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A history of Rhodesian sleeping sickness in the Lambwe Valley

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The main events in the spread of Rhodesian sleeping sickness around the eastern shores of Lake Victoria during the 1930s and 1940s are summarized and the history of the disease in the Lambwe Valley area of western Kenya is described since its appearance there in 1959. The area was very receptive to the introduction and dispersal of T. rhodesiense *on account of a close association between human communities and their domestic livestock, a large tsetse* (Glossina pallidipes) *population, and game animals. The possible origins of the first Lambwe Valley disease focus and the epidemiological significance of the main elements of the Lambwe environment (man, tsetse, game animals) are discussed in relation to the consolidation and spread of the disease throughout the area.*

Between 1968 and 1971 there was a marked decline in the incidence of the disease, probably as a result of tsetse-control operations that included ground and aerial application of insecticides, bush clearance, and the efforts of the Kenya Game Department to enforce the by-laws of the Lambwe Valley Game Reserve. However, it is considered that the situation remains potentially dangerous, mainly because populations of tsetse are recovering from the effects of aerial spraying and because there is evidence that the tsetse habitats are encroaching on farming land.

Sleeping sickness caused by virulent strains of *Trypanosoma rhodesiense* has affected the human population of the Lambwe Valley area, South Nyanza District, Kenya, for little more than a decade, having first been detected in the area in 1959 (Willett, 1965).[2] Operating from a field station at Lambwe between the years 1968 and 1971, staff of the WHO Trypanosomiasis Project,[3] in cooperation with officials of the Kenya Government, studied many aspects of the disease, its vector, and its reservoirs. During that period there was a marked decline in the incidence of the disease, which was thought to be largely concomitant with the activities of the Project and with tsetse-control operations conducted by the Tsetse Survey and Control Unit of the Kenya Ministry of Agriculture.

This account is concerned with the historical background to the appearance of the disease in South Nyanza, with epidemiological aspects of its spread in the Lambwe Valley area, and with the factors thought to have been responsible for its decline.

ANTECEDENTS TO THE INTRODUCTION OF *T. RHODESIENSE* INFECTION INTO SOUTH NYANZA

Events in the Central Nyanza District of Nyanza Province

Sleeping sickness caused by *T. rhodesiense* was first recorded in Kenya in 1942 among people living in the Samia *Glossina pallidipes* fly belt just south of the Uganda–Kenya border (Ford, 1971). As the Samia fly belt was an eastward extension of the South Busoga belt of south-eastern Uganda, and because the disease had first appeared in Uganda in 1940 at the western end of the Busoga belt (Mackichan, 1944), the disease had probably spread into Kenya via the contiguous Busoga and Samia fly belts. Alternatively, it could have been introduced by Samia fishermen who fished around the shores of Lake Victoria in south-eastern Uganda (Robertson, 1963). At that time, the rest of Central Nyanza

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[2] Also Willett, K. C. et al. (1965) *Joint WHO/FAO African Trypanosomiasis Information Service Report* (unpublished document Tryp./Inf./2.65).

[3] Jointly supported by WHO and a special fund of the United Nations.

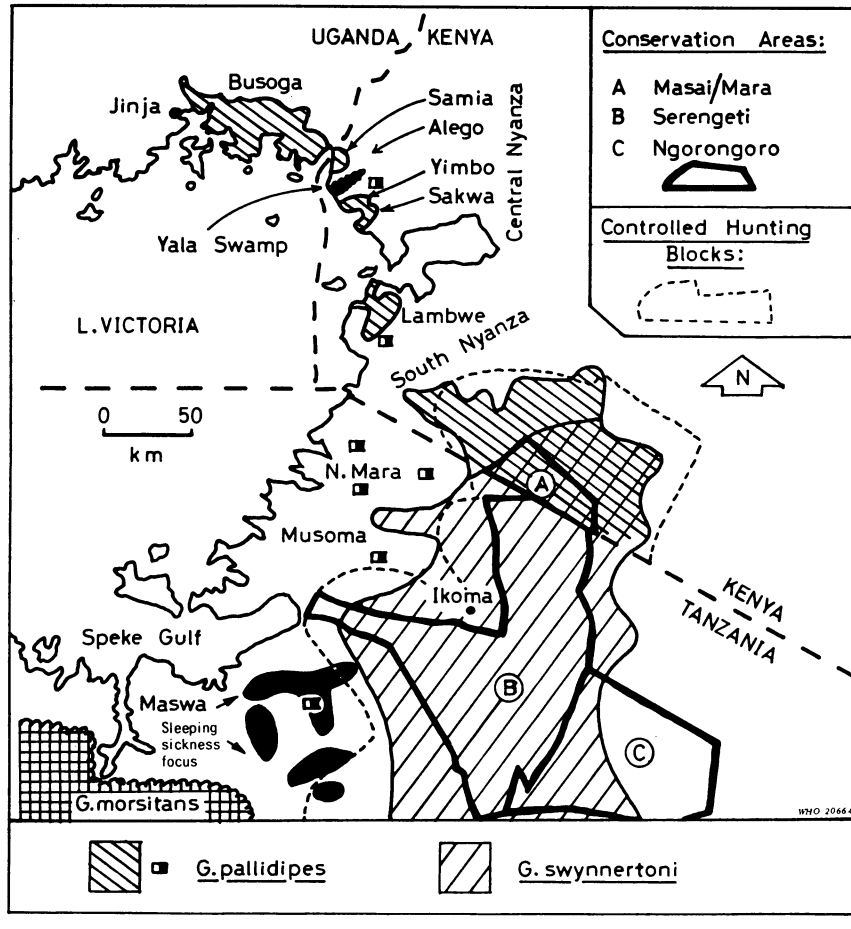

Fig. 1. The distribution of *G. pallidipes* and *G. swynnertoni* in the east Lake Victoria coastal plains (based on Survey of Kenya, 1962, and Ford, 1963).

District was thought to be free of *G. pallidipes*, although suitable thicket habitats were known to exist south of Samia in the Yimbo and Sakwa locations (Glasgow, 1947). *G. pallidipes* was first recorded in the Sakwa Location in 1945 but it is possible that the fly had already entered the Yimbo Location at that time (Wijers, 1969).

The spread of *T. rhodesiense* sleeping sickness southwards from Samia was first suspected in 1953 when sleeping sickness cases did not respond to treatment with the arsenical drug tryparsamide (there was already a history of infection with *T. gambiense* in the area). At that time, the failure of patients to respond to arsenical drugs was regarded as diagnostic of infection with *T. rhodesiense* (Ford, 1971). Confirmation that the disease had spread southwards came in 1954 when *T. rhodesiense* was isolated from a Sakwa sleeping sickness case (Willett, 1955). In the following year the same trypanosome was identified in *G. pallidipes* collected on Sakwa Peninsula,[1] some 30 km south-east of Samia (Fig. 1). These events must have been viewed with grave concern by the authorities at that time, who had doubtless believed that the extensive Yala swamp south of Samia was an efficient barrier to any southward extension of both the disease and its vector.

Although *T. rhodesiense* was identified in man and in *G. pallidipes* in the Sakwa Location around 1954,

[1] See footnote 2 on p. 699.

it has been suggested by Wijers (1969) that *T. rhodesiense* sleeping sickness was established in the Yimbo Location (between Samia and Sakwa) in about 1945. Wijers (*op. cit.*) reported that the inhabitants of Yimbo had described a new form of sleeping sickness that was very different from the form traditionally known to them, i.e., *T. gambiense* sleeping sickness, and much more rapidly fatal. The symptoms described strongly suggested Rhodesian sleeping sickness and, in view of the subsequent findings, there is little doubt that the new type of infection reported by the Yimbo people was in fact caused by *T. rhodesiense*.

Very soon after the confirmation of *T. rhodesiense* in the human and *G. pallidipes* populations of the Sakwa Location, Heisch et al. (1958), working in the Utonga Ridge area of the same location, successfully isolated a strain of *T. rhodesiense* infective to man from a bushbuck (*Tragelaphus scriptus*). The epidemiological implications of this discovery were far-reaching. For many years it had been suspected that animal reservoirs of infection might be involved in *T. rhodesiense* transmission cycles (see Fairbairn, 1948) and experimental evidence was available from the Tinde Experiment (Ashcroft, 1959) to support the hypothesis, but this was the first time that an animal reservoir had been incriminated under natural conditions.

During the 1950s *T. rhodesiense* infection persisted in the Central Nyanza District at a low endemic level, although there was a tendency for localized outbreaks to occur periodically (Ford, 1971). New outbreaks were presumably caused by infected persons moving about the district and introducing the infection into previously uninfected populations of *Glossina*.

This situation in Central Nyanza persisted well into the 1960s (by which time the disease had been reported in South Nyanza), and one of the most important consequences was the introduction of the disease into the Alego Location. An entirely new development in the Alego Location was the cyclic transmission of the disease through *G. fuscipes fuscipes*, which resulted in the explosive epidemic of 1964 (Onyango et al., 1965). The Alego epidemic has been so well documented that a further description of it is unwarranted. However, some aspects of the epidemic may be mentioned briefly here because at least one discovery made while the outbreak was being studied and controlled was relevant to subsequent findings in the Lambwe Valley.

Intensive epidemiological investigations conducted at Alego revealed not only that *T. rhodesiense* was being transmitted by peri-domestic *G. fuscipes fuscipes* under conditions of close contact between man and tsetse, but also that the cattle population was acting as a reservoir of infection. Two *T. brucei* subgroup isolates from cattle were inoculated into human volunteer subjects; one subject rapidly exhibited symptoms of sleeping sickness and *T. rhodesiense* was subsequently confirmed as the causative organism (Onyango et al., 1966).

The position in Central Nyanza at the beginning of the 1960s, i.e., at approximately the time when *T. rhodesiense* infection appeared in South Nyanza, can therefore be summarized as follows. The disease was largely of low endemicity, but was becoming more widely dispersed. In some localities, *T. rhodesiense* infection in tsetse flies was maintained by strains being passed through cattle and at least one species of wild animal, the bushbuck.

