Exploring Disease in Africa Sleeping Sickness

Exploring Disease in Africa: AIDS Sleeping Sickness Small Pox

A curriculum for advanced high school +College students

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SLEEPING SICKNESS: ANCIENT SCOURGE, MODERN PROBLEM ECOLOGY AND AFRICAN KNOWLEDGE

AN ANCIENT AND MODERN DISEASE

Possibly the earliest record of death due to sleeping sickness came in 1373 when a traveler noted that the Sultan of Mali suffered from lethargy, "a disease that frequently befalls the inhabitants." European slave traders were aware of the disease and would check for swollen lymph glands in the neck—an early symptom of sleeping sickness. Unfortunately, sleeping sickness was not just an ancient disease, it continues today. The World Health Organization (WHO) estimates that 50-60 million Africans live in areas where sleeping sickness might infect them. There are different hypotheses about why sleeping sickness remains a scourge today: the sophistication of the parasite, the tolerance of the parasite to drug treatment, the socioeconomic status of most Africans, and the complex ecological nature of the disease. In this section, we'll explore the disease of sleeping sickness, which continues to plague the rural and poor across large parts of Africa.

THE TWO DISEASES

The term "sleeping sickness" is used to refer to two distinct forms of human trypanosomiases. In this curriculum, we will be focused more on *Trypanosoma brucei rhodesiense*, which is the form that occurs across parts of Eastern and Southern Africa. *TB rhodesiense* is transmitted by tsetse flies that live in woodland regions. It is particularly acute; typically killing infected individuals in a matter of weeks. These traits are in contrast to the other forms of sleeping sickness that affect parts of Central and Western Africa. That form of sleeping sickness (*TB gambiense*) is spread by tsetse flies that reside near water sources, and resembles more of a chronic disorder. People infected with *TB gambiense* have been known to live for years after first being bitten by an infected tsetse fly, although the ultimate outcome of both forms of sleeping sickness is death. The table below shows the major differences.

	Trypanosoma brucei rhodesiense	Trypanosoma brucei gambiense
Туре	Acute	Chronic
Region	Eastern and Southern Africa	Western and Central Africa
Incubation Period	5-7 days	Weeks, months, or years
Spread by	Savanna tsetse fly	Riverine tsetse fly
Spread at	Savanna woodlands	Lowland rainforests; gallery forests along waterways
Reservoirs	Wild and domestic animals	Wild and domestic animals
Transmission linked to	Occupation: Searching for wood, hunting, fishing, honey gathering, cattle keeping	Location: Water sources when fishing, bathing, collecting water
More often affects	Men and boys	Women and children
Time until death	6-18 weeks	2-3 years

SYMPTOMS. There are two stages to the disease, an early stage and an advanced stage. In the early phase, the symptoms are quite nondescript and include swollen lymph glands, aching muscles and

joints and headaches. For the acute form of sleeping sickness in East Africa (rhodesiense), the first phase may only last a few days or weeks, and cannot be accurately diagnosed by sight. For the chronic form of the disease in West Africa, a person may live for years with low levels of parasites in the blood and without the symptoms progressing.

Once the advanced stage sets in, a variety of complications begin. Patients sometimes develop anemia in addition to heart or kidney problems. The central nervous system is eventually affected. Victims often suffer extreme mood swings and are sometimes violent or show signs of mental breakdown. Those who are infected are sometimes such a danger that they are chained to poles or houses as the picture to the right shows. It is at this stage that people eventually become lethargic and fall into comas that give this disease its name. Left untreated, sleeping sickness always results in death.



TREATMENT. Since the 1920s there have been drugs to treat sleeping sickness. The problem with the earliest drugs and those that have come since is both their limited efficacy and many painful side effects. The first treatment used was atoxyl, an

arsenic based drug that was toxic for 38% of the patients who received it. More worrisome was one of the side effects: it caused blindness in nearly 30% of those who were treated. Treatments have been improved upon since the 1920s, and while the cures of today do not cause blindness, they do cause other uncomfortable side effects.

