



VCU

Virginia Commonwealth University
VCU Scholars Compass

Theses and Dissertations

Graduate School

2015

African Sleeping Sickness in British Uganda and Belgian Congo, 1900-1910: Ecology, Colonialism, and Tropical Medicine

dana bivens

Follow this and additional works at: <https://scholarscompass.vcu.edu/etd>



Part of the [Arts and Humanities Commons](#)

© The Author

Downloaded from

<https://scholarscompass.vcu.edu/etd/3723>

This Thesis is brought to you for free and open access by the Graduate School at VCU Scholars Compass. It has been accepted for inclusion in Theses and Dissertations by an authorized administrator of VCU Scholars Compass. For more information, please contact libcompass@vcu.edu.

©Dana L. Bivens _____ 2015

All Rights Reserved

African Sleeping Sickness in British Uganda and in Belgian Congo, 1900 – 1910:

Ecology, Colonialism, and Tropical Medicine

A Thesis submitted in partial fulfillment of the requirements for the degree of Master of Arts in History at Virginia Commonwealth University

By
Dana Lauren Bivens

Master of Arts in History from Virginia Commonwealth University May 2015
Masters of Public Administration with a Concentration in Environmental Policy from Virginia Commonwealth University December 2013
Bachelor of Arts in History from Virginia Commonwealth University May 2010

Director: Dr. Karen Rader
Associate Professor in History, Department of History and STS

Virginia Commonwealth University
Richmond, Virginia
May, 2015

Table of Contents

Abstract.....	iii
Introduction.....	1
Chapter 2: The British Response to Sleeping Sickness in Uganda, 1900-1910.....	21
Chapter 3: The Colonial Response to Sleeping Sickness in Belgian Congo, 1900-1910...56	
Conclusion.....	88
Bibliography.....	97
Appendix.....	102

Abstract

AFRICAN SLEEPING SICKNESS IN BRITISH UGANDA AND BELGIAN CONGO, 1900-1910: ECOLOGY, COLONIALISM, AND TROPICAL MEDICINE

By Dana Lauren Bivens, MA History.

A thesis submitted in partial fulfilment for the requirements for the degree of Master of Arts in History at Virginia Commonwealth University.

Virginia Commonwealth University, 2015

Major Director: Dr. Karen Rader, Associate Professor in the Department of History and STS

This thesis deconstructs the social, ecological, and colonial elements of the 1900-1910 Human African Trypanosomiasis (African Sleeping Sickness) epidemic which affected British Uganda and Belgian Congo. This paper investigates the epidemic's medical history, and the subsequent social control policies which sought to govern the actions of the indigenous population. In addition, this paper argues that the failure to understand and respect the region's ecological conditions and local knowledge led to disease outbreaks in epidemic proportions. Retroactive policies sought to inflict western medical practices on a non-western population, which resulted in conflict and unrest in the region. In the Belgian Congo, colonial authorities created a police state in which violence and stringent control measures were used to manage the local population. In Uganda, forced depopulation in infected regions destabilized local economies. This thesis

compares and contrasts the methods used in these regions, and investigates the effects of Germ Theory on Sleeping Sickness policy and social perceptions during the colonial period in Africa.

Introduction

African Sleeping Sickness (SS) is a disease that has persisted in sub-Saharan Africa for thousands of years. During the colonial period on the continent, European economic activities fundamentally destabilized the delicate ecological balance established between indigenous groups, domesticated animals, and disease hosts, arguably exacerbating the impact of SS on affected populations. The historical record shows that outbreaks of SS in British Uganda and the Belgian Congo at the turn of the twentieth century coincided with increased economic activities in the region after the formal colonization of the areas by the United Kingdom and Belgium respectively. Understanding disease, however, requires both a biological and social analysis of the factors which caused the epidemic, and the ways in which the human population interpreted and confronted the challenge.¹ SS is a unique disease because of its history on the continent. It is a nondiscriminatory pathogen which affects both animals and humans. Additionally, once contracted, the disease is 100% fatal unless treatment is quickly administered. It continues to affect sub-Saharan Africa, and has presented a social and logistical dilemma for healthcare workers and governments who wish to combat an extremely virulent and historically complex disease.

SS, or Trypanosomiasis, is caused by the predatory protozoa known as the Trypanosome. The disease is spread by the tsetse fly (most common species is *glossina palpalis*) which acts as a vector: the fly transports the disease between organisms without showing symptoms or being affected by the protozoa. The fly acts as an “intermediary reservoir in domestic and wild

¹ Charles E. Rosenberg, *Framing Disease: Studies in Cultural History*, (New Brunswick: Rutgers University Press, YEAR), XVI – XXVI and E. Russell, *War and Nature: Fighting Humans and Insects with Chemicals from World War I to Silent Spring* (Cambridge: Cambridge University Press, 2001).

animals”² and can cause infection in livestock and humans alike. This disease is unique to Africa and has affected regions through sub-Saharan Africa for centuries. As a result, indigenous populations have evolved to avoid tsetse fly habitats, which include woody savannah or regions covered in dense brush. The correlation between the fly’s existence and the decimation of livestock and human populations was understood as early as 5,000 years ago on the continent.³ “In Africa, because humans and game co-evolved, many animals evolved techniques that allowed them to remain more numerous in other parts of the world. In addition, some diseases such as Trypanosomiasis... made it difficult for humans to live in certain African environments and these environments in turn protected animal populations.”⁴ It has even been suggested that the legacy of SS on the continent affected the evolution and spread of African Hominids and *Homo sapiens*, indicating that the disease history extends beyond the written record.⁵ “Sleeping sickness was [also] responsible for the absence of the plow and animal-drawn cart in pre-European Africa,”⁶ which negatively impacted economic development, agricultural production, and demographic increase. SS also decreased the biodiversity of domestically raised animals, curtailing efforts for mixed farming and thus diminishing the resilience of existing herds during other disease outbreaks such as rinderpest in cattle. SS in Africa, therefore, demonstrated the influence of a disease pathogen on the development, dispersal, and diversification of indigenous populations. Humans and animals evolved alongside this disease and actions and habits were shaped by the ability to adapt to the regions where the flies were less prevalent, and to vacate regions where SS was common. This evidence also suggests that the indigenous people

² C Gregory Knight, “The Ecology of African Sleeping Sickness,” *Annals of the Association of American Geographers* 1, no. 1 (March 1971) : 23-30.

³ Gregory H. Maddox, *Sub-Saharan Africa: An Environmental History*, (Santa Barbara: ABC-CLIO, 2006), 40.

⁴ *Ibid.*, 29

⁵ Knight, “The Ecology of African Sleeping Sickness,” 23.

⁶ *Ibid.*, 23

understood the connection between the fly and SS. That is not to say that a microbiological understanding existed prior to European colonization, but it was clear that when humans and livestock lived among the fly, disease and death soon followed.

Because of this threat, communities tended to develop in regions where woodlands were relatively scarce, or away from other areas populated by the tsetse fly. Scientists have recognized twenty-two species of the tsetse fly, and eleven of the species are capable of transmitting trypanosomes. While these different species occupy different habitats, “woodland is required during some part of the life cycle for all tsetse species.”⁷ In Uganda and the Belgian Congo, fly populations generally existed alongside rivers and lakes that were close to this woody brush, making transportation networks and trade centers also epicenters for disease after colonial trade took advantage of these waterways. This inclination to avoid regions where the disease may be prevalent affected travel patterns, the existence of civilizations, farming practices, and other important economic functions. Prior to colonial rule on the continent, the rates of SS outbreaks were relatively stable as these unspoken rules regarding areas to avoid were traditional practices within these ancient communities and tribes. The upset of habitat barriers and human migration into these previously uninhabited zones during colonial exploitation of Africa’s natural resources resulted in the infection and the death of millions of Africans.⁸

The disease pathogens are protozoa “of the genus *Trypanosoma*. Trypanosomes live in the blood and tissues of hosts such as [humans], cattle, horses, sheep, goats, and pigs. When trypanosomes invade the human nervous system, the characteristic lethargy of sleeping sickness

⁷ Ibid., 30

⁸ Michael P. Barrett and Francesco Checchi, “African Sleeping Sickness,” *British Medical Journal* 33, no. 7646 (March 29, 2008): 679.

results.”⁹ Once the trypanosomes have been introduced into the blood stream, the pathogen infects the red blood cells and lymph nodes causing severe flu-like symptoms including vomiting, diarrhea, high fever, chills, and disorientation. This early stage, called the haemolymphatic stage, occurs directly after infection. When the trypanosomes are isolated to the blood stream, available treatments are more effective and recovery rates are generally good.¹⁰ Unfortunately, it is difficult to definitively diagnose the disease, as malaria, influenza, and many other illnesses present with the same initial symptoms. One of the greatest challenges for modern health groups such as the World Health Organization (WHO) is to spread awareness for the disease and to diagnose patients before it has progressed to later stages. The logistics of providing these resources to rural groups is extremely challenging, and early detection has been a major problem in combatting SS in modern Africa.¹¹

When the trypanosomes cross the blood/brain barrier, “severe headaches, mental dullness, apathy, trembling, spasms, and sleepiness results.”¹² This late stage is known as the meningocephalitic stage. When neurological symptoms commence, SS treatments are less effective and the associated side effects present considerable risks to the patients. Unfortunately, because the disease’s characteristic lethargy does not present until this stage, many cases are often misdiagnosed and go untreated. Patients are unable to feed or care for themselves, resulting in emaciation and overall poor hygiene. Survival rates are further diminished by secondary infections and other problems which arise after the onset of

⁹ Knight, “The Ecology of African Sleeping Sickness,” 25

¹⁰ BMJ, “African Sleeping Sickness,” *The British Medical Journal* 336, no. 7646 (Marcy 29, 2008), 679.

¹¹ World Health Organization, “Trypanosomiasis, human African (Sleeping Sickness). <http://www.who.int/mediacentre/factsheets/fs259/en/>, updated March 2014.

¹² Knight, “The Ecology of African Sleeping Sickness,” 28

neurological symptoms. The key to successful treatment is awareness and early diagnosis, both of which are logistically challenging among remote tribal groups in sub-Saharan Africa.¹³

Contemporary medicine still struggles to treat and abate the effects of this disease. Due to the complexity of available testing procedures and the scarcity of primary health care for many citizens, many cases are still not identified until the late stage of the disease cycle. The WHO recommends early stage screening for at risk populations. This requires blood samples and physical exams which in turn require the presence of a medical establishment and healthcare professionals.¹⁴ Late stage diagnosis requires a Serological Card Agglutination Test (CATT) and the close analysis of cerebro-spinal fluid. Performing these tests in the field is difficult due to equipment sensitivity and the wide areas affected by SS. Additionally, this test requires repeat follow up screenings as the presence of trypanosomes varies, and the results of the CATT test can be controversial if blood and spinal fluid demonstrate negative parasitology. Screening and diagnosis, therefore, require ambulatory medical establishments that are equipped with the materials and expertise to identify the disease at different stages. Additionally, ongoing screening and retesting is required to definitively establish the existence of trypanosomes in relatively asymptomatic cases.¹⁵

If identified and treated during the initial stages of infection, chances for recovery are very good. However, if the disease is left untreated, risk of death is nearly 100% and it generally

¹³ BMJ, "African Sleeping Sickness," 679.

¹⁴ WHO, "Trypanosomiasis, human African," World Health Organization, last modified March 2014, <http://www.who.int/mediacentre/factsheets/fs259/en/>.

