Zusammenfassung: Schlafkrankheit in Kenia

Im tropischen Afrika ist die Trypanosomiasis (Schlafkrankheit beim Menschen, Naganaeuche beim Vieh) von hoher ökonomischer Bedeutung, hat diese Krankheit doch annähernd 10 Millionen km² und damit etwa ein Drittel des Kontinents mehr oder weniger stark in seiner wirtschaftlichen Entwicklung behindert. Am Beispiel von Südnyanza, Kenia, werden in der folgenden Studie die im Verlauf der vergangenen Jahrzehnte unternommenen Anstrengungen zur Eindämmung der Trypanosomiasis bei Menschen und Tier dokumentiert.

Nachdem die Schlafkrankheit zum erstenmal im Jahre 1902 im Untersuchungsgebiet ausgebrochen war, wurden zunächst Versuche unternommen, den Überträger der Krankheit, die Tsetsefliege, auszurotten und die infizierten Personen medizinisch zu betreuen. Der Lebensraum der Tsetsefliege wurde eingeengt durch die Rodung der Buschvegetation und die Anlage von Streifen offenen Landes um die Siedlungen. Diese Maßnahmen waren jedoch wenig erfolgreich, weil die geringe Bevölkerungsdichte und damit der Mangel an Arbeitskräften für die erforderlichen Rodungen ein Wiederaufkommen von Gehölzen begünstigte und wirksame Heilmittel fehlten.


Introduction

Tropical Africa’s dismal agricultural performance especially over the past two decades cannot be explained merely in terms of domestic policy constraints, low food crop pricing, and foreign exchange rates. Agricultural production is a complex also involving human-environmental circumstances in which disease is an important factor. An estimated one-third or about 10 million km² of the continent, an area capable of supporting over 100 million people, now lies unused or underused because of trypanosomiasis or sleeping sickness and its tsetse vector (Fig. 1).

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In Africa there occurs both human trypanosomiasis, sometimes called African sleeping sickness, and livestock trypanosomiasis or nagana. Kenya is one African country with both human and animal trypanosomiasis. Although human sleeping sickness may exist in an endemic form in parts of East Africa, only in South Nyanza and sections of Busia and Siaya districts in the Lake Victoria basin is the disease known to occur actively. Tsetse distribution roughly covers one-quarter of Kenya's land area. Despite a
history of fighting the disease and tsetse spanning several decades, the war has yet to be won. This is probably because of the very complicated sleeping sickness epidemiology which involves not only the disease germs (trypanosomes) and the tsetse vector but also vegetation, soils, and human ecology (Ford 1971).

The purpose of this paper is to reconstruct carefully the history of tsetse and trypanosomiasis in the South Nyanza district of Kenya. The paper explains how measures to fight tsetse and the disease led to almost complete control of one species of tsetse, *G. palpalis* in the riverine areas of the district while unwittingly leading to the expansion of the savanna *G. pallidipes*. This reconstruction is necessary because trypanosomiasis and its insect vector persist especially in the Lambwe valley area of the district despite repeated efforts at control. The South Nyanza case will add local detail to our knowledge of trypanosomiasis and provide pointers to the management of a significant African problem.

**Background**

Up to about 1960 South Nyanza patients were diagnosed to suffer from the chronic form of the disease (Gambian sleeping sickness) (Morris 1960, Willett 1965). Since then, the cases of the disease reported appear to be mainly those of Rhodesian or acute sleeping sickness. Associated with this change in the form of sleeping sickness were changes in the tsetse vectors involved. Until the late 1950s, the riverine and lakeshore *G. palpalis* species, the main vectors of Gambian sleeping sickness were predominant. Control measures employed up to this period appear to have successfully reduced *G. palpalis*. *G. pallidipes* proved elusive. In fact *G. pallidipes*, the major vector of Rhodesian sleeping sickness, steadily expanded in territory and by 1960 appears to have replaced *G. palpalis* as the major vector of the disease in South Nyanza (Morris 1960, Willett 1965). *G. pallidipes* which also is a vector of nagana is reported to have been confined to a small area between Gembe and Gwassi hills at the beginning of this century (Ford 1971: 270). Its expansion in later years may be attributed to the increase in suitable habitat for that species of tsetse. Below, I turn to the details of these changes in disease and tsetse and consider government response.

**Early period, 1900 – World War II**

South Nyanza district which currently has a land area of 5714 km² and counted 817600 people in the 1979 Kenya national census, has a history of sleeping sickness going back several decades. The origins of the disease in the district have been documented (Morris 1960). It appears that the disease was carried from Uganda by fishermen and traders operating on Lake Victoria. By 1902 *Trypanosoma gambiense* infection was well established in South Nyanza. Its vector was the *G. palpalis* group, tsetse which appear to have been prevalent along the Lake Victoria coastline and the river systems in the area (Ford 1971, Morris 1960). The disease had developed into an epidemic by 1906 and was killing many people in a wide area along the lakeshore up to the Sondu river, lower and middle Gucha valley and the Migor river valley (Nyanza Provincial Annual Report 1905–6, cited in Morris 1960).

