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HUMAN TRYPANOSOMIASIS IN SOUTH-EAST UGANDA

1. A STUDY OF THE EPIDEMIOLOGY AND PRESENT VIRULENCE OF THE DISEASE

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In south-eastern Uganda (Fig. 1) trypanosomiasis has existed since the devastating epidemic at the beginning of the century (CHRISTY, 1903 ; COOK, 1934, 1945). At that time, though *Glossina pallidipes* was known to exist in Busoga (NABARRO and GREIG, 1905), *G. palpalis* was probably the sole vector (BRUCE, NABARRO and GREIG, 1903) and the disease was controlled by removal of persons from the lakeshore distribution of this tsetse and the supervision of fishing activities (BELL, 1908, 1909 ; BAGSHAWE, 1909). During the epidemic

FIGURE 1. Map of northern shores of Lake Victoria. The limits of the epidemiological unit discussed in this paper are outlined.

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and after, when the disease persisted in a low endemic form around Mjanji (Fig. 2), the trypanosomes isolated had avirulent characteristics in laboratory animals typical of *T. gambiense* (BRUCE, NABARRO and GRIEG, 1903 ; DUKE, 1928) even though the virulence in man varied (CHRISTY, 1903 ; VAN HOOF, 1926).

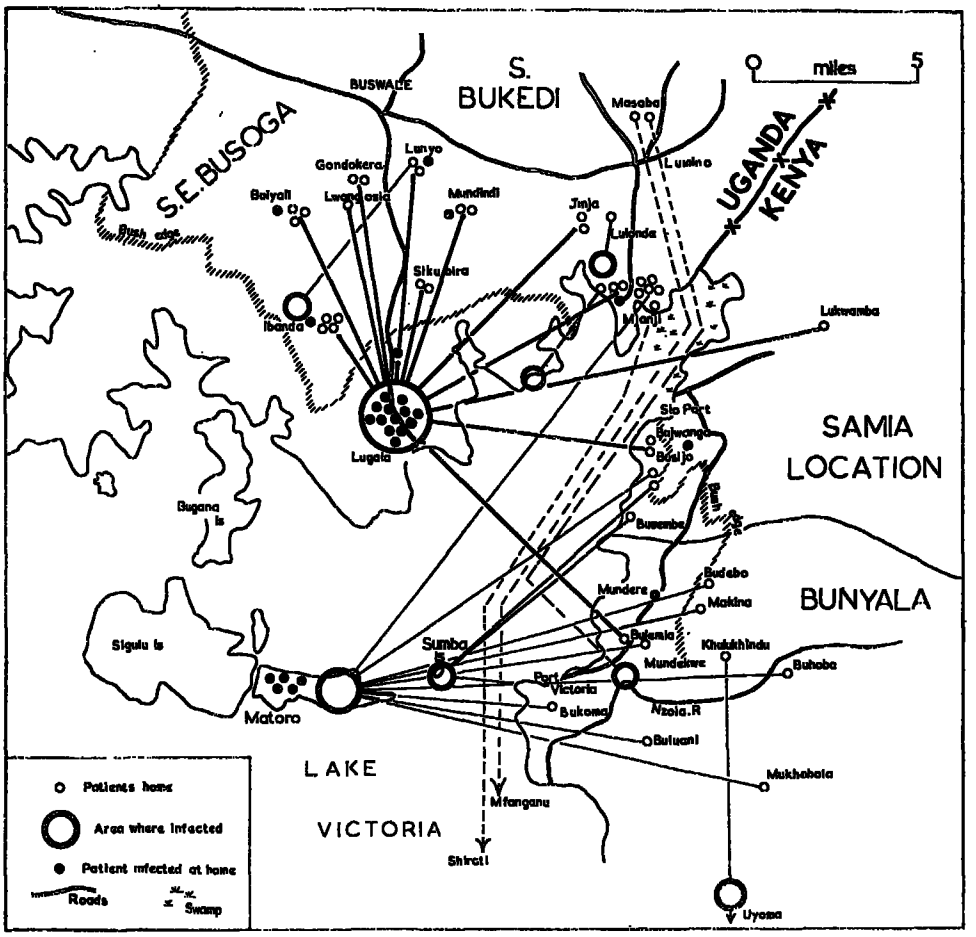

FIGURE 2. Map of S.E. Busoga and S. Bukedi in Uganda and the Samia and Bunyala Locations of Central Nyanza in Kenya, illustrating the epidemiology of sleeping sickness in this area. A continuous line joins the home of each patient to the place of infection. Each black dot refers to a patient infected where he was living permanently or semi-permanently. Interrupted lines indicate a journey taken by the patient, during which he became infected, the exact locality of his infection being uncertain. (Cases seen between 16/9/56 and 30/4/57.)