¹⁵ Francois Chappuis, Elisa Stivanello, Karen Adams, Solon Kidane, Anne Pittet and Patrick A. Bovier, "Card Agglutination Test for Trypanosomiasis (CATT) end dilution titter and cerebrospinal fluid cell count as predictors of Human African Trypanosomiasis among Serologically Suspected Individual in Southern Sudan," *The American Journal of Tropical Medicine and Hygiene*. Accessed March 30, 2015, <http://www.ajtmh.org/content/71/3/313.short>.

takes one to four years for this to occur.¹⁶ Late stage treatment is also less effective and poses the risk of potentially deadly side effects. “Only two drugs are available for treatment of late stage disease. The first is a derivative of arsenic [known as] melarsoprol. In areas where resistant parasites may be prevalent... melarsoprol has a cure rate of less than 70%. About 3-5% of patients die from drug induced encephalopathy.”¹⁷ The second drug, Eflornithine or α -difluoromethylornithine, is as effective as melarsoprol but is considered safer with fewer deadly side effects. The disease relapse rate is of concern, however, as those who relapse have an even smaller chance of recovery. α -difluoromethylornithine is distributed by the WHO in two week treatment kits, and is more widely used to treat late stage infections of SS. Even in the best case scenario, thirty percent of late stage patients die, and a smaller percent of survivors have drug-related complications.¹⁸

SS is spread by the African tsetse fly, which acts as a vector and carries trypanosomes from infected organisms to new victims as it feeds. The trypanosome develops within the fly and is transferred to the host organism when the fly bites that organism. Transmission is, however, relatively unlikely between host and disease vector; “a mature human trypanosome infection in tsetse is rare. Less than ten percent of tsetse flies biting people with Gambian sleeping sickness eventually become infected, and only one percent of flies biting Rhodesian sleeping sickness victims become similarly capable of spreading the disease.” That is to say, only one to ten percent of infected flies (depending on the SS strain) are able to transmit the disease as the protozoa need to be at a specific point in its life cycle within the fly. After the trypanosomes enter the tsetse fly, the protozoa mature in the fly’s gut and eventually travel to the

¹⁶ BMJ, “African Sleeping Sickness,” 679.

¹⁷ Ibid., 679

¹⁸ Ibid., 679

salivary glands which enable the pathogen to enter a new host when the fly bites another organism. An important part of the pathogen's life cycle occurs in the gut, and thus the tsetse fly is both a disease vector and a symbiotic organism. The second part of the protozoan life cycle occurs within the host organisms that are eventually infected by the disease.¹⁹

Other transmission methods between host and vector do exist. However, these methods are statistically less likely. Laboratory tests indicate that "mechanical transmission, in which the trypanosome is carried directly to a new host without the cyclical development in the tsetse," is possible but very rare. This would require a fly to bite another organism within an hour or two of its encounter with trypanosomes (such as if a meal were interrupted etc.) and the protozoa would enter the new organism before developing in the gut of the fly.²⁰

Evidence suggests that flies other than the tsetse are capable of spreading the disease, but this generally occurs in livestock populations and not with humans. "Blood-eating village flies like *mucosa sorbens*, which frequent open wounds and ulcers, have been known to excrete or vomit infected blood after feeding on an infected host. Such blood may infect the new host." Again, while this transmission method is possible, it requires such an exact set of circumstances that its rates are relatively low. For the purposes of this essay, the tsetse fly transmission method is considered the most relevant and that which is the most hazardous to human populations.²¹

Two strains of SS affected both the Belgian Congo and British Uganda: Gambian Sleeping Sickness (*Trypanosoma gambiense* or *T. b Gambiense*) and Rhodesian Sleeping Sickness (*Trypanosoma rhodiense* or *T. b Rhodiense*). Named for the regions in which they

¹⁹ Knight, "The Ecology of African Sleeping Sickness," 27.

²⁰ Ibid., 27

²¹ Ibid., 27. It is important to note that this transmission method was not understood during the time period covered in the body of this essay. As a result, the implications of such transmission methods did not influence the scientific and historiographic analysis of past sources and are not included in this paper.

originated, these two strains spread to affected populations throughout sub-Saharan Africa, and presented symptoms over different timelines. Gambian sickness enters the blood and can exist in this state for up to two years before crossing the blood/brain barrier. At this point, the neurological symptoms manifest, and the victim usually succumbs to the disease within one to two years after this occurs if treatment is not available. The Gambian strain generally is fatal after two to four years.²² The disease causes death from fatal heart lesions, general debility, or complications including pneumonia, dysentery, or other secondary illness. Treatment is generally successful in the first stages of the disease, but dramatically less effective after the protozoa has entered the neurologic phase.²³

The Rhodesian strain is more acute. Although difficult to contract, this strain kills much more quickly than the Gambian strain. Unlike other forms of SS, Rhodesian SS can become fatal when the protozoa are still within the blood stream and before it enters the central nervous system. Generally death occurs within a year and the available treatments are more effective if used in the early stages.²⁴ Unlike the Rhodesian strain, which was more commonly seen in Uganda, the Gambian strain of the disease (which was more common in the Belgian controlled region of the Congo) presented a unique problem to colonial authorities. Because the disease took years to become fatal, the economic capacity of the affected worker could be extended beyond infection. Therefore, management policies attempted to capitalize upon this and allowed those in the early stages of the disease to remain in the company of other workers in order to perform their economic function within the colony.²⁵ The economic implications of disease

²² Ibid., 27-28

²³ Ibid., 28

²⁴ Ibid., 28 - 30

²⁵ Maryinez Lyons, "From Death Camps to Cordon Sanitaire: The Development of Sleeping Sickness Policy in the Uele District of the Belgian Congo, 1903 - 1914." *The Journal of African History* 26. no 1 (1985): 78.

diagnosis, control, and treatment will be discussed in later chapters. The Rhodesian strain was not definitively identified until 1910, and the sources suggested that this strain and the Gambian strain were present in Uganda during the 1901 Epidemic. However, microbiological evidence was not conclusive as scientists were unable to differentiate between the strains at this time.

A third strain of the disease, *T.b Brucei* affects animals but not humans, and was known locally as Nanga. This form of animal Trypanosomiasis was especially detrimental to African economics due to the importance of cattle and other livestock in the survival and trade among and between groups.²⁶ Nanga was also spread by the fly, and once infected livestock became increasingly emaciated and listless. The historical record shows that tribes in south and central Africa knew of the connection between the tsetse fly and sickness in livestock, and made efforts to avoid fly habitats when traveling or transporting their herds. Europeans traveling in the region made note of these warnings, and such insight was instrumental in establishing a scientific link between the tsetse fly and the spread of trypanosomes to both humans and animals.

Diseases, however, cannot be understood strictly from a biological standpoint. The cultural and historical contexts in which diseases are discovered, understood, and treated dramatically affect the nature of disease spread and control or lack thereof. Culture is an important component of medicine. “The variable of culture has become more significant in medical sociology; many researchers now consider biomedicine as a product of culture, as well as a culture within itself.”²⁷ SS epidemics during the colonial period in Africa history offered

²⁶ Geoff Hide, “History of Sleeping Sickness in East Africa,” *Clinical and Microbiological Reviews* 12, no 1 (January 1999): 113.

²⁷ Martha O. Loustaunau and Elisa J. Sobo, *The Cultural Context of Health, Illness, and Medicine*, (London: Bergin & Garvey, 1997), 3.

historians a window through which conceptions of Germ theory, hygiene, racial differences, and imperial goals were understood within an early twentieth century cultural context.

While SS has been part of the African continent's ecology for thousands of years, Europeans first recorded the disease and its symptoms starting in the eighteenth century. An Arabian historian, Ibn Khaldun, verified the disease in 1374 when it presumably killed the Mali King Mansa Djata in that year after a two year battle. John Atkins, a surgeon in the British navy who worked on slave ships on the West African coast in the 1720's and 1730's, recorded symptoms of "Negro Lethargy." Atkins noted that among the newly enslaved Africans who became ill "Their Sleeps are sound, and Sense of Feeling very little; for pulling, drubbing, or whipping, will scarce stir up a Sense and Power enough to move; and the Moment you cease beating, the Smart is forgot, and down they fall again into a State of Insensibility, drivling [sic] constant from the Mouth, as if in a deep Salivation." Europeans at this time noted that the disease was more prevalent among the African populations and attributed this susceptibility to a weakened mental state from lack of use and physical inferiority.²⁸ The assumed cause was poor sanitation and bad water in the Congo region, as Europeans had yet to discover the association between fly and disease, despite the fact that Africans knew of this link. This disregard reflected the diminished respect Europeans had for native disease interpretations. In southern Africa especially, local groups understood that when cattle and other livestock lived in the tsetse habitat, the animals quickly succumbed to the Nanga disease, leading these groups to understand that the fly was detrimental to the health and wellbeing of livestock herds and by extension, villages and local communities.

²⁸ John J. McKelevy, Jr, *Man Against the Tsetse: Struggle for Africa*, (Ithaca: Cornell University Press, 1973), 5, 7, 8-9

Albert A. Gore, M.D., F.R.C.S.I., Surgeon-major and a senior medical officer in the British army, documented his knowledge of what was then known as “African Lethargus” in a letter to published in the January 1875 edition of the *British Medical Journal*. This summary of the disease and its symptoms was designed to forewarn those traveling to the region of its signs, and to spread awareness off this mysterious ailment. The following excerpt outlines the disease symptoms and its understanding in the late nineteenth century. Lethargy in this sense is defined as:

Mental and corporeal turpitude, with deep quiet sleep. The occasional causes being congestion or effusion in the brain by violent mental commotion, as that of fright or furious anger; by retrocedent gout, or repelled examthem; but more generally by long continued labour of body or severe excise of mind, cerebral exhaustion... it is not unfrequently a strictly nervous affection connected with an irregular or debilitated state of mind... In a case mentioned by Cook, in his treatise on *Nervous Diseases*, the paroxysms ultimately ended in the derangement of the mind. Some of the symptoms of that convulsion of the mind known as *melancholia attonita*, or its paralysusu, as seen in acute or chronic dementia, an impassive or motionless body, vacant stupid expression, involuntary passage or urine and feces, and a passive resistance to the actions of others, are occasionally seen in the last stages of African lethargus.²⁹

SS, as Gore described, was initially understood as an indigenous disease that affected the mentally inferior African. Because it affected those who entered the fly habitat on a regular basis (generally the African laborers conscripted by European settlers), SS seemed to reaffirm the notion that cultural practices (such as food production and dwellings) and poor sanitary conditions in the region played a major role in the infection and spread of SS within the indigenous population.

This thesis will argue that the SS epidemics which affected sub-Saharan Africa during the first decade of the twentieth century were fundamentally caused by European Colonialism.

²⁹ Albert A. Gore, M.D., F.R.C.S.I., Surgeon-major. “The Sleeping Sickness in Western Africa,” *The British Medical Journal*, (January 2, 1875): 5-6.

Disease mitigation policies also provided colonial governments with cause for exercising social control among African populations, while experimenting with mitigation and treatment methods. Colonialism effectively spread the disease which had previously existed in a sustainable capacity, through the expansion of trade networks and the development of cities and towns. Forced labor and migration into tsetse habitats (for example, to harvest rubber in the Belgian Congo), placed workers in close proximity to the disease vector which resulted in increased infection rates. Additionally, the spread of the disease and the fear created by epidemics resulted in drastic measures for control by the colonial government which included forced treatment and isolation in SS camps, abandonment of infected areas, and monitored movements within the colonial jurisdiction. The indigenous people, many of whom were already wary of colonial control and rejected western conceptions of medicine, often did not report cases or hid relatives in order to avoid forced confinement. In Belgian Congo, this resulted in a relative police state where the government monitored individuals' travel, forced admission to camps, and administered Atoxyl injections to treat sick. In Uganda, colonial authorities forcibly removed entire communities from traditional living spaces. These disruptive measures were often met with passive and active resistance which made control and compliance logistically complicated. In both regions, colonial control superseded indigenous lifestyles, and often disrupted economic practices in the area, preventing communities from providing themselves with adequate nutrition and other materials needed for sustaining life.

Thus, in the case of African sleeping sickness, culture, ecological change, and biological factors all combined to create a disease epidemic that claimed millions of lives during the colonial period. At the turn of the twentieth century, European expansion into the continent centered on resource exploitation and the export of raw materials for profit in the industrializing

western world. Affected natural resources included wildlife, forests, minerals, land, and the indigenous populations who were (in many cases) forced into labor. During the late 19th and early 20th century, the European view on environmental management centered more on ecological control and manipulation to suit the needs of the human enterprise, including such practices as preserving game for hunting, eliminating species which were a threat to human security, or extracting natural resources from the earth with no concern for future uses. The European perspective also placed blame for degradation and epidemics, such as the encroachment of the African tsetse fly and SS, on the misuse of natural resources by native Africans who failed to properly manage and 'improve' their surroundings. Colonial officials dismissed traditional disease control practices in order to interject a western approach to epidemic management. These approaches, however well intentioned, were largely ineffective due to a combined failure to respect traditional knowledge and a lack of understanding for the ecological forces which governed local habitats.

