agglutination.
(7) Other reactions.
(8) Mode of transmission.
(9) Iconography.

(1) *Morphology.*—The minimum length was 18 microns, the maximum length was 36 microns, the variation being 18 microns. The minimum breadth was about 1 micron, the maximum breadth was 2·5 microns measured across the widest part.

The average length of 1,000 non-dividing trypanosomes measured in the usual way was 25 microns. The distribution according to length of 1,000 non-dividing forms measured by one hundred per diem from the blood of an infected monkey, *Lasiopyga callitrichus* (I. Geoffroy 1851), and drawn by means of a camera lucida at a magnification of 1,000 diameters and measured by the tangent method, is set forth in Tables I, II and III, and in Chart I.

The history of the monkey is as follows :—

It was inoculated subcutaneously on January 8, 1914, with citrated blood of a gerbil, *Gerbillus pygargus*, which was in the early stages of the disease. Ten days later, *i.e.*, on January 18, for the first time the monkey showed a heavy infection and the count was started and completed on January 27. The monkey died on February 9.

YEI STRAIN.

Graphical representation of 1,000 Trypanosomes from one monkey, *Lasiopyga callitrichus*. (I. Geoffrey, 1851.)

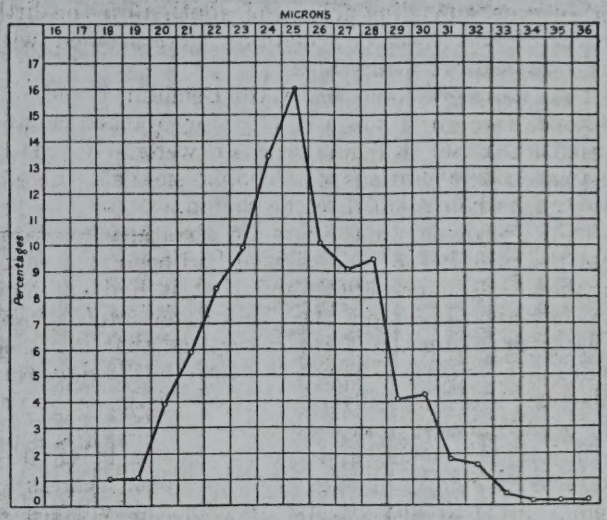


CHART I.

The measured trypanosomes were therefore taken from the tenth to nineteenth day inclusive of an infection lasting about thirty-three days.

A posterior nuclear position has, so far, never been observed by us in this trypanosome.

(2) *Animal Reactions.*—Briefly it may be stated that its virulence in dogs, cats, rabbits, gerbils, jerboas, white rats and monkeys is distinctly less than that produced by *Trypanosome rhodesiense* Stephens and Fantham, but more marked than that recorded for *T. nigeriense* Scott-Macfie.

The average duration of life in infections with this parasite is as follows :—

Dog	Incubation, 12 days; average length of life, more than 40 days, often several months.
Monkey	Incubation about 9 days; average length of life, 35·5 days.
Gerbil	Incubation, 7 days; average length of life, 14·6 days.

(3) *Immunity.*—A dog was rendered immune, *i.e.*, its peripheral blood had failed to show trypanosomes for more than sixty-one days after receiving its fifth inoculation with the Yei strain. The last tested gerbil inoculated with this dog's blood failed to develop an infection.

The serum of the dog, when fully immune, destroyed the *Trypanosome from Yei* in twenty minutes *in vitro*, but had no effect on *T. rhodesiense*, after one hour, *i.e.*, the serum destroyed the homologous but not the heterologous trypanosome. In these observations we confirm the work of Mesnil and Ringenbach as quoted by Stephens and Fantham.

When partially immune the serum was taken and

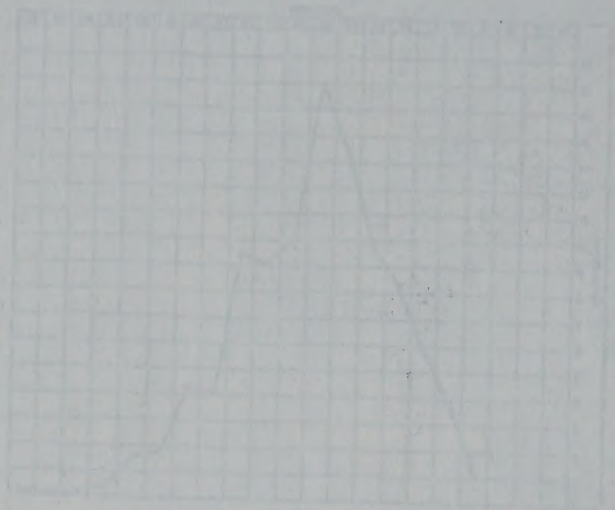


TABLE I.—DISTRIBUTION IN RESPECT TO LENGTH OF 1,000 NON-DIVIDING INDIVIDUALS OF YEI STRAIN OF TRYPANOSOMES IN A SINGLE MONKEY, *Lasiopyga callitrichus* (I. Geoffroy 1851).