The biggest problem however, was, and continues to be, access to the drugs. The access is limited on both sides of the equation: from those who supply the drug (pharmaceutical companies) and those who demand the drug (rural, poor Africans). On the supply side, the drug therapies available today to cure sleeping sickness are expensive. A course of treatment costs approximately \$1,000. On the demand side are hundreds of thousands of Africans who are in need of the drug, but have no way to pay for it. From the perspective of the drug company, there is little incentive to provide the drug in these regions, since there are very few people or organizations willing to purchase it. (There is also little incentive to develop new treatments since the drugs will only be used and purchased in poor areas.) In response to public criticism of such conditions, some pharmaceutical companies have agreed to donate drugs. This is only a temporary solution since infections will continue and the donations will eventually run out.

Unfortunately there are problems in addition to just procuring the actual drugs. Most sleeping sickness cases occur in rural areas, where there are few hospitals and health clinics. The drug treatment requires technically and medically competent staff, something that is often in short supply in rural areas.

A sleeping sickness victim in the Belgian Congo. Taken from Maryinez Lyons, The Colonial Disease.

Finally, the drugs themselves are also coming under attack. Due to a resurgence of sleeping sickness since the 1960s, cases of drug resistance are beginning to appear.

As will be explained in the following section that describes the cycle of transmission, one of the difficulties of drug treatment is that treatment for the disease does nothing to prevent re-infection. And, since the infection often occurs in the midst of routine, necessary, activities (such as hunting, grazing cattle, gathering wood), people are liable to become re-infected even after being cured. The next section will show that despite a century of research on this disease, and attempts to both eradicate and control it, this disease has persevered and may even have grown worse.

CYCLE OF TRANSMISSION

The concept of disease ecology is particularly helpful when trying to untangle a complicated disease like sleeping sickness. If we take an ecological approach to evaluating sleeping sickness it means that we have to pay attention not just to the parasite, or the victim, or even to the tsetse fly that transmits the parasite. Rather, in order to understand the way the disease functions we have to look at the complex web of factors that interact and allow sleeping sickness to thrive. In practical terms, it requires that we are conscience of the geography, climate, rainfall patterns, flora, fauna, human settlement and working patterns, in addition to tsetse fly behavior, man-fly contact, and man-animal contact. The ecological approach to studying disease grew during the 1900s as colonial researchers tried to focus on only one aspect, only to discover that they were unable to control, or even understand the disease without looking at all the necessary components.

What are those necessary components? Sleeping sickness involves at least five different actors: tsetse fly, trypanosomes, wildlife, livestock, and humans. These are only the active participants needed to transmit the disease. The environment also plays a significant role since each of these actors must have a livable environment. In order to understand the cycle of transmission better, each of the five actors involved in spreading the disease will be explained.

VECTOR—tsetse fly. A vector is the living thing that transmits a disease from one living thing to another.

The tsetse fly is the insect that transmits sleeping sickness, and is much disdained for its role in spreading this deadly disease. But to see the tsetse as being a malevolent character would be wrong. The tsetse fly is only trying to fulfill basic living needs: to eat, to rest, to reproduce.

The problem is that while fulfilling these basic requirements the fly first becomes the unwitting receptacle for a deadly parasite, and second, becomes the vehicle to move the parasite around. It



could be useful to think of the tsetse fly as a kind of hop on-hop off rural bus. In East Africa, it trolls up and down the savanna woodlands, stopping when it gets hungry and food is nearby. When the fly stops to suck the animal or human blood it needs to live, it also picks up the parasites that reside in the blood.

Those parasites are the tsetse bus passengers. Once those parasites enter into the tsetse fly, a variety of things can happen. Some of the parasites die off because they can't survive inside the fly's body. Some of the parasites—like the trypanosome— thrive inside the fly's gut and begin reproducing.

When the tsetse fly bus makes another stop to feed, some of the parasites can hop off. If the tsetse fly is feeding on an animal, the parasite hops off and infects the blood of a cow or wild animal without causing any ill effects. (This infected animal can now be thought of as a reservoir for the disease.) The fly may instead feed on a human, though. And if that fly sucks blood from a human, the trypanosome parasite will hop off into the human blood. Once the parasite has entered into the human blood stream, infection with sleeping sickness has occurred.