Historians and ecologists have begun to understand the negative effects associated with dramatic topographic changes, and how failure to understand complex ecological systems led to unforeseen side effects. As a result, historians have begun to redefine the role of the indigenous population from that of the habitat abuser to the ecological preserver. Cultural understanding and traditional customs incorporated environmental and economic components, which served to benefit the individual as well as protect the natural resource. Despite the vastness of the African continent and its seemingly endless bounty, ecosystem fragility was a concern for settlers and Africans alike, as the negative ramifications of colonial encroachment, wars, and land modification began to reverberate across colonial boundaries.

African environmental historians who wrote in the 1970's and 1980's demonstrated this concept in detail as the negative externalities of colonial expansion, and the resulting native demographic decline were deconstructed through an academic lens which awarded more cultural and ecological dignity to indigenous groups. Historian Helge Kjekshus discussed in great detail the human-induced tsetse fly outbreak at the turn of the nineteenth century, in his 1977 publication titled *Ecology Control and Economic Development in East African History*. Kjekshus argued that colonial environmental change, as well as a lack of understanding for the disease vectors and lifestyles, undermined the indigenous peoples' understanding of and respect for the disease. This created an ecological imbalance which caused SS epidemics as well as other environmental and anthropogenic disasters. For example, Kjekshus described a culture of 'game depletion' surrounding human settlement which fundamentally altered the African ecology.³⁰

John M. Mackenzie agrees with Kjekshus' thesis in his 1988 work *Empire of Nature*. Spending a great deal of time discussing the SS issue, Kjekshus and Mackenzie demonstrated the European desire to control the environment and to promote characteristics which were beneficial to humanity and to destroy those which were harmful, with little or no concern for biodiversity or ecological decline. Mackenzie based his argument on the earlier work of John Ford, who studied Trypanosomiasis and published his findings in 1971. He concluded that prior to colonial invasion, indigenous Africans had over time established an ecological equilibrium which effectively managed the spread of SS. Inhabitants understood the areas infested with the tsetse fly and systematically avoided settlement. Additionally, cattle herding and cultivation, along

³⁰ Helge Kjekshus, *Ecology control and Economic Development in East African History*. (London: Heinemann Educational Books Ltd, 1977), 142, 679-681.

with hunting, prevented bush encroachment and limited the ideal habitat for the Tsetse fly.³¹ European efforts to control the environment were designed to bolster economic output of newly established colonies, to preserve large game species for hunting and sport, and to create transportation and other necessary infrastructure. Failure to understand the area's ecology and to respect the natural world for its own vitality resulted in ecological decline and led to issues such as disease outbreaks, famine, and habitat loss.³²

Mackenzie emphasized how this equilibrium ended "With the arrival of Europeans... A whole series of disasters – Rinderpest, smallpox, the plague of jigger fleas, drought, the wars of 'pacification' and finally the First World War – led to the destruction of stock, the failure of harvests, and population collapse on a large scale."³³ Biodiversity loss and a weakened ecosystem thus led to the depletion of necessary resources, and severe casualties occurred for both indigenous peoples and their domesticated livestock. The tsetse fly thrived on a human population whose ability to control its habitat diminished under conflict, famine, drought, and colonization.

Historically, Nanga or the animal strain of Trypanosomiasis, influenced domestic species distribution. Some indigenous groups understood the connection between the tsetse fly and Nanga disease in livestock which were, in many communities, the basis for tribal economic exchange. Animal Trypanosomiasis "caused fever and progressive deterioration in the health of livestock, especially cattle. They knew that it was transmitted by tsetse flies.... Cattle headers in East Africa avoided tsetse-infested areas or set fire to bush in order to clear areas of flies and of

³¹ John M. Mackenzie, *Empire of Nature: Hunting, Conservation, and British Imperialism*, (Manchester: Manchester University Press, 1988), 226-235.

³² William Beinart, "African History and Environmental History," *African Affairs* 99, no. 395 (April 200), 269-302.

³³ Mackenzie, *Empire of Nature: Hunting, Conservation, and British Imperialism*, 235.

wart-hogs, bush-pigs, and other wild animals whose blood the flies fed on.”³⁴ Livestock herders in southern Africa were well aware of regions where their cattle became ill, and these groups avoided brush, riverside areas, and certain landed regions which traditionally led to the death of their livestock. Africans knew of the link between fly and disease livestock because livestock could survive when flies were not present, but perished when bitten by the fly. Descendants of Dutch settlers who occupied present day South Africa fled the cape region in the 1830’s to the Transvaal in the northwest of the country to escape British domination in the colony, experienced and recorded their interactions with Nanga. These settlers chose their routes north to specifically avoid fly regions and thus protect their livestock. Roualeyn Gordon Cumming, a Scottish hunter who lived in South Africa, recorded similar difficulties with travel and the fly populations. In 1843 Cumming attempted to travel to Limpopo against the advice of local groups, and subsequently lost his entire oxen and horse population to the Nanga disease.³⁵ Other stories from the mid nineteenth century surfaced from European travelers who had similar problems with the fly and livestock infection.

SS epidemics in the Belgian and British colonies were financially detrimental to imperial investments. The disease drastically affected economic output through trade network disruption and workforce loss, and as a result, colonial governments invested heavily in SS research in an attempt to control disease epidemics. During the late nineteenth and early twentieth century, Germ theory and the idea that diseases were caused by specific pathogens, motivated scientists and doctors to uncover the biological explanation for SS. From a medical standpoint, Germ theory assumed that microorganisms caused most diseases, and finding these pathogens would eventually lead to the development of chemotherapeutic treatments to combat specific ailments.

³⁴ Headrick, Daniel R. “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940.” *PLOS Neglected Tropical Diseases* 8, no. 4(April 2014): 1.

³⁵ McKelevy, *Man Against Tsetse: The Struggle for Africa*, 15-16.

This approach to disease prevention and control revolutionized social conceptions of disease and transmission. French chemist and biologist Louis Pasteur was a major contributor and developer of germ theory. Pasteur developed a vaccine for rabies in 1885 which “demonstrated the validity of germ theory of disease”³⁶ and contributed to a scientific fervor for identifying and finding cures for some of the more deadly public health threats. Other scientists made tremendous progress in disease cure and prevention alongside Pasteur. “In Germany, Robert Koch had identified the pathogens for cholera, anthrax, and tuberculosis. In Britain, Patrick Manson and Ronald Ross identified the *Anopheles* mosquito as the vector for malaria.”³⁷ The rhetoric in the scientific community touted the strength of microbiology and diagnostics. This culture of discovery influenced the methods by which researchers studied SS, and different groups tested subsequent treatment methods. Robert Koch, for example, who worked on the Sese Islands in Lake Victoria (a heavily infested area), systematically tested arsenic remedies on natives to determine the most effective treatment and dosage. Research missions led by the London School of Hygiene and Tropical Medicine and the Liverpool School of Tropical Medicine in Uganda and the Congo respectively, studied the blood and spinal fluid of thousands of SS victims to identify trypanosomes and to confirm that the pathogen in fact caused the disease.

Imperialism and tropical medicine are two other important historical developments which contribute to the understanding and framing of SS epidemics in east and central Africa in the first decade of the twentieth century. European imperial initiatives coincided with an industrial era in which resources and economic expansion directly correlated with national pride and power. Africa, during this time period, was viewed as a source for raw materials to fuel the industrializing west. The field of tropical medicine developed in response to the growing

³⁶ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940.” 2.

³⁷ Ibid., 2

number of Europeans who traveled into areas where tropical diseases affected both the European colonialists and the economic output of these new and growing international enterprises. The field of tropical medicine reflected many of the goals and priorities of germ theory in that doctors and researchers treated field research stations as a laboratory, and endeavored to identify specific pathogens to combat disease. Additionally, sanitation, miasma theory, and the concern for infection from the tropical climate motivated the policies and perceptions of Europeans abroad.³⁸ In an attempt to protect personal and economic interests, westerners sought to modify the natural environment to suit the colonialists' racial concepts and to impose sanitation and other western notions of disease management on populations which developed outside of the European tradition. Germ theory argued that disease control stemmed from pathogen identification and treatment measures, coupled with increased sanitation in tropical regions. Medical officers and social theorists in the nineteenth and twentieth century argued that "that the boundaries within which an individual could stay healthy and comfortable coincided with the region in which his race had long been situated. To venture beyond this natural realm in any circumstances seemed hazardous; to go abroad to fight a war on treacherous ground was to court disaster."³⁹ As a result, Europeans abroad perceived the tropical climate as fundamentally at odds with the white race, and this perception further deepened the rift between the colonizer and the colonized, between the imperialist and the indigenous population, and between the sick and the healthy, as germ theory and social theory collided within the new field of tropical medicine.

Margaret Humphrey highlighted the challenges associated with germ theory and vector control in *Yellow Fever in the South*. In this work, the historian stipulated that germ theory and notions of sanitation permeated public health policy and social conception during the late

³⁸ Warwick Anderson, *Colonial Pathologies: American Tropical Medicine, Race, and Hygiene in the Philippines*, (Durham: Duke University Press, 2006), 20-25.

³⁹ *Ibid.*, 41

nineteenth and early twentieth century. Scientists became focused on identifying and categorizing disease pathogens in an attempt to develop a medical solution to the problem. Humphrey highlighted that germ theory attempted to impose a scientific solution on a natural problem, and this failure to incorporate an ecological component was detrimental to disease control. That is to say that the laboratory and treatment aided in identifying disease pathogens and treating the sick, but ultimately controlling the disease vector and human behavior were at the heart of curtailing epidemics such as Yellow Fever or SS.⁴⁰ Controlling SS in British Uganda and the Belgian Congo ultimately relied on separating humans from tsetse territory, and controlling exposure to the disease vector rather than identifying an effective treatment as a primary barrier to infection and spread.

This thesis demonstrates how the ecological disruption, conflict, and retroactive policies resulted in the SS epidemic which ravaged Sub-Saharan Africa during the colonial period. Because social history and medicine cannot be understood in isolation, it is necessary that the perspectives of African historians be considered when deconstructing medical history during this time period. Rosenberg articulates that diseases are framed by cultural situations, and thus any investigation must incorporate qualitative social elements with quantitative scientific detail. SS and other tropical diseases threatened the colonial enterprise which was designed to bolster international prowess and bring economic gain to host countries. This thesis will show that culture is an important component of medicine, and colonial governments framed SS policies within the context of both culture and science.⁴¹ The colonial response reflected these goals and in many cases disregarded the African experience during research, testing, and disease management. This episode in African history offers a window through which contemporary

⁴⁰ Margaret Humphrey, *Yellow Fever in the South*, (New Brunswick: Rutgers University press, 1992), 17-44.

⁴¹ Martha O Lousaunau and Elisa J. Sobo, *The Culture of Health, Illness, and Medicine*, (London: Bergin & Garvey, 1997), 2-3.

