

*Rachel Galun*

## THE INFLUENCE OF THE TSETSE FLY ON THE HISTORY OF AFRICA

Prof. Rachel Galun is Professor Emerita of Entomology at the Medical School of the Hebrew University of Jerusalem. Her main interest is in the physiology and behavior of blood-sucking insects. In the years 1977–2000, she served as Director of Regional Scientific Cooperation at the Israeli Academic Center in Cairo. For twelve years she headed a joint project between Ain Shams University and the Hebrew University dealing with insect-transmitted diseases.

The tsetse fly is not much bigger than the housefly, and it used to be thought that the two belonged to the same family. Unlike the housefly, however, the tsetse fly has been a significant player in the history of Africa from ancient times to the present, both as the carrier of “sleeping sickness” (trypanosomiasis), a disease deadly to humans, and even more so as the carrier of *nagana*, deadly to domestic animals and beasts of burden.

Although externally the two flies look alike, biologically they are very different. The house fly, like most other insects, reproduces by ovipositing several hundred eggs, from which the larvae emerge, grow through several molts, pupate, and emerge as adults – all within a couple of weeks. Thus, after two weeks a single female may have some 200 adult offspring. The tsetse fly is much more “human”: A single egg is deposited into what we call the uterus and develops there through three moltings. After about ten or more days, when the mature larva is ready to

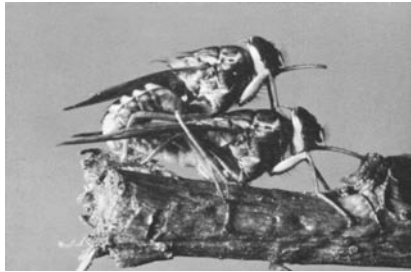
pupate, the female “gives birth” to it in moist soil. The larva digs into the soil and pupates there within one hour. It remains in the soil for 30 days or more, until the adult emerges from the pupa and crawls out of the soil.

The adult fly feeds exclusively on blood, and it has to take a blood meal every other day. Of the 30 different species of the genus *Glossina*, to which the tsetse fly belongs, none prefers humans as a source of blood, but they will do when no other source is available. The fly can live as long as three months, during which it will produce six to eight offspring. Thus, its population density is more similar to that of rodents than to that of insects. This manner and rate of reproduction may explain why, unlike many other insects, it has never spread out of tropical Africa. Compared to mosquitoes or ticks, the tsetse population is negligible. Why, then, has it had such a great impact on African history?

The importance of the tsetse fly results from what is called its

“vectorial potential” – its ability to transmit the pathogenic *Trypanosoma* protozoan from one person or animal to another. When the fly takes a blood meal from a person or animal infected with the trypanosome, the parasites are ingested into the fly’s gut, where they pass through several developmental stages and reach its salivary glands. In one of the fly’s next meals, the trypanosomes are injected into a new individual.

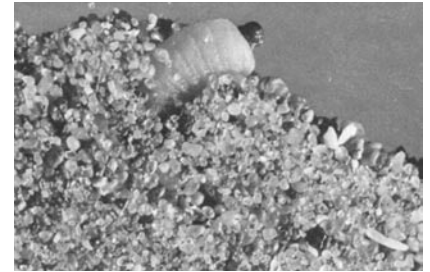
There are several species of *Trypanosoma*. In humans, the disease is caused by *Trypanosoma brucei*. The disease has two forms. Gambian sleeping sickness, *Trypanosoma b. gambiense*, has a chronic, malignant nature, giving rise to the torpor and eventually the coma and death classically associated with sleeping sickness. Over the course of several years, the trypanosomes gradually leave the bloodstream to enter first the lymph glands and then the spinal fluid and the tissues of the central nervous system. If the patient receives no treatment, s/he becomes comatose and dies. By



Mating



Birth of a larva.



Larva burrowing.



Color change during formation of the puparium.



Fly emerging from puparium.



Newly emerged fly.



Fly with unexpanded wings.



Mature fly after its first blood meal.

Fig. 1: The tsetse fly life-cycle. From: *The Tsetse Research Laboratory*, brochure published by the University of Bristol, 1979.