		In Microns																					Average	
		14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	Of each 20	Of each 100
1st day	1	..	..	..	..	..	2	2	4	3	..	4	2	2	1	..	..	..	..	..	..	..	..	..	22·65	
	2	..	..	..	..	..	1	1	5	5	2	2	2	1	1	..	..	..	..	..	..	..	..	..	22·55	
	3	..	..	..	..	..	..	3	5	2	4	2	3	1	..	..	..	..	..	..	..	..	..	..	22·50	22·74
	4	..	..	..	..	1	..	3	..	4	8	2	1	1	..	..	..	..	..	..	..	..	..	..	22·45	
	5	..	..	..	..	..	..	2	3	4	–	2	5	2	1	1	..	..	..	..	..	..	..	..	23·55	
2nd day	1	..	..	..	..	1	–	3	2	3	1	5	1	2	1	1	..	..	..	..	..	..	..	..	23·05	
	2	..	..	..	..	–	–	–	–	2	4	5	5	2	1	1	..	..	..	..	..	..	..	..	24·40	
	3	..	..	..	..	..	..	2	1	1	6	3	2	1	1	1	–	2	..	..	..	..	..	..	24·20	24·14
	4	..	..	..	..	..	1	–	3	1	3	5	3	2	–	2	..	..	..	..	..	..	..	..	23·80	
	5	..	..	..	..	..	..	..	..	..	5	3	4	2	4	1	–	1	..	..	..	..	..	..	25·25	
3rd day	1	..	..	..	..	..	..	1	1	2	5	2	4	1	–	3	–	1	..	..	..	..	..	..	24·40	
	2	..	..	..	..	..	..	1	2	2	1	6	4	1	2	–	1	..	..	..	..	..	..	..	24·10	
	3	..	..	..	..	1	–	–	–	5	3	2	3	3	2	1	..	..	..	..	..	..	..	..	24·00	23·99
	4	..	..	..	..	..	1	–	1	3	1	3	5	3	1	2	..	..	..	..	..	..	..	..	24·35	
	5	..	..	..	..	1	–	2	1	4	5	1	3	1	1	1	..	..	..	..	..	..	..	..	23·10	
4th day	1	..	..	..	..	..	1	1	–	1	–	2	2	1	6	–	2	1	1	2	..	..	..	..	26·50	
	2	..	..	..	..	1	–	–	2	2	2	6	4	–	–	1	–	2	–	–	..	..	..	..	24·10	
	3	..	..	..	..	1	–	3	2	–	–	2	1	4	3	2	–	1	1	..	..	..	..	..	24·75	25·05
	4	..	..	..	..	..	..	1	1	1	3	4	4	1	1	1	1	1	1	..	..	..	..	..	24 95	
	5	..	..	..	..	1	–	–	1	1	–	3	7	1	4	1	1	–	–	..	..	..	..	..	24·95	
5th day	1	..	..	..	..	..	..	1	–	1	2	1	4	2	3	3	1	2	–	..	..	..	..	..	25·90	
	2	..	..	..	..	..	..	..	3	2	2	3	1	1	3	3	–	1	1	..	..	..	..	..	25·25	
	3	..	..	..	..	..	..	..	1	2	1	2	3	2	1	1	4	2	–	–	1	..	..	..	26·35	25·87
	4	..	..	..	..	1	–	–	–	2	1	2	2	5	2	4	1	..	..	..	..	..	..	..	25·40	
	5	..	..	..	..	..	..	..	..	2	1	2	5	–	2	2	2	3	1	..	..	..	..	..	26 45	
6th day	1	..	..	..	..	..	..	1	2	–	–	1	6	2	3	4	–	–	1	..	..	..	..	..	25·60	
	2	..	..	..	..	..	..	..	1	3	–	4	5	–	3	1	1	1	–	1	..	..	..	..	25·40	
	3	..	..	..	..	..	..	1	1	–	2	4	1	5	1	1	–	3	1	..	..	..	..	..	26·20	25·51
	4	..	..	..	..	..	..	2	1	1	4	2	1	3	2	2	2	–	..	..	..	..	..	..	24·70	
	5	..	..	..	..	..	..	..	1	2	2	1	4	1	5	1	1	2	..	..	..	..	..	..	25·65	
7th day	1	..	..	..	..	..	..	1	2	1	4	3	4	1	–	1	2	–	1	..	..	..	..	..	24·55	
	2	..	..	..	..	..	..	..	..	2	–	3	4	5	–	3	–	1	1	1	..	..	..	..	26·15	
	3	..	..	..	..	1	1	–	–	–	2	3	2	2	4	2	2	–	1	..	..	..	..	..	25·50	25·46
	4	..	..	..	..	..	..	..	1	–	1	1	5	3	2	2	2	2	1	..	..	..	..	..	26·50	
	5	..	..	..	..	..	1	1	–	1	2	6	3	2	1	1	1	1	..	..	..	..	..	..	24·60	
8th day	1	..	..	..	..	..	1	4	1	4	5	1	2	2	..	..	..	..	..	..	..	..	..	..	22·45	
	2	..	..	..	..	..	..	..	1	–	2	3	6	–	3	3	1	1	..	..	..	..	..	..	25·15	
	3	..	..	..	..	1	1	–	2	3	1	5	3	1	2	1	..	..	..	..	..	..	..	..	23·55	23·69
	4	..	..	..	..	..	1	1	3	3	3	2	2	2	1	1	1	..	..	..	..	..	..	..	23·55	
	5	..	..	..	..	..	..	1	2	3	1	5	6	1	–	1	..	..	..	..	..	..	..	..	23·75	
9th day	1	..	..	..	..	..	..	..	..	..	2	2	3	3	6	2	–	–	–	2	..	..	..	..	26·45	
	2	..	..	..	..	..	..	..	1	1	–	5	4	3	–	4	1	–	1	..	..	..	..	..	25·65	
	3	..	..	..	..	..	..	..	..	..	2	1	3	5	4	4	–	1	..	..	..	..	..	..	26·25	26·31
	4	..	..	..	..	..	..	..	1	2	1	1	4	1	2	2	4	1	–	1	..	..	..	..	26·30	
	5	..	..	..	..	..	..	..	..	1	1	–	1	3	5	7	1	1	..	..	..	..	..	..	26·90	
10th day	1	..	..	..	..	..	..	..	..	..	..	1	2	4	–	1	4	2	2	2	..	1	..	1	28·90	
	2	..	..	..	..	..	..	..	..	..	1	–	2	1	1	4	1	4	1	3	1	..	1	..	29·10	
	3	..	..	..	..	..	..	1	–	–	–	1	3	2	1	8	–	1	–	1	2	..	..	..	27·50	28·05
	4	..	..	..	..	..	..	..	..	1	2	2	1	3	1	4	–	2	2	2	..	..	..	..	27·20	
	5	..	..	..	..	..	..	..	..	..	..	2	2	3	2	4	3	3	–	1	..	..	..	..	27·55	
Totals		..	..	..	..	10	11	39	58	83	98	133	159	100	90	96	40	43	17	16	4	1	1	1	Average length of 1,000 is 25·071	