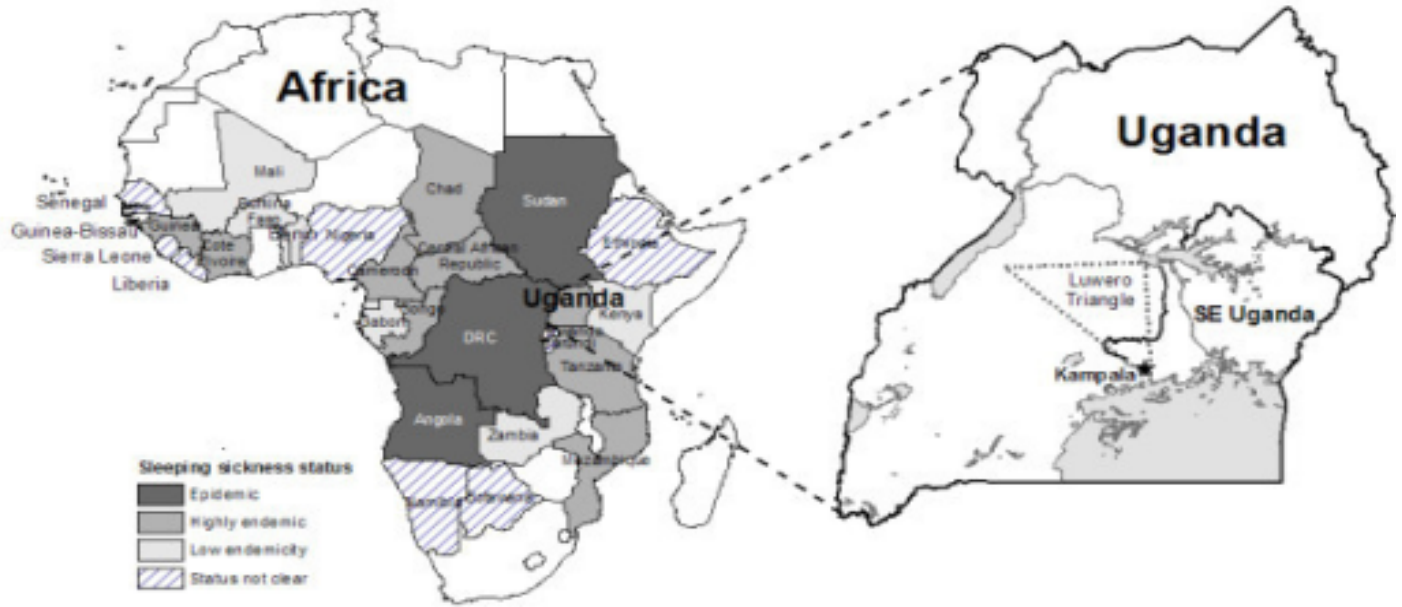
scholars can understand the current political and economic crises across the region, and how disease, famine, conflict, and colonialism in many ways negatively impacted the political and economic independence of an entire continent.

Chapter 2 – The British Response to Sleeping Sickness in Uganda, 1900-1910

The SS epidemic which affected the British African colony of Uganda in the early twentieth century, created an enormous financial and social dilemma. High mortality rates, widespread infection, and diminished economic capacity made studying and controlling the disease a priority of the British colonial government. Early twentieth century notions of race and class motivated control measures which disregarded native culture and traditions by imposing disruptive actions such as forced relocation and limited access to economic resources. The epidemic also allowed historians to understand the social context in which the disease was understood and addressed, and by extension how the British attempted to control the African population. germ theory rhetoric and ideologies dictated research strategies and subsequent experiments which provided policy makers with the political clout to enact restrictive laws and population control strategies in the infected regions.

Following the Berlin Conference of 1884-1885, Great Britain formally gained control of present day Uganda in East Africa. This region of the continent is comprised of bushy scrub, woody savanna, and open plains. Unlike the thick jungles of central Africa, Uganda boasted an environment more conducive to agricultural exploitation, and also supported a larger and denser indigenous population. While SS was known in other regions of the continent, it was not experienced in epidemic proportions in the Ugandan region until the first major outbreak in 1901. “Sleeping sickness was probably endemic in the region and flared into epidemics as the expansion of colonial rule increased trade and migrations throughout Africa. This dramatic social change triggered outbreaks of sleeping sickness in several areas of east and central

Africa.”⁴² The 1901 epidemic was probably imported from the Congo region or from Sudanese Soldiers who traveled east. The first cases were reported in 1900, and the disease quickly reached epidemic proportions a year later.⁴³ The lack of previous epidemics in the region and the limited cultural notions of tsetse fly and habitat indicated that prior to colonialism, the Ugandan region was relatively safe from infection.⁴⁴



Map 1: Uganda in Central Africa (2015)⁴⁵

The map above shows the regions of Uganda most affected by the disease. The depicted isolated pockets represented areas in which the native habitat supported the fly, and it also reflected areas where dense populations of domestic and wild animals and humans provided a

⁴² Daniel R. Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900 – 1940." *PLOS Neglected Tropical Diseases*, vol 8, no 4 (April 2014), 1.

⁴³ George C. Low and Aldo Catelli M.D, "Report on Sleeping Sickness from its Clinical Aspects," *London School of Tropical Medicine*, June 1903.

⁴⁴ T. Koerner, P. de Raadt, and I. Maudlin, "The 1901 Ugandan Sleeping Sickness Epidemic Revisited," *Parasitology Today* 11, no. 8, (1995): 1-4.

⁴⁵ Lea Berrang Ford, "Civil Conflict and Sleeping Sickness in Africa in Genreal and Uganda in Particular," accessed March 1, 2015, http://openi.nlm.nih.gov/detailedresult.php?img=1851948_1752-1505-1-6-1&req=4.

sufficient food source. Just to the west of Uganda, major outbreaks in the Congo region arguably resulted in the transfer and disease into the east Africa region. The Congo, controlled at this time by Belgium, established a sophisticated transportation network which capitalized on the areas abundant and navigable waterways, by which people, animals, resources, and Trypanosomiasis travelled to regions previously protected from the disease.⁴⁶

In Uganda, Lake Victoria's shore and islands were particularly affected by SS. The image below shows this region in greater detail:



Map 2: Uganda and Lake Victoria. Sleeping Sickness affected the northern lake shore and its islands.⁴⁷

⁴⁶ Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa," 1-8

⁴⁷ Planet Earth Online, "Seeing Great Sorrows: Climate and Disease Linked by Missionary Medical Records," Planet Earth Online, last modified November 29, 2007, <http://planetearth.nerc.ac.uk/features/story.aspx?id=149&cookieConsent=A>.

The colony's south-eastern territory consisted of a habitat which supported the tsetse fly along the river banks and lake shores, and in the woody vegetation that survived in this lake side region. Additionally, growing trade and travel between neighboring areas by way of the lake and the rivers that fed its waters resulted in the transfer of disease into and around the lake at an unprecedented rate. Dr. Cuthbert Christy, one of the scientists who participated in the London School of Hygiene and Tropical Medicine expedition to Uganda, wrote in November of 1903:

In studying the Map of the Distribution of Sleeping Sickness, it will be seen at a glance that the disease is connected in some way with the great lake or its waters. In no case has the infection spread far inland, 30 or 40 miles being its limit. It shows no tendency to spread along the Nile source or to other lakes or river; 3. Or 40 miles, in fact, is beyond the limit. My observations led me to believe that most cases to be found further than 10 or 15 miles from the lake are cases which have become infected near the shores of the lake. The nearer one approaches the shores of the lake the more prevalent is the disease.⁴⁸

In 1901, an outbreak along the shores and on the islands in Lake Victoria resulted in widespread infection and high rates of mortality. The epidemic began in the Busoga region of the lake and travelled to Entebbe, the seat of the government, and the islands near the mainland (especially the Sese Islands in the west and the Kavirondo Islands in the east).⁴⁹ The lake was a major commercial and transportation center and the consequences of tsetse infestation were tremendous. For example, nearly 2/3rds of the population on the the Buvuma Islands in Lake Victoria died from sleeping sickness infection.⁵⁰ By 1905 nearly 200,000 of the lake areas roughly 600,000 inhabitants had perished.⁵¹ Nearly one tenth of the country's population

⁴⁸ Cuthbert Christy, "Reports of the Sleeping Sickness Commission of the Royal Society, no III," in *Reports of the Sleeping Sickness Commission*, (London: Harrison and Sons, 1903), 3.

⁴⁹ George C Low and Aldo Castellani M.D "Reports on Sleeping Sickness from its Clinical Aspects," in *Reports of the Sleeping Sickness Commission*, (London: Harrison and Sons, 1903,) 15.

⁵⁰ Christy, "Reports of the Sleeping Sickness Commission of the Royal Society, no III," 3.

⁵¹ Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940," 1

succumbed to SS during this epidemic. Colonial officials generated these estimates, and relied on the self-reporting of local leaders and chiefs regarding the population loss in their communities. Some scholars estimate even greater casualties from the outbreak, but these reports are difficult to substantiate. The fact remains that the epidemic's economic and social consequences were widespread and pervasive, and the disease posed a monumental threat to the colony's economic vitality.

Contemporary scholars have blamed the outbreak on increase transportation and trade between regions, as well as the negative externalities associated with European expansion into the region. One major precursor was the Rinderpest outbreak in 1889. This disease was highly infectious and deadly, and affected cattle populations. In this region of Africa, cattle husbandry was a major economic and cultural activity. Cattle represented a food source through meat and milk, as well as an economic currency as the trade and possession of cattle facilitated transactions within and between indigenous groups. In 1889, the Italian invasion of Eritrea in Eastern Africa, north of Uganda along the red sea, brought Rinderpest to the continent. The disease decimated 90-95% of cattle populations, and the infection spread along established trade routes to the south and west. Rinderpest also infected wildlife populations, and thus severely diminished food sources for the tsetse fly. This led the fly to feed more heavily on the available human population, and also led fly habitat expansion as cattle fields were overtaken by woody brush.⁵²

Additionally, the loss of such an important food and economic resource drove many communities to the brink of starvation. Those who did not succumb to hunger relied on hunting wildlife to procure meat and other forms of sustenance. As a result, greater numbers ventured

⁵² Ibid., 1

into the bush to find food. This exposed more individuals to the fly and to Trypanosomiasis. Poor nutrition and low body weight as a result of the rinderpest outbreak, coupled with greater exposure to the tsetse, led to an unprecedented rise in the number of SS cases in eastern Africa. This example illustrates one of the many ways in which European colonial exploits disrupted the delicate balance established in these regions through centuries of human civilization and evolution alongside the natural barriers which hindered travel and population growth. Breaching these barriers unleashed a torrent of diseases and epidemics which inequitably affected the native populations who were disproportionately exposed to disease pathogens.⁵³

The SS epidemic quickly became an international crisis, and its severity and persistence prompted European colonial governments to launch research missions to determine the source and causes of these epidemics in hopes of finding a treatment. Motivations to solve this problem predominately resulted from the potential economic losses of wide scale mortality rates of the areas labor force, as opposed to the loss of human life and native suffering. Colonial officials and Europeans were less affected by the disease because they were less frequently exposed to the fly and the habitats in which they proliferated. They relied on the natives to control the spread of brush, to provide manual labor in agricultural fields, and to transport products to trading epicenters. As a result, the epidemics were financially detrimental to the European colonial enterprises who had invested heavily in the regions to procure a profitable product, but disproportionately affected Africans versus Europeans. “The Busogan district commissioner’s primary concern lay with the financial cost of avoiding an epidemic in the region. A sleeping-sickness epidemic might threaten the availability of African labor before it could be put to

⁵³ Ibid., 1-4

British advantage.”⁵⁴ Because SS also affected livestock, using large animals to replace human labor was logistically difficult. A human work force was therefore essential to the economic stability of these newly established colonies. As a result of this financial threat, the British government invested heavily in disease control methods. These methods focused on three predominant areas of control: investing in scientific research to discover a cure to the disease, forced depopulation of fly-infested areas, and attempts to eradicate fly populations through habitat clearing, livestock and wildlife slaughter, and removal of human sources for food.⁵⁵

The cultural climate in the European scientific community at the time was abundant with germ theory rhetoric. Study and discovery in the late nineteenth and early twentieth century resulted in the identification and in some cases, treatment of many ailments which in previous decades and centuries had been deadly. The turn of the twentieth century proved to be an exciting time for the field of microbiology and the growing field of tropical medicine. Scientific achievements, such as those of Louis Pasteur and Robert Koch who developed a vaccine for anthrax and identified the tuberculosis bacilli respectively, shaped the conception of disease study and treatment. Scientists were now looking for specific pathogens such as bacteria or parasites, which were responsible for associated diseases. Once identified, these pathogens could theoretically be treated with the use of new medications and other methods for control. The widespread economic impact of SS prompted European nations to dispatch teams of scientists and doctors to study and identify the cause of the mysterious “negro lethargy.”⁵⁶

⁵⁴ Kirk Arden Hoppe, “Lords of the Fly: Colonial Visions and Revisions of African Sleeping-Sickness environments on Uganda lake Victoria, 1906-61,” *Journal of the International African Institute* 67, no. 1 (1997): 86.