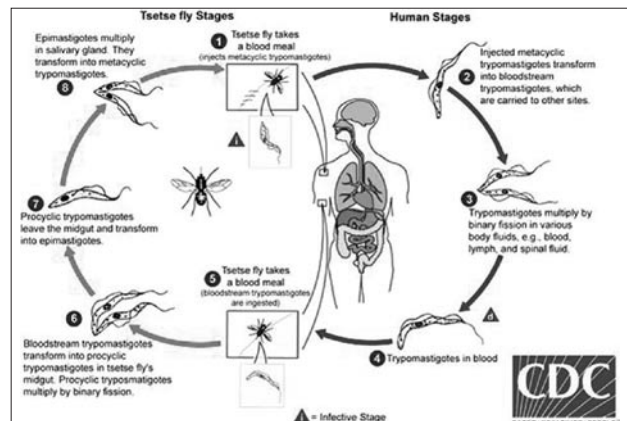


Fig. 2: Trypanosomiasis life cycle. From: Centers for Disease Control and Prevention, National Center for Infectious Diseases, Division of Parasitic Diseases: [http://www.dpd.cdc.gov/dpdx/HTML/ImageLibrary/TrypanosomiasisAfrican\\_il.htm](http://www.dpd.cdc.gov/dpdx/HTML/ImageLibrary/TrypanosomiasisAfrican_il.htm).

contrast, Rhodesian sleeping sickness, *Trypanosoma b. rhodesiensis*, kills its victims within weeks or months.

The ecological setting – the landscape epidemiology – of each variety is different. The Gambian variety is generally restricted to the humid forests bordering the lakes and rivers of west and central Africa, while the Rhodesian variety is endemic to the dry savanna woodlands of east



Fig. 3: A person with sleeping sickness. From: *The Tsetse Research Laboratory* (see Figure 1).

and central Africa. Only a limited number of tsetse species transmit human sleeping sickness, and so, although the disease exists from 14° north to 29° south of the equator, its distribution is patchy. We find about 200 foci, exposing more than

50 million people in 37 countries to the disease.

Although sleeping sickness has certainly been in Africa for hundreds or even thousands of years, the first written record of it is from the fourteenth century, when Ibn Khaldoun, in his *History of the Berbers*, wrote: “Sultan Djata of the Kingdom of Meli [now Mali] was stricken by a lethargy that killed him.” Caravanners recognized the signs of sleeping sickness, which had often been observed in travelers to southern kingdoms. There is information from the nineteenth century on sleeping sickness in West Africa, especially in the Congo. It is estimated that half a million people died of the disease in the decade from 1896 to 1906, including a third of the population of Uganda, some 200,000 people, who died in a very famous epidemic on the north shore of Lake Victoria – an area not previously known to have been infected – between 1902 and 1906. The disease may have been brought into the region by Congolese veterans who settled in Bugosa (Uganda) after taking part in the 1887 expedition of Sir Henry Morton Stanley to relieve Emin Pasha, Governor of Equatoria (Southern Sudan), who had been cut off from Egypt by the campaign of Muhammad Ahmad

(known as the Mahdi) to liberate Sudan from Egyptian rule. It was in this same period that the British offered the Zionist Congress a part of Uganda as a place to create the Jewish national home, and I speculate that, in the back of their minds, they hoped that the introduction of educated people into this epidemic-stricken area might help solve the problem.

Until 1902, no one had any idea what caused sleeping sickness in humans. Some African tribes were convinced that the disease was contagious, and they turned the sick out of their villages. While the locals related nagana in cattle to the tsetse fly, they did not realize that sleeping sickness in humans was a related disease. Even after Sir David Bruce’s 1895 discovery of the relationship between the tsetse fly and *nagana*, it took seven more years until he was able to demonstrate, in 1903, that *T. gambiense* was the organism that caused the disease in humans, and *G. palpalis* was its vector.