TABLE II.—SUMMARY OF MEASUREMENTS (IN MICRONS) OF LENGTHS OF 1,000 INDIVIDUALS OF *T. yei* STRAIN FROM A SINGLE MONKEY, *Lasiopyga callitrichus*.

		Maximum	Minimum	Averages of each 100	Averages of each 20	Range of averages of each 20
	1	27	19		22·65	
	2	27	19		22·55	
1st day	3	26	20	22·74	22·50	1·10
	4	26	18		22·45	
	5	28	20		23·55	
	6	28	18		23·05	
	7	28	22		24·40	
2nd day	8	30	20	24·14	24·20	2·2
	9	28	19		23·80	
	10	30	23		25·25	
	11	30	20		24·40	
	12	29	20		24·10	
3rd day	13	28	18	23·99	24·00	1·3
	14	28	19		24·35	
	15	28	18		23·10	
	16	32	19		26·50	
	17	30	18		24·10	
4th day	18	31	18	25·05	24·75	2·4
	19	31	20		24·95	
	20	29	18		24·95	
	21	30	20		25·90	
	22	31	21		25·25	
5th day	23	33	21	25·87	26·35	1·2
	24	29	18		25·10	
	25	31	22		26·45	
	26	31	20		25·60	
	27	32	21		25·40	
6th day	28	31	20	25·51	26·20	1·3
	29	29	20		26·70	
	30	30	21		25·65	
	31	31	20		24·55	
	32	32	22		26·15	
7th day	33	31	18	25·46	25·50	1·95
	34	31	21		26·50	
	35	30	19		24·60	
	36	26	19		22·45	
	37	30	21		25·15	
8th day	38	28	18	23·69	23·55	2·7
	39	29	19		23·55	
	40	28	20		23·75	
	41	32	23		26·45	
	42	31	21		25 65	
9th day	43	30	23	26·31	26·25	1·25
	44	32	21		26·30	
	45	30	22		26 90	
	46	36	24		28·90	
	47	35	23		29·10	
10th day	48	33	20	28·05	27·50	1·9
	49	32	22		27·20	
	50	32	24		27·55	
					Range =	29·10
						22·45
						6·65