The disease persisted near Mjanji until 1933 when no further cases were notified until 1939 (Uganda Protectorate Medical Reports). During 1940 - 43 an epidemic occurred in Busoga and south Bukedi, which spread to involve persons beyond the range of *G. palpalis*, and showed the virulent features of *T. rhodesiense* in man and laboratory animals. Jackson isolated *T. rhodesiense* from *G. pallidipes* in the Lugala area at this time (MACKICHAN, 1944).

Since the epidemic of Rhodesian sleeping sickness, the disease has persisted in south Bukedi, mainly, and in Busoga with an incidence varying between 10 and 97 cases annually. It is the purpose of this paper to define the main foci of infection in S.E. Uganda, to present data on the virulence of strains of trypanosomes now found and to discuss epidemiological factors governing the incidence of the disease and the possible routes of its further spread.

DETAILS OF PRESENT EPIDEMIOLOGICAL STUDY

Since September, 1956, it has been possible to investigate cases of sleeping sickness fully in a hospital attached to the laboratories of the East African Trypanosomiasis Research Organization at Sukulu (Fig. 1). As the majority (85/111) of these patients have been in an early stage, accurate details of the origin of their infections have been more easily obtained.

The area from which this series of 111 cases was drawn includes the districts of south-eastern Busoga and south Bukedi in Eastern Uganda, and the adjoining Samia and Bunyala Locations of Central Nyanza in Kenya (Figs. 1 and 2). It may be noted that the Samia people live in the Samia location of Central Nyanza as well as in south Bukedi and in the gombolola (chiefdom) of Buswale in Busoga.

The cases may be divided into two groups ; those occurring between September, 1956 and April, 1957, (72 cases), and those occurring during May and June, 1957 (39 cases).

1. *September*, 1956 — *April*, 1957

(*A*) *The main endemic foci*

From the map (Fig. 2) and Table I it is clear that there are two main foci of infection :

(i) The Lugala area, including the road south of the Lunyo crossroads to the fishing camp and market at Lugala itself ;

(ii) Sigulu Island (including Matoro).

(i) *Lugala*

The road from Lunyo passes through the completely uncleared *G. pallidipes* habitat of mixed deciduous and evergreen thicket, which reaches to within 200 yards of the shore and extends about four miles inland.

The lake shore vegetation provides the habitat of *G. palpalis* and *G. brevipalpis.*

The permanent population of Lugala has increased recently. In 1953 there were only 32 persons but by May, 1957, there were 88 permanent settlers of whom seven contracted sleeping sickness between November, 1956, and May, 1957. The new settlers had come from places as far away as Bunyala, the Samia Location, Mfanganu Island in Kenya and Shirati and Bukoba in Tanganyika as well as from inland villages nearby. The visiting of these places by the settlers from time to time may result in the introduction and spread of *T. rhodesiense.* In addition to these permanent settlers there were (in May, 1957) 64 fishermen living in a semi-permanent camp, whose homes were mainly on the inland fringe of the *G. pallidipes* fly-belt, together with 25 E.A.T.R.O. staff. Of the latter, five contracted sleeping sickness between January and April, 1957. In addition there was a daily average of 30-40 fish-traders coming to Lugala and returning home on the same day.

(ii) *Sigulu Island*

This island consists of two main parts, the settled eastern portion separated from the " officially " uninhabited western part by a papyrus marsh. The eastern part, Matoro,

TABLE I. Place of origin of infection in 72 persons infected with sleeping sickness in S.E. Uganda and the adjacent region of Kenya during the months of September, 1956, to April, 1957.