Although the riddle of the disease’s complex transmission had been solved, there were no means of curing it or killing the vector. The only way of preventing sleeping sickness was to move the people to fly-free zones. Because rural African populations rarely have the means to obtain water from distant sources, communities tend to form along the banks of rivers and lakes, where bathing, washing, drawing water, and fishing take place at the water’s edge, making for intense man-fly contact. To break it, communities had to be moved away from the water. After the big epidemics in the Bugosa area on Lake Victoria, the place was almost deserted. A resettlement attempted in the 1940s was followed by a new epidemic, and once again

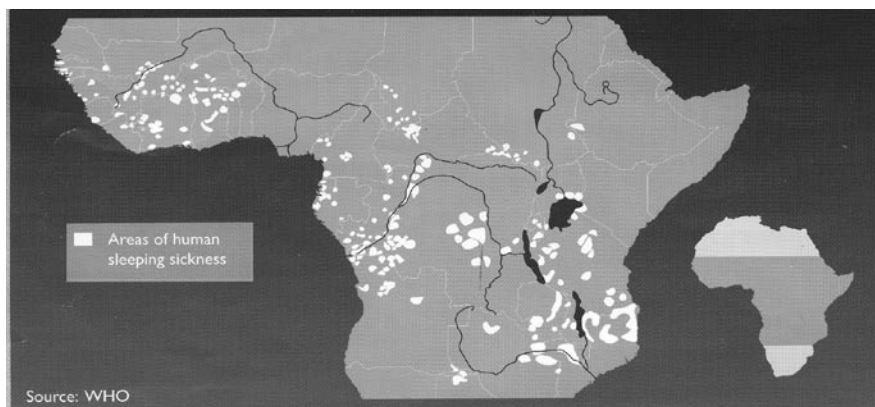


Fig. 4: Areas of Human Sleeping Sickness (2001). Source: World Health Organization.

the inhabitants deserted the place. People settled where vegetation was cleared. Flies do not cross vegetation-free areas, as they can not fly without resting on tree trunks.

The first drugs against trypanosomiasis were developed in the 1920s and 1930s. The colonial health services managed to reduce the number of cases dramatically in the 1950s, and the low level of infection was maintained for about a decade. Protection against the disease in west and central Africa was based on monthly checking of the whole population, using swollen lymph nodes as the criterion for drug application. A medical team would reach a village, call all

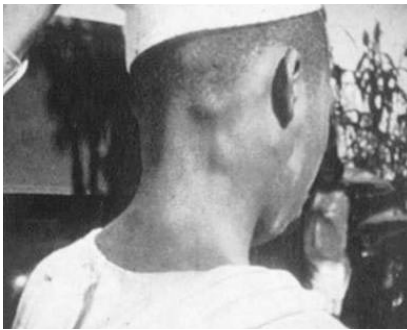


Fig. 5: Infected man, showing swollen lymph node (courtesy of the author).

the people by way of the tam-tam drums, and treat the sick ones. In east Africa, the disease was held back by tsetse-control, which became much more effective with the availability of DDT.

After most of the African countries became independent in the 1950s, human and financial resources were no longer available to keep up the effort to control and monitor the disease. This explains the new outbreaks that have been reported in the last thirty years, both in old foci areas that had been free of infection and in new areas.

Compared with malaria, which

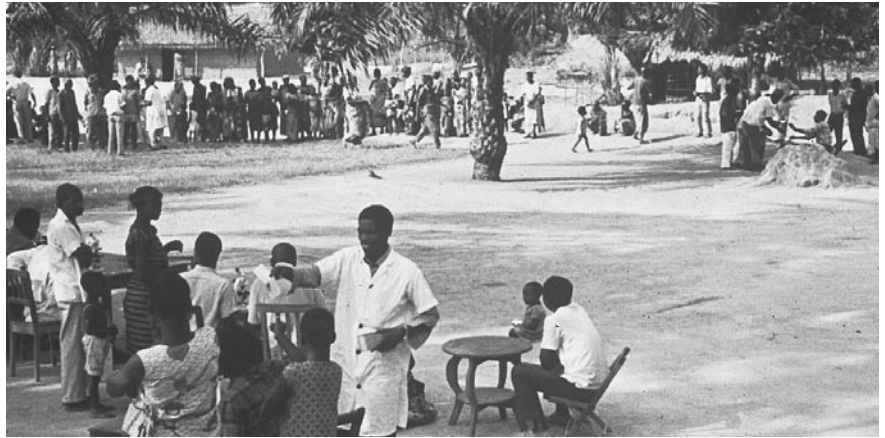


Fig. 6: Belgian Congo, 1950s: Villagers called by tam-tam drums assemble to be treated for sleeping sickness (courtesy of the author).