inoculated into gerbils *immediately* after mixture with the trypanosomes. The results were as follows:—

Trypanosome of Yei — An infection resulted from which the animal was recovering when it died of heat stroke, on the same day with forty-nine other animals, *i.e.*, on the thirty-ninth day after inoculation.

T. rhodesiense — An infection resulted which killed the animal on the seventh day after inoculation.

When completely immune the serum was taken and after being in contact with the trypanosomes (1 c.c. of the serum to 0·1 c.c. of infected blood, both infections being as nearly as possible of the same apparent strength) for thirty minutes, was inoculated into gerbils:—

Trypanosome from Yei — No trypanosomes seen in the peripheral blood of the inoculated gerbil, but the animal was accidentally killed two days fourteen hours after inoculation. It showed no trypanosomes in the internal organs, but peculiar bodies were seen in the cells of lung smears, comparable with the granules found by Archibald in the spleens of kala-azar patients and of animals inoculated with kala-azar. These granules are quite different from the infective granules described by Fry and Ranken.

T. rhodesiense — The gerbil became infected and died in ten days.

TABLE III.—*T. yei* STRAIN IN WHICH THE TRYPANOSOMES ARE ARRANGED IN BRUCE'S THREE GROUPS: (*a*) 13—21 μ; (*b*) 22—24 μ; (*c*) 25 μ AND UPWARDS.

Day	1	2	3	4	5	6	7	8	9	10	Totals
(*a*) Stumpy, 13—21 μ	32	13	12	15	6	10	8	19	2	1	118
(*b*) Intermediate, 22—24 μ	44	47	45	27	26	26	29	41	19	10	314
(*c*) Long, 25—36 μ	24	40	43	58	68	64	63	40	79	89	568
Totals	100	100	100	100	100	100	100	100	100	100	1,000

TABLE IV.—IMMUNITY EXPERIMENTS IN VITRO.

Animal	Immune serum + *Trypanosome from Yei*	Immune serum + *T. rhodesiense*
Dog immunized against *Trypanosome from Yei* in the Lado.	All trypanosomes dead in twenty minutes.	Trypanosomes alive at end of one hour.
Goat immunized against *T. rhodesiense*, original strain of Stephens and Fantham 1910.	Trypanosomes alive at end of one hour.	All trypanosomes dead in twenty minutes.

TABLE V.—IMMUNITY EXPERIMENTS IN VIVO.

Immune serum	Gerbil inoculated with immune serum + *Trypanosome from Yei*	Gerbil inoculated with immune serum + *T. rhodesiense* original strain
From dog partially immunized against the *Trypanosome from Yei*. The inoculation of the gerbils was made immediately after mixing with the immune serum.	Inoculated 9.4.14; showed trypanosomes 15.4.14; good infection 17.4.14; after which trypanosomes diminished and the animal was in apparently good health on 17.5.14, when it died of heat stroke with forty-nine other healthy and inoculated animals, *i.e.*, thirty-nine days after inoculation.	Developed severe trypanosomiasis and died on seventh day after inoculation.
From dog completely immunized (*i.e.*, gerbil inoculated with its blood did not develop trypanosomiasis) against the *Trypanosome from Yei*. Trypanosomes left for thirty minutes in contact with serum before injection into gerbils.	Did not show any trypanosomes but was killed accidentally two days and fourteen hours after inoculation. No trypanosomes to be found in internal organs, but peculiar bodies in lung cells identical with those found by Archibald in spleens of kala-azar patients and of animals inoculated with kala-azar.	Developed severe trypanosomiasis and died on the tenth day after inoculation.
From goat immunized against *T. rhodesiense*, original strain. Trypanosomes left for twenty minutes in contact with serum before inoculation into gerbils.	Developed severe trypanosomiasis and died on fourth day after inoculation.	Alive and apparently in its usual health one month after inoculation, and has not shown trypanosomes in its peripheral blood. The gerbil was now killed and films made from the lungs, spleen and liver, but no trypanosomes were found.