The last important thing to remember about the tsetse fly is that once infected with the sleeping sickness parasite, it remains infected for the rest of its life, which typically spans one to six months. So although the fly does operate as a bus where the parasite can hop on and then hop off, it never truly disappears. This means that once infected, every time the tsetse fly bites it can spread the parasite into another human or animal.

PARASITE—trypanosome.

Sleeping sickness is caused by a protozoan parasite, called a trypanosome. The parasite is complex enough to have foiled all attempts to create a vaccine to prevent against sleeping sickness. The parasite can reside without causing any ill effects in the blood of wild ungulates (hoofed mammals such as antelope and wild pigs) and domestic cattle. It is transmitted from animal reservoirs to human hosts by tsetse flies. Inside the tsetse fly, the parasite is able to survive and reproduce, infecting the fly for the duration of its life. In humans, the parasite quickly begins to reproduce and in the advanced stage of the disease, it attacks the central nervous system.

RESERVOIR—wild animals, cattle. A reservoir refers to the animal where a vector-borne disease is maintained. A reservoir is not negatively harmed by being infected with the disease, and it is a place where the disease can be maintained over long periods of time.

One of the things that make sleeping sickness so difficult to eradicate is that the disease has multiple reservoirs. The parasite is able to reside in two distinct populations: wild animals and domesticated animals. Wild animals are abundant and a regular source of the tsetse fly's blood meals. These wild animals are potent reservoirs for sleeping sickness since they are so abundant, share so much of the tsetse fly's habitat, and have long life spans. People would come in infrequent contact with these animals except when venturing into the forest areas to collect wood or hunt.

The other common reservoir for sleeping sickness is cattle. Being a domesticated animal, humans are in regular and sustained contact with them. Cows are typically brought in to rest near a village at night, and



A victim of sleeping sickness, in the advanced phases. Taken from Maryinez Lyons, The Colonial Disease.

herders are around them during the day, regularly moving them from place to place. Cows are usually grazed away from villages since adequate grass is needed, which often brings herders to the edges of the forest or "bush" (land unmanaged by humans).

In terms of transmission, one of the most important questions is how much contact there is between reservoirs and flies; flies and humans; and reservoirs, flies and humans in the same place. As should be apparent by now, sleeping sickness can only be spread when: a tsetse fly bites an infected animal; the fly becomes a carrier of the sleeping sickness parasite; and the infected fly then bites a human.

HOST—humans. A host refers to the person or animal where a vector-borne disease is living. A host is typically negatively affected by being infected.

Unfortunately for humans, they are the hosts for sleeping sickness, and in this case, host is just another word for victim. A person is at risk for sleeping sickness when two conditions are met. First, the person has to be bitten by a tsetse fly. This means that the person has ventured outside of the village into a savanna forest area to do something like graze cattle or gather wood. The bite of the tsetse fly isn't enough to cause infection, though. The second condition is that the fly must be infected with the trypanosome parasite. The fly is more likely to be infected if there is a large reservoir of infected cattle or wild animals. Alternately, if there is an epidemic going on and there is a large number of infected humans for tsetse flies to bite, the humans can also serve as a short-term reservoir for the disease.

PRECOLONIAL AFRICAN METHODS

Prior to colonization beginning around 1900, many different groups of Africans had developed methods meant to minimize or prevent disease. In the case of a complicated disease like sleeping sickness, the transmission cycle was not fully understood. But incomplete knowledge about the disease didn't prevent very pragmatic techniques from emerging. Below are some of the strategies used in Africa in the pre-colonial period.

One of the first things that could be done to avoid infection with sleeping sickness was to carefully locate villages and farms to minimize contact between man and the tsetse fly. Going back to at least the 1860s, Africans in the Eastern and Southern parts of the continent had determined that the fly was responsible for causing infection. They also realized that by avoiding the locations where the fly lived was one of the simplest ways to prevent the disease. The only problem with this method was that not all man-fly contact could be prevented. The East African form of sleeping sickness is spread by tsetse flies living in woodland areas. Humans had to enter the forests occasionally to hunt, fish and herd cattle, which exposed some of the population to the bite of the tsetse fly.