kills between 1 and 2 million people every year, or with AIDS, which in several African countries is carried by 30–40% of the population, sleeping sickness looks like a relatively minor problem. The greater problem, in terms of African political and economic history, is not human sleeping sickness but *nagana* – animal trypanosomiasis. While only *T. brucei* is pathogenic in humans, several species of trypanosomes, such as *T. brucei*, *T. vivax*, and *T. congolensis*, are lethal to all domestic animals. Thus, wherever there is tsetse, we find *nagana*.

The map shows that the

distribution of cattle in Africa is the converse of that of the tsetse fly. An area of some 10 million sq km, with enough rain, potentially, to carry 125 million head of cattle, is unavailable for cattle breeding because of the presence of the fly. All domestic animals, including cattle, sheep, goats, horses, camels, and even dogs, are sensitive to *nagana*.

Trypanosomes also reproduce in the blood of wild animals, but over thousands years of natural selection, those native to the area developed resistance and survived, because the pathogens do not cross their blood-brain barrier. They thus

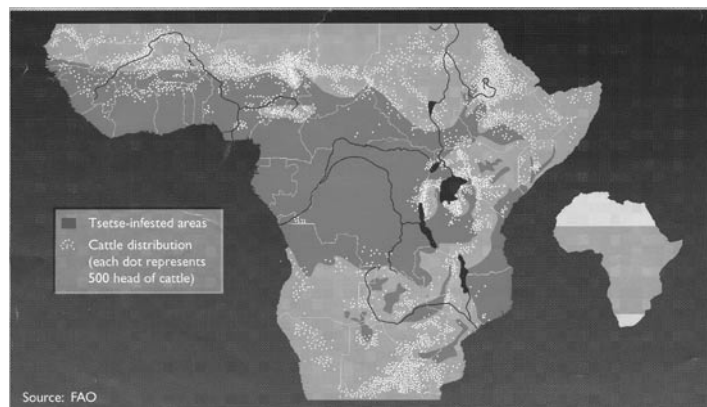


Fig. 7: Tsetse infested areas (shaded) and cattle distribution (light dots) in Africa (1974). Source: Food and Agriculture Organization of the United Nations (FAO): Program Against African Trypanosomiasis (poster).

become asymptomatic carriers of the disease, transmitting it to domestic animals via the flies that feed on them.

Some cynical ecologists look at the inability to breed domestic animals in this area as a blessing, because maintaining a high population of wild animals unique to Africa creates a major tourist attraction. However, in areas of high population density, such as Nigeria, the lack of cattle causes a serious shortfall of protein supply.

Since the trypanosomiasis complex was recognized only at the beginning of the twentieth century, historical data on *nagana* are limited. In the mid-fifteenth century, Prince Henry the Navigator of Portugal sent Captain Cadamosto to look for the source of the gold that, for centuries, had been transported by caravans across the Sahara to Morocco and Tripoli and was reputed to come from a mythical place called Wangara. Cadamos to failed to find the gold, but he learned about the "silent trade." He described how camel caravans carrying salt crossed the Sahara to Timbuktu and Mali, where they were unloaded and the great slabs of salt broken up into human head loads. From there, an army of men on foot would transport the salt a great distance, where it would be traded for the gold. The reason for this, he heard, was that the pasturage in the area was very unsuitable for four-legged animals, since they all died – suggesting the presence of *nagana*, which is endemic to the area.

As related by the historian Abderahman es Sadi, El Mansur, the Sheriff of Morocco, also wanted to discover the source of the gold, and so, in 1590, he sent his army across the Sahara. They conquered Gao, capital of the Songhai empire east

of Timbuktu, and then continued along the Niger river to Dahomey. There, however, they had to call for help, because they had lost all their horses. In 1857, Livingston mentioned in his book *Missionary Travels* how he lost 43 oxen, which he had used in his expedition to replace the human caravans, as part of his attempt to fight slavery. The Africans, he noted, related the loss of the oxen to the tsetse fly, but he could not see that they had died after being bitten.