(4) *Cross Immunity.*—The immunized animal was used for a cross immunity experiment which we propose to detail in a subsequent paper.

(5) *Cytolytic Sera.*—We have tried the effect of normal human blood serum upon several strains of trypanosomes.

The technique used was to take 0·5 c.c. of the serum and to add to it 0·025 of the infected blood containing as far as possible equivalent numbers of trypanosomes. The experiments were conducted at room temperature, *i.e.*, 102° F.

The results may be summarized briefly by saying that no trypanolysis, worthy of record, took place with two strains of mule trypanosomes, with *T. rhodesiense* original strain, or with the *Trypanosome from Yei* which we are considering.

The human serum certainly slowed the movements of *T. rhodesiense* but it was not observed to destroy any during the space of one hour.

It did not appear to be beneficial to the mule strains.

With regard to the Yei strain the human serum appeared to have a distinctly beneficial action, as at the end of one hour's microscopical examination *in vitro* the trypanosomes were in excellent condition and more active than at the commencement of the experiment.

This beneficial action of human serum on the Yei strain of trypanosome may perhaps explain, at all events in part, the epidemic character of the attack in the Eastern Lado as well as the high mortality in that region.

The only sera which we have observed to produce trypanolysis have been strongly immune sera which destroyed the homologous trypanosomes in a most remarkable manner but did not act on heterologous trypanosomes.

(6) *Agglutination.*—We have observed strong though incomplete agglutination of a strain of trypanosomes from a gerbil and derived originally from a mule by mixing 0·025 c.c. of the infected blood with 0·5 c.c. of normal human blood serum.

If this is admitted it is obvious that agglutination, as a specific test, is useless for the recognition of a trypanosome.

(7) *Other Reactions.*—We have not used such methods as phagocytosis, attachment, complement deviation, &c., as other observers have found them to be unsuitable for the purpose of the differential diagnosis of a trypanosome. We have only performed a few experiments with trypanolytic drugs and quickly came to the conclusion that this form of research would not help our present purpose.

(8) *Mode of Transmission.*—We have made no experiments under this heading, nor are, in our opinion, any necessary if we are correct in our recognition of the species of trypanosome (*vide infra*) which we have received from Yei as the brilliant discoveries of Sir David Bruce and his co-workers have sufficiently proved that it is spread by *Glossina palpalis* (Robineau-Desvoidy, 1830).

TRYPANOSOMA RHODESIENSE.

Graphical representation of 1,000 Trypanosomes from one Rat (white). (Stephens and Fantham, 1913.)

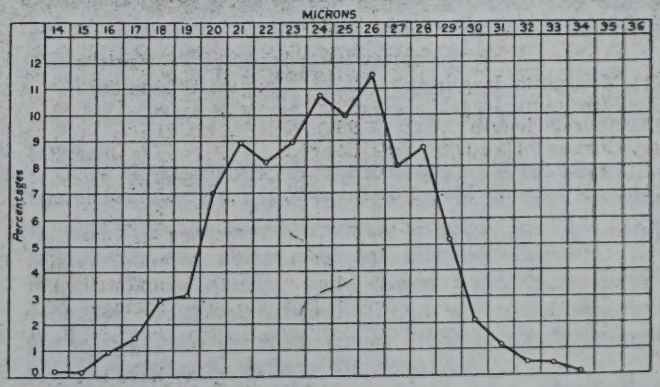


CHART II.

(9) *Iconography.*—We give no photomicrographs of this strain as we believe it (*vide infra*) to be the

same as the Uganda strain which has been so beautifully depicted by Lady Bruce in plate 13 of vol. 84, series B, of the *Proceedings of the Royal Society* for 1911.

Comparison with other Trypanosomes.—The differences and the similarities of this trypanosome with the other known human trypanosomes will now be discussed in the following order:—

(I) *T. rhodesiense.*
(II) *T. nigeriense.*
(III) *T. gambiense*, Congo strain.
(IV) *T. gambiense*, Uganda strain.