Perhaps before discussing the reactions both of the local people and government to sleeping sickness infestation, it is appropriate to add something more about the vector which made possible the transmission of the disease. Tsetse appear to have a long history in South Nyanza. Local legend has it that tsetse first appeared in the areas of Ruri mountains and Kakingri at about 1860. It is said that tsetse originated in the northern shores of Lake Victoria and was brought to South Nyanza by “swimming” elephants (Anonymous 1935). But Ford (1971) has claimed that the presence of forest and bush tsetse such as *G. fuscipennis* and *G. brevipalpis* appears to go back to the Pleistocene period. He notes that up to the end of the nineteenth century these two species of tsetse plus *G. pallidipes* were restricted to isolated forests and thickets around human settlements (Ford 1971: 270). Here, such wild animals as bushpig, buffalo, hippopotamus and so on served as food sources. According to Ford, only the riverine and lakeshore *G. palpalis* group came into contact with human beings. This would happen at river fords or when collecting water for domestic purposes and taking livestock to water.

Ford (1971: 272–273) notes that *G. pallidipes* which was previously restricted began to extend into a larger area. This was encouraged by the increase in suitable

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1) Place names referred to in the text may be found in Fig. 2.
habitat for that fly. It is to be recalled that the initial sleeping sickness epidemic led to the depopulation of a wide area. Survivors moved away as well. This made it difficult to keep bush and wildlife at bay any more. Consequently “G. pallidipes that, hitherto, had been confined to limited zones in forest or thicket patches above the level of human occupation”, began to spread (Ford 1971: 270).

The outbreak of sleeping sickness in South Nyanza in 1902 drew attention of the colonial authorities. The British government sent a Royal commission on sleeping sickness to East Africa during the same year to investigate the extent of the problem. Dr. Christy of the team reported the disease to be widespread on the eastern shores of Lake Victoria. This report led to the establishment in 1904 of rules under the Infectious Diseases Ordinance to prohibit the eastward movement of people from infected areas of Nyanza province (Medical Research Laboratory 1943, hereinafter MRL). There was however no way of enforcing the rules and the problem continued.

The British government, worried about the disease, entered into a protocol with the Germans who now occupied Tanganyika to fight sleeping sickness. The ensuing Anglo-German Sleeping Sickness Agreement of 1909 was meant to stop population movements across the Kenya-Tanganyika border (MRL 1943), part of which was South Nyanza district.

Another government response to the initial epidemic of sleeping sickness in the area was the establishment, in 1909, of an isolation and treatment camp at Kanyamkago under a medical officer. This camp was located in “one of the worst infected areas” in South Nyanza (District Commissioner, South Kavirondo 1911, hereinafter DCSK) \(^{33}\). There are conflicting reports on the number of local people who turned up for treatment at the camp. Some reports claim that a large number were treated (Anonymous 1935: 2) while others note that people were reluctant to submit to treatment (DCSK 1911: 18). During the first half of 1909, some 260 cases were seen by the medical officer (MRL 1943).

If the South Nyanza residents were reluctant to seek medical help, it was because the treatment was “admittedly ineffectual” (DCSK 1911: 18). The provincial commissioner rightly noted that “in the absence of a cure for the disease the natives exhibit no particular interest in our efforts” (Provincial Commissioner, Nyanza 1912: 28, hereinafter PC). Lack of patients seeking treatment led to the permanent closure of the camp in March 1912 (DCSK 1912: 19).

Without a medical solution to the sleeping sickness menace, people took the only option open to them – abandoning their farms and moving to uninstructed areas. Such emigration especially from Gwassi and Lambwe valley led to the development of bush and encouraged farther advance of tsetse (Anonymous 1935: 1, 4). Further, the presence of wild animals provided a food source for tsetse and trypanosomases. One early resident observed the changes taking place in the Lambwe valley as follows: “In a matter of a decade ... the drying up of the Valley combined with the spread of the tsetse fly Glossina pallidipes, has changed the valley from an oasis for cattle into a veritable death trap. Where once there were thousands of cattle today there might be a few hundred at the most” (Blunt, 1935: 3).

Thus the first sleeping sickness epidemic which lasted up to 1912 inflicted a heavy toll on the local population both in human and livestock losses. For the next decade, sleeping sickness remained endemic in South Nyanza except for occasional isolated outbreaks.

In the period between 1920 and 1930, a number of government surveys were conducted in South Nyanza to determine the extent of sleeping sickness. Cases of the disease were found in Homa and Gwassi areas and along the Miriu and Gucha rivers. These surveys led to various suggestions on control. These included selective bush clearing, especially at watering places and river crossings, evacuation of people from infected areas, control of fishing and canoe traffic, and the creation of a treatment camp at Kendu Bay (MRL 1943).

Some of these recommendations were effected from the beginning of the 1930s. The medical headquarters in Nairobi decided on bush clearing along the Gucha river at certain fords and in Lambwe valley (DCSK 1932). Clearing was to combine the block system and barrier clearings. The latter involved clearing bush to isolate tsetse-infested areas from human settlements. The aim was to break tsetse-livestock and tsetse-human contact. Tsetse could then be reduced in the isolated blocks by trapping and hand-catching.