Whether or not *T. rhodesiense* infection was introduced into South Nyanza from localities in Central Nyanza, e.g., Yimbo and Sakwa, or whether it came from farther afield, e.g., the Busoga focus, is debatable. However, it is worth describing the antecedent events in Central Nyanza, because they appear to be so clearly reflected in the epidemiological situation that we now know to have existed in the Lambwe Valley in recent years. In all probability, *T. rhodesiense* was introduced from one or both of the northern foci. If so, the environmental similarity between those foci and the Lambwe Valley focus may have been a contributing factor of considerable importance to the rapid spread of the disease through the Lambwe area.

Events in northern Tanzania

Sleeping sickness caused by *T. rhodesiense* was first recorded in northern Tanzania in the 1920s, when it spread northwards from the Maswa focus to the Ikoma and Musoma Districts and then to the west of the Serengeti Plains (Fig. 1). Although the disease was established in this area (in the *G. swynnertoni* and *G. pallidipes* fly belts) about 1925, it did not reach its peak there until about 1932. At a low level of endemicity the disease subsequently spread farther afield and finally entered the Mara District of Kenya via the Mara River Valley (Willett, 1965).

Although sleeping sickness does not appear to have been a problem in Mara District, probably because of the small human population—much of the area having been set aside as the Mara–Masai National Park, as controlled hunting blocks, and as

game reserves—the disease obviously persisted on account of the large *G. pallidipes* and *G. pallidipes–G. swynnertoni* fly belts and the large mammal populations resident there. At its nearest point, the Mara *G. pallidipes* belt was only some 35 km from the Lambwe fly belt, and its shape suggests that at some time in the past the two belts were contiguous. It is perhaps significant that only a few decades ago seasonal migrations of game took place between the Lambwe Valley and the Mara–Serengeti Plains across the 35-km gap between the two fly belts, and that the latter area was populated by elephants that lived in the Lambwe Valley before they were exterminated or driven from the Valley some 30 years ago (Lamprey et al., 1967).

Today, movements of game between Lambwe and the Mara–Serengeti Plains can no longer occur because of a heavily settled area that separates the two areas. In recent years, however, there appears to have been an appreciable movement of people, sometimes accompanied by livestock, into South Nyanza from Mara District and from the North Mara and Musoma (including Ikoma) Districts of Tanzania. Many of these people would have been exposed to *G. pallidipes* and *G. swynnertoni* fly populations, both of which harbour *T. rhodesiense*. Some of them may even have entered South Nyanza with subclinical infections.

It may be of no significance, but it is worth noting that 2 of the 6 sleeping sickness cases diagnosed in the Lambwe Valley in 1970 (Watson, 1972) involved immigrant Maragoli tribesmen who had recently driven their cattle into the area from the Musoma District of Tanzania. These settlers could have contracted the disease in the Lambwe Valley or they could have travelled to the Valley with subclinical infections of Tanzanian origin.

However, while the hypothesis that *T. rhodesiense* entered the Lambwe area from a Mara–Serengeti focus is an interesting one, as will be seen later, the available evidence tends to indicate a Central Nyanza–Uganda origin.

CONDITIONS IN THE LAMBWE AREA AT THE TIME *T. RHODESIENSE* INFECTION WAS INTRODUCED

The principal objectives of this account are to describe the ecological conditions existing in the Lambwe Valley area in the period 1959–60 and to show that a number of environmental factors were potentially favourable for the introduction and spread of *T. rhodesiense* infection.

It was not difficult to reconstruct many aspects of the overall situation during the period, partly because the Survey of Kenya maps in current use were made from aerial photographs taken shortly after the period in question, and partly because the writer had an opportunity to carry out aerial reconnaissance and photographic surveys of much of the area, and to compare his findings with the East African Tsetse and Trypanosomiasis Research and Reclamation Organization vegetation map (1954).[1]

In making the preliminary synopsis, it became clear that each epidemiological factor would have to be analysed separately. The value of graphic representation of the data for this purpose is apparent and in the following sections dealing with the environmental factors the data are presented mainly in the form of distribution maps. Unfortunately, it was not possible in the time available to make a precise determination of the distribution of livestock in the study area; therefore, no livestock distribution map is included.

Factors contributing to the general receptiveness of the Lambwe Valley area to the establishment and dissemination of *T. rhodesiense* infection are considered below under separate headings.

Man, the succumbing vertebrate host

The area is dominated by peoples of Nilotic origin, mainly belonging to the Luo tribe. The origins and occupations of these peoples are described elsewhere (Allsopp & Baldry, 1972). Administratively, the area was divided into the sublocations of Rusinga Island, Kasigunga, Kaksingiri, Gwasi, Nyokal (North, East, and West), and Konyango (East and West). The Lambwe Valley today incorporates part of each of those sublocations, with the exception of Rusinga Island (Fig. 2). The Lambwe Valley sublocation as such was not created until 1960–61.

Human settlement in the area involves 6 distinct zones (Fig. 3), as follows.

(1) *Rusinga Island.* Being largely unsuitable for agriculture, this island is almost entirely populated by fishermen. Economic requirements would have necessitated regular traffic between the island and the mainland. It was most significant that on account of overfishing in the Kavirondo Gulf, the fishermen pursued their activities as far afield as the shores of the Busoga District of Uganda (W. P. Langridge, personal communication).

[1] Compiled by the Division of Tsetse Survey and Control, Kabete, May 1954.

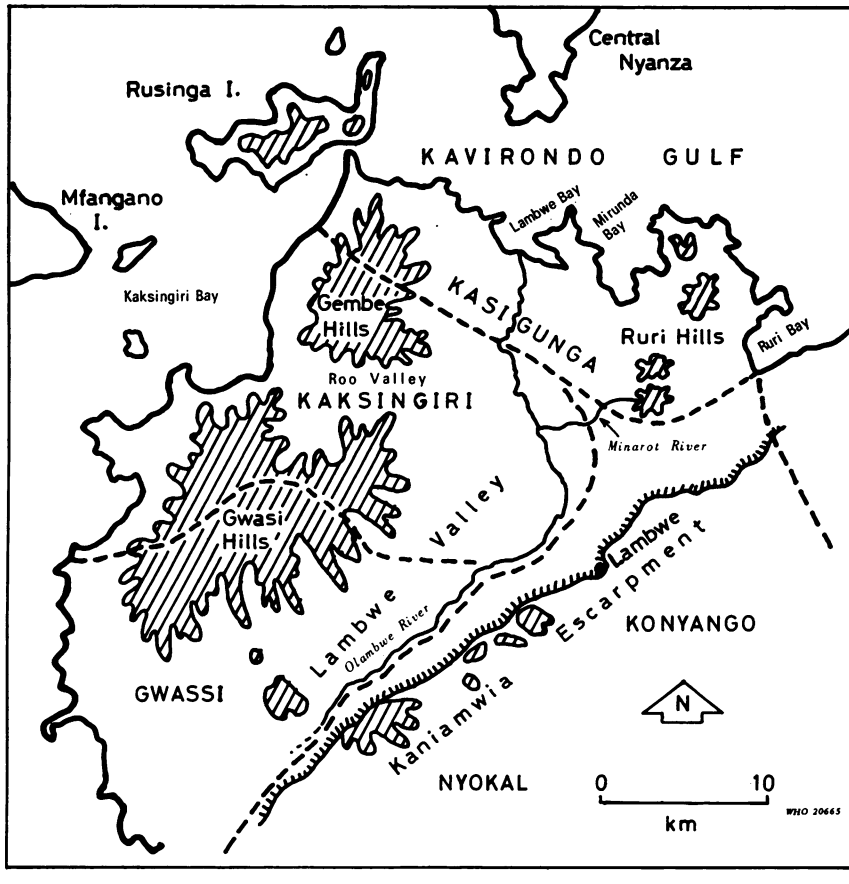

Fig. 2. Sublocation boundaries in the Lambwe Valley area at the end of the 1950s (based partly on Willett, 1965).

(2) *Kaksingiri plains.* This narrow coastal strip is bounded on the west by the lake and on the east by the Gwasi massif. Fishing and subsistence agriculture are the main occupations of the population, together with the manufacture of baskets and mats from the papyrus reeds that fringe the lake. As is usual in this Province, cattle are kept as convertible assets. Population growth probably forced this community to encroach on the thicketed tsetse-infested slopes of the Gembe Hills.

(3) *Plains adjoining the Kavirondo Gulf.* This broad low-lying region, approximately equivalent to the Kasigunga sublocation, displays an agricultural maturity that reflects competent farming over a long period. There are plantations of sugar and bananas in addition to the usual subsistence crops of maize, etc. Perhaps on account of seasonal flooding, this area has remained free of the dense semideciduous thickets that characterize the upper reaches of the Olambwe River. Instead, the area is dominated by grasslands and *Acacia* woodland with *Balanites aegypticum.* Riverine vegetation occurs along the seasonal watercourses ramifying this flood plain.