⁵⁵ *Ibid.*, 86-88

⁵⁶ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940, 2 and Christoph Gradmann, *Laboratory Disease: Robert Koch’s Medical Bacteriology*, (Baltimore: Johns Hopkins University Press, 2009).

The British government invested heavily in research for tropical disease treatment and control. The Liverpool School of Tropical Medicine was founded in 1898 and led by Ronald Ross. The London School of Hygiene and Tropical Medicine was founded in 1899 and directed by Patrick Manson.⁵⁷ These schools dispatched research missions to affected regions, and scientists assessed sanitary conditions, performed experiments to determine pathogens, and experimented with treatments and control methods to mitigate disease virulence. Research missions included doctors, epidemiologists, bacteriologists, and other scientists whose interest in the natural sciences and desire to combat these tropical pathogens motivated them to undertake these potentially life-threatening assignments. The United Kingdom was particularly invested due to their widespread colonial enterprise. Of the fifteen research missions sent to Africa between 1901 and 1913, eight were British.⁵⁸

In 1902 and 1903 the London School of Hygiene and Tropical medicine dispatched two groups to Uganda by request of the Sleeping Sickness Commission of the Royal Society. The first group was led by pathologist George C. Low, bacteriologist Aldo Castellani, and epidemiologist Cuthbert Christy. The group successfully identified a pathogen existing in the spinal fluid of SS victims. Castellani speculated as to whether this pathogen was a protozoa or bacteria. The 1903 expedition was led by David Bruce who identified that the SS pathogen was in fact a protozoa and he named it *T. b. gambiense* (*T. b. Rhodiense* would migrate north from southern and south eastern Africa later in the decade, and was officially discovered by pathologist John W. Stevens and Harold B. Fantham when working in Rhodesia in 1910). The expedition also definitively showed that the disease was transmitted by the tsetse fly and that the fly lived in the woody brush and vegetation along riverbanks, lakeshores, and on Lake Victoria's

⁵⁷ Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940, 2.

⁵⁸ *ibid.*, 2

many islands. This explained the increase in SS cases around the lake, and promoted measures to eradicate the disease from these regions.⁵⁹

The *British Medical Journal* regularly published testimonies and letters from doctors and other medical officials regarding the latest scientific research, treatment, and biological understanding of the disease. While the epidemic in Uganda officially began in 1901, British scientists and medical professions had been researching and writing on SS for decades. Albert A. Gore, M.D., a surgeon-major in the royal government, wrote on this mysterious disease in a letter published January 2, 1875. Known as African Lethargus or simply lethargy, observed symptoms included “the derangement of the mind. Some of the symptoms of that convulsion of the mind, known as *melancholia attonita*, or its paralysis, as seen in acute or chronic dementia, an impassive or motionless body, [and a] vacant and stupid expression.”⁶⁰ Other indications included lack of control over bodily functions, inability to remain awake even during eating or other such activities, and an overall decline in mental clarity.

Working at the time in Western Africa, Gore experienced the disease for the first time in 1866 in Portuguese Senegambia. A native, presumably affected by lethargus, came to the doctor with enlarged neck glands. This initial indication became predictive of subsequent disease symptoms, and native healers treated this by removing or draining the glands to potentially ward off disease. Gore observed many indigenous people with numerous scars on their neck and other parts of their bodies.⁶¹ Local healers believed that draining swollen glands freed the body of SS

⁵⁹ *ibid.*, 2-4

⁶⁰ Albert A. Gore., M.D. FRCSI., Surgeon-major. “The Sleeping Sickness in Western Africa.” *The British medical Journal* (January 2, 1875), 6.

⁶¹ *Ibid.*, 6

and provided relief to its symptoms. Unfortunately, the treatment had little effect on disease mortality rates.

This curious disease piqued the doctor's interest, and Gore subsequently researched army medical records and found evidence of similar infections in the early nineteenth century. The Army's first recorded case was in 1833 in Sierra Leone. Assistant Surgeon McDonald of the Royal African Corps treated a private in the regiment who had uncontrollable tendency to fall asleep while on duty, during meals, and in the middle of conversations. In March of 1836, Private George Gannys of the West India Regiment was hospitalized with symptoms of lethargus. Treatments for his ailments included purging, doses of colocynth and calomel, leeches applied to forehead and temples, and blisters upon his scalp. The patient recorded periods of recovery where the lethargy decreased and he was released from the hospital, only to return with more severe symptoms. The private died in May of that same year.⁶²

Gore also found evidence that lethargus existed in the West Indies. Private J.M of the 1st West India regiment was hospitalized with dementia and lethargy in Nassau, Bahamas on June 27, 1865. More sporadic cases describing similar symptoms dated from the 1830's onward, suggesting that lethargus had affected colonial and military officials for decades. Gore concludes his letter with a synopsis of available treatments at that point in time. The following excerpt outlines these attempted remedies:

The treatment of African lethargy has been, as a rule, eminently unsatisfactory in the severer forms. Stimulants, alternatives, depurants, and counterirritants have equally failed to arrest the onward progress of the disease. Celsus, who contemplated lethargus as a nervous affection, confined himself to external and internal pungents, shaving the head, forming it, and afterwards applying rubefacient epithems. Others, such as Good, advised cupping, blisters, purgatives,

⁶² Ibid., 6

the voltaic current from the occiput to sacrum, and metallic tonics. If the glandular enlargement observed were due to leuchaemia and hypertrophy of the spleen, conditions sometimes associated with lethargic symptoms, quinine, ferruginous tonics, and antiperiodic medicines might be of value.... The etiology of the disease is still curiously obscure.⁶³

Because the link between the protozoa and infection had not yet been established, attempts to treat clinical symptoms were unsatisfactory and proved to do nothing to abate the disease infection and spread.

David Livingston, writing in 1858 of his travels (1847-84) in present day Botswana, recorded a potential treatment for animal Trypanosomiasis,. He outlined his experimental treatment of a horse that was bitten by the tsetse fly and began to show symptoms of the Nanga disease. Livingston fed the animal arsenic in its daily rations and noticed an improvement in its conditions. The author described the animals coat as being dull and coarse prior to treatment. After a week of arsenic treatments, a sore resembling small pox appeared, and Livingston discontinued the arsenic. In his letter, Livingston observed that after a time the sores healed and the “animal’s coat became so smooth and glossy that I imagined I had cured the complaint.”⁶⁴ The horse’s condition began to decline and it became listless and its coat again dull and dry. When Livingston attempted to treat with arsenic he was unsuccessful because the mare would not eat, and she eventually perished from malnutrition six months after the initial bite.⁶⁵ This source suggests that arsenic was successful, but perhaps the dosage was not sufficient to eradicate the trypanosome within the body of the animal; the small arsenic dosage temporarily abated infection and caused short-term improvement and presumably extended the animal’s life before relapse and secondary complications led to death. Livingston’s arsenic experiment would

⁶³ Ibid., 6-7

⁶⁴ David Livingstone, “Arsenic as a Remedy for the Tsetse Bite,” *The British Medical Journal* 1, no 70 (May 1, 1858): 360.

⁶⁵ Ibid., 360

later be applied to human treatment, and would prove to be one of the most effective methods to treat Trypanosomiasis in the early twentieth century.

While the observed link between the tsetse fly and infection was not revolutionary, a laboratory-based scientific justification for disease eluded scientists until the turn of the twentieth century. J.A.E. Ferguson, writing about a speech given by Dr. Patrick Manson in December of 1889, wrote “In regards to the pathology, Dr. Manson admits that the condition remains a puzzle; its etiology is equally obscure; and it is not to be wondered at that this “sleeping sickness,” so far has proven to be incurable.”⁶⁶ A break through occurred in 1901 when Dr. Robert Forde, British colonial surgeon “observed ‘worms’ in the blood of a sleeping sickness patient. The following year, physician Joseph Everett Dutton identified them as protozoa *T. gambiense* (now *T.b. gambiense*).”⁶⁷ Aldo Castellani, the pathologist who traveled to Uganda with the 1902 London School of Tropical Medicine research expedition, identified pathogens in the spinal fluid of a SS patient. Castellani was unsure if these were bacteria or protozoa, but postulated that the organisms caused SS. Dr. David Bruce led the second expedition which traveled to Uganda in 1903. This expedition solidified the information gathered by the former scientists, and identified that *T. gambiense* was the protozoa responsible for Human African Trypanosomiasis. Additionally, this expedition confirmed scientifically that the disease was transmitted by the tsetse fly which made its home in the brush along river and lake shores.⁶⁸

Some of the key scientific actors who contributed greatly to the discovery and study of trypanosomes, and their relationship to Trypanosomiasis, were Dr. J. Everett Dutton, Dr. John L.

⁶⁶ J.A.E Ferguson. “The Sleeping Sickness.” *The British Medical Journal* 1, no 1988 (Feb 4, 1899): 315.

⁶⁷ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940,” 2 and BMJ, “The African Sleeping Sickness,” *The British Medical Journal* 2, no. 3651 (December, 1930): 194.

⁶⁸ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940,” 2.

Todd, and Dr. Cuthbert Christy. Dutton was perhaps the most experienced, having spent years in the past studying tropical diseases including Trypanosomiasis. He “was a member of the southern and northern Nigeria expedition of the Liverpool School (1899-1900), the leader of the Gambia expedition (1901-1902), and of the Senegambia expedition (1902-1903).”⁶⁹ Dutton’s expertise and notoriety in this field grew when he became the first to identify trypanosomes in a SS patient’s blood in 1901. Dr. Todd, a Canadian educated scientist who travelled to England in 1901 to work with the Liverpool School of Tropical Medicine, worked with Dutton in Senegambia studying Trypanosomiasis. Dutton and Todd would later travel together to the Belgian Congo, by request of King Leopold, to study SS in that region.⁷⁰ Dr. Christy joined Dutton and Todd in Uganda and the Belgian Congo as a member of the Commission of the Royal Society and the Liverpool School of Tropical Medicine respectively.⁷¹ These scientists were instrumental in the discovery, analysis, and understanding of the trypanosome’s effects on the human body, and also made recommendations for treatment and control.

One of the commission’s initial goals was to determine a way to definitively diagnose SS, as its early symptoms resembled that of the flu or malaria. Fever, weakness, tremors of the hands, and other flu-like symptoms were observed in many cases which later evolved the neurological symptoms of lethargy and disorientation which had become indicative of the disease. Castellani writing in November of 1902 stated that “The diagnosis in early cases may be exceedingly difficult, as the typical features of the disease are generally absent. The most important fact on which to base the diagnosis in this state is the evening rise of temperature and

⁶⁹ BMJ, “The Liverpool School of Tropical Medicine and Trypanosomiasis,” *The British Medical Journal* 2, no. 1 (August 22, 1903): 427.

⁷⁰ Barbara Lawson, Pamela Miller, and Kristen Dobbin. *Dr. John Lancelot Todd*, (Montreal: McGill University, 2011), accessed April 1, 2015, <https://www.mcgill.ca/library/files/library/osler-toddbibliography.pdf>.

⁷¹ “Dr. Cuthbert Christy,” *Journal of the Royal African Society* 31, no 124 (July, 1932), 339.

the increased pulse rate.”⁷² It was essential, therefore, that the research team find a method by which the disease could be identified and differentiated from the aforementioned ailments. In this way, it would be possible to isolate these groups and, as later sources indicate, experiment with treatment methods.

In Uganda by the end of 1902, the colonial doctors had experimented with several different treatment methods, none of which succeeded in curing patients. To date, all cases had been fatal with a 100% death rate that was unprecedented for many of these doctors and natives. The commission doctors treated patients with iron, quinine (which was effective against malaria) and arsenic. Quinine proved only effective when the disease was complicated by malaria, and arsenic helped to reduce symptoms while the patient was receiving treatment, but the disease returned as soon as treatment ended. These methods prolonged the life of certain cases, but did not eliminate the pathogen from the victim. Later studies would show that arsenic had to be processed in a particular way, and given at extremely high doses in order to successfully kill trypanosomes in the body. This treatment method will be discussed later in this chapter. Doctors also tried purging, relieving constipation with magnesium sulphate and castor oil, blistering the head and spine, and providing clean food and lodging to the victims. The commission reported that these methods alleviated some discomfort and may have prolonged life, but did not eradicate the disease.⁷³ The group, therefore, focused on isolating the pathogen, diagnosing the disease microbiologically, and then would later work toward testing a treatment. However, during this initial stage, disease identification and understanding its distribution and spread were priorities.