It had been planned that cleared areas should be resettled in order to keep down the bush. This plan followed the experiments on tsetse eradication in Shinyanga, Tanganyika. From the very beginning the clearing programme was destined to fail. The major problems included lack of labour, unavailability of tools, and insufficient supervision (DCSK 1932). The tsetse eradication work was slow and costly. However

\(^{33}\) South Kavirondo district later changed its name into South Nyanza. Until 1961, South Nyanza district consisted of the present district by that name and Kisii district.
tsetse control became the “biggest single measure of public health . . . in the district” (DCSK 1938: 33). By the end of 1940, about 518 km² of land previously unsettled had been cleared of *G. palpalis* and opened up for settlement for several families (MRL 1943). But the inability to obtain labour on a continuous basis meant that bush could regenerate and tsetse could reinfest. Further, as it is now well known, tsetse are quite adaptable to new environments provided that animal hosts are present. Consequently, people moved their homes and livestock to even uncleared areas which had earlier been abandoned because of tsetse (DCSK 1939). This population movement to tsetse-infested areas was responsible for isolated epidemics in the area in the 1930s.

By the beginning of World War II, three schemes dealing with sleeping sickness were operational (Fig. 2). The methods used in each of them were aimed at eradicating tsetse and through it the disease. The first was the Gucha river which was supervised by the medical department. In 1939, the Gucha scheme focused at *G. palpalis* leaving *G. pallidipes* alone...
Bush clearing continued until the end of the war. A second project was on the Oyani river, a tributary of the Gucha. The Oyani scheme received financing from the Local Native Council and was later joined up with the major Gucha project (Gordon 1946). The Oyani scheme successfully reclaimed land through bush clearing. The third scheme was the Lambwe valley. This tsetse eradication project was closed down at the beginning of the war when the officer in charge was recalled for war duties and funds from the Colonial Development Fund were discontinued.

It would appear that government prohibition of Africans from killing wild animals, the food source especially for the so-called savanna tsetse, contributed to the inability to reduce or control sleeping sickness. The DC noted a “surprising large amount of game (sic) of all sorts in parts of the district in which human habitation is scarce on account of tsetse fly infestation”. He named those areas to be the lower Gucha river and its major tributaries of Migori and Oyani, Lambwe valley and Kasigunga (DCSK 1939: 39; Fig. 2).

During the war years, some bush clearing went on. Cattle began to be inoculated with Phenanthrindinium. This drug became popular despite its high cost of 75 cents a dose. It nonetheless offered no immunity (PC 1945: 18) and cattle exposed to the trypanosomiasis risk could once again contract the infection.

In the early 1940s, human sleeping sickness was building up to an epidemic level especially in the river Gucha area. This was particularly serious in view of the fact that the government did not have a trained tsetse person in the field at the time to check infection (Director of Medical Services 1941). In addition, no work had been done against G. palpalis in the lower Gucha where there was a considerable amount of Gambian sleeping sickness among the residents (Bax 1943). Sleeping sickness incidence increased from 78 cases in 1943 to 1012 cases treated and diagnosed in 1944 (PC 1944: 9).

The epidemic appears to have been caused by frequent human-tsetse contact. People moved to disease-free areas at the outbreak of an epidemic but soon returned to former homes when the disease had subsided. They would go around with a certain level of infection enough only to keep the disease in an endemic form. Once the people returned to tsetse-infested areas, repeated fly bites added to the level of parasitemia in human blood to lead to a subsequent epidemic (Morris 1960, Moloo et. al. 1970). Moreover, a person infected with the chronic T. gambiense could stay for a long time before the disease symptoms show up.

By 1945, the scourge of sleeping sickness remained as it had been during the first epidemic. The provincial administration in Nyanza called for funds which might aid in instituting a more efficient sleeping sickness control service in the future (PC 1945). But why did the failure occur after so much effort? It might be noted that the methods of tsetse control through bush clearing and land reclamation were borrowed from the Tanganyika tsetse research department – dubbed the Shinyanga people. The research at Shinyanga emphasized “land reclamation on the grand scale” and was “of little immediate benefit to the medical and veterinary people” (Chief Secretary, East African Governors 1942).

Post-World War II period, 1946-1960

At the end of World War II, both human and animal trypanosomiasis remained as insuperable problems in South Nyanza. Tsetse were widespread. The disease was raging. This period saw an intensification of efforts to deal both with the disease and its vector. However, the methods remained almost unchanged. The medical department continued with its work of treating patients once funds and personnel permitted. The bush clearing and tsetse-catching schemes were carried out by the East African Tsetse Research department. The veterinary department’s postwar agenda of fighting sleeping sickness was based on tsetse control. The department set to conduct surveys on tsetse and trypanosomiasis, reclaim currently tsetse-occupied areas, and conduct more research into the behaviour of tsetse and trypanosomes (Director of Veterinary Services 1943).