The historical and economic effect of *nagana* ensues from the wide spectrum of domestic animals for which it is fatal. The ancient technique of using oxen to pull a plough, practiced in Africa as in other lands, was unfeasible throughout the tsetse belt – leaving the farmer with a hoe and shovel; and the same was true of transportation by horse- or ox-driven wagons, or by camels, mules, or donkeys. Thus, until the development of mechanical transportation, human caravans were the only means of haulage and communication, as we saw above in connection with the transport of salt and gold in west Africa. In east Africa, human caravans also carried ivory (elephant tusk), rhinoceros horns (known as an aphrodisiac), and animal hides from the interior to the coastal areas.

In the early nineteenth century, the Sultan of Zanzibar, Seyyid Said (previously known as the Imam of Oman), considered himself ruler of the whole interior of east Africa. Yet he remained on the coast, sending human

caravans to the far-removed sources of his supplies. A typical caravan consisted of several hundred men, mostly slaves, whose use continued despite Seyyid Said's agreement with the British to help eliminate slavery. The slaves were treated brutally and deserted when sick; on a typical expedition, which could last over two years, less than 20% of them came back, usually to be sold overseas. Slavery continued on a large scale in Africa until mechanical transportation was introduced at the beginning of the twentieth century. The first train inland, the "Lunatic Express" from Mombassa to Kampala on Lake Victoria, was completed in 1902.

The Islamic invasion marked a turning point in the history of Africa. The split of Islam into the Shia and Sunni sects, immediately after the death of Mohamed in 632, produced a bitter conflict that sent waves of Sunni migration to east Africa. There are records of Arab settlement in the Lamu Archipelago as early as seventh century. These



Fig. 8: Map of Islam in Africa.

Arabs, known as Zini, established a dozen city-states along 2,000 miles of the eastern coast. They enjoyed robust economic health, cultivated the land, harvested the sea, and developed foreign commerce – but they never entered the interior, and so had hardly any problems with trypanosomiasis. Egypt and central North Africa were conquered by Islam during the reign of Khalif Omar in 634–644, and the rest of North Africa in 661–750. Cairo was founded in 973 by the Fatimids, as was its university, El Azhar, where not only religion but also sciences and medicine were taught.

Islam also crossed the Sahara, and in the sixteenth century the University of Sankore in Timbuktu was a Muslim center of learning in Africa. As late as the mid-nineteenth century, a Muslim cavalry of the Fulanic tribes in northern Nigeria tried to conquer and convert the big populations along the Niger River, but they were thwarted by the death of their horses. Thus, Islam and its culture were impeded from invading tropical Africa until the end of the nineteenth century, and they are not there even today. The introduction of western knowledge must have been obstructed by the same disease. The Portuguese started creeping down the west coast of Africa in 1435, and they also established fortresses on the east African coast to secure their connection with India. However, in contrast to their inland invasion of South America in the same period, their efforts to invade the higher inland plateaus of east Africa failed, because their transport animals always died.

The interior of tropical Africa was the scene of constant warfare. When the crops had been harvested, different tribes would raid their enemies and take slaves. This spurred

previously dispersed populations to concentrate in larger, safer communities. By clearing the surrounding vegetation for farms and firewood, they incidentally reduced the number of tsetse flies entering the compound. The entry of European powers into tropical Africa suppressed the wars and slave raiding, leading to a reversal of the old pattern of settlement: Once law and order were established, the people tended to move out of the defended communities, with their exhausted farmlands, and form small hamlets in the tsetse-infested bush. Sleeping sickness started to build up and spread to new foci.

Modern western agriculture changed the environments in these areas. Where the new crops were unsuitable for tsetse flies, they disappeared. However, coffee, cocoa, and rubber plantations created suitable conditions for the fly.

Many modern methods for tsetse control have been developed in recent years: traps and attractants, the use of sterile males, chemical control, and application of drugs to cattle. But after more than a century of field and laboratory studies, and despite its vulnerable biology, the tsetse fly is still with us.