	Place of infection	Uganda residents (64% Basamia)	Kenya residents Banyala	Kenya residents Basamia	Total cases
Uganda	Lugala	33 (46%)	1	3	37 (51%)
	Matoro Island	11 (15%)	6(8%)	1	18 (25%)
	Mjanji area	2	0	0	2
	Lulonda	1	0	0	1
	Mundindi	1	0	0	1
	Lunyo	1	0	0	1
	Bulyali	1	0	0	1
	Ibanda	2	0	0	2
	Unknown	1	0	0	1
	Total infected in S.E. Uganda	53 (74%)	11	(15%)	64 (89%)
Kenya	Port Victoria to Mundere area	1	1	0	2
	Bujwanga	0	0	1	1
	Visit to Mfanganu	1	0	0	1
	Sumba Island	0	0	2	2
	Uyoma	0	1*	0	1*
	Total infected in Kenya area	2 (3%)	5	(7%)	7 (10%)
Tanganyika	Visit to Shirati	1	0	0	1 (1.5%)

* *T. gambiense.*

has a partially cleared fishermen's camp on its northern shore, which has recently been further cleared on the advice of the Sleeping Sickness Inspector, who has allowed a small settlement to develop on the understanding that continued clearing is carried out. This settlement extends across the central portion of Matoro, which was previously occupied at the beginning of the century. Settlement started in August, 1956, and by 1st March, 1957, consisted of 17 family groups, a total of 170 persons.

The settlers consist mainly of those whose fathers left the island during the great epidemic to live at Mfanganu Island, Bunyala, Shirati and elsewhere. Irregular illegal settlements of a semi-permanent nature are to be found on the shores of the island.

The vegetation is similar to that of Lugala. *G. pallidipes*, *G. brevipalpis* and *G. palpalis* are to be found.

(*B*) *The incidence and distribution of cases*

The predominance of men (91 per cent.) was striking. Only 6 per cent. of infected persons were women and 3 per cent. children under 12 years. The main groups of male subjects were fishermen and fish-traders (64 per cent.) and peasant-cultivators (21 per cent.). The cultivators had been infected mainly on casual visits to buy fish at Lugala. This high incidence among males and especially fishermen resembles closely the incidence described by VAN HOOF (1926) in the era of *T. gambiense.*

During the period under consideration (Sept., 1956—April, 1957), only occasional cases had been infected at or near their villages on the inland fringe of the fly-belt. Furthermore, during the months of January to April, 1957, there were 50 new cases; the total number which occurred during these months in the previous 3 years together was only 39.

This increase constituted a minor epidemic and was probably due to the resettlement at Matoro, the considerable increase in the Lugala population, both permanent and semi-permanent, and the resulting increase in the local fish trade.

Only five cases occurred among the permanent settlers at Matoro; three of the persons infected were father, mother and son. Among those living permanently or semi-permanently at Lugala, 13 cases occurred, five of which were in employees of E.A.T.R.O. Though these figures are small it is certain that persons in the Lugala area are more at risk than the larger numbers of Matoro settlers. However, it can be seen from the map (Fig. 2) that the Mjanji fishermen were mainly infected at Sigulu; this may not reflect the danger to which the Matoro settlers are exposed as it is known that the Mjanji fishermen are not always cordially received by the settlers and it is likely that these visiting fishermen have their own illegal semi-permanent camps elsewhere on the island or on the nearby Sumba Island where fly contact may be heavy.

The incidence among the people of Samia and Bunyala Locations of Central Nyanza is important, constituting 22 per cent. of all cases (Table I). Although at least one of these subjects, a schoolboy, contracted the disease near Port Victoria, the majority of infections occurred as a result of contact at Lugala or Sigulu. The continuous traffic to and from Lugala and Sigulu Island by fishermen and fish-traders could lead to a higher incidence of *T. rhodesiense* in these Locations as the population lives in intimate contact with *G. pallidipes*. Furthermore, persons from Sigulu and Lugala, as well as those from Bunyala, travel to many places on the shores of Lake Victoria, especially to Mfanganu Island and Shirati, from where many of the Matoro and Lugala settlers have come.

CARPENTER (1925) drew attention to the need for a uniform policy on the control of the waters of Lake Victoria divided between Kenya and Uganda and he recognized (1928) Sigulu Island as a reservoir for Gambian trypanosomiasis.

The conclusion is clear: both Sigulu Island and Lugala now form important foci on main fishing trade routes for the more readily transmissible and highly virulent Rhodesian trypanosomiasis.

Only one case of trypanosomiasis due to *T. gambiense* was seen, a woman from Khulukhindu in Bunyala, who was infected in Uyoma where she had been living for several years (Fig. 2).