⁷² George C. Low and Aldo Castellani, “Report on Sleeping Sickness from its Clinical Aspects,” In *Reports of the Sleeping Sickness Commission of the Royal Society* (London: Harrison and Sons, 1903), 34.

⁷³ *Ibid.*, 34-37

One of the expedition's major goals was to determine a way to definitively diagnose SS through the identification of parasites in infected victims. Castellani was instrumental in developing a method by which researchers were able to isolate and identify trypanosomes in the cerebro-spinal fluid of SS patients. During the first expedition which landed in Uganda in May of 1902, Castellani standardized this method, and set the stage for subsequent studies which confirmed his initial findings. Castellani identified a patient who showed the classic symptoms of SS and then extracted fifteen cc's of cerebro-spinal fluid via lumbar puncture. He put this fluid in a centrifuge for fifteen minutes, after which the sediments in the fluid separated from the remaining liquid. After pouring off excess liquid, Castellani observed the remaining sediments under a microscope. The scientist discovered that large samples and often numerous observations of the same sample were needed in order to definitively identify trypanosomes in the cerebro-spinal fluid. Additionally, the parasite was not always present. In his first progress report, Castellani performed this experiment on thirty four individuals who were admitted to hospital with confirmed cases of SS. Of these, twenty showed trypanosomes in the cerebro-spinal fluid.⁷⁴

In March of 1903, the London School of Hygiene and Tropical Medicine sent a second expedition Entebbe, Uganda to continue the research begun by the first group in the previous year because Castellani's method proved to be a useful way to diagnose patients. Dr. Lieutenant Colonel David Bruce and Dr. Davis Nabarro led this second expedition. They repeated the cerebro-spinal fluid experiments begun by Castellani and made some important observations. First, healthy spinal fluid was consistently clear in color with no sediments in the fluid, while the fluid from SS patients was consistently yellow or cloudy with floating particulates. Bruce and

⁷⁴ Aldo Castellani, M.D, "Reports of the Sleeping Sickness Commission of the Royal Society," in *Reports of the Sleeping Sickness Commission of the Royal Society* (London: Harrison and Sons, 1903), 2-8.

Nabarro found trypanosomes in these sediments, some of which contained minute traces of blood cells and lymphocytes. Subsequent observations showed similar findings: 70-80% of SS patients had trypanosomes in the cerebro-spinal fluid, and a higher percentage showed trypanosomes in the blood.⁷⁵

These experiments were reminiscent of the Robert Koch's standardized method for establishing a link between a disease pathogen and the arrival of disease symptoms. Koch theorized that, in order to prove that an organism caused a specific disease, the bacteria or protozoa must be isolated from an infected host and identified in that host, and then subsequently transferred to a test animal where the pathogen is again found in that organism's body fluids.⁷⁶ Castellani's methods showed that the trypanosome existed in both the blood and spinal fluid of infected parties and could be transmitted to other organisms. Earlier research established the link between the fly and the disease, and now a new level of understanding pertaining to SS transmission led to policies which directly dealt with fly populations and human access to tsetse habitats.

The scientist from both the first and second Uganda expedition wished to continue this experiment in order to compile more concrete data to prove the relationship between the trypanosome and SS. Dr. Wiggins, a colonial government employee working at a SS hospital at Kisumu in Kavirondo, was called upon to test his patients' cerebro-spinal fluid. Dr. Wiggins's findings were as follows: He used five healthy patients as a control group, and no trypanosomes were found in their cerebro-spinal fluid. Wiggins then tested four patients who were not yet

⁷⁵ David Bruce and Davis Nabarro, "Progress Report on Sleeping Sickness in Uganda," in *Reports of the Sleeping Sickness Commission of the Royal Society* (London: Harrison and Sons, 1903), 12-14.

⁷⁶ Robert Koch, "The Aetiology of Tuberculosis," in *Source Book of Medical History*, Compiled by Logan Clendening M.D. (New York: Dover Publications, 1942), 398-399.

diagnosed, and again found no trypanosomes. The doctor then tested twenty five first stage SS patients, and twenty had the parasite. Finally, Wiggins tested thirteen second stage patients, and all thirteen positively showed trypanosomes in their cerebro-spinal fluid. The final group of seven 3rd stage patients showed the same results. Dr. Wiggin's found that 80% of his cases presented trypanosomes in spinal fluid.⁷⁷

The Uganda research mission made another important observation: trypanosomes were often found in the blood of early stage patients, but not in the cerebro-spinal fluid of these same patients. Generally these early stage patients showed symptoms which included fever with temperature fluctuations, swollen glands, and mild tremors in the hands. Often these initial fluctuations in temperature subsided after the first week and the temperature returned to a normal range, and the patients felt relatively normal. At this time, many were able to return to work and live a normal life until symptoms progressed. Because the majority of advanced stage cases consistently presented trypanosomes in the cerebro-spinal fluid, the question arose as to the correlation between advanced neurological symptoms and the presence of the parasite in the spinal fluid. The following excerpt demonstrates this confusion as the researchers attempted to make sense of this important finding:

From these cases it will be seen that these trypanosomes do not visibly affect the health of their native hosts. All the men are at duty and say they feel strong and well. It is a curious fact that although the parasites are found in the blood they have never been seen in the cerebro-spinal fluid. The question seems to resolve itself into this: either the trypanosomes found in sleeping sickness and those found in Trypanosoma fever belong to different species and give rise to different diseases, or they are one in the same, and if confined to the blood give rise to slight feverish symptoms, whereas if they gain entrance to the cerebro-spinal fluid they give rise to sleeping sickness.⁷⁸

⁷⁷ Bruce and Nabarro, "Progress Report on Sleeping Sickness in Uganda," 14.

⁷⁸ Ibid., 24

Germ theory argued that, if the latter part of this observation as correct, then animals inoculated with infected serum would show trypanosomes in the blood and subsequently in spinal fluid with the onset of neurological symptoms. The group performed experiments to test their theories using white faced monkeys. This species was abundant in the region and its availability and natural susceptibility to SS made it an ideal test animal.⁷⁹ First, the researchers injected the monkeys with fluid, and then tested the animals' blood to determine how long the trypanosomes took to appear. Some of the monkeys received injections with the fluids from confirmed SS patients, and others with the fluids from Trypanosoma fever. Trypanosoma fever proved to be a name given to this first stage of the disease when the blood tested positive for parasites, but the symptoms were milder than those of the diagnosed SS patients who showed neurological symptoms. After infection, researchers tested the monkeys' blood, and when the parasites appeared, the group studied and compared their findings under a microscope. The report included a statistical analysis of the parasites, which showed that the trypanosomes in SS patients were slightly shorter. However, the researchers noted that variations in the length and shape of *T.b Brucei* (animal Trypanosomiasis) were well documented, and thus this statistical analysis was not conclusive and did not prove that the researchers were in fact dealing with two different diseases.⁸⁰

Subsequent observations and experiments would determine that the difference in Trypanosoma fever and SS was not microbiological, but were in fact two different stages of the same disease. Trypanosomes in the blood showed that the patient had in fact been infected, and the resulting fever and swollen glands were the body's initial immune response. When the

⁷⁹ Colonel David Bruce, "Weekly Evening Meeting: Sleeping Sickness in Uganda" (paper presented at the Weekly Evening Meetings for the Ugandan Colonial Government, Uganda, April 22, 1904).

⁸⁰ Bruce and Nabarro, "Progress Report on Sleeping Sickness in Uganda," 25-35.

trypanosomes crossed the blood-brain barrier, the disease rapidly progressed in symptoms and severity, resulting in the lethargy, lack of coordination, inability to focus, and difficulty speaking and moving which became characteristic of late stage cases. This finding was a major step in understanding both the lifecycle of the disease, as well as the nature of parasitic interactions within the body.

Another major finding of these first two expeditions to Uganda dealt with the distribution of the fly and the habitats. The disease was most common along the shores of Lake Victoria, its islands and tributaries, and in the major economic centers established in the region.

Additionally, the disease spread along established transportation routes, and the pathogen often entered areas which had previously been unaffected by SS. Bruce and Nabarro observed that cases were generally found along the northern lake shore within a fifteen mile wide strip.

Northern lake shore islands were also particularly susceptible to the disease. The group blamed disease spread on “a constant interchange of commodities and the intercourse between natives.”⁸¹

Initially the London School of Hygiene and Tropical Medicine’s progress report assumed that SS was not native to Uganda because the tsetse fly which had been charged with spreading Nanga was not present. However, closer observation indicated that the fly was in fact present, and its population was particularly numerous near the lakeshores and other bodies of water. The research group asked local leaders and missionaries to collect fly samples for definitive identification, and citizens collected thousands of tsetse flies which they presented to the research group who later identified them as the Trypanosomiasis vector. The researchers, therefore, established that the fly habitat

⁸¹ Ibid., 36

surrounded the lakeshores and its islands, and this correlated with the disease epidemic epicenter.⁸² The evidence supported the hypothesis that the disease was imported to the region as a byproduct of war and colonial expansion, and this was exacerbated by a traditional culture which was unprepared for dealing with the fly. Other groups in central, western, and southern Africa had coexisted with nanga and SS for centuries, and evolved alongside the disease, learning to avoid the tsetse fly habitat. As a result, epidemics of this magnitude were arguably absent from pre-colonial African history. Researchers from the London School of Hygiene and Tropical Medicine discovered the scientific link between the fly and infection in the Uganda region, the correlation between fly habitat in epidemic epicenters, and the nature by which the disease progressed within each victim. These findings heavily influenced SS policies in the colony in subsequent years. The British colonial government focused predominately on removing individuals from the fly habitat, and eradicating food sources to limit the population of the tsetse fly and by extension the trypanosome.

These findings in SS diagnosis and understanding drastically altered mitigation policies in the Uganda Colony. The British colonial government implemented several methods for control after researchers established this definitive scientific link between the tsetse fly and human Trypanosomiasis in the early twentieth century. The first line of defense against SS infection was to remove human populations from infected areas and to declare occupation of these areas illegal. The second involved clearing the brush and woody scrub that created the fly habitat. The third strategy involved forcibly testing potential SS patients to determine if the disease was present, and then the subsequently removing and isolating these individuals in designated camps

⁸² Ibid., 36-37

and hospitals.⁸³ The British approach to control relied heavily on the cooperation of local leaders and chiefs as the logistical challenges of vacating large areas of land became apparent.

Additionally, self-reporting of infections and death rates in these communities was essential in order for the colonial government to estimate the impact of the epidemic on its populations.

Compliance was a major issue as the scientific and economic approach to disease control conflicted with indigenous beliefs and customs.

Fly depopulation policies proved to be challenging for both the colonial government and for those affected by forced relocation. The government identified areas of infection and, save for the confirmed SS victims who were isolated in camps, the healthy population was required to relocate to designated infection-free areas. “By 1910, there were six declared ‘infected areas in Uganda’ with a major concentration of infection within “a two-mile-wide strip running the entire southern length of Uganda, along the lake Victoria shore, through the kingdoms of Buganda and Busoga, including all Ugandan Islands in the lake.”⁸⁴ This densely populated area with its lake shore access was an important zone for fishing, trading, and transportation between communities in and around the lake. Additionally, hundreds of thousands of native peoples lived in this area, and their customs, traditions, and cultural legacies were tied to a way of life along the lake shores, and the methods of subsistence and trade which had governed these communities for generations. As a result, these groups actively resisted relocation. “British policy-makers perceived no important links between people and specific lands, or between production specialization and access to specific resources,” and interpreted such resistance as a reflection of

⁸³ Kirk Arden Hoppe, “Lords of the Fly: Colonial Visions and Revisions of African Sleeping Sickness Environments on Ugandan Lake Victoria 1906-61,” 87. And Peter Mac Queen, “Sleeping Sickness in Uganda,” *Bulletin of the American Geographical Society* 43, no. 3 (1911), 181-183.