(I) *T. rhodesiense* Stephens and Fantham 1910. The trypanosome from Yei differs from *T. rhodesiense* in that:—

	Trypanosome from Yei		*T. rhodesiense*
(1) Maximum length	36	against	34 microns.
(2) Minimum length	18	,,	14 ,,
(3) Average length ..	25	,,	24 ,,
(4) Curve of 1,000 lengths	*Vide* Chart I.	,,	*Vide* Chart II.
(5) Posterior nucleation	Not observed	,,	Present.
(6) Animal reactions	Less virulent	,,	More virulent.
(7) Yei immune serum reactions—			
(*a*) *In vitro*	Destruction of trypanosomes	,,	No destruction.
(*b*) *In vivo*..	Destruction of trypanosomes	,,	Development of disease and death.

TRYPANOSOMA NIGERIENSE.

Graphical representation of 1,000 Trypanosomes from one Guinea-pig. (Scott Macfie, 1913.)

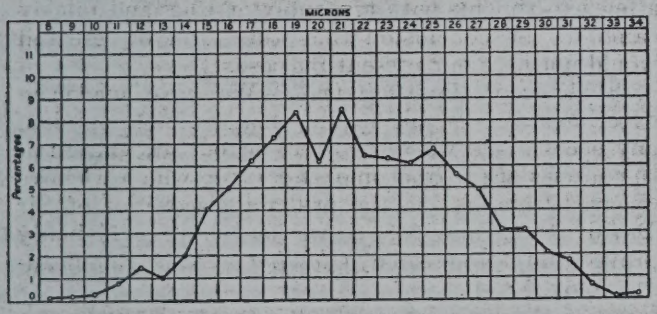


CHART III.

(II) *T. nigeriense* Scott-Macfie 1913. It differs from this trypanosome in that:—

	Trypanosome from Yei		*T. nigeriense*
(1) Maximum length	36	against	34 microns.
(2) Minimum length	18	,,	8 ,,
(3) Average length ..	25	,,	21 ,,
(4) Curve of 1,000 lengths	*Vide* Chart 1	,,	*Vide* Chart 3.
(5) Anterior nucleation	Not marked..	,,	Marked in small forms.
(6) Animal reactions	More virulent; monkeys die in about 36 days after inoculation	,,	Less virulent; monkeys alive and well on an average 127 days after inoculation.

In making these comparisons decimal figures have not been considered.

We have found no records of immunity and transmission experiments with *T. nigeriense* except two observations by Scott-Macfie, indicating a possible development of *T. nigeriense* in the gut of Stomoxys.

We have thus shown that the trypanosome from Yei is neither *T. rhodesiense* nor *T. nigeriense.*

TRYPANOSOMA GAMBIENSE.

Graphical representation of 1,000 Trypanosomes in one Rat (white). (Stephens and Fantham 1913.)

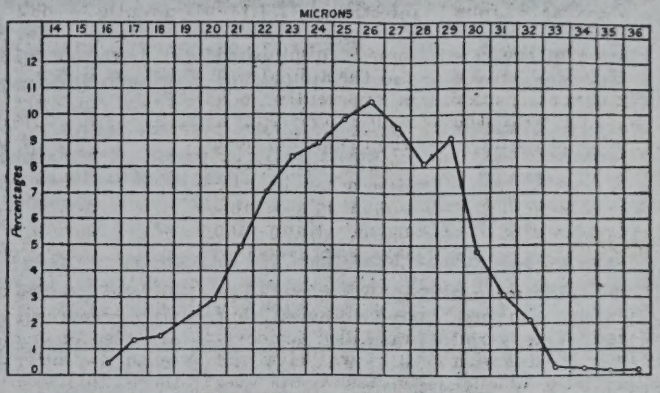


CHART IV.

(III) *T. gambiense, Congo strain.*—It is now necessary to compare this trypanosome with a known strain of *T. gambiense* (*sic*) and this can be done by taking the strain ably described by Stephens and Fantham in the "Annals of Tropical Medicine and Parasitology," 1913, vol. vii, No. 1, p. 27, which, according to Professor Stephens, was obtained from Professor Mesnil in 1905 who, according to Dr. Fantham, obtained it from a case of sleeping sickness from the French Congo.

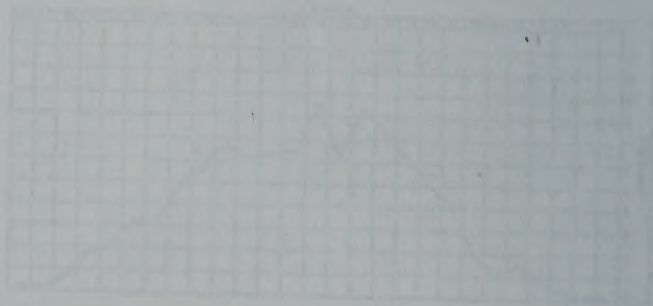
	Trypanosome from Yei		*Trypanosome from Congo*
(1) Maximum length ...	36	against	36 microns
(2) Minimum length ...	16	,,	16 ,,
(3) Average length ...	25·017	,,	24·867 ,,
(4) Curve of lengths ...	Chart I.	,,	Chart 4

Mesnil and Ringenbach have demonstrated that the immune serum protects against the homologous but not against the heterologous trypanosome when *T. gambiense* (*sic*) is compared with *T. rhodesiense* and *vice versa.* With regard to their immunity experiments it is not definitely known whether the strain of *T. gambiense* (*sic*) used was the same as that described by Stephens and Fantham.