Once a village had been established, if residents were being plagued by sleeping sickness they would utilize another practice: bush burning. In addition to realizing tsetse flies spread the disease, Africans had also observed that the fly resided in wooded areas. They correctly surmised that if they burned all the brush near the village, the tsetse flies would be forced to move further afield. The other advantage to this practice was that it was good for farming since it cleared new land for agricultural fields and the ash from the burned trees and bushes served as fertilizer.

Another technique that was sometimes used was the killing of animals such as wild pigs. While no one could, or would, have called these animals "reservoirs" in the eighteenth and nineteenth century, people had already grasped that wild animals played a role in the transmission of the disease. So, when cases of sleeping sickness increased, animals would be culled, reducing the overall reservoir for the disease.

If all of these techniques failed to bring an end to an epidemic or to lessen the strain of endemic sleeping sickness, there were a few more extreme tactics available. One brutal method was to physically segregate or isolate all sleeping sickness victims. By banishing those infected people into the bush, far away from the village, the other citizens were minimizing the human "reservoirs" in their midst. If a village continued to be terrorized by disease, another dramatic step was to abandon the site and look for a new location. In this case, villagers would begin their search again for a place where good agricultural land and water was available without the assault of the tsetse fly.

Despite the assorted techniques used by Africans to control sleeping sickness, almost all of this was overlooked or ignored by Europeans. After committing themselves to eradication attempts starting after the 1900 epidemics, colonial scientists nearly always missed the successful strategies already being employed. John Ford, who worked as an entomologist for the British colonial government in East Africa wrote that his scientist colleagues, "almost entirely overlooked the very considerable achievements of the indigenous people in overcoming the obstacle of trypanosomiasis to tame and exploit the natural ecosystem of tropical Africa by cultural and physiological adjustment both in themselves and [in] their domestic animals." While there were some notable exceptions to this blindness (some articles from the 1920s praised the practice of bush burning), the techniques pushed by the colonial governments were not typically those used historically.

COLONIZATION AND INCREASING SLEEPING SICKNESS

One of the sad ironies of colonization of Africa was that counter to colonial claims that they were going to rid the continent of disease, colonial intrusion actually increased the prevalence of sleeping sickness. In general, we can think about the start of the colonial period, around the turn of the century, as a time of intense biological, social and economic upheaval. Ford considers colonization to be an "ecological disaster" and refers to "the biological catastrophe of the colonial impact".

The start of the colonial era coincided with terrible epidemics of sleeping sickness in Eastern Africa. In the British Protectorate of Uganda, between 200,000-300,000 people died over a ten year period. In the Belgian controlled Congo, nearly half a million people perished. As people continued to die, colonial governments were spurred to action. New colonial policies and laws were created, meant to lessen the incidence of sleeping sickness. Unfortunately many of these practices were misguided and did more to exacerbate the situation than alleviate it.

Based on our understanding of how sleeping sickness is spread, there is at least one explanation for why the disease increased around the turn of the century. Colonization brought with it warfare and social disruption. It also happened to coincide with a number of ecological catastrophes such as years of drought, plagues of locusts and the resulting famines. Because of these events, people were

abandoning farms and sometimes whole villages. As people moved away, the forests regrew. With the forest came wildlife reservoirs and tsetse flies. The implied relationship here is that it was the presence of humans, clearing land and controlling the onslaught of the forest, that impeded the spread of sleeping sickness. When humans leave a place, the environment needed to spread the disease begins to grow back and can even take over land that was formerly free of sleeping sickness.

COLONIAL METHODS OF CONTROL

As the colonial government committed to the control of sleeping sickness, and hoped to be able to eradicate it entirely, they used a variety of strategies. There were different approaches taken by the colonial powers.

The British in East Africa tended toward environmental solutions. Their goal was to eradicate the tsetse fly, so there would no longer be a vector to transmit the disease. In order to kill the flies, they advocated bush burning (to destroy habitat) and also spraying with insecticides. Unfortunately neither of these strategies were effective, and the goal has remained elusive. Although the complete eradication of a disease vector would stop all transmission, this is a concept that has not worked out well in reality. (The only exception to this rule has been in unique closed environments, such as islands. More information is provided about this in activity #2.)