⁸⁴ Hoppe, “Lords of the Fly: Colonial Visions and Revisions of African Sleeping Sickness Environments on Ugandan Lake Victoria 1906-61,” 87.

the native peoples' inferior mental status and failure to understand the consequences of remaining in tsetse areas.⁸⁵ This approach to tsetse control created tensions between groups and affected the success of such policies.

The British assumed that forced relocation of human inhabitants would suffice to reduce the infected tsetse fly population due to a lack of available food sources. "Theoretically, if denied the human reservoir of trypanosomes, fly populations would eventually become infection-free," and the disease would naturally subside. British officials and researchers believed that depopulation would allow the environment to purify itself and if there were no infected humans to transmit trypanosomes, then the flies would no longer harbor the disease and act as disease vectors, purifying the area. The close proximity between the fly habitat and the indigenous populations further cultivated the belief that Africans became infected due to their 'savage' nature or lifestyles, and this deepened the cultural rift between the perceived behaviors of the 'civilized' British populations and that of the native tribes who lived among the flies. This theoretical assumption, however, did not factor in the availability of wildlife as potential tsetse food sources and trypanosome vector.⁸⁶

Initially, scientists estimated that evacuating a region for a few days would effectively rid the tsetse population of the disease as it was believed that the fly retained infectivity for up to forty eight hours. However, by the end of the first decade of the twentieth century, Bruce and his team determined that the fly could remain infected for the rest of its life, and as a result, areas needed to remain vacant for a minimum of eighty days. The researchers admitted that concrete evidence did not exist pertaining to the lifespan and infectivity within the species, and as a result,

⁸⁵ Ibid., 89

⁸⁶ Ibid., 89

many areas were vacated indefinitely until the research team was able to determine that flies in the region were no longer harboring trypanosomes. In some cases, permanent abandonment followed by resource and housing destruction deterred inhabitants from returning to home sites for months or years.⁸⁷

The colonial government made attempts to educate the population to sway public support for relocation policies. In 1906, the government published the *Explanatory Address on Sleeping Sickness to the Natives of the Uganda Protectorate*. This document outlined disease transmission, the importance of keeping roads and villages clear of bush, and the potential health consequences of policy violations.⁸⁸ The address, written in both English and the local dialect of Luganda, was dispersed to local leaders and citizens. Dr. Hodges (the medical officer in Busoga), the main author on the address, stressed the importance of colonial regulations and the need for universal compliance. These efforts reflected an attempt to expose the indigenous population to a western conception of disease control and prevention, arguably due to a lack of adequate resources to effectively police and enforce depopulation. Colonial authorities needed the cooperation of local groups in order to eradicate the disease from the shores of Lake Victoria. Unfortunately, it was not yet understood that the flies could also feed upon and infect wildlife with Trypanosomiasis, so these efforts at forced depopulation did not succeed in eliminating trypanosomes from the flies and from their food sources.

In this same year, Ugandan governor Hesketh Bell took an authoritative stance on forced removal and “ordered all Africans to move to fly-free areas two miles or more away from the

⁸⁷ David Bruce, A.E. Hamerton, H.R Bateman, and F.P Mackie, “Sleeping Sickness in Uganda – Duration of the Infectivity of the *Glossina Palpalis* after the Removal of the Lake-Shore Population.” *Proceedings of the Royal Society of London*, 82, no. 552 (December 20, 1909): 56-57.

⁸⁸ Hoppe, “Lords of the Fly: Colonial Visions and Revisions of African Sleeping Sickness Environments on Ugandan Lake Victoria 1906-61,” 90.

lakeshore and the islands of Lake Victoria and forbade fishing and the sale or possession of fish. Hunting and gathering firewood in the infected area was forbidden. Kampala Port remained accessible to Africans, but only to those who had been medically examined and registered. In Bell's words "we must withdraw from the insects the source of their infection. The whole country must be depopulated. There seems to me to be no other course than to remove *everyone* from the reach of the fly for an indefinite period."⁸⁹

The government offered other incentives to encourage compliance with SS policies. For example, in force removal cases, colonial authorities promised to compensate the natives for their homes and fields, and exempted the affected parties from taxes for one to two years. In some instances, the British government actively destroyed these homes and fields in order to make occupation impossible when relocation was not done in a prompt and complete manner.⁹⁰ These measures did not, however, make up for the difficulty in forced removal and the resulting decreased ability for these communities to support themselves. Many were unable to feed and clothe themselves after their crops and homes and traditional hunting and fishing grounds were no longer accessible. Additionally, conflict arose when groups were forced to assimilate with each other despite traditional and cultural differences which historically kept groups separated. These issues proved to be more immediate and more pressing than potentially contacting SS for some groups, and trespassing, illegal fishing, and illegal reoccupation resulted. The logistical challenge of enforcing the depopulation of such a large area made these activities both possible and widespread.⁹¹

⁸⁹ Daniel Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940," 4

⁹⁰ Hoppe, "Lords of the Fly: Colonial Visions and Revisions of African Sleeping Sickness Environments on Ugandan Lake Victoria 1906-61," 91.

⁹¹ *Ibid.*, 89-93

Pervasive lack of compliance eventually forced the colonial government to financially cut off the diseased areas along the lake shore. Any fishing, travel, or other activities along the lake were illegal, and the government outlawed the sale of fish and other foodstuffs generated from these regions. The areas that were successfully depopulated were quickly overtaken by brush and thus the tsetse habitat in many of these lake communities increased dramatically without human occupation to control this growth.⁹² Those who did remain did so in direct contradiction to colonial policy. This form of passive resistance was the most popular form of action among most of the native groups in the area. Rather than fight, the subversive simply resumed business as usual and continued to trade, travel, and exist in a more traditional manner.

Passive resistance and a quickly diminished economic vitality in the region prompted the colonial government to grant limited access to some regions. It was eventually understood that “A complete quarantine would be economically disruptive, undermining British ability to police and politically control Uganda.”⁹³ The colony’s main purpose was to produce products for sale in European and abroad. SS policies which directly affected this economic premise were thus not reasonable in the eyes of the colonial government. Colonial authorities interpreted mitigation strategies through an economic lens to minimize the effects of epidemics while maximizing the region’s potential monetarily.

The colonial government designated cleared roads and other access areas for controlled entrance to lakes and to the shores. These areas were cleared of brush and declared “tsetse-free areas.” Authorities granted approved tsetse-free citizens regulated access to the lake shore. The colony also appointed a designated SS officer who was in charge of drafting and disseminating

⁹² Ibid., 94

⁹³ Ibid., 95

policies on tsetse control, determining restricted and accessible areas, and monitoring compliance of local tribes and communities. The overall theme of these methods for control centered on limiting access between humans and the fly habitat, and reporting the affected to health officials for quarantine and later treatment with experimental methods.⁹⁴ “Ideas for controlled human resettlement both reflected and affected British ideologies of African environments – and Africans’ place in them. Settling Africans back into their ‘natural’ environment, as David Bruce wrote, to ‘restock it with healthy natives,’ relied on British confidence in their ability to control African behavior, presumably, in order to assure a sustainable natural order.”⁹⁵ These conceptions of healthy environments stemmed from the limited knowledge of the fly, its habitat, and the methods by which Trypanosomiasis was spread. These guidelines for control evolved with scientific understanding, but a respect for and understanding of the natural ecology from the natives’ standpoint was noticeably absent. Europeans viewed the natives as Trypanosomiasis vectors, food sources for the fly, and economic apparatus of the colonial enterprise. Functions and positions outside of these areas were not well received by colonial officials and often led to conflict.

While passive resistance and reoccupation were common, the historical record also shows episodes of active resistance. In 1908 the colonial government began to focus its attention on the lake islands where SS was especially prevalent. The Ugandan side of the lake consisted of thirty three islands, all of which were inhabited by different groups. Health officials ordered the evacuation of twenty if these islands, mandating that the displaced groups occupy the remaining thirteen habitable areas. This created conflict between groups and competition for resources as

⁹⁴ Ibid.,94-97

⁹⁵ Ibid.,100

the populations on these thirteen islands grew tremendously and all the dispossessed were in need of food, shelter, and a way to survive.⁹⁶

In April of 1909 the colonial administration closed all islands and ordered the occupants to vacate within two months. Many did not obey these mandates and by July of that year only “2,300 Sese islanders, out of an estimated total 12,000, and thirty four of an estimated 11,000 total Bavuma” had relocated. Many of the islanders met the mandates with armed resistance and in some cases, attempted to negotiate with the colonial government. Chief Weba of the Sese and Chief Mbubi of the Buvuma demanded answers regarding duration of resettlement, means of survival, and monetary compensation for the forced removal. The groups suggested a three year deadline, after which they would be allowed to repopulate their homes. The colonial government, however, rejected this proposal and withheld monetary compensation until the groups complied with policy. The chiefs and many followers reluctantly relocated to the mainland in 1911, but many deserted to smaller islands to avoid colonial manipulation. After the settlements were abandoned, British officials burned homes and destroyed crops to discourage repopulation.⁹⁷

This episode in colonial history outlines some key concepts which are in many ways responsible for the Ugandan SS outbreak and the limited success of European control policies. The Europeans viewed the indigenous people as childlike, uncivilized, and simple-minded compared to Europeans. Enacting SS policies proved a way in which the British government could consolidate control over these populations, and by limiting traditional economic functions, could force the groups into labor for the colonial enterprise. “Sleeping sickness provided an

⁹⁶ Ibid., 92

⁹⁷ Ibid., 92-93

opportunity for the colonial state to articulate and enact visions of African environments”⁹⁸ and to manipulate populations into regions and positions which were deemed priority by the state. The British habitually dismissed the cultural importance of many of the lands which were vacated, and the natives often disregarded or disrespected policies that reflected this lack of respect for native tradition and culture. The British sought to dictate orders and demanded compliance without understanding the cultural nuances which separated the traditions and actions of individual groups and their way of life. This fundamental conflict between western authority and native resistance (may it be passive or armed) resulted in limited success in SS policies in the region. Despite the efforts made to control the native population the British colonial government and “British scientists would never completely control sleeping sickness, tsetse flies, or African behavior.”⁹⁹

In addition to strategies for control, scientists and researchers worked vigorously to identify and treat Trypanosomiasis from a microbiological perspective. While researchers understood the pathogen and its transition methods after the 1902 and 1903 London School of Tropical Medicine expeditions, the logistics of definitively diagnosing SS in the field, and finding a treatment for the disease, proved to be extremely challenging. In 1906, Dr. Robert Koch, a famous German physician and microbiologist, led a scientific mission to east Africa. Sponsored by the German government after outbreaks of SS in that nation’s east African colonies, Koch’s mission was to experiment with different treatments for SS on the Sese Islands, a heavily infected area of Lake Victoria.¹⁰⁰ This location proved to be ideal for experimentation for several reasons. First, the population on the island was relatively isolated from the

⁹⁸ Ibid., 87

⁹⁹ Ibid., 86

¹⁰⁰ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940,” 3.

surrounding regions and remained confined in a small area. Second, SS was extremely prevalent on the islands. By 1906, over 20,000 or nearly 2/3rds of the population died of Trypanosomiasis, with new cases occurring regularly. Finally, the isolation and fear associated with the islands' epidemics resulted in minimal disturbances from British colonial officials. Koch and his team were essentially able to do as they wished with the affected population. As a result, the Sese islands became German's east African SS laboratory, and the natives became test subjects.¹⁰¹

Koch and his team isolated the sick and set up observation and treatment stations on the island. Patients wore wooden signs which displayed their patient number, and they received gland punctures, blood tests, spinal taps, and other invasive test procedures. The scientists administered experimental treatments to confirmed cases, and Koch's team observed the results and adjusted doses and concentrations to identify the minimum and maximum allowable amounts of each substance. Koch's work in developing Germ Theory and identifying specific disease pathogens resulted in a type of treatment known at the time as chemotherapy: the scientists attempted to identify a specific treatment which would directly attack the disease pathogens affecting a sick population. "Before 1906, it was known, through animal experiments, that the organic arsenical Atoxyl which –because of its poisonous side effects – had been used against syphilis – could also kill trypanosomes. Additionally, Atoxyl and other arsenicals played an important role in the young and progressive chemotherapeutic research, especially that being done by Koch's disciple, Paul Ehrlich, in Frankfurt."¹⁰² Ehrlich also worked on the islands and worked with Koch's team to test these treatments. Many scientists at the time believed that animal testing for tropical diseases was ineffective, and the only definitive way to understand the

¹⁰¹ Wolfgang U. Eckart, "The Colony as Laboratory: German Sleeping Sickness Campaigns in German East Africa and Togo, 1900-1914," *History and Philosophy of Life Sciences* 24, no 1 (2002): 69- 73.