It is concluded that the trypanosome from Yei is not dissimilar from the trypanosome of the French Congo.

(IV) *T. gambiense, Uganda strain.*—With regard to the trypanosome found in Uganda, it is not possible to compare the measurements exactly, as the 1,000 trypanosomes measured by Surgeon-General Sir David Bruce, F.R.S., were taken from man, chimpanzees, monkeys, oxen, antelope and rats, whereas our measurements were made from a single animal on ten successive days of its infection.

Notwithstanding this, there is a curious similarity, the proportions being the same, only there is everywhere a difference of 3 microns and the curves very much resemble one another.



	Trypanosome from Yei	*Trypanosome from Uganda*	Difference
(1) Maximum length ...	36	33	– 3 microns
(2) Minimum length ...	16	13	– 3 ,,
(3) Average length ...	25·0	22·1	– 3 ,,
(4) Curve of length ...	Chart I.	Chart V.	..

One cannot help being surprised at the remarkable similarity of the result, considering the very different manner in which the two sets of results were obtained.

TRYPANOSOMA GAMBIENSE.

Graphical representation of 1,000 Trypanosomes in man and various animals. (Surgeon-General Sir David Bruce, F.R.S., 1911.)

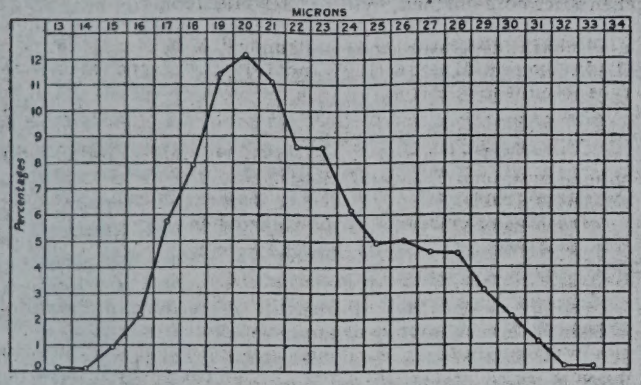


CHART V.

The explanation of the systematic smaller size of the Uganda trypanosome is obtained by comparing our results with those of Sir David Bruce and Stephens and Fantham when divided into Sir David Bruce's three classes :—

Strain	Short, stumpy, 13-21 microns	Intermediate 22-24 microns	Long and slender, 25 microns and upwards
Uganda strain ...	51·2 ...	23·1 ...	25·7
Congo strain ...	18·2 ...	27·0 ...	54·8
Yei strain	11·8 ...	31·4 ...	56·8

It is seen that Sir David Bruce's strains have a preponderance of short stumpy forms but in our opinion this does not prevent them from being the same trypanosome as that examined by Stephens and Fantham and by ourselves, and we are supported in this view by the measurement of the breadth, our minimum being about one micron against Sir David Bruce's 1·5 microns and our maximum 2·5 microns against Sir David Bruce's 2·5 microns.

We have made a preliminary study of the variations in length of a given trypanosome and, with all reserve, we have provisionally come to the conclusion as the result of our observations that the same trypanosome in the same animals may show at times an excess of long and slender forms and at other times an excess of short and stumpy forms.

We are inclined to think that inoculations made from recently infected animals tend to produce increased numbers of long and slender forms while inoculations made from late infections tend to produce short and stumpy forms; we also consider that it may require more than one passage to produce the result.

If we are correct in this, it might partially explain the uniform discrepancy in the measurements given by Sir David Bruce, and a further explanation might be the number of different hosts used by Sir David Bruce, as compared to one host used by Stephens and Fantham and by ourselves, and lastly perhaps the difference in technique (*e.g.*, the compass versus the tangent method, &c.) may also help to explain the difference.