Another strategy that the British used was to resettle whole villages out of tsetse fly zones. These massive movements often came in the midst of disease outbreaks, and decisions would be made without local input. Entire villages were forced to pack up and move to an uninfected area. Although this was a strategy also used precolonially, it was a last-ditch strategy. The manner in which this occurred during the colonial period fostered much ill will.

The Belgian and French colonial government tended toward medical solutions that tried to eliminate the human hosts for the disease. In the Belgian Congo, medical missionaries and colonial doctors worked together to identify and treat those infected. This required hugely invasive tactics since many Africans tried to avoid being identified and sent to government hospitals where they would be treated with only partially-effective therapies. It also involved physically invasive practices in order to diagnose the disease. The standard method was to perform a lumbar puncture, a painful procedure where a needle is introduced into the spinal cord. A bit of lymph fluid is extracted, which then



can be analyzed to see if the typanosome parasite is *A Belgian doctor performs a lumbar puncture on a* present. A picture of this procedure being performed is *patient suspected of having sleeping sickness. Taken* shown at the right. (More information is provided about *from Maryinez Lyons,* The Colonial Disease. this in activity # 3.)

MODERN METHODS OF CONTROL

Since the end of the colonial era in the 1960s, sleeping sickness has remained a major problem. In general, there are a few different ways people have thought about control. The goals of various campaigns have been to reduce the number of flies; reduce contact between flies and humans; and treat infected people to eliminate human hosts. To date, none of these strategies have proved successful, but it is the idea of reducing the fly population that has generated the most interest.

Potentially one of the most promising strategies, fly eradication attempts have not been very successful. Tsetses are remarkable in their ability to reproduce and repopulate areas. As an example, one type of tsetse fly was eradicated from the area around Lake Victoria (in East Africa) between 1954 and 1957. Despite complete eradication of the fly in 1957, tsetses had returned and repopulated the area by 1967.

Another technique used in the 1950s-1970s and again today is the spraying of pesticides to kill the flies. During the 1950s, when there was a campaign to eradicate malaria, the pesticide DDT was sprayed across wide swaths of Africa. The poison killed not only mosquitoes, but also the tsetse fly. DDT was eventually banned because of other negative side effects it caused in the environment. Since then, naturally derived insecticides (pyretheroids) have been used. Finally, "trap and target" is another strategy that was being used in Zimbabwe. Tsetse traps were set up on the edges of regions where the flies lived in order to stop their spread into populated areas and decrease the overall population. While initial results showed some success, one difficulty with this method is that it required a great deal of manpower to set up and maintain. Thus, despite being quite effective this practice has not caught on and is not widespread.

Discussion Questions

- If you were trying to control a sleeping sickness epidemic occurring today, what techniques would you advocate? Is there any additional information you would need before deciding on the best approach?
- Sleeping sickness today is a curable disease, yet many people still die each year. Effective drugs have been found, yet they are not available in many regions of Africa. Do you think there's anything that can be done about this?
- There is an inherent tension between public health and medicine. Explain that tension and explore it in the case of sleeping sickness. What policies are good for the community, and which are good for the individual? Are they mutually exclusive?

Activities

1. John Ford was an entomologist who worked for the British colonial government in East Africa for many years. While there, he participated in much of the research about sleeping sickness. Eventually, in the 1970s, he wrote a book about sleeping sickness and colonial policies, and came to some surprising conclusions about the efficacy of colonial interventions. Discuss the following quote:

"It seemed that my efforts and those of my colleagues were, perhaps, very misdirected. We were feebly scratching the surface of events that we hardly knew existed, and if we achieved anything at all, it was often to exacerbate the ills of the societies we imagined ourselves to be helping. Unfortunately, with very few exceptions, it was psychologically impossible for men and women concerned in imperial expansion in Africa to believe that their own actions were more often than not responsible for the manifold disasters in which they found themselves caught up."