2. *May and June*, 1957

There was a further increase in the number of cases, 24 occurring in May and 15 in June. The sex distribution and their origins of infection are shown in Table II. A significant increase is evident in the proportion of infected women (28 per cent.) compared with the low incidence (6 per cent.) during the previous 8 months. Fishermen and fish-traders constituted only 31 per cent. of the males, the majority (54 per cent.) now being cultivators. It can be seen (Table II) that the majority of people were infected in their villages. During the 1940 - 43 epidemic there was a similar high incidence among women. (Records of a group of 300 cases treated during 1942 at Lumino showed that the population

TABLE II. Origin of infection and sex incidence in 39 cases of trypanosomiasis occurring during May and June, 1957.

Groups	Place where infected	Age and sex distribution		
		Men	Women	Children
(i)	Lugala (a) buying fish, or visiting	3	0	0
	(b) resident or fishing continuously	7	0	0
(ii)	In the " villages " on the inland fringe of the fly-belt in S.E. Busoga and S. Bukedi	11	9	2
(iii)	Matoro Island (a) visiting	1	0	0
	(b) resident	4	2	0
	Totals	26	11	2

as a whole were involved, 35 per cent. of the cases being in males, 37 per cent. in females and 28 per cent. in children).

The high proportion of people given as infected in these villages could be caused, in part, by denials of having visited Lugala, because a picket had been placed on the Lunyo-Lugala road on 8 May, 1957, to reduce traffic to Lugala, but the high proportion of females supports the belief that infections were actually contracted in the villages. Of these people (infected in their villages) all were peasant cultivators, apart from a schoolmaster and a pupil of his school at Lwangosia.

During April and May, especially the former, the rains had been abnormally heavy and it is probable that this caused an unusual increase in the numbers of *G. pallidipes* and a greater spread inland from its dry-season habitat. This spread is important to remember when deciding limits for settlements or placing institutions such as schools, which might be safe during the dry season but in danger during the wet. MOGGRIDGE (1936) has indicated that *G. pallidipes* may spread out as much as six miles from its dry-season habitat during the wet season.

On 4 June, 1957, the bush along the Lunyo-Lugala road was power-sprayed with Dieldrin emulsion by the Uganda Tsetse Control Department, in an attempt to reduce the number of flies along this road. It is as yet too early to say whether this has had any effect on the incidence of the disease in this area.

(*C*) *The present species of trypanosome and its virulence*

Strains of trypanosomes were isolated from patients immediately on admission, by intraperitoneal inoculation of 1 ml. of blood into albino rats, and, from 53 consecutive attempts, 50 strains were established. A rough measure of their virulence was obtained from the lengths of life of the rats inoculated which varied from 10 - 77 days (Fig. 3).

The three cases in which the trypanosome could not be isolated were an aparasitaemic late case, an aparasitaemic melarsen oxide relapse and an antrypol relapse. Strains were isolated readily in all early cases.

Of the 50 strains isolated, 22 (18 in early and four in late cases) were examined in greater detail. The incubation period and length of life of rats infected with 500,000 parasites from the original rat (inoculated with the patients' blood) were determined, as also was the response of the established infection to tryparsamide in single doses of 80 and 160 mg./kg. given by intraperitoneal injection.

In four of the rats infected with each strain, the percentages of stumpy and posteronuclear forms were determined. Short broad trypanosomes with or without a very short free flagellum were considered as stumpy forms, and those in which the distance from the kinetoplast to the posterior border of the nucleus was equal to, or less than, the nuclear diameter were classified as posteronuclear (P.N.) forms.

In addition to these 22 strains the following four others were tested in the same way :

(i) *Strain Lugala* 2. A polymorphic trypanosome isolated from wild *G. pallidipes* in the Lugala area (RENNISON, 1956). Its infectivity to man was established by Dr. K. C. Willett. It was isolated in September, 1955, and maintained by fly-transmission in the laboratory chiefly through guinea-pigs. This strain had only one syringe passage during its maintenance. It was tested twice, once in March, 1956, and again in February, 1957, after one passage through man by Dr. M. T. Ashcroft at Tinde Laboratory.

(ii) *Sakwa* 21. A polymorphic trypanosome isolated from *G. pallidipes* by Mr. J. C. McMahon, in the Sakwa peninsula of Central Nyanza, between 7 and 9 November, 1956. Its infectivity to man was established by Dr. M. T. Ashcroft.