Dispensaries for treating sleeping sickness were established near Kabwoch forest, Ndhia, and a temporary one in Kadem. As the data on Table 1 show, there was a considerable number of sleeping sickness cases in the post-World War II decade. In the Lambwe valley area alone, a survey of all villages showed a 10 percent incidence of sleeping sickness (District Commissioner, South Nyanza 1950, hereinafter DCSN). But a large number of patients did not go for treatment despite propaganda campaigns from local chiefs (DCSK 1948: 21). Sleeping sickness now consisted of 3 antrypol injections on alternate days followed by 12 bi-weekly or weekly injections of tryparsamide (DCSK 1949: 33). Towards the late 1950s the record shows a reduced incidence of sleeping sickness. Was this a result of more effective control measures or was
it because of under-reporting? I will return to this issue later in the paper.

Animal trypanosomiasis was widely prevalent in South Nyanza during this period. Veterinary treatment against nagana was done with innoculations of Dimidium bromide. In 1950, 16,000 cattle were treated (DCSN 1950). The number of cattle inoculated in the following year remained nearly the same (DCSN 1951: 12). However, the disease was becoming increasingly resistant to the drug and cattle had to be given double doses (DCSN 1951: 12, DCSN 1954: 17). In many areas of the district, cattle could be kept only by continual innoculations with dimidium (DCSN 1952: 14).

Apart from the treatment measures outlined above which were aimed at the disease germs, the indirect method of vector control through the destruction of favourable habitat and the direct methods of tsetse trapping and hand-catching were all in operation. Post-War II measures to fight trypanosomiasis via tsetse really focused on eliminating *G. palpalis* in the River Gucha basin, and along the lakeshore and fighting *G. pallidipes* in the Lambwe valley area (Fig. 2). It may be noted that four species of tsetse occurred in South Nyanza - *G. palpalis*, *G. pallidipes*, *G. brevipalpis* and *G. fusca*pleuris. However, the latter two were restricted and of minor economic significance compared with the other two (Wilson 1952).

The discussion below inevitably emphasizes *G. palpalis* and *G. pallidipes*.

**Control campaign against *G. palpalis***

The East African Tsetse and Trypanosomiasis Research and Reclamation Organization (EATTRO) which was formed in 1948 to coordinate sleeping sickness control in East Africa planned that a scheme be commenced to rid the lakeshore, Gucha river delta, lower Gucha and Migori rivers of *G. palpalis* (Fig. 2). The use of insecticidal spraying as a method of tsetse control was started in 1955. Bush clearing continued as before. The veterinary department wanted to isolate tsetse breeding areas by clearing and then eradicate tsetse between the cleared areas by spraying (DCSN 1955: 21). This programme eliminated tsetse from 2590 km² of the district at a cost of £50,000 (DCSN 1959: 34).

In 1959 the local government voted funds to eradicate *G. palpalis* from the entire coastline of South Nyanza (DCSN 1958: 29). Spraying with 2 percent Dieldrex was done a number of times in the following year along the Sare river, and along the lakeshore between Nyandiwa and Sindó (DCSN 1960). It appears that by 1960 the Gucha river system was largely free from *G. palpalis* (DCSN 1960: 26–27). But a scatter of these flies and *G. fusca*ipes remained along the lakeshore.

The combined campaigns of bush clearing, tsetse hand-catching and trapping, and spraying reduced *G. palpalis*. And together with medical treatment, these measures affected the number of sleeping sickness cases reported in the late 1950s (Table 1). In contrast, it will be shown below that *G. pallidipes* heavily infested the locations of Gwassi, Kaskingirí and Kasiguná during this period. What follows is a discussion of the Lambwe valley scheme to reveal the difficulties of eliminating tsetse and sleeping sickness from this part South Nyanza. This is the area where most of the sleeping sickness cases come from today.

**Table 1: South Nyanza: new cases of human sleeping sickness notified, 1946–1959**

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1946</td>
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</tr>
<tr>
<td>1947</td>
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<td>1956</td>
<td>16</td>
</tr>
<tr>
<td>1959</td>
<td>14</td>
</tr>
</tbody>
</table>

* Most of these cases came from the Gucha river basin


**The Lambwe valley tsetse eradication scheme**

During the years 1949 and 1950 several attempts were made to reduce the tsetse population by eliminating wildlife from Lambwe (Tsetse Fly and Trypanosomiasis Committee, no date, hereinafter Tsetse). This is because effective transmission of Rhodesian sleeping sickness requires human-tsetse-wildlife contact. There were large herds especially of buffalo, elephant, topí, impala, hartebeest and roan antelope. Blunt estimated 500 elephant, 300 topí, 200 impala, 150 hartebeest, 100 roan antelope, 75 reedbuck, 70 waterbuck, and 40 buffalo in the Lambwe valley in
1935 (Blunt 1935; see also Tsetse 1949). The programme to remove wild animals was based on the experience at Shinyanga where the elimination of wildlife appeared to considerably reduce tsetse population (Tsetse 1949). Wildlife extermination was a preliminary step in the efforts to set up a resettlement scheme. This plan was not carried out. Although the failure was attributed to lack of manpower (DCSK 1949: 20), the unwillingness of the government to decimate wildlife was certainly a contributing factor.