With regard to animal reactions we scarcely meet on common ground, as the conditions under which our animals live must be very different from those under which the Uganda animals lived. Besides this, we can only find one common animal, viz., *Lasiopyga callitrichus* (I. Geoffroy 1851). The duration of infections observed in this monkey may be tabulated as follows :—

Strain	Incubation period	Duration of life
Uganda (Sir D. Bruce) ..	9-40 days ..	2½-12 months
Uganda (Bentmann and Günther)	10 ,, ..	82 days
Yei	9 ,, ..	36 ,,

In other words, the incubation of the disease more or less agrees, but the trypanosome from Yei appears to be more virulent. This, however, may be only apparent and not real, being simply due to the trying climatic conditions under which the animals were compelled to live in Khartoum.

We cannot find records of immunity and cross immunity experiments in which the Uganda strain (definitely stated) is compared with strains from other regions.

Conclusions.—We consider we have brought forward sufficient evidence to show that the trypanosomes which we found in the infected animal sent by Captain Ranken, R.A.M.C., and the Congo strain are the same, and that in all probability they and the Uganda strains are also the same. There being no data, that we know of, to compare these strains with *T. gambiense* Dutton 1902, we are of the opinion that at all events provisionally it would be safer to keep the name

"*Trypanosoma castellanii* Kruse 1903"

for these strains until more light is thrown upon the complicated problem of "What is *T. gambiense* Dutton 1902?"

It would appear to us as though the sleeping sickness of Africa could be divided into the following categories :—

(*A*) *Southern sleeping sickness* caused by *T. rhodesiense* Stephens and Fantham 1910, and spread by *Glossina morsitans* Westwood 1850.

(*B*) *Equatorial sleeping sickness* caused by *T. castellanii* Kruse 1903, and spread by *G. palpalis* (Robineau-Deavoidy 1830).

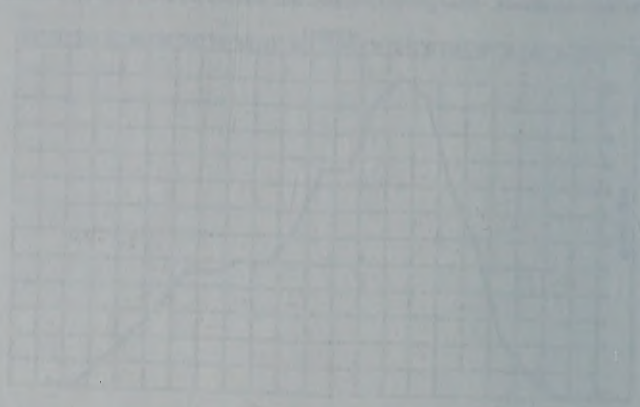
(*C*) *Northern sleeping sickness* which may be caused by as yet imperfectly known trypanosomes named—

(*a*) *T. gambiense* Dutton 1902,

(*b*) *T. nigeriense* Scott-Macfie 1913, and perhaps also by some as yet unknown trypanosomes.

With regard to Sir David Bruce's method of measuring and charting a large number of trypanosomes our observations support the view that this method, if carefully carried out, of comparing these parasites one with another is probably of distinct value and not merely a matter of coincidence as has been maintained recently by Yorke and Blacklock.

Laveran and Mesnil's methods of differentiation by immunization and cross immunization are also, in our opinion, of distinct value.



Further Investigations.—There is, however, a complication to be remembered with regard to the Lado Enclave which, stated in the form of a question, is as follows:—

Why are so many of the cases exceedingly chronic, while others are very acute?

This question is capable of being answered in two ways:—

(*a*) Because the disease has been for some time endemic in the western part of the Lado in which the chronic cases are found and more newly introduced into the part on the east where the acute cases occur. This is supported by evidence given to the writers by Captain Archibald, R.A.M.C., and Captain Ranken, R.A.M.C., and is probably the solution.

(*b*) Because there are two different forms of sleeping sickness. This is not so likely.

Steps have already been taken to enable work to be done to attempt to elucidate these points.

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Illustrations.

(A) Map of the Lado Enclave belonging to the Mongalla Province.

(B) Charts of Lengths of Trypanosomes.

Chart I.—Yei strain of trypanosome	*T. castellanii* Kruse 1903.
,, II.—*T. rhodesiense* made by Stephens and Fantham	*T. rhodesiense* Stephens and Fantham 1910.
,, III.—*T. nigeriense* made by Scott-Macfie	*T. nigeriense* Scott-Macfie 1913.
,, IV.—*T. gambiense* made by Stephens and Fantham	*T. castellanii* Kruse 1903.
,, V.—*T. gambiense* made by Sir David Bruce, F.R.S.	,, ,, ,,