(iii) *Maswa strains.* Of four strains isolated from man by Dr. F. I. C. Apted, two were fully tested — Maswa 2 and Maswa 3. These two strains were isolated on 28 December, 1956, from patients infected during the recent recurrence of *T. rhodesiense* in Maswa, Tanganika, in August, 1956 (APTED, 1956). The original outbreak appeared in the latter part of 1920 and its vector was identified as *G. swynnertoni* (DUKE, 1923). Cases continued to occur until 1938 when there was a prolonged gap until this 1956 recurrence.

Maswa 2 and 3 had been syringe-passaged about 10 times before testing.

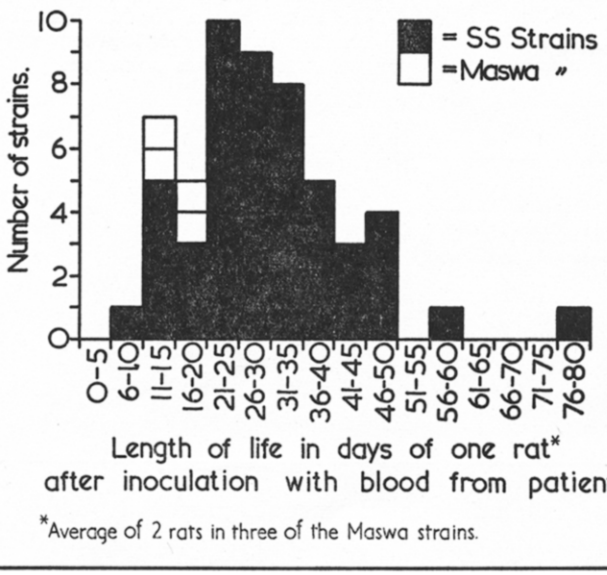

FIGURE 3. Histogram showing the length of life of rats inoculated with 1 ml. of blood from 50 patients from the north-east shore of Lake Victoria, and from four patients from Maswa, Tanganyika.

(a) *Results of the examination of the strains*

The strains consisted of four main groups: those infections originating in Sigulu or its adjacent island of Sumba ; those certainly infected at Lugala ; those infected at various inland villages on the fringe of the *G. pallidipes* fly-belt in Uganda ; and a miscellaneous group of strains including the two strains of *T. rhodesiense* isolated from *G. pallidipes* and two Maswa strains (Fig. 4).

The 50 strains inoculated from cases originating in Sigulu Island, Lugala and the south-east Uganda area were established readily in rats producing a high parasitaemia characteristic of *T. rhodesiense*. Furthermore it is seen that *T. rhodesiense* has occurred within the Samia and Bunyala Locations of Central Nyanza (Fig. 2). It is thus clear that

the *T. gambiense* which was prevalent in south-east Uganda and the adjacent islands before 1940 has been completely replaced by *T. rhodesiense* without greatly altering the epidemiological picture. The focal form of the disease remains unaltered, and it predominantly involves the same groups of people.