In 1949, EATTRRO, after field investigations in Lambwe submitted a proposal for a major scheme for eradicating tsetse by progressive bush clearing and human settlement. EATTRRO’s ultimate aim was not resettlement per se; it was to conduct experiments to find the best means of reclaiming land from tsetse on a large scale (Tsetse 1948). It was hoped that the resettling of people into Lambwe valley, an area of 518 km² would lead to bush clearing and the elimination of tsetse (PC 1950: 17). The settlement programme was predicated on the belief that several locations of South Nyanza such as Karachuonyo, Kanyada, and North Nyokal were suffering from population pressure (DCSN 1950: 16) and that there would be no shortage of settlers to see the tsetse eradication programme through.

Work on Lambwe valley reclamation project would be done in two phases. The first one involved the area outside the valley itself. This was divided into four blocks – Samunyi, Komato, Achol, and Nyamaji (Fig. 3). Resettlement would begin with Samunyi and end with Nyamaji. The second phase of the project would start after tsetse had been eradicated in the phase one areas. Phase II would involve the settling of Lambwe valley itself containing Ruma bush, the centre of tsetse infestation in the area (Fig. 3; South Nyanza Gazetteer 1943–1955).

In order to make the area attractive for settlement, water and other amenities such as roads were to be supplied. Other incentives included establishing a shop, a school and a plan to plough about one hectare free of charge for each settler. The land was subdivided into plots and each family was allocated 20 hectares. Plot owners were required to clear bush. Initially, settlers were not allowed to take cattle into the settlement area ostensibly because of tsetse and nagana.

A barrier clearing planned in the western part and another one from Sikiri to Sokolo were completed in the mid-1950s (Fig. 3). These were intended to protect the settlement blocks from tsetse and make it possible to keep cattle. In fact, EATTRRO had by 1950 completed a protective barrier of about 800 hectares separating the area earmarked for settlement...
from the remainder of Lambwe valley (Director EATTRO 1952). Although EATTRO’s clearing scheme almost always kept ahead of settlement, there were problems of getting free communal labour to do the job.

Settlement started with Samunyi, the eastern arm of Lambwe. Some clearing was done beginning in 1950 by residents of neighbouring locations. By 1953 there were 320 settlers in residence mainly from East Konyango and Kasigunga (PC 1953, South Nyanza Gazetteer 1943–1955).

One problem which plagued the Lambwe reclamation scheme from the very beginning was the difficulty of getting settlers. This was partly because of the ban on cattle (PC 1951: 14). It became necessary to reduce the acreage per family from 20 to 10 hectares in 1955. But with an estimated number of 930 plots covering an area of 12700 hectares in that year, there were only 677 settlers (South Nyanza Gazetteer 1943–1955). After the mid-1950s it was clear that the Lambwe tsetse project was making little progress. The number of settlers decreased from 677 to 620. The lack of population pressure and several stringent rules (for example settlers were allowed only 10 head of cattle per family from 1955 onwards) discouraged potential settlers. Also, the few cattle which were on the scheme suffered considerably from trypanosomiasis (DCSN 1957).

Towards the end of the decade the Lambwe Valley scheme appeared a complete failure. Discussions to encourage people from other districts especially the Maragoli and the Luo from Siaya and Kisumu did not lead to any action. The success of the Lambwe project was dependent on a high level of settlement. This did not happen. In 1958 there were only 450 settlers meaning that more than 200 people had returned to their original homes in the previous three years after a brief sojourn in Lambwe (DCSN 1958).

Yet conflicts within government on the actual purpose of the scheme contributed partly to its failure. As shown above the Lambwe scheme was the brainchild of EATTRO. It was intended to be a tsetse reclamation project. Settlers were meant to keep bush from regenerating. The agriculture department however conceived the scheme as a settlement programme for improved agriculture and good husbandry. It argued that the government could not spend large sums of money on rehabilitating land for traditional land use. On their part, EATTRO wanted settlement to follow traditional land use patterns and practices.

Evaluated as an agricultural settlement, Lambwe had a number of limiting factors outside tsetse. Lack of sufficient water, seasonal waterlogging and poor soils hindered cultivation. In many parts of the valley, livestock raising was possible if and when permanent clean water could be provided. Also, the areas earmarked for bush clearing and resettlement in the phase one programme did not represent the typical physical conditions of Lambwe valley. Samunyi, for example, was fertile country suitable for intensive cultivation (see for example, Pasture Research Officer 1951). Planning for its settlement required to take a different line.

At the end of the 1950s, the Lambwe scheme was a white elephant. The scheme failed both as a tsetse reclamation project and as an agricultural improvement experiment. Large areas both in the valley, Gwassi, West Nyokal and parts of the Lake Victoria coastline had sleeping sickness. A number of locations which adjoined Lambwe, Gwassi, Kaksingri, and Kasigunga, also continued to have bushes which had always attracted a heavy infestation of tsetse.