¹⁰² *Ibid.*, 70

disease pathology and treatment was to observe treatment and responses in a live human population. As a result, Koch's team tested Atoxyl and other arsenic-based compounds on the native population in the Sese island laboratory.¹⁰³

During the experiment, Koch and his team tested Atoxyl and other arsenic-based treatments. These drugs included *Natrium Arsenicosum* (an arsenic acid), Nucleogen (which contained less arsenic than Atoxyl), Arsenferratin, Trypanred, and Afridolblue. The latter treatment proved to be extremely painful when administered, and Ehrlich discontinued its use after the side effects proved to be too painful. The team tested each substance at varying dosages, noting the relationship between trypanosomes in the blood and spinal fluid, and the severity of side effects from arsenic poisoning. At the height of the experiment, over 1000 patients were treated per day with Atoxyl, with doses varying from 0.5g to 1.0g per treatment. Koch remained in the colony until October of 1907 when complications from sand fleas forced him to return to Germany. Dr. Professor Kleine of the research group was appointed as his successor and continued experimenting on natives after Koch's departure.¹⁰⁴

After months of experimentation, Koch reported to the German colonial ministry that "The drug he found most effective and least toxic was Atoxyl, or aminophenyl arsenic acid. First synthesized by the French chemist Antoine Béchamp in 1859, it proved to be effective in the short term but left patients blind"¹⁰⁵ in up to 20% of cases. Atoxyl was also a toxic substance and could cause death in patients if overdose occurred. Conversely, the maximum allowable dose also proved to be the minimum dose effective against trypanosomes.¹⁰⁶ Other side effects

¹⁰³ Ibid., 69-73

¹⁰⁴ Ibid., 69- 74

¹⁰⁵ Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940," 3.

¹⁰⁶ Ibid., 3

from Atoxyl included vertigo, sickness, and colic.¹⁰⁷ The treatment's negative externalities made it unpopular among the indigenous people, and financially risky in terms of preserving a viable work force. Additionally, this treatment was only successful if administered to patients who were in the early stages of the disease. Atoxyl was ineffective for late-stage cases, thus making self-reporting and widespread disease awareness essential components to disease treatment.

Some discrepancies existed over the use of this treatment as a method to eradicate Trypanosomiasis. Koch's expedition was unsuccessful in finding a chemotherapeutic treatment to eradicate Trypanosomiasis in patients, but did determine the maximum allowable limits to some of the more toxic treatments. Atoxyl, while effective in alleviating symptoms and effective in early stage cases, was not a definitive cure and was extremely dangerous to give. Koch reported that traditional isolation methods and ecological interventions were the primary safeguards against epidemics, and suggested Atoxyl as a supplementary treatment. Koch's recommendations also included establishing SS concentration camps where infected natives were forcibly removed to fly-free areas. Koch asserted that "simply getting the infected out of the way was an ideal method for protecting the rest of the population" and was far more logistically feasible than relocating an entire population (as the Ugandan colonial government attempted to do in infected regions).¹⁰⁸ His cold, clinical tone, and the assertion that death for these groups was inevitable anyway, eroded any dignity awarded to the native groups who were seen as a cause of the epidemic and a potential barrier against eradication. The team understood this field laboratory setting as an ideal location to further their knowledge and understanding of SS and arsenic treatments in tropical medicine.

¹⁰⁷ Eckart, "The Colony as Laboratory: German Sleeping Sickness Campaigns in German East Africa and Togo, 1900-1914," 72.

¹⁰⁸ *Ibid.*, 73

As a result of Koch's findings, German east Africa did set up SS camps where the government isolated the sick and treated them with Atoxyl. The Belgian Congo, which effectively became a police state during the SS epidemic, replicated this policy and also set up isolation camps. The Belgian government established *cordone sanitaires* (or isolation camps) where infected natives were deported and given Atoxyl injections. This policy will be discussed in greater detail in the next chapter, but its significance can be drawn from the policy initiated set forth by Koch in German East Africa, and the disregard for native well-being in the face of clinical scientific research.

In 1910, John W. Stevens and Harold B. Fantham who worked with Dutton in the Liverpool School of Medicine, discovered a second, more acute strain of SS (which was later named *T.b Rhodiense*) when working in Rhodesia and Nyasaland in 1910 and 1911.¹⁰⁹ The scientists noticed abnormalities in the blood of a rat that had been inoculated SS from an infected male patient. Stephens noticed that the disease progressed more rapidly than the traditional SS infections. This strain generally killed its host in less than a year, and quickly progressed to the second stage. In the Gambian strain, second stage disease progression often took over a year, and death occurred after two to five years.¹¹⁰ Stephens and his colleague Fantham studied the trypanosomes' morphology and discovered that it differed structurally from *T. b. Gambiense*. In an article published in the British Medical Journal, Stephens and Fantham state:

In sheep and goats the difference in evolution, symptomatology, and gravity of the two infections is quite remarkable; whereas *T. Gambiense* infections in these animals often gave rise to no symptoms except fever.... And usually end in recover, *T. Rhodiense* infection led to an acute disease, with high fever, odema,

¹⁰⁹ J. W. W Stephens, H. B. Fantham, F. K. Kleine, F. Mesnil, Allan Kinghorn, Warrington Yorke, S. B. Wolbach and C. A. L. Binger, "Papers Dealing with Trypanosomiasis," *The British Medical Journal* 2, no. 2705, (Nov 2, 1912): 1182-1185.

¹¹⁰ Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940," 3.

and keratitis, and death was invariably the end after a relatively short duration... our view is that we are dealing with a new species of human trypanosome.”¹¹¹

Drs Kinghorn and Yorke who worked with Fartham and Stephens, determined that *T.b Rhodiense* was transmitted by *Glossina Morsitans*, while *T. b Gambiense* was transmitted by *Glossina Palpalis*. Additionally, experiments performed with *T. b Rhodiense* demonstrated in a laboratory setting that the fly can transmit the disease between species (in this case monkeys and sheep) and that the rates of infection and the severity of the disease varied depending on the animal species. This is important because for the first time researchers were able to observe SS transmission in a laboratory setting. Prior to this, scientist observed the correlation between tsetse habitat and disease epicenters. Yorke and Kinghorn also conducted surveys in which they tested the blood serum of wild animals caught within the surrounding environment, and found that *T. b Rhodiense* affected about sixteen per cent of wild game in Northern Rhodesia. These discoveries were important in understanding the virulence and life cycle of trypanosomes within both the fly and its human and animal hosts.¹¹²

The morphology, rapidity of infection, and virulence of the disease led researchers to determine its fundamental difference from *T. b Gambiense*. The Rhodesian strain affected areas of eastern and southern Africa, and cases were later found around the Lake Victoria region in Uganda.¹¹³ Scholars debated over whether it was this strain that affected the communities in Uganda in the 1901 epidemic. The disease foci of the Gambian strain and the Rhodesian strain are only 200km apart within Uganda,

¹¹¹ Stephens, Fantham, Kleine, Mesnil, Kinghorn, Yorke, Wolbach, Binger, “Papers Dealing with Trypanosomiasis,” 1183.

¹¹² *Ibid.*, 1182-1187

¹¹³ *Ibid.*, 1182-1188

making SS diagnosis and treatment in this region more challenging than in the Belgian Congo where only *T. b Gambiense* was found.¹¹⁴

While research and experimentation with different remedies continued for decades, the most successful methods for disease control proved to be prevention and removal of humans from tsetse habitats, or the eradication of tsetse habitat and thus a decline in the fly population. The methods used to forcibly remove Africans from infected areas resulted in widespread unrest, passive and armed resistance, and negatively affected the economic stability and livelihoods for thousands of citizens. Despite the logistical challenges in universal implementation, “the results were remarkable. Whereas in the years 1900 – 1904, at the height of the epidemic, 200,000 out of the 300,000 inhabitants of the infected areas died of SS, between 1905 and 1909, fewer than 25,000 died. By 1910 the epidemic had tapered off and Africans began returning to their former homes.”¹¹⁵ The invasive and reactionary policies implemented by the Ugandan colonial government did successfully reduce exposure between humans and the disease vector.

This epidemic in British Uganda represented several key concepts in colonial history, history of medicine, and environmental history. First, from an environmental standpoint, the influx of European inhabitants and their associated diseases exposed livestock and humans to new pathogens which diminished the native population and forced these groups to look for alternative food sources and living spaces. European efforts to control and change the natural landscape also disrupted the ecology of local habitats. Second, trade and transportation resulted in the exchange of goods and diseases across natural barriers which had traditionally limited human and tsetse exposure. Finally, western perceptions of African indigenous populations

¹¹⁴ Lea Berrang Ford, “Civil Conflict and Sleeping Sickness in Africa in General and Uganda in particular,” *Conflict and Health* (2007), 2.

¹¹⁵ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940, 4.

motivated policies which were authoritative and paternalistic in nature, and often overlooked personal and cultural consequences. The historical record for this time period is saturated with testimony from colonial officials, doctors, scientists, and Europeans, but lacks references to the personal experiences of those native groups who were forced to operate under SS policies. Even though the laboratory-based methods proved successful in the end, arguably the epidemics were caused by colonial expansion, and thus the treatment and attempts for disease control were reactive responses to a European-induced epidemic. It would take years for the epidemic to subside to pre-colonial levels.¹¹⁶

¹¹⁶ John M. Mackenzie, *Empire of Nature: Hunting, Conservation, and British Imperialism*. (Manchester: Manchester University Press, 1988), 236-237.

Chapter 3 – The Colonial Response to Sleeping Sickness in the Belgian Congo, 1900-1910

Sleeping sickness ravaged the Belgian Congo during the first decade of the twentieth century. Like Uganda to the east, the Congo region experienced widespread economic decline as a direct result of the epidemic. Unlike Uganda, however, the Belgian colonial government adopted a more stringent policy for control, and effectively established a police state in which native actions, occupations, and movements were directly controlled by the colonial government. Belgian SS policies included forced relocation to quarantine camps and treatment with an arsenic-based medication with dangerous side effects. In the region, SS afforded the Belgian government a way in which the European power could exercise control over the native population and dictate behavior through the establishment of a military state.

In 1887, American Henry Shelton Sanford led an expedition to the Belgian Congo to assess the quality of land after the formal acquisition of the territory in the Berlin Conference in 1884-1885. Shelton, a tycoon and diplomat, aided King Leopold II in gaining international recognition for the territory, and thus he was awarded the honor and prestige of leading such an excursion. Shelton described colonization as a positive and morally just enterprise, reflecting the dominant western justification for European acquisition and control of foreign territory and peoples. Belgian regional control was “for the benefit of white peoples and for the uplifting of the Africans themselves.”¹¹⁷ This statement illustrated the paternalistic viewpoint adopted by the Belgians toward the native African groups that resided in the Congo region. The colonial government viewed the indigenous people as a subaltern group and thus implemented policies to manage labor and later to manage SS in the region.

¹¹⁷ John J. McKelevy, Jr. *Man Against the Tsetse: Struggle for Africa*, (Ithaca: Cornell University Press, 1973), 39.

Shelton observed SS during his travels and initially the travelers and researchers attributed the disease to poor sanitation and bad water in the many rivers which traversed the region. The expedition attempted to treat the sick with Quinine (an effective treatment at the time for malarial fever), but the results were discouraging. Shelton also noted that local doctors lanced the swollen glands and boils which were later understood to be indicative of the disease, in an attempt to drain the sickness from the body. These