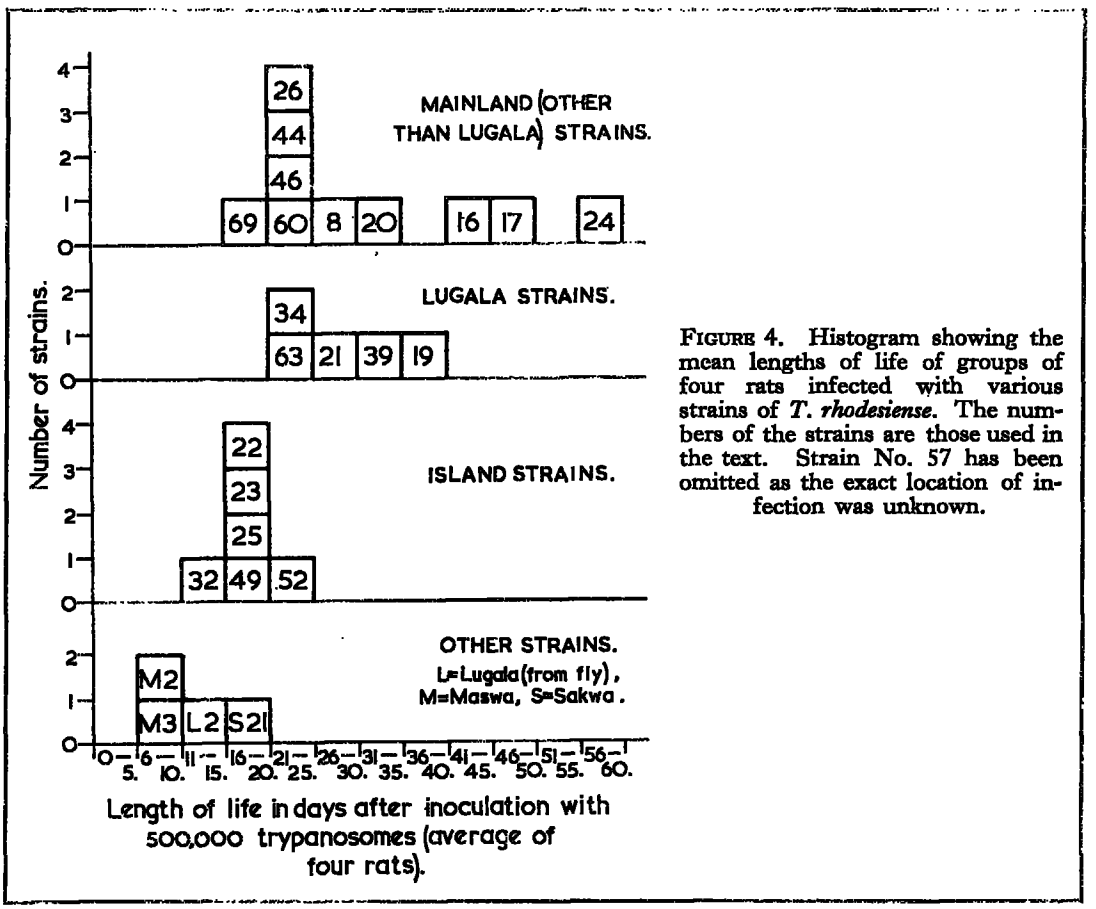

FIGURE 4. Histogram showing the mean lengths of life of groups of four rats infected with various strains of *T. rhodesiense*. The numbers of the strains are those used in the text. Strain No. 57 has been omitted as the exact location of infection was unknown.

The results showed that there is a wide range of virulence (as measured by length of life of infected rats) among strains of *T. rhodesiense* (Fig. 4). The strains isolated from patients from Sigulu or Sumba islands were among the more virulent, and the Maswa strains were more virulent than any encountered locally. The latter had, as previously mentioned, at least 10 syringe passages before testing so did not have the same handling as the strains from our patients. Those strains isolated from *G. pallidipes* (Lugala 2 and Sakwa 21) were also among the more virulent. Fairly wide variation occurred from rat to rat and many tolerated a very high parasitaemia for several weeks : in one strain the length of life of rats varied from 13 to 59 days.

The incubation period was variable (2 - 7 days), though in a single strain a 3-day difference between members of the group of eight rats receiving the same dosage at the same time was the greatest encountered. The length of the incubation period was not absolutely related to the virulence as determined by the length of life of rats.

Great variation was noted in the proportion of stumpy and P.N. forms. In the Mainland, Lugala and Island groups of strains (Fig. 4) the mean maximum proportions of P.N. forms (in four rats) varied between 5 per cent. and 21 per cent. of all forms, and of stumpy forms from 7 per cent. to 50 per cent. The two highly virulent Maswa strains had mean maxima of only 4 per cent. and 2.5 per cent. stumpy forms, and 1.5 per cent. and 0.75 per cent. P.N. forms. In some rats infected with Maswa 3, neither of these forms was ever seen. The two virulent strains isolated from *G. pallidipes* (Lugala 2 and Sakwa 21) had maxima between 6 per cent. and 22 per cent. stumpy forms and between 2 per cent. and 6 per cent. postero-nuclear forms only.

In all the miscellaneous strains, Maswa 2 and 3, Lugala 2 and Sakwa 21, the resistance to 80 and 160 mg./kg. of tryparsamide was absolute. In other groups the majority of strains were absolutely resistant, and in some a temporary remission occurred which never exceeded 5 days. In some individual rats tryparsamide exerted a temporary effect, while no effect was shown in others of the same group infected with the same strain.

(b) *Clinical associations of strains of* T. rhodesiense.