(4) *Roo Valley.* This steep-sided, thicketed, and tsetse-infestated valley links the Kaksingiri Plains with the Lambwe Valley and the adjoining Kavirondo coastal planis. The western extremity of the valley has been extensively cleared for cultivation, and thickets with their attendant fauna, including tsetse, have been confined to the Gwasi hillslopes. To the east, settlement has become more diffuse and interspersed with lowland thickets—an arrangement favourable for contacts between large mammals and *G. pallidipes.* Major settlement has occurred in the Ponge Dam–Kwoyo Market area, which is a point of convergence of most of the roads and tracks that

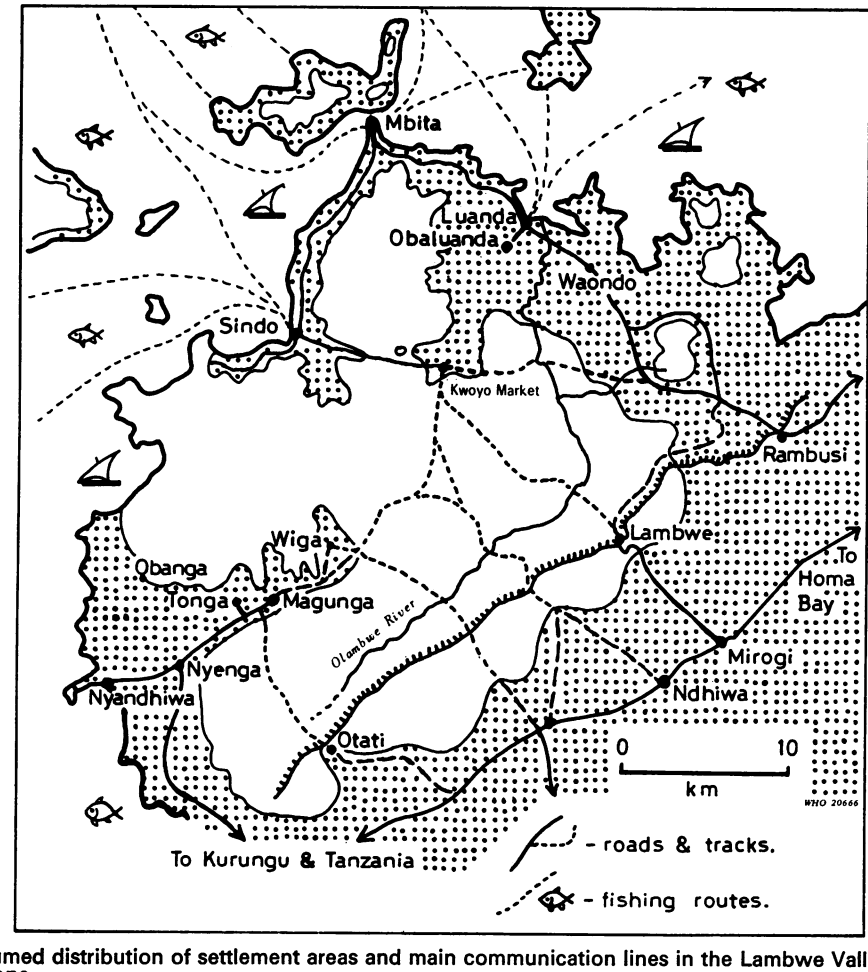

Fig. 3. Presumed distribution of settlement areas and main communication lines in the Lambwe Valley area at the end of the 1950s.

pass through the Gwasi massif. Subsistence farming, charcoal production, and probably hunting are the main occupations.

(5) *South-western coastal plains (Gwasi).* This area of subsistence agriculture and fishing communities is relatively densely populated. A main road between Kwoyo Market, Magunga, and Nyenga, which then continues to the Tanzanian border, crosses the area. Settlement extends well into the Magunga and Wiga valleys of the Gwasi hills. A medical dispensary at Magunga serves a large proportion of the population.

(6) *Nyokal–Konyango uplands.* Situated on the dip slope of the Kaniamwia escarpment, this area is densely populated except in the immediate proximity of the escarpment. The slope is drained by permanent and semipermanent tributaries of the Kuja River system, some of which are fringed with dense vegetation.

Good lines of communication between this area and the Lambwe Valley are used by charcoal-burners, wood gatherers, and fishermen during seasonal flooding of the Olambwe River. A main all-season road connects Home Bay township with Karungu, and eventually with Tanzania. The road passes through Ndhiwa, which contains a medical centre and agricultural and veterinary offices.

The general trend in the Lambwe Valley population was one of steady movement away from the densely populated coastal areas to the low-lying

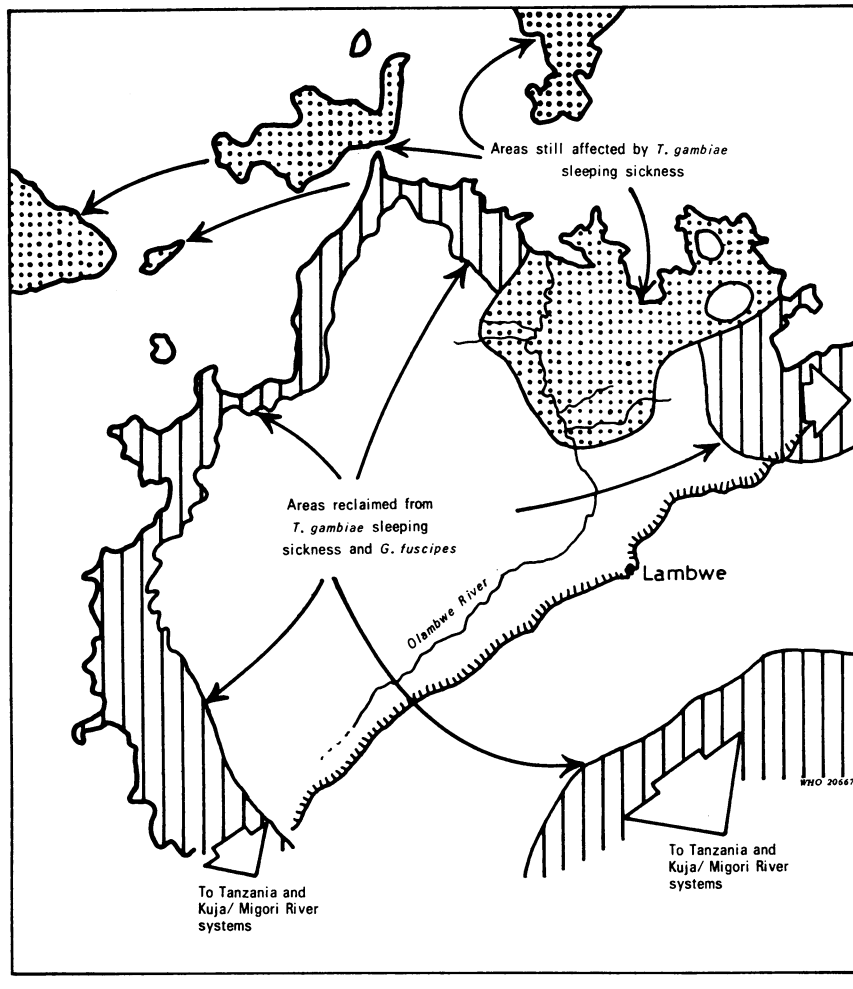

Fig. 4. Areas affected in Lambwe Valley by, and reclaimed from, *T. gambiae* sleeping sickness after the 1955–57 insecticide campaign (from Glover, 1962).

fertile grasslands of the Lambwe Valley and its side valleys. Although the people knew that their cattle would succumb to trypanosomiasis transmitted by the large populations of *G. pallidipes* in the thicketed areas (Fig. 6), the Lambwe Valley appealed to subsistence farmers because, owing to its bimodal distribution of rainfall, they could grow more crops there than in many of the coastal areas, which experience a single wet season only. In addition, the prospect of settling close to the largest population of game animals in the whole of South Nyanza (Fig. 5) must have been attractive.

Some of the people may also have believed that by retreating from the lake shore they were removing themselves from exposure to *T. gambiense* infection transmitted by lacustrine populations of *G. fuscipes*. In fact, Gambian sleeping sickness had been largely exterminated except around the mouth of the Olambwe River by the treatment of *G. fuscipes* habitats with insecticide during 1955–57 (Glover, 1962) (see Fig. 4). These people obviously did not realize that by moving into the Lambwe Valley they were placing themselves in close contact with certain environmental factors (a large *G. pallidipes* population and game animals) that could become extremely dangerous if the disease to which they had never been exposed—Rhodesian sleeping sickness—were introduced into the area.

Table 1. Ungulate species of game animals in [the Lambwe area, and their preferred habitats

Species	Habitat [a]
bushbuck (*Tragelaphus scriptus*)	I, C
buffalo (*Syncerus caffer*)	I, C
bushpig (*Potamochoerus porcus*)	I, C
grey duiker (*Sylvicapra grimmia*)	I, C
impala (*Aepyceros melampus*)	G, I
Jackson's hartebeest (*Alcelaphus buselaphus jacksonii*)	G, I
oribi (*Ourebia ourebi*)	G, I
bohor reedbuck (*Redunca redunca*)	G, I
roan antelope (*Hippotragus equinus*)	G
topi (*Damaliscus korrigum*)	G, I
defassa waterbuck (*Kobus defassa*)	G, I, C

[a] G, grassland; I, isolated thicket clumps; C, continuous thickets.

Livestock : potential reservoirs of infection

Cattle, sheep, and goats were kept by the farmers in some areas, mainly as convertible assets. The susceptibility of cattle to trypanosomiasis was undoubtedly the main factor restraining people from moving into the Lambwe Valley.

It has been suggested above that conditions in the Lambwe Valley were potentially dangerous to people settling there. The simultaneous introduction of cattle, already proved to be natural reservoirs of *T. rhodesiense* (Onyango et al., 1966), increased the potential danger.

Game animals ; potential reservoirs of infection and agents of dispersal

Wild ungulate animals inhabiting the Lambwe Valley area and their preferred habitats are listed in Table 1. With the exception of one or two species, e.g., waterbuck (*Kobus defassa*) and impala (*Aepyceros melampus*), these animals can be classified either as " bush-dwelling " or " plains " game, the former being found in the thicketed areas and the latter roaming the adjacent grasslands.

The main concentrations of bush-dwelling species were in the thickets of the Lambwe Valley, the Roo Valley, the western foothills of Gembe, and West Nyokal. Plains game was largely restricted to the lush grasslands of the Lambwe Valley and the mouth of the Roo Valley (Fig. 5).

Of the bush-dwelling species, the most important in the present context was the bushbuck (*T. scriptus*). This shy, largely nocturnal animal was probably ubiquitous in the Lambwe area, making use of the minimum amount of cover to conceal itself in settled areas. It was also known to be a reservoir of *T. rhodesiense* (Heisch et al., 1958), to be the preferred host of *G. pallidipes* (Weitz, 1963), and to have the ability to survive in settled areas in close association with man and livestock.