Sleeping sickness in recent time, 1961–1986

The foregoing analysis has shown that cases of Gambian sleeping sickness especially in the Gucha river basin and parts of the lakeshore had gradually gone down by the end of the 1950s. This was achieved through a sustained fight against, and drastic reduction in, G. palpalis. In contrast, the picture in the non-riverine areas of South Nyanza and especially Lambwe valley and its environs was quite different. It was noted earlier that G. pallidipes actually expanded in territory particularly in the World War II period. This was partly a result of ineffective control measures.

The increase in the incidence of G. pallidipes led to regular human-tsetse contact and escalated Rhodesian sleeping sickness by 1960 (Glover 1962 cited in Willett 1965). Consequently, the Lambwe valley and the surrounding areas of Gwassi, Kaksingri, and Gembe became important foci for the Rhodesian disease. This led to serious outbreaks of sleeping sickness in the area between 1962 and 1964 (Willett 1965: 375) and in later years. Although the 1962–1964 epidemic was soon controlled by insecticidal spraying, bush clearing and treatment, this did not last for long. There was a widespread outbreak of the disease during 1966/1967 (DCSN, Kiri 1970: 28). In 1966 alone, 97 cases of sleeping sickness were diagnosed and treated in the district.

Before explaining the control measures employed to quell the outbreak, a number of conditions which
enhanced the survivability and multiplicity both of the disease germs and the tsetse vector must be explained. In early 1966, the South Nyanza county council gazetted an area of about 109 km² in the Lambwe valley as a game reserve (DCSN, Kirui 1970). The object was to preserve the area’s natural habitats for future generations. Lambwe had been home of many wildlife species including the rare roan antelope for a long time. The county council planned also to introduce other animals into the reserve once the boundary had been established and fencing installed. For the future, the national reserve would pose difficulties of eliminating sleeping sickness.

Tsetse sustenance and expansion in Lambwe was also helped by the establishment, in 1965, of Kanyamwa escarpment forest. This forest was, in later years, to become heavily infested with G. pallidipes (Challier a. Turner, no date). Kanyamwa forest covers an area of about 5 km² and contains the two tree species Pinus patula and Cupressus pinus. Tsetse were apparently attracted to Kanyamwa by an initial protective hedge of Euphorbia tirucalli (Challier a. Turner, n.d.). In subsequent years, indigenous scrub grew in the area further enhancing the ecological suitability for G. pallidipes.

Following the 1966–1967 epidemic in Lambwe area there was a mass campaign by the United Nations, and the Kenya government Ministries of Agriculture and Health to eradicate tsetse. Experts from the World Health Organization, Food and Agriculture Organization, and the Kenya veterinary department arrived in the area in 1967 to mount the campaign (DCSN Kirui 1970, Willett 1972). Their work was a combination of spraying, cattle treatment, and bush clearing. The Lambwe valley area including Sikiri peninsula, Gembe, Kaskingri and Gwassi locations (but excluding the game reserve) were sprayed.

Patrol bases were established in the Lambwe valley and at Sindo and Lwanda (Kirui 1970: 28). The WHO and FAO team conducted experiments on aerial spraying by helicopter over the game reserve. Bush clearing and spraying helped reduce the tsetse menace but at a cost, and only for a time. For three years up to 1970, 86,000 head of cattle had been treated with Berenil and Ethidium bromide (Kirui 1970, DCSN 1968, 1970).

The work of the WHO and FAO experts and the veterinary department continued until early 1971. Its short term effect of tsetse reduction made one over-

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**Table 2: Human sleeping sickness in South Nyanza: positive cases according to location of residence, 1967–1984**

<table>
<thead>
<tr>
<th>Year</th>
<th>Total cases</th>
<th>Kaskingiri</th>
<th>Gembe</th>
<th>Gwassi</th>
<th>Lambwe</th>
<th>W. Nyokal</th>
<th>Kanyamwa</th>
<th>Others</th>
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**Totals**

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<th>545</th>
<th>105</th>
<th>22</th>
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<th>115</th>
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<td>In per cent</td>
<td>100.0</td>
<td>19.3</td>
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<td>39.1</td>
<td>21.1</td>
<td>2.9</td>
<td>3.7</td>
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**Source:** These data are worked from the records of the Division of Vector-Borne Diseases of the Homa Bay District Hospital.
zealous DC to believe that “with the elimination of tsetse fly it can be said with certainty that no other significant cause of disease is left in the valley” (DCSN 1973: 43). Despite this assertion, sleeping sickness persisted in the district (Table 2).

Distribution of infection

Table 2 shows the source areas within South Nyanza of the positive cases of sleeping sickness diagnosed between 1967 and 1984. It reveals a reduction in the number of cases identified from Kaksingri and Gembe locations while Gwassi shows a dramatic increase. This appears to have been a result of considerable tsetse reduction in the first two locations by the efforts of the WHO, FAO, and the veterinary department between 1968 and 1971. Also, as will be seen below, relative increases in population density may have contributed to reducing tsetse. Table 2 shows also that the incidence of sleeping sickness in Gwassi location increased almost steadily from 1972 onwards. Of the 545 cases recorded between 1967 and 1984, 39.08 percent were from Gwassi. Further, Kubia East sublocation of Gwassi alone accounted for 35.23 percent (or 192 cases) of the total number of cases during the period. Lambwe location has also recorded an almost steady figure.