In Case No. 69 the original infection had been diagnosed at a native dispensary 883 days before the patient's admission to this hospital and the isolation of his strain of *T. rhodesiense*. On his first admission to the dispensary he had been ill for about 4 months. He had had no antrypol for at least a year before admission in an advanced stage, without wasting but with marked mental symptoms. This strain, though harboured by him for about 33 months, was among the more virulent (Fig. 4), which suggests that passage through man does not reduce the virulence of *T. rhodesiense*. Furthermore, the danger of such cases as carriers is obvious ; many persons with central nervous system involvement incorrectly treated with antrypol only, may enjoy apparent health for a long time before developing symptoms of the late stage. One case was found which, though trypanosomes were present 10 weeks after antrypol treatment, was entirely symptom-free.

A boy of 16 years of age, Case No. 46, is of special interest in that he resembled a healthy carrier such as was described by Ross (1956). He was admitted for observation suffering from a schizophrenic-like state and had been ill for about 5 days. No trypanosomes could be found on microscopic examination of the blood or glands and his C.S.F. showed no abnormality. He was discharged after 3 days when his condition had improved and he returned home, being able shortly afterwards to continue at school. A rat, which had been inoculated with his blood, became positive. He was re-admitted after 67 days when he was entirely symptom-free, though scanty parasites were found in the gland juice. His C.S.F. protein was still within normal limits and his cell count had reached 10/c.mm. It is probable that he would have developed the late stage insidiously, but his history is given as it does indicate that persons may have long asymptomatic periods and exhibit a carrier-state even though they may later suffer from the effects of the disease. The strain isolated from this boy was among the more virulent (Fig. 4).

In general the virulence of a strain, as determined by animal inoculation, was not related to the severity of the symptoms, and individual variations in susceptibility appeared more responsible for the type of symptoms produced.

(c) *Comparison of the* T. rhodesiense *strains with some of* T. gambiense.

One patient was sent from Pagaya in northern Uganda by the Uganda Medical Department for investigation of the nature of the trypanosome causing the recent epidemic in

Lango, Uganda. This patient had grossly enlarged glands, a characteristic of *T. gambiense* disease, and various animals were inoculated with his blood and gland juice. Rats and guinea-pigs either failed to become infected or developed scanty and intermittent parasitaemia, whereas monkeys (*Cercopithecus aethiops*) were always infected. Two immature monkeys died of trypanosomiasis within 4 weeks of their inoculation, but one adult monkey developed only a scanty parasitaemia. This monkey was apparently cured by the intramuscular injection of tryparsamide (160 mg./kg. body weight).

Another strain of *T. gambiense* from a woman infected at Uyoma in Kenya, seen at Kisii hospital, produced a similar result in an adult monkey, killing it in 168 days. This strain consistently established itself in monkeys, and also infected a 25-days-old rabbit (which died after 178 days). It failed to infect a 6-weeks-old rabbit, a kitten and several mice. Tryparsamide given to an immature infected monkey, 40 mg./kg. body weight, intramuscularly, cleared the blood for 55 days ; the monkey then relapsed but, following a second remission, remained negative for over 4 months. A suspected *T. gambiense* from a third patient (p. 341) failed to become established in adult or suckling rats.

The most important and constant criteria of use in distinguishing *T. rhodesiense* from *T. gambiense* are (1) the ability of the former to establish itself in rats and to produce a high persistent parasitaemia, and (2) its complete resistance to a high dosage of tryparsamide (160 mg./kg.) or its rapid relapse (within a maximum of 10 days) after such treatment. *T. gambiense* either fails to become established in rats or, if it does, it produces a low intermittent parasitaemia. Posteronuclear trypanosomes, often cited as being diagnostic of *T. rhodesiense*, were occasionally seen in monkeys infected with the strain of *T. gambiense* from Uyoma, forming a maximum of 3 per cent. of the trypanosome population. Similar results were reported by LAVIER (1926) for strains of *T. gambiense*. It may be noted that the virulent strains of *T. rhodesiense* from Maswa produced relatively few posteronuclear forms — in some rats, in fact, none was seen.