None of the plains game species was as important as the bushbuck in relation to sleeping sickness but the reedbuck (*Redunca redunca*) may have been involved to a lower extent. Although *G. pallidipes* rarely fed on reedbuck (Weitz, 1963), these game animals frequently visited settled areas and it has been shown that they are reservoirs of *T. rhodesiense* (Robson et al., 1972). The species may, therefore, also have contributed, with the bushbuck, towards creating conditions in the Lambwe area that were potentially dangerous to the human population.

Several species of game animal may have influenced the movements of *G. pallidipes* thus facilitating the dispersal of various strains of trypanosome. In addition to bushbuck and reedbuck, some game animals may also have acted as reservoirs of infection.

Glossina pallidipes : disease vector

According to E. A. Lewis (in Ford, 1971) *G. pallidipes* was first recorded in the Lambwe area in 1910, " probably in the lakeside section of Roo ". It was therefore present in the area long before it spread from Samia into the Yimbo and Sakwa areas of Central Nyanza, a fact that tends to support the hypothesis that the Lambwe and Mara fly belts were previously contiguous.

G. pallidipes, which is a thicket-dwelling species of the *morsitans* group, prefers to feed on bushbuck and, in western Kenya, bushpig (*Potamochoerus porcus*) (Weitz, 1963), but also takes enough blood meals from man and domestic livestock to make it an important vector of both human and animal trypanosomiasis.

Much has been written about *G. pallidipes* (e.g., Swynnerton, 1936; Buxton, 1955; Ford, 1971), but this account is limited to those aspects of its behaviour and ability to transmit trypanosomes that are relevant to the occurrence of the species in the Lambwe area a decade or so ago. Wherever dense continuous thickets grew below a height of 1 520 m there were medium–high density populations of *G. pallidipes*. In areas where such thickets were less

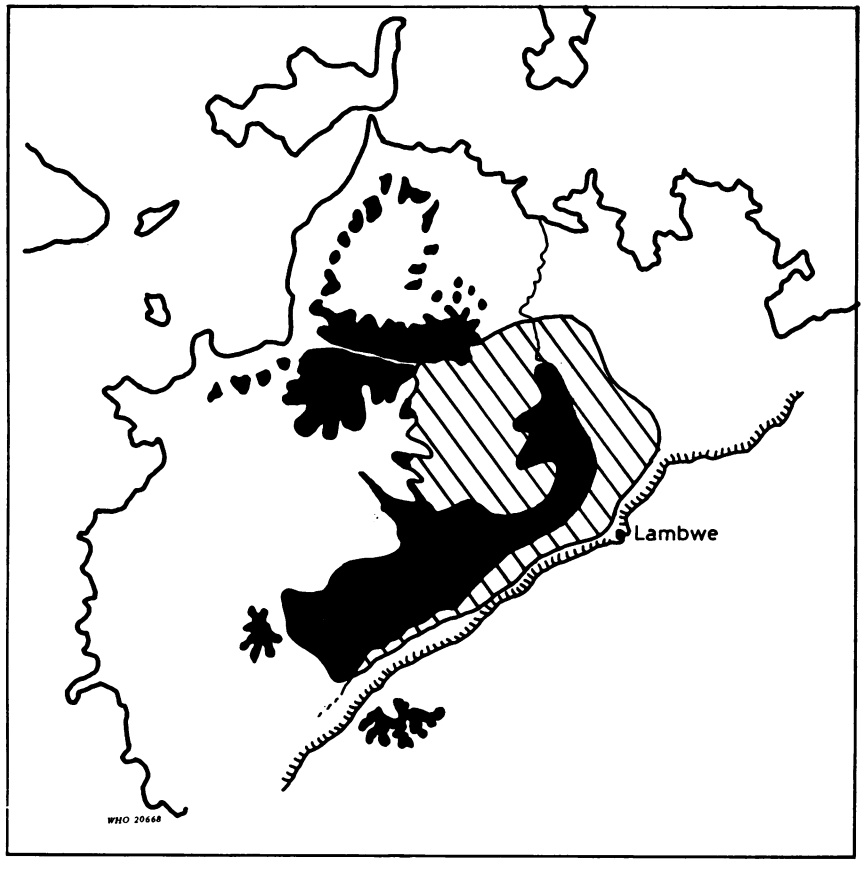

Fig. 5. Presumed lowland distribution of game animals in the Lambwe Valley area at the end of the 1950s (information from R. Allsopp, unpublished data). Solid shading, bush-dwelling species of game animals; diagonal shading, game species of the plains.

dense or where they were replaced by isolated clumps of thicket (see Allsopp & Baldry, 1972), *G. pallidipes* was correspondingly less numerous.

With the help of data on the Kasigunga area supplied by Willett (1965), it has been possible to prepare a fairly accurate map showing the lowland distribution of dense continuous thickets with heavy infestations of *G. pallidipes*, and thin scattered thickets with or without light infestations of *G. pallidipes* (Fig. 6). It will be seen that the heavily infested areas were along the centre of the Lambwe Valley, in the Roo Valley, and in the western foothills of the Gembe Hills (Kasingunga and Kaksingiri sublocations). Subsidiary infestations occurred in the Obaluanda area north of Kwoyo Market and in the West Nyokal sublocation above the Kaniamwia escarpment.

From an intensive investigation carried out at Lugala in the South Busoga fly belt of Uganda, it was known that *G. pallidipes* was active from before dawn until after dusk (Harley, 1965). It is also reported to be active during the night (Harley, *op. cit.*) and to feed at night (Chorley & Hopkins, 1942; Moggridge, 1948). There is no evidence to show that *G. pallidipes* in the Lambwe area behaved differently. It can therefore be assumed that, provided weather conditions were suitable, people and their livestock living close to thickets were exposed to the risk of being bitten by *G. pallidipes* during the hours of daylight. Livestock may also have been attacked at night.

The findings of Harley (1966) on trypanosome infection rates in *G. pallidipes* are also relevant—namely, the overall infection rates at Lugala were

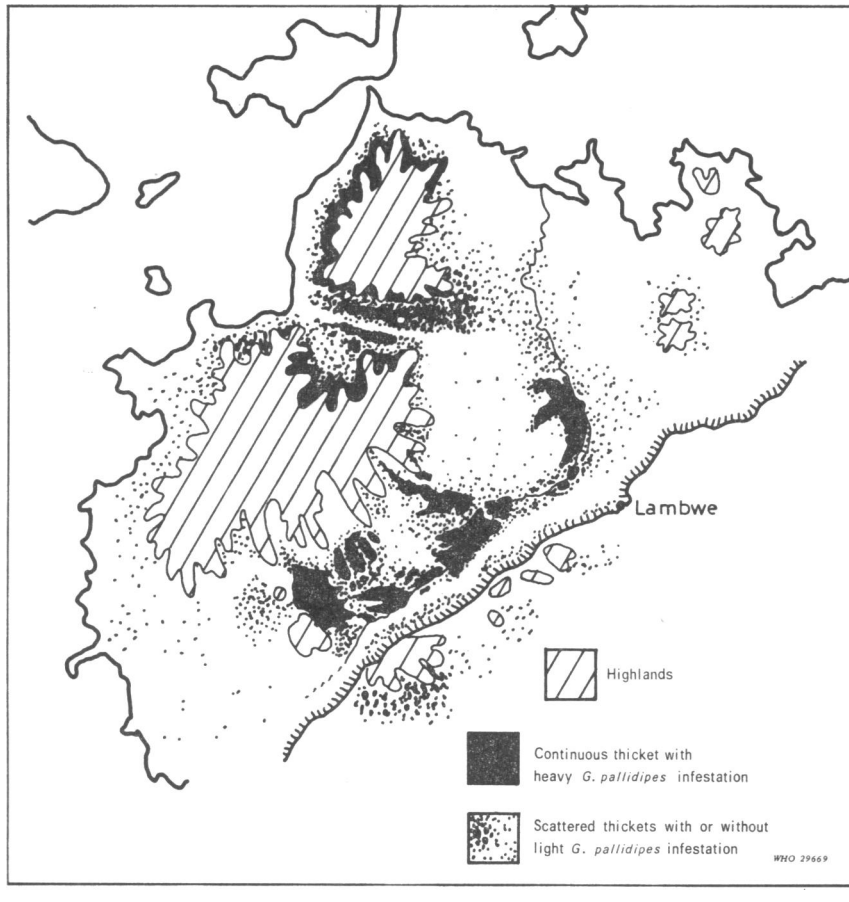

Fig. 6. Presumed associations between *G. pallidipes* and lowland thickets in the Lambwe Valley area at the end of the 1950s.

highest during, or immediately after, months with the greatest rainfall. Recent, though less intensive, studies in the Lambwe Valley have indicated that the same pattern occurs there. The explanation appears to be that under more favourable wet-season conditions, flies live longer and the chance that they will be infected with trypanosomes, particularly of the *brucei* subgroup, is greater.

Areas particularly receptive to the establishment and spread of T. rhodesiense infection

The basic requirements for the cyclic transmission of Rhodesian sleeping sickness in the Lambwe Valley area appear to be man, nonhuman reservoirs (certain game and domestic animals), *G. pallidipes*, and *T. rhodesiense*. It can therefore be assumed that where the trypanosome is absent, but where the other requirements are satisfied, situations suitable for the introduction and establishment of the pathogen are created. Under optimum conditions, the various factors can become so closely associated that the disease in man may reach epidemic proportions. It is apparent, therefore, that in any area for which reliable data are available on the human population, potential reservoirs of trypanosomes, and vectors, the localities within the area most receptive to *T. rhodesiense* can be identified.