Increases in human population density may have produced different results in the sleeping sickness-affected locations of South Nyanza (Table 3). In Kaksingri and Gembe, for example, increased population density appears to have led to more effective bush management as more land was put under cultivation. In contrast, the relative increase in population density in Gwassi saw also a greater incidence in disease cases. It is probable that increased population density in Gwassi led to an increased exposure to tsetse because of the proximity of the wildlife conservation area. Intensifying population pressures in the neighbouring locations of Gembe, Kaksingri, Kanyamwa and West Nyokal may preclude in-migration from the severely affected locations. This means that the traditional method of coping with sleeping sickness outbreaks by moving away can no longer be practised.

Both Kubia East sublocation and Lambwe lie next to the Ruma national park, the centre of tsetse infestation in South Nyanza. Also, the ecology of bushes and thickets at the southeastern part of Ruma national park also characterizes Kubia East. This maintains tsetse infestation. In fact some 300 hectares of bush have recently been cleared at Nyaboro in Gwassi to act as a barrier between the game park and human settlement (PC 1982: 24). In general, data presented in Table 2 suggest that a preponderant number of cases of human sleeping sickness in South Nyanza over the past twenty years have come mainly from Lambwe valley and the surrounding areas.

It is clear that by 1974, cattle trypanosomiasis was on the increase in the district. Apparently, after the tsetse and trypanosomiasis campaign of the late 1960s and the early 1970s, bush regenerated and tsetse increased. The veterinary department did not have funds to continue with bush clearing, spraying, and treatment (DCSN 1974: 23). Trypanosomiasis persisted in the Lambwe valley as the tsetse survey and control people focused their activities more on the lakeshore and the river systems (PC 1976: 85, 131) where there still exist some G. fuscipes and G. palpalis.

In 1980 yet another epidemic of the human disease erupted in Lambwe after a period of decline (Table 2). A cattle sample in Lambwe at the time showed a 40 percent infection rate by T. brucei (PC 1980: 161). There was a high tsetse infestation at 104 per trap and the infection rate of the fly was high (PC 1981: 116).

The 1980 epidemic in Lambwe led the Kenya Trypanosomiasis Research Institute (KETRI) into the area. An operation of aerial spraying (with endosulfan) was done by the Desert Locust Control of East Africa with the support of the Lake Basin Development Authority (LABDA). Meanwhile a veterinary department group helped in ground

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Table 3: South Nyanza population density: selected locations

<table>
<thead>
<tr>
<th>Location/sublocation</th>
<th>Area (km²)</th>
<th>1969 Total population</th>
<th>1969 Density</th>
<th>1979 Total population</th>
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<tr>
<td>Kaksingri</td>
<td>183</td>
<td>6364</td>
<td>30</td>
<td>12341</td>
<td>67</td>
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<tr>
<td>Gembe</td>
<td>158</td>
<td>9613</td>
<td>57</td>
<td>17705</td>
<td>111</td>
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<tr>
<td>Gwassi</td>
<td>253</td>
<td>15885</td>
<td>59</td>
<td>23402</td>
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<tr>
<td>Kubia East</td>
<td>87</td>
<td>4725</td>
<td>42</td>
<td>7013</td>
<td>79</td>
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<tr>
<td>Lambwe</td>
<td>334</td>
<td>8527</td>
<td>29</td>
<td>14459</td>
<td>43</td>
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<tr>
<td>West Nyokal</td>
<td>231</td>
<td>22532</td>
<td>93</td>
<td>25643</td>
<td>110</td>
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<tr>
<td>Kanyamwa</td>
<td>234</td>
<td>22191</td>
<td>93</td>
<td>25885</td>
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spraying hill and valley areas which could not be adequately covered by aerial sprays (Provincial Veterinary Officer, Nyanza 1981, hereinafter PVO).

The campaign to suppress the 1980 epidemic reduced sleeping sickness cases from two per two weeks to one case in two months (PVO 1981: 18). A post-spraying survey conducted by KETRI showed a 99 percent reduction in the tsetse population “but the infection rate remained the same as before spraying” (PVO 1981: 18). It was also noted that old tsetse population increased dramatically suggesting the movement of flies from non-treated to treated areas. More daunting was the fact that post-spraying monitoring showed that the tsetse population almost fully recovered within twelve months (PVO 1981: 19). This may be attributed to the inability of the tsetse field staff doing much follow-up work due to lack of funds and insecticide (PC 1981: 117). It appears that sustained tsetse reduction in Lambwe area requires constant spraying and/or bush control. When there was no large scale spraying in late 1981 the tsetse population increased and so did human and livestock cases of trypanosomiasis (PVO 1982: 17).

In the contemporary time, the government has tsetse survey and control camps covering the entire Lake Victoria coastline from Uganda to Tanzania border. In South Nyanza, these camps are located at Homa Bay, Sindo and Karungu. They use traps in tsetse survey and organise control via bush clearing and spraying. The Sindo camp is responsible for tsetse control in the Lambwe area. In addition these camps check on G. palpalis which exist along the lakeshore and the main rivers.