(*D*) *The vector of* T. rhodesiense *in south-east Uganda*

Since 1903 (BRUCE, NABARRO and GREIG, 1903), when the association between the distribution of *G. palpalis* and sleeping sickness was noted, this fly has been presumed to be the sole vector of trypanosomiasis both in Busoga and elsewhere on the shore of Lake Victoria until the 1940 epidemic of *T. rhodesiense*. In the Report of the League of Nations International Commission of 1926, the importance of *G. palpalis* only is considered with no mention of *G. pallidipes*, even though in 1903 specimens of the latter were found in Busoga (NABARRO and GREIG, 1905), and identified by AUSTEN (1905). *G. pallidipes* was recognized as important in animal trypanosomiasis only. KLEINE (1926) failed to isolate *T. gambiense* from 8,900 *G. palpalis* at Homa Point, Kavirondo Gulf (Kenya), in 1926. *T. rhodesiense* has been isolated from *G. pallidipes* in or near S.E. Uganda on three occasions (Jackson quoted by MACKICHAN, 1944 ; RENNISON, 1956 ; and MCMAHON, pers. commun.), so there can be no doubt that this fly is involved in the spread of the disease.

It is necessary to decide whether *G. palpalis* plays an important rôle in transmission. As the epidemiological picture of the incidence and foci of infection have remained almost unchanged since *T. gambiense* was endemic, presumably transmitted by *G. palpalis*, it might be suggested that this fly may play a major rôle in the spread of *T. rhodesiense* here. Other evidence, is, however, against this. This evidence is outlined below :

(i) RENNISON (1956) failed to isolate *T. rhodesiense* from 3,706 *G. palpalis* caught at or near Lugala and fed on rats.

(ii) Mr. J. Ford pointed out the importance of the site of trypanosome chancres in determining the species of the infecting fly. It is well known that *G. pallidipes* tend to bite below the knee, whereas *G. palpalis* prefer the body, head and neck. In order to test this, JOHNSEN (pers. commun.) noted the parts of the body from which the two species of flies were caught during a " fly-round " at Lugala : his results were as follows :

Site	*G. palpalis*	*G. pallidipes*
Head	4	0
Neck	1	0
Arms	6	2
Body	11	6
Legs	6	73
(On the ground nearby	0	11)
	28	92

These results show that *G. pallidipes* does bite preferentially on the legs.

Of 34 trypanosome chancres seen on patients admitted to our hospital, two were on the head or shoulder, one on the upper arm, two on the forearm, one on the dorsum of the hand, two on the thigh, and 26 on the leg below the knee. This evidence strongly suggests that *G. pallidipes* is now the major vector of trypanosomiasis. It is possible, however, that *G. palpalis* may play a minor part.

Determination of the infection rates of flies in the field by the isolation of strains of trypanosomes of man has never yielded sufficient data to assess the relative importance of two possible vectors, and is unlikely to do so because of the low infection rate of these trypanosomes in tsetse flies.

SUMMARY AND CONCLUSIONS

1) The virulence of the present strain of trypanosome in south-east Uganda is described and it is shown that *T. gambiense* has now been completely replaced by *T. rhodesiense*, possibly since the epidemic of 1940.

2) The site of trypanosome chancres provides evidence that *G. pallidipes* is the major vector of *T. rhodesiense* in this area.

3) Although *T. rhodesiense* has replaced *T. gambiense*, and the major vector is *G. pallidipes*, the disease still remains concentrated in the same foci and affects the same class of people (fishermen and fish-traders) as did *T. gambiense* before 1940.

4) During and immediately after the rains, in May and June, 1957, the incidence increased in the villages on the inland fringe of the *G. pallidipes* fly-belt due to the seasonal extension of the range of this fly.

5) The focal distribution of the disease suggests that game is not a major reservoir in this area and that man-fly-man passage is continuous. It is thought that asymptomatic cases and those relapsing after incorrect use of antrypol constitute the main reservoir. Two such cases are described.

6) No evidence was found that the virulence of a strain of *T. rhodesiense* was reduced by remaining a long time ($2\frac{1}{2}$ years) in a man.

7) As Lugala and Sigulu (Matoro) are main foci of *T. rhodesiense*, and as these places are visited constantly by fishermen and fish-traders, they constitute an important risk to neighbouring territories. *T. rhodesiense* has occurred in Bunyala, Kenya, and it is probable that further cases may occur as the population lives in intimate contact with *G. pallidipes*.

The eastern shore of Lake Victoria is mainly at risk but more remote spread is liable to occur as the fishermen and fish-traders of Bunyala travel widely in their dhows.

8) The most important criteria for distinguishing *T. gambiense* from *T. rhodesiense* in the laboratory are the constant ability of *T. rhodesiense* to produce a high parasitaemia in rats and its complete resistance to a high dosage of tryparsamide (160 mg./kg.) in rats, or its rapid relapse (within a maximum of 10 days) after such treatment.

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