This idea has been applied to the Lambwe Valley area as it is considered to have been at the end of the 1950s. By superimposing data on human settlement zones, distribution of game animals, and *G. pallidipes* habitats (Fig. 3, 5, and 6), it has been possible to plot the distribution of areas that were potentially dangerous at the time when *T. rhodesiense*

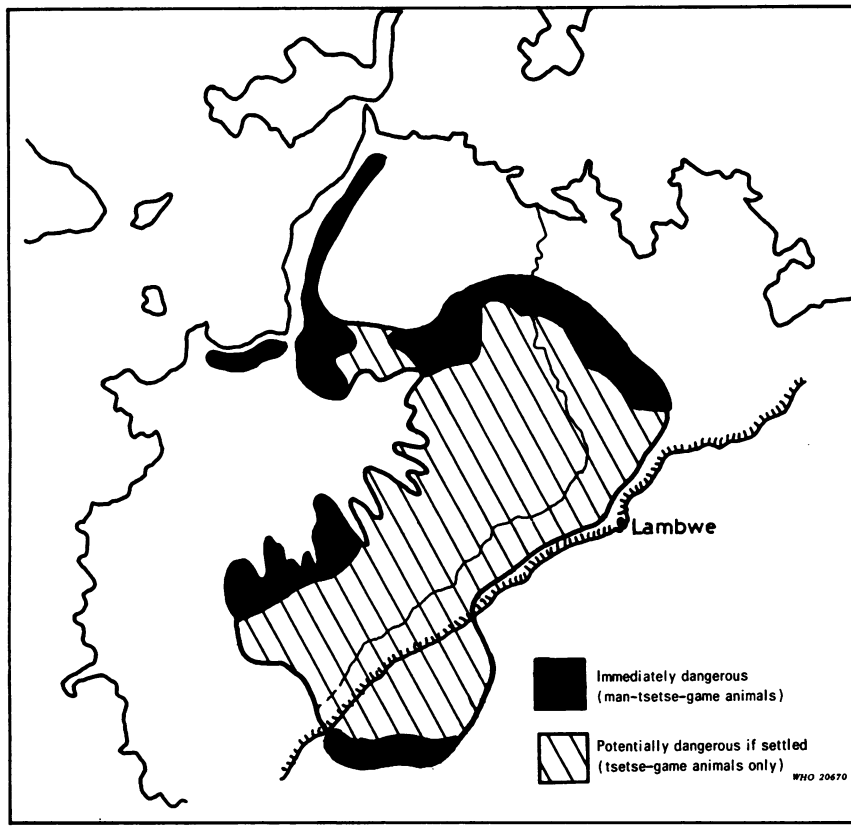

Fig. 7. Presumed distribution of primary and secondary receptive centres of sleeping sickness transmission in the Lambwe Valley area at the end of the 1950s.

was introduced (Fig. 7). Two types of potentially dangerous situation are recognized. In the first type (black areas in Fig. 7), human societies co-existed with heavy infestations of *G. pallidipes* and large populations of game animals (mainly bush-dwelling species). In the second type (shaded areas in Fig. 7), vector and reservoir elements were the same but there was no human population; these areas could therefore only become dangerous if there were human settlement or frequent passages. For convenience, these two types of situation are referred to as primary and secondary receptive areas, respectively.

A closer inspection of the data presented in Fig. 7 shows that there were probably 6 primary and 2 secondary centres.

Primary receptive centres:

(1) the western Gembe foothills in Kasingunga and Kaksingiri sublocations;

(2) the north-western coastal foothills of the Gwasi Range in Kaksingiri sublocation;

(3) the Roo Valley in Kaksingiri sublocation, contiguous with (4);

(4) the plains to the north and north-east of Ruma thicket, also in Kaksingiri sublocation;

(5) the eastern and south-eastern foothills of the Gwasi Range, largely inside Gwasi sublocation; and

(6) on top of the southern Kaniamwia Escarpment, in West Nyokal sublocation.

Secondary receptive centres:

(1) the centre of the geographical Lambwe Valley; and

(2) the area on top of the southern Kaniamwia Escarpment, in West Nyokal sublocation.

The importance of some of these areas in relation to the spread of *T. rhodesiense* infection is pointed out in subsequent sections of this report.

Table 2. Analysis, by location, of all cases of *T. rhodesiense* sleeping sickness recorded in the Lambwe Valley area between 1959 and 1970

Location	No. of cases (by year)												Totals
	1959	1960	1961	1962	1963	1964	1965	1966	1967	1968[a]	1969[a]	1970[a]	
Kaksingiri	1	2	3	8	7	22	16	15	39	8	4	3	128
Kasigunga	—	1	13	43	22	26	22	16	16	—	—	2	161
Rusinga	—	—	1	—	—	—	—	—	—	—	—	—	1
Gwasi	—	3	—	17	6	13	4	9	4	—	—	—	56
Lambwe Valley	—	—	1	6	8	28	35	31	35	14	8	5	171
Nyokal and Konyango	—	—	—	4	1	—	2	3	3	6 (1)[b]	4	1	25
totals	1	6	18	78	44	89	79	74	97	29	16	11	542

[a] WHO/UNDP Trypanosomiasis Project (Kenya) 2301 in operation.
[b] Case from the Gucha River area near Kadem, 20 km south of the nearest Nyokal focus.

RHODESIAN SLEEPING SICKNESS IN THE LAMBWE VALLEY AREA, 1959–71

The annual numbers of *T. rhodesiense* infections, diagnosed in the various sublocations during the period 1959–71 according to information provided by the Ministry of Health, Kenya, and Watson (1972) are shown in Table 2.

For the period 1959–67 the precise settlement locations of sleeping sickness cases are not known but it is possible to reconstruct the general course of events from the data presented in Table 2 and from the observations of scientists who visited the area during the period.

The introduction and spread of the disease between 1959 and 1963

The first case of Rhodesian sleeping sickness was diagnosed in 1959 from the Kaksingiri sublocation. No indication is given by Willett (1965) or Willett et al. (1965) [1] that the first patent was an immigrant, indicating that the infection had been acquired from an already infected *G. pallidipes* population.

If it is believed that *T. rhodesiense* and *T. gambiense* are variants of *T. brucei*, and that the terms " Rhodesian " and " Gambian " refer only to differences in the clinical manifestation of the disease (see, for example, Hoare, 1965), it could be argued that a virulent *rhodesiense* strain of *T. brucei* emerged by some selective mechanism (exerted by the vector or the game reservoir or both) from the area's existing pool of nonpathogenic *T. brucei* strains. However, in view of events in Central Nyanza and northern Tanzania, it seems much more probable that the disease was introduced from another disease focus. It should be stressed that the disease may have been introduced on more than one occasion and from more than one established focus.

In theory, the disease could have been introduced by man, tsetse, livestock, or game animals. Since the area is bounded on one side by Lake Victoria and on the other by settled land, it is unlikely that game animals were responsible. Alternatively, livestock, particularly cattle, could have introduced the infection because the 1959 Lambwe Valley Settlement Scheme undoubtedly resulted in an influx of domestic animals from Central Nyanza and possibly the Mara Districts of Kenya and northern Tanzania. Many of these animals may have been exposed to tsetse before they were moved. The distances between the Lambwe Valley *G. pallidipes* populations and those in Central Nyanza (Sakwa and Yimbo) and in the Mara region (Fig. 1) seem to be too great for the disease to have been introduced by infected *G. pallidipes* from one of those fly belts.

The most likely hypothesis is that the disease was brought into the Lambwe Valley area by man. Fishermen from Kasigunga and Kaksingiri, whose activities took them as far as south-eastern Uganda, could have been infected by lacustrine *G. pallidipes*

[1] See footnote 2 on p. 699.

populations if they went ashore at places such as Sakwa, Yimbo, Samia, and Busoga.

The 1959 Lambwe Valley Settlement Scheme evidently attracted settlers from all over Central Nyanza and from the south as far away as northern Tanzania. Any of these settlers coming from or passing through sleeping sickness foci could have carried the disease into the Lambwe Valley.

For many decades the Lambwe Valley, with its large population of game animals, had been a traditional hunting area for people from South Nyanza, Central Nyanza, and northern Tanzania. In those days, game conservation regulations were inadequate and undoubtedly people travelled regularly over great distances to hunt in the valley. Evidently, many of these people came from areas where the combination of an impoverished fauna with a fly population (*G. pallidipes* and in some cases *G. swynnertoni*) more closely associated with man had resulted in high levels of endemic Rhodesian sleeping sickness. Such people may have played an important part in the introduction of the disease.

Events in the Lambwe Valley area immediately after the introduction of *T. rhodesiense* will now be considered. If Table 2 is examined in conjunction with some of the accompanying figures, it becomes apparent that the disease quickly became established in the area to the west and south of the Gembe Hills. It is also apparent that the disease spread very rapidly into the Lambwe Valley and finally into the Nyokal/Konyango area. Willett (1965) reported that many cases were found in the Roo Valley in the very early stages of the establishment and spread of the disease. Presumably, the highest incidences of the disease were in this valley and along the Kaksingiri/Kasigunga lake shore, because thickets on the coastal plains from Sindo to the mouth of the Olambwe River were sprayed with dieldrin in 1961. However, there was very heavy rain at that time, and the insecticide deposits were rendered ineffective (Glover, 1962). After the detection of 17 cases of sleeping sickness in 1961, Glover (1962) reported as follows on the *G. pallidipes* situation in the Lambwe Valley. " Fortunately this infestation of *G. pallidipes* is isolated but cases of *T. rhodesiense* sleeping sickness are likely to continue to occur in that area." This hypothesis very quickly became a reality—probably much more quickly than Glover had expected—when the number of cases for the whole area quadrupled in the following year.

Reference to Fig. 7 shows that during these early years the main disease foci coincided with what have been described as primary receptive centres (1, 2, and 3). A contributing factor to the spread of the disease, which may have been very important, was the weather during that period.

In 1960, the short wet season, which usually lasts from October to December, failed; in the following year there was also a failure of the main wet season, which normally lasts from March to May. The resulting 12-month period of severe drought was then followed, not by the expected short wet season, but by sustained torrential rain, which resulted in extensive flooding in many of the lowland thicketed areas. In addition, there was an appreciable rise in the level of Lake Victoria, which, according to Glover (1962), completely inundated the spray lanes used by his control teams along the Kaksingiri/Kasingunga coastal plains. The effects of the two types of climatic extreme, i.e., severe drought and excessive rainfall, on the transmission of sleeping sickness are considered separately.

During the period of drought, *G. pallidipes* would have been concentrated in the densest of the blocks of continuous thicket, where the greatest concentration of game animals would have been found, particularly in the grasslands penetrating the edges of the thicket. It is difficult to determine to what extent man intruded into this close association between the vector and the natural reservoir of the disease; it is certain, however, that the longevity of *G. pallidipes* was greatly reduced as a direct results of sustained high saturation deficits and extreme diurnal variations in temperature, under both the macro- and micro-climatic conditions. The reduced lifespan of *G. pallidipes* would have had profound consequences with regard to the uptake, development, and subsequent transmission to man of infective trypanosomes.

During the period of excessively heavy rainfall late in 1961, the lifespan of *G. pallidipes* must have been greatly extended and the chances of flies becoming infected with *T. rhodesiense* considerably increased. Populations of both flies and game animals would have dispersed from the confines of the continuous thickets into more sparsely thicketed and settled areas. Therefore, man and domestic livestock must have been more frequently bitten by *G. pallidipes*, increasing the chances of *T. rhodesiense* being dispersed and introduced into new areas. A resumption of fish-trapping activities along the thicketed banks of the Olambwe River and its tributaries would also have brought man into closer contact with *G. pallidipes*.

Collectively these factors must have played an important role in expanding the transmission cycle, which led to the marked increase in the number of cases of sleeping sickness recorded in 1962, and to the appearance of the disease in the Lambwe Valley and Nyokal/Konyango sublocations between 1961 and 1962.

The further spread of the disease between 1964 and 1967

After an initial rapid increase in the number of cases of sleeping sickness during the period 1964–67 there was little variation in the level of disease transmission in most of the sublocations, e.g., Kasigunga, Gwasi, and Nyokal/Konyango (Table 2). The most notable events were the irregular rise in the number of cases in the Kaksingiri sublocation, largely in the Roo Valley (92 cases in the period), and the sudden sustained increase of the disease in the Lambwe Valley itself (129 cases in the period). The explanation for this probably lies in the fact that the largest primary receptive centres of the disease were located in these two adjoining areas (Fig. 7).

In other locations, which had more restricted primary receptive centres, contacts between man and tsetse were probably at such a level that the disease had reached its limit of expansion. This explanation appears to account for the levelling out of the number of cases recorded in the Lambwe Valley; however, it is difficult to believe. In all probability the creation of the Lambwe Valley Game Reserve in 1966 influenced the course of events. Although anti-poaching regulations were not enforced, few people were allowed to settle in the valley bottom. At the time, the local authorities responsible for the allocation of land undoubtedly made serious attempts (not alway successful) to settle newcomers in areas such as the Magunga and Wiga valleys, which are outside the boundary of the Game Reserve.

The disease between 1968 and 1971

During this period there was a marked decline in the incidence of Rhodesian sleeping sickness in all parts of the Lambwe Valley area and a complete disappearance of the disease from the Gwasi sublocation (Table 2). The distribution of cases in relation to the settlements, based on the data of Watson (1972), is shown in Fig. 8. Most of the cases came from within or near the primary receptive centres, but a few cases occurred far from *G. pallidipes* infestations. Clearly, the latter infections were contracted by people passing frequently through fly-infested areas or making frequent visits to them to poach game, collect firewood, fish, etc.

The very welcome decline in the incidence of sleeping sickness can probably be attributed to the activities of the WHO Trypanosomiasis Project, the Tsetse Survey and Control Division of the Kenya Ministry of Agriculture, and the Kenya Game Department. It is also possible that there may have been a natural decline in the prevalence of the disease during this period, but this cannot be known with certainty.

The WHO Trypanosomiasis Project. As part of the programme of research on aerial spraying techniques for the control and/or eradication of *G. pallidipes*, 4 *G. pallidipes*-infested thicket areas in the Lambwe Valley were sprayed from the air with dieldrin. Most of the thickets were at the northern end of the valley adjacent to the eastern Roo Valley and the Ruma primary receptive centres of sleeping sickness transmission (Table 3; Fig. 9). More than any other individual activity, the spraying of Ruma thicket is considered to have been the main factor in controlling sleeping sickness in the lower Lambwe Valley.

Activities of the Kenya Ministry of Agriculture Tsetse Survey and Control Division. During the 1968–69 period ground-spraying techniques were used to apply dieldrin to tsetse habitats in the northern end of the Lambwe Valley, i.e., part of the area left unsprayed by Glover (1962), and the eastern foothills of the Gembe Hills.

In May 1969, work began near Sindo on the hand-clearing of *G. pallidipes*-infested thickets in the Roo Valley. The area covered by this campaign was estimated to be about 57 km^2; the operation was completed late in 1970. Towards the end of 1970, some thicketed areas along the Kaksingiri and Luanda coastal plains were also sprayed from the ground with dieldrin.

Activities of the Kenya Game Department. Although the Lambwe Valley Game Reserve was gazetted in 1966, the by-laws regulating its management and development did not come into force until 1968 and enforcement of the by-laws by the Game Department through its Homa Bay administration did not really become effective until 1969–70.

During this period, Game Department rangers took up residence in the Lambwe Valley, and an Assistant Game Warden was given responsibility for the welfare of the Reserve. Efforts were immediately made to enforce anti-poaching regulations (contra-

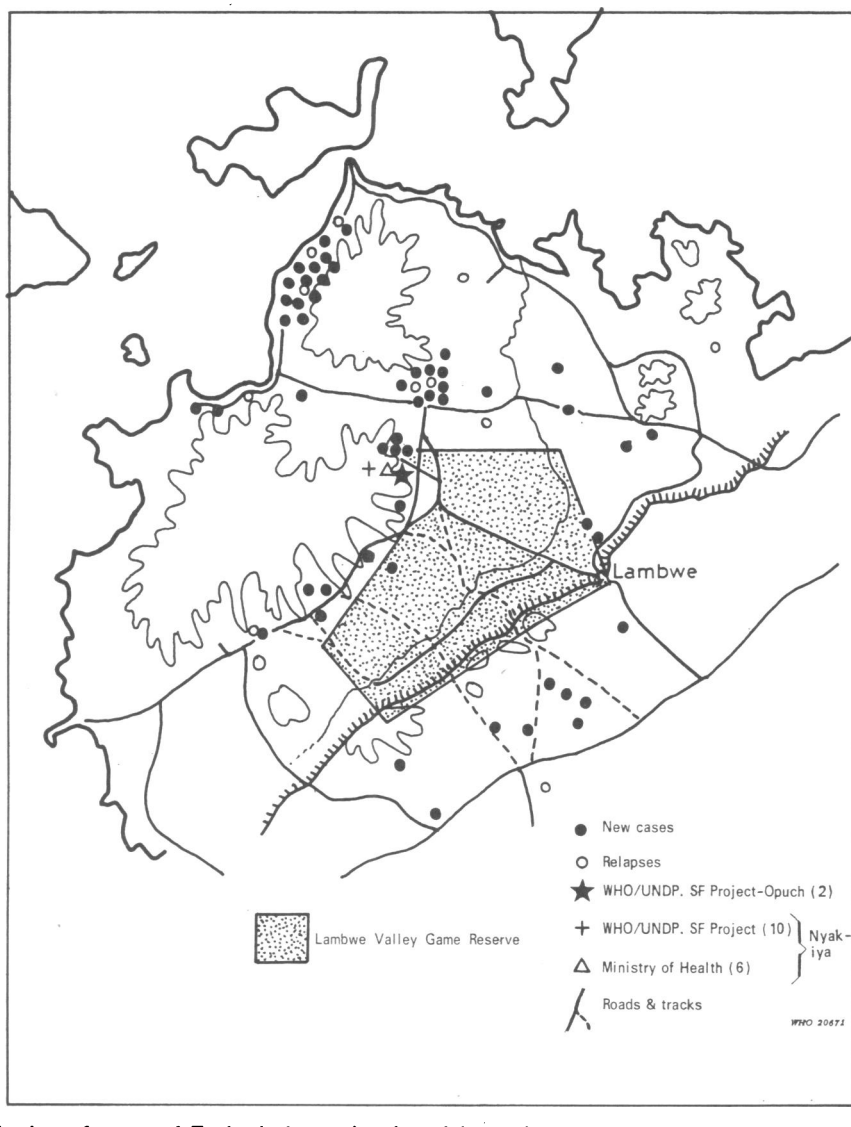

Fig. 8. Distribution of cases of *T. rhodesiense* sleeping sickness in the Lambwe Valley area in 1968–70 (based on Watson, 1972).

vention of which could be punished by a fine or imprisonment) including the collection and destruction of game-trapping devices, and the prevention of settlement in, and translocation of people from, the Game Reserve. These activities and also regular patrolling were greatly facilitated by the establishment of boundary tracks and by the efforts of the Trypanosomiasis Project to construct tracks within the Reserve that would be suitable for motor vehicles. These various activities had two effects on the sleeping sickness situation: the control or eradication of *G. pallidipes* in areas most seriously affected, and the reduction of contact between man and the remaining fly-infested areas.

Rickman & Robson (1970a, 1970b) described a simple test, the blood incubation infectivity test, for distinguishing *T. brucei* from *T. rhodesiense*. Without this test it would have been impossible to assess the

Table 3. Aerial spraying of dieldrin: operations in the Lambwe Valley, 1968–70

G. pallidipes-infested thickets	Month of spraying	Insecticide applied by:
Ruma	October 1968	helicopter
Ruma	January 1969	helicopter
Ruma	March 1969	helicopter
Masangala	January 1970	fixed-wing aircraft
Masangala	February 1970	fixed-wing aircraft
South Riamkanga	May 1970	fixed-wing aircraft
South Riamkanga	July 1970	fixed-wing aircraft
Nyaboro	September 1970	fixed-wing aircraft
Nyaboro	October 1970	fixed-wing aircraft

incidence of *T. rhodesiense* in livestock, game animals, and tsetse populations.

During the period under review, 56 cases of sleeping sickness were diagnosed in the Lambwe Valley area in a population of about 6 000 people (Watson, 1972). The incidence of the disease, based on parasitological diagnosis, was 0.48% in 1968, 0.26% in 1969, and 0.18% in 1970. However, serological examinations of the population indicated that the incidence was in fact higher (Binz, 1972).

The incidence of *T. rhodesiense* in game animals and livestock (Table 4) for the area as a whole tended to be higher than in the human population (Rickman, 1971). In areas such as the Rari Valley,

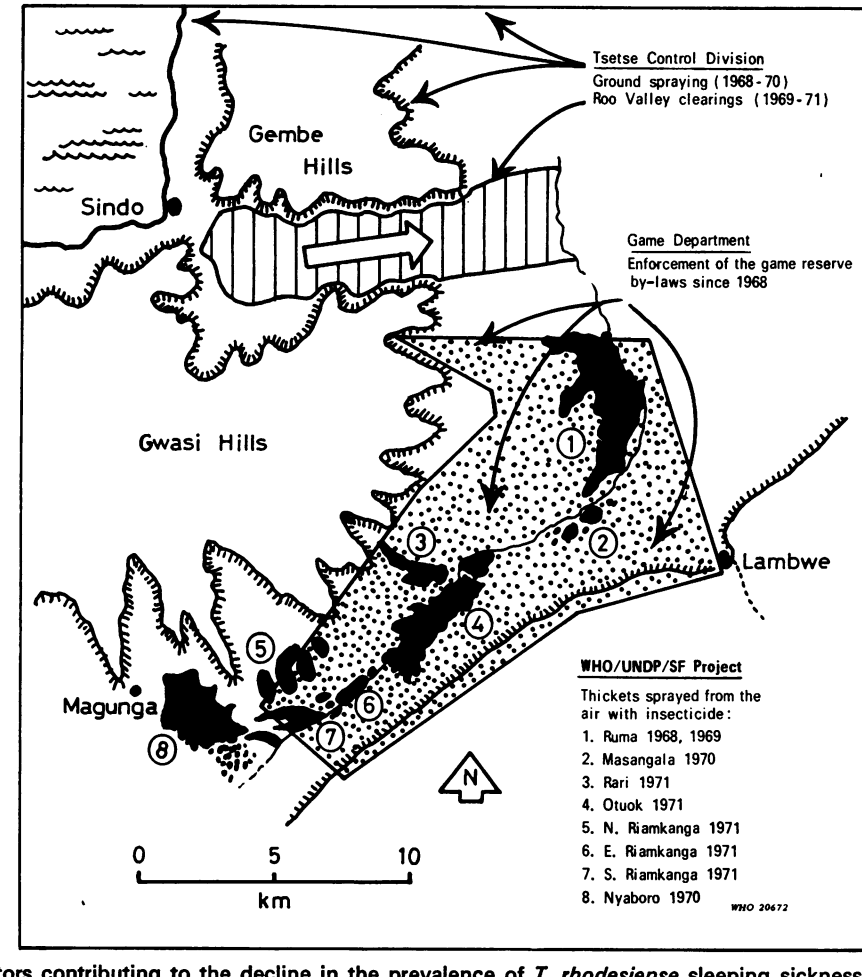

Fig. 9. Factors contributing to the decline in the prevalence of *T. rhodesiense* sleeping sickness in the Central Lambwe Valley in 1968–70.

Table 4. Incidence of *T. rhodesiense* in wild and domestic animals in the Lambwe Valley area, 1968-1970 (from Rickman, 1971)

Reservoir	No. examined	No. (and percentage) of animals infected	
		T. rhodesiense	Intermediate forms[a]
reedbuck	37	1 (2.7)	—
cattle	1 686	14 (0.8)	10 (0.6)
sheep	208	1 (0.5)	—
goats	636	—	1 (0.2)

[a] Intermediate between *T. brucei* and *T. rhodesiense* according to the blood incubation infectivity test.

where, over a period of months, there was increased contact between man, livestock, game animals, and *G. pallidipes*, the incidence of trypanosomes in game animals and livestock showed a corresponding increase (Rickman, *op. cit.*) (Table 5).

It is worth noting that one of the cases of sleeping sickness found in the Rari Valley in 1970 (Watson, 1972) was in a primary receptive focus and the other was in the main secondary receptive focus. Although by definition there was no human settlement in secondary receptive centres, these centres could become dangerous if, after the introduction of the pathogen, human settlement took place. In fact, in contravention of the Game Reserve by-laws, a small farming community settled inside the Reserve along the edge of the Rari thicket and the second case of sleeping sickness was found among these people. More cases would probably have occurred in this community if it had not been evicted from the Reserve by the Game Department.

Table 5. Incidence of *T. rhodesiense* in wild and domestic animals in the Rari Valley in April and in September 1970 (from Rickman, 1971)

Reservoir	Incidence of *T. rhodesiense* (%)	
	April	September
reedbuck	0	7.1
cattle	2.4	2.8
sheep	0	3.0
goats	0	7.1

The disease situation at the present time

During the period from 1 January to 30 April 1971, the Homa Bay Hospital examined 22 persons suspected of being infected with *T. rhodesiense*. Early in May 1971 no positive case had been diagnosed.

In February and March 1971, dieldrin invert emulsion was applied aerially to thickets infested with *G. pallidipes* in the Lambwe Valley that had not previously been sprayed—namely, Rari, Otuok, and North and East Riamkanga (Baldry et al., 1972) (Fig. 9). However, some recent events, to be described, indicate that the disease situation may deteriorate in the near future.

The recovery of G. pallidipes populations in dieldrin-sprayed thickets. Appreciable recoveries of the *G. pallidipes* populations in the Masangala, South Riamkanga, Nyaboro, and Ruma thickets were noted early in 1971. The populations inhabiting the latter two thickets are considered to be the most dangerous. The situation in the southern end of the Lambwe Valley could become dangerous not only to farming communities on the periphery of the Nyaboro thicket, but also to travellers who have converted an old aerial-spraying marker line across the middle of the thicket (see Baldry et al., 1972) into the main thoroughfare from areas south of the Lambwe Valley to Wiga and beyond.

It appears that the Ruma population of *G. pallidipes* has been assisted in its recovery by the influx of flies from the progressively shrinking Roo Valley thickets, the clearing of which was completed towards the end of 1970. Hillside thickets in the Gwasi foothills adjacent to the Ruma thicket remain a potential hazard, being within the dispersal of the Ruma *G. pallidipes* population, and doubtless having been invaded by flies from the displaced Roo Valley fly population.

The extension of G. pallidipes habitats. A comparison of serial photographs of the Lambwe Valley taken in the early 1960s and in the early 1970s indicates that there has generally been little change in the shape and size of the blocks of continuous thicket in the valley. However, some thickets, particularly Nyaboro and Rari, have been reduced in size, presumably as a result of the bush-clearing activities of local farmers.

It is disturbing that in several parts of the valley there have been considerable extensions of woodland and isolated thicket clumps. This development is particularly noticeable to the east of the Ruma and Masangala thickets, and to the north-west of the North Riamkanga thicket area. The encroachment of thicketed woodland could probably be checked by the correct use of bush fires (see Hopkins, 1966), but this is unlikely to occur as long as local people fire the bush regularly without producing a fire sufficiently fierce to destroy young thicketed and woodland vegetation.

Encroaching thicketed woodland is probably not suitable at present as a permanent habitat for *G. pallidipes*, although in parts of the valley it undoubtedly provides a very acceptable dispersal habitat. In the near future, permanent occupation by *G. pallidipes* is more than likely.

Settlements inside the Game Reserve. To the east of the Ruma and Masangala thickets there are still a number of occupied settlements inside the recently erected Game Reserve fence. The position of these people, who claim to be awaiting compensation for being translocated from established farms to newly allocated land outside the Reserve, is becoming progressively more dangerous. Their settlements are not only within the dispersal zone of *G. pallidipes*, but are also in one of the areas most affected by encroaching thicket and woodland.

In contravention of the by-laws, a number of new settlements have suddenly appeared at the foot of the Kaniamwia escarpment, to the east of the Masangala and Otuok thickets. When the Otuok *G. pallidipes* population recovers from the effects of a 1971 aerial spraying trial these illegal settlers will also be in a dangerous position.

A recent outbreak of sleeping sickness. On 17 May 1971, a farmer (J. T.) who lived in the Opuch–Nyakiya area of the Gwasi foothills at the north-east corner of the Game Reserve (Fig. 8), and who had contracted sleeping sickness in 1970, reported that he suspected that his son was suffering from the same disease. The next day, a wet-blood film prepared from the son (M. O.), a 30-year-old farmer from the same settlement, was found to be positive for *T. rhodesiense*. On the suggestion of J. T. that other people in the same area were probably suffering from the disease, 28 people who complained of being unwell were examined between 21 and 26 May; 9 of these people including 2 children (aged 4 and 10 years) and a man aged 70 years were found to be infected. Trypanosomes could not be detected by the examination of wet-blood films or stained thick and thin blood films from the remaining 18 people. These people were referred to Homa Bay Hospital, where 2 of them were subsequently found to be infected with *T. rhodesiense*. Early in June 1971, before tsetse-control operations commenced, a Ministry of Health survey team diagnosed a further 6 cases, bringing the total to 18.

At the end of May 1971, a sample of 194 *G. pallidipes* was collected, using the tsetse traps described by Langridge (1972), in farmland adjacent to residual thicket in the Opuch–Nyakiya area. All the flies were dissected by the method of Lloyd & Johnson (1924) and the trypanosome infection rate was found to be 13.4%, which was normal for that locality. Analysis of the infections showed that 18 were caused by the *T. vivax* group, 7 by the *T. congolense* group, and 1 by a member of the *T. brucei* group. The latter was identified by the blood incubation infectivity test of Rickman & Robson (1970) as *T. brucei*.

Most of the inhabitants of the area were reported to have arrived there since September 1970 from Central Nyanza District and northern Tanzania. The newness of houses, land clearance, and cultivation supported the reports.

This outbreak of sleeping sickness has been reported at some length because it illustrates two points. First, it shows that although there has been a marked decline in the prevalence of sleeping sickness in recent years, the situation could very rapidly deteriorate in some parts of the Lambwe Valley area. Second, it clearly demonstrates the value of comprehensive epidemiological mapping, illustrated in Fig. 7, for predicting where outbreaks of sleeping sickness may occur. In this case, settlement resulted in the conversion of the area concerned from a secondary receptive centre to a primary receptive one.

The present situation in the Lambwe Valley can be summarized as follows: Rhodesian sleeping sickness persists there at a low endemic level but it could become a more serious problem in the near future. Although the cycle of transmission of *T. rhodesiense* to man is quiescent, it can be assumed that some strains of this trypanosome have persisted in wild and domestic reservoirs and that these strains are now being introduced into *G. pallidipes* populations that are recovering from the effects of aerial spraying with insecticide.

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RÉSUMÉ

HISTORIQUE DE LA MALADIE DU SOMMEIL À *TRYPANOSOMA RHODESIENSE* DANS LA VALLÉE DE LA LAMBWE

L'auteur expose en premier lieu les principaux faits qui ont marqué la propagation de la trypanosomiase à *Trypanosoma rhodesiense* du district de Busoga (Ouganda) vers le district du Nyanza central (Kenya), et du nord de la Tanzanie vers le district de Mara (Kenya) avant que la maladie ne fasse son apparition dans la vallée de la Lambwe, dans le district du Nyanza du sud (Kenya), en 1959. C'est au cours de ce cheminement de l'affection vers le sud à travers le Nyanza central qu'ont été faites, depuis 1955, plusieurs découvertes épidémiologiques importantes comme l'isolement de *T. rhodesiense* à partir d'un céphalophe (*Tragelaphus scriptus*), du bétail et de *Glossina fuscipes*.

On évoque ensuite les conditions écologiques relatives à l'homme, aux animaux sauvages et à la mouche tsé-tsé qui existaient probablement au moment de l'apparition de *T. rhodesiense* dans la vallée de la Lambwe en 1959-60. On peut concevoir deux types de régions infestées par *G. pallidipes* mais différant par leur réceptivité à l'introduction et à la transmission de *T. rhodesiense*. Dans les unes, directement menacées, coexistaient *G. pallidipes*, des animaux sauvages et des groupements humains; l'introduction de *T. rhodesiense* y représentait un danger immédiat pour l'homme. Les autres, exposées à un risque « secondaire », hébergaient des populations de *G. pallidipes* et d'animaux sauvages; elles devenaient dangereuses si, après l'introduction de *T. rhodesiense*, l'homme s'y installait ou y faisait de fréquentes incursions.

On peut s'interroger sur les voies de pénétration de la trypanosomiase à *T. rhodesiense* dans la vallée de la Lambwe à partir des foyers voisins. Il est possible que l'infection ait été introduite par des pêcheurs ou par des fermiers immigrant avec leur bétail en provenance des districts de Busoga et du Nyanza central ou de la Tanzanie; cependant il semble plus probable qu'elle a été apportée par des groupes de population venant de l'un des foyers du Nyanza central. On décrit la progression de la maladie dans la vallée de la Lambwe en montrant l'importance du rôle joué par les régions à risque « primaire » dans son implantation et sa dissémination ultérieure. Le nombre des cas de maladie du sommeil diagnostiqués dans la vallée de la Lambwe était en 1959 et 1960 de 1 et 6; en 1961 et 1962, il atteignait 18 et 78. Cette augmentation rapide du taux de transmission est peut-être imputable en partie aux pluies très abondantes de la fin de 1961 qui ont fortement accru la durée de vie de *G. pallidipes* et les possibilités d'infection par *T. rhodesiense*. Peu après 1963, l'augmentation régulière du nombre des cas de trypanosomiase dans les vallées de la Lambwe et de la Roo est due vraisemblablement à une consolidation de l'emprise du parasite sur les populations de vecteurs et d'animaux sauvages dans ces régions. Par la suite, l'incidence de la maladie se maintient à un niveau relativement constant; ce fait est peut-être à mettre en rapport avec la création d'une Réserve dans la vallée de la Lambwe contribuant à réduire la fréquence des nouvelles occupations de terres dans la plaine où la densité des populations de *G. pallidipes* était la plus forte.

Entre 1968 et 1971 se marque une nette diminution de l'incidence de la trypanosomiase à *T. rhodesiense*, due principalement à la mise en œuvre d'une série de moyens de lutte: *a*) épandages de dieldrine, par voie aérienne, sur les habitats de *G. pallidipes* dans la vallée de la Lambwe; *b*) traitement au sol des habitats situés sur les rives du lac Victoria et débroussaillement de la vallée de la Roo; *c*) efforts du Kenya Game Department visant à limiter les activités de chasse et l'occupation de terres dans la Réserve et à expulser les occupants illégaux. Par leur association, ces mesures ont abouti à réduire les populations de *G. pallidipes* et à raréfier les contacts homme/tsé-tsé dans une bonne partie de la région.

Actuellement, dans la vallée de la Lambwe, l'endémicité de la maladie du sommeil à *T. rhodesiense* est faible. La situation ne doit cependant pas être considérée comme entièrement satisfaisante et elle pourrait se dégrader rapidement. On constate en effet que les populations de *G. pallidipes* se reconstituent dans les zones traitées par épandanges aériens d'insecticides; la végétation basse favorable à l'installation du vecteur empiète sur certaines régions de la vallée et menace de lui fournir de nouveaux habitats; nombre de terres de la Réserve proches de secteurs infestés par *G. pallidipes* sont occupées illégalement. On a assisté, en 1971, à une flambée soudaine de la maladie du sommeil dans l'un des nombreux foyers résiduels où persiste un danger virtuel d'épidémie.

L'auteur insiste sur la nécessité d'établir une carte épidémiologique aussi complète que possible des foyers de la maladie afin d'identifier les endroits les plus directement menacés.

REFERENCES

Allsopp, R. & Baldry D.A.T. (1972) *Bull. Wld Hlth Org.*, **47**, 691

Allsopp, R. et al. (1972) *Bull. Wld Hlth Org.*, **47**, 795

Ashcroft, M. T. (1959) *Ann. trop. Med. Parasit.*, **53**, 137

Binz, G. (1972) *Bull. Wld Hlth Org.*, **47**, 751

Buxton, P. A. (1955) *The natural history of tsetse flies*, London, Lewis, p. 816

Chorley, T. W. & Hopkins, G. H. E. (1942) *Proc. roy. ent. Soc. Lond. A*, **17**, 93

Fairbairn, H. (1948) *Trop. Dis. Bull.*, **45**, 1

Ford, J. (1963) *Bull. Wld Hlth Org.*, **28**, 653

Ford, J. (1971) *The role of the trypanosomiases in African ecology: a study of the tsetse-fly problem*, Oxford, Clarendon, p. 530

Glasgow, J. P. (1947) *Notes on the history of* G. pallidipes *in Samia* (quoted by Ford, 1971)

Glover, P. E. (1962). In: *Report of the 9th Meeting of the International Scientific Council for Trypanosomiasis Research*, Conakry, p. 253 (Commission for Technical Co-operation in Africa South of the Sahara, Publication No. 88)

Harley, J. M. B. (1965) *Bull. ent. Res.*, **56**, 141

Harley, J. M. B. (1966) *Bull. ent. Res.*, **56**, 595

Heisch, R. B. et al. (1958) *Brit. med. J.*, **2**, 1203

Hoare, C. A. (1965) In: *Discussion of* Willett, K. C. (1965)

Hopkins, B. (1966) *Forest and savannah: an introduction to tropical plant ecology with special reference to West Africa*, London, Heinemann, p. 100

Lamprey, H. F. et al. (1967) *E. Afr. Wildl. J.*, **5**, 151

Langridge, W. P. (1972) In: *Report of the 12th Meeting of the International Scientific Council for Trypanosomiasis Research*, Bangui (in press)

Lloyd, Ll. & Johnson, W. B. (1924) *Bull. ent. Res.*, **14**, 265

Mackichan, I. W. (1944) *Trans. roy. Soc. trop. Med. Hyg.*, **38**, 49

Moggridge, J. Y. (1948) *Proc. roy. ent. Soc. Lond. A*, **23**, 87

Onyango, R. J. et al. (1965) In: *Report of the East African Trypanosomiasis Organization, July 1963–December 1964*, Nairobi, East African Community, p. 54

Onyango, R. J. et al. (1966) *Trans. roy. Soc. trop. Med. Hyg.*, **60**, 175

Rickman, L. R. (1971) In: *Report of the 13th Meeting of the International Scientific Council for Trypanosomiasis Research*, Lagos, Organization of African Unity

Rickman, L. R. & Robson, J. (1970a) *Bull. Wld Hlth Org.*, **42**, 650

Rickman, L. R. & Robson, J. (1970b) *Bull. Wld Hlth Org.*, **42**, 911

Robertson, D. H. H. (1963) *Bull. Wld Hlth Org.*, **28**, 627

Robson, J. et al. (1972) *Bull. Wld Hlth Org.*, **46**, 765-770

Survey of Kenya (1962) *Atlas of Kenya*, Nairobi, Survey of Kenya, p. 46

Swynnerton, C. F. M. (1936) *The tsetse flies of East Africa, Trans. roy. ent. Soc. Lond.*, special supplement to volume No. 84, p. 579

Watson, H. J. C. (1972) *Bull. Wld Hlth Org.*, **47**, 719

Weitz, B. (1963) *Bull. Wld Hlth Org.*, **28**, 711

Wijers, D. J. B. (1969) *Trop. geogr. Med.*, **21**, 323

Willett, K. C. (1955) In: *East African Tsetse and Trypanosomiasis Research and Reclamation Organization* Annual Report 1954–55, Nairobi, East African High Commission, p. 31

Willett, K. C. (1965) *Trans. roy. Soc. trop. Med. Hyg.*, **59**, 374