Total tsetse control has, however, proved difficult. One of the government field staff concerned with the problem summarizes the situation this way: “The much recommended method of bush clearing and spraying with dieldrin is time consuming and expensive especially when applied in an area like Lambwe Valley. But so far it is the only effective method . . . Aerial spray with pyrethroid compound was tried but did not work as it only killed the mature flies and not the pupae” (Nyanza Provincial Director of Veterinary Services 1983: 15).

An ongoing programme started in 1984 involves ground spraying in Lambwe valley and the surrounding areas (MIREGA 1985). This programme is pulling together the resources of the Kenya government veterinary, wildlife, and forest departments. International agencies involved in the tsetse control project are the WHO and the International Atomic Energy Agency. This project involves four spray applications in an area at 28-day intervals to ensure that tsetse pupae are also killed. The current programme will last for five years.

The veterinary department will go ahead with its drug treatment and the atomic energy agency will experiment with the male sterilization technique. The forest department is to clear the undergrowths in Kanyamwa forest plus help in spraying the forest itself.

**Discussion**

A study of sleeping sickness in South Nyanza reveals a history of fragmentary and uncoordinated efforts to reduce the disease incidence. The sleeping sickness problem in the district and especially in the Lambwe valley is both a medical and veterinary issue. But seldom have the two departments mounted sustained joint programmes.

It is already established that since 1900, the greatest efforts to fight the disease come only after an epidemic outbreak. These ignore the fact that the disease is endemic. Moreover the ad hoc reactions of the government to the disease outbreaks have almost always ignored the complicated epidemiology of the disease. This analysis has shown that considerable measures have been taken to eliminate tsetse through destroying their habitat. It was believed that sleeping sickness would be eradicated once the tsetse were exterminated. There was nothing done to influence cultural practices which enhance the survivability of trypanosomes. Local people have never been involved in the search for a permanent cure to the problem. Isolated treatment of human cases and inoculations of cattle are insufficient to contain trypanosomiasis. Also, wildlife have been scarcely brought into the picture.

This study has also shown that tsetse reduction especially after an epidemic leads to a reduced incidence of the disease. This suggests that in South Nyanza, the disease is caused by direct contact with tsetse and that there is perhaps little mechanical transmission of the trypanosomes. In fact, results of an analysis of the 1980 sleeping sickness epidemic in Lambwe area suggest that the outbreak was a localized problem without connection, for example, with the Busoga, Uganda epidemic which occurred at about the same time (Kenya 1980: 5). Thus the greatest incidence of both human and animal sleeping sickness is at those locations in which tsetse are prevalent – Gwassi and Lambwe.

The inability to clear both Gwassi and Lambwe of tsetse is related both to the failure of Lambwe settle
ment scheme and the establishment of the Ruma national park. The former could not attract a level of settlement necessary to eliminate the tsetse habitat and through it the disease. This is because, in ordinary circumstances, a certain threshold population density may be required to ward off sleeping sickness (Matzke 1979).

A big chunk of land in Lambwe and parts of Gwassi was allocated to people during the 1950s and early 1960s. Much of this land however remains fallow; some owners have not even seen the land. Part of the reason for this is the fear of tsetse and sleeping sickness. Also, it is because, in Kenya, land tends to be acquired for its asset rather than productive value (Kenya 1979). Some people may misuse, underuse or not use it at all. But they will keep it.

It should be noted again that within the Lambwe valley area, tsetse have expanded in territory even over the past two decades. This has been encouraged by the expansion and preservation of suitable tsetse ecology as shown above. Formerly tsetse-free grassland on the floor of the valley has been replaced by Acacia woodland much of which is resistant to fire. Tsetse expanded from their initial concentration in the heavy thicket area in the middle of the valley (Ruma bush) to both the eastern and western flanks of the valley and into the rest of the valley floor (Challier a. Turner, n. d.).

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Das Internationale Phänologie-Symposium, die erste Veranstaltung dieser Art, fand an der Universität für Bodenkultur zu Wien statt. Unter der Leitung von Herrn Prof. Dr. A. Baumgartner (München) und Frau Prof. Dr. I. Dirmhirn (Wien) und mitveranstaltet von der Arbeitsgemeinschaft Internationale Phänologische Gärten (IPG) wurden an zwei Sitzungstagen vor allem eine Bestandsaufnahme, die Arbeitsweisen und -ergebnisse sowie die Zukunftsperspektiven der Phänologie diskutiert. Die Zusammenfassungen der Vorträge dieser Tagung, auf der Teilnehmer aus acht Ländern Europas sowie der USA vertreten waren, sind in den Mitteilungen der Arbeitsgemeinschaft IPG, ARBORETA PHAENOLOGICA, Nr. 31, Offenbach 1986, erschienen.


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INTERNATIONALES PHÄNOLOGIE-SYMPOSIUM
WIEN, 17.–20. SEPTEMBER 1986
Ein Tagungsbericht
WALTER ERLENBACH UND HERBERT WEBER