- 2. Read the PDF document included in the appendix about the eradication of tsetse flies on the island of Zanzibar. Discuss the pros and cons of taking such an approach. Also, try to determine if this is a strategy that could work in other parts of the continent. Finally, is there anything that is worrisome about this approach, or that you'd like to know more about?
- 3. Read the information about the medical missionary Stanley George Browne that is included in the appendix. He was stationed in the Belgian Congo and participated in sleeping sickness control measures in the 1930s-1950s. After you've read the introduction and the excerpts from his own writings consider the following questions:
- Does anything stand out about the methods the Belgian government used to control sleeping sickness?
- Do you think the strategies described to punish those who refused to be examined were fair?
- Why do you think people did not want to be examined and were willing to go to jail instead?
- Is there a tension when it comes to sleeping sickness between public health and individual liberties? What is that tension, and why does it matter?

Primaries Human sleeping sickness foci in East Africa^{*}



- 1. Busoga focus (Uganda and Kenya), epidemics, 1900, 1942, 1953 to 1960, and 1964, in Alego, Kenya, 1976 to 1983; and 1988 to 1992 in Tororo District.
- 2. Lambwe Valley focus (Kenya), epidemics, 1960 and 1980.
- 3. Gilo River focus (Ethiopia), epidemic, 1970.
- 4. Gambela focus (Ethiopia), epidemic, 1967.
- 5. Kasulu focus (Tanzania), epidemics, 1930s and 1957 to 1960.
- 6. Tabora focus (Tanzania), epidemics, 1930s and 1957 to 1960.
- 7. Rungwa River focus (Tanzania), epidemic, 1920 to 1921.
- 8. Maswa focus (Tanzania), epidemic, 1920.
- 9. Matandu River focus (Tanzania), epidemic 1925.
- 10. North Luangwa Valley focus (Zambia), epidemic 1970.
- 11. South Luangwa Valley focus (Zambia), epidemic, 1970.
- 12. Kafue River focus (Zambia), epidemic, 1960 to 1968.
- 13. Okavango Swamp focus (Botswana), epidemics, 1939 to 1942 and 1957 to 1971.
- 14. South Malawi focus (Malawi and Mozambique), epidemic, 1912.
- 15. West Nile (T. b. gambiense) focus (Uganda), epidemic, 1930.

^{*} Graphic and caption taken from Geoff Hide, "History of Sleeping Sickness in East Africa," *Clinical Microbiology Reviews,* January 1999, 12, 1: 112-125.

16. Southern Sudan, recent epidemic, 1997 onward.



Trypanosomiases Transmission Cycle, Option 1

Trypanosomiases Transmission Cycle, Option 2



Sleeping Sickne	ss Control Techniques	
Technique	Logic	Used by

Technique	Logic	Used by	Used when	Efficacy
Forced resettlement schemes	Separate people from flies by moving villages	British in E. Africa	Colonial era	Partially effective, did lessen infections, but led to growth of tsetse belts
Bush Burning	Destroy tsetse habitat and decrease the overall number of flies	Africans in E. and S. Africa	Pre-colonial	An effective practice, but must be timed right
Vaccines	Prevent the disease with a vaccine	Belgians and French in Congo and W. Africa	Colonial era	Ineffective. Has been impossible to create a vaccine
Game Control	Kill wild animals that serve as reservoirs for the disease	Africans in E. and S. Africa; British in E. Africa	Pre-colonial Colonial era	Effective, but human hosts & domestic animal reservoirs remains
Settlement Patterns	Carefully locate villages to minimize human-fly contact	Africans in E. and S. Africa	Pre-colonial	Partially effective, but impossible to avoid all sites of infection
Movement Patterns	Don't travel in tsetse- infested regions when flies are biting; travel at night	Africans in E. and S. Africa	Pre-colonial	Partially effective, but travel at night presents risks of other diseases (malaria)
Isolate/Treat Infected People	Lessen the number of human hosts by isolating or treating the infected	Africans in E. and S. Africa; Belgians and French	Pre-colonial Colonial era	Partially effective, but animal reservoir remains; dependent on locating all infected
Aerial and ground spraying of DDT	Decrease the number of flies, or eradicate them entirely		1950s-1970s Today	Partially effective, but flies often return; environmental side effects of DDT
"Trap and Target"	Reduce the overall population of tsetse by trapping flies	Zimbabwe	Today	Effective, but difficult to set up and maintain over time
Sterile Male Technique	Release sterilized male tsetse flies into a location so that the entire population will be eradicated	Zanzibar	Today	Effective, but requires that no new (non- sterilized) flies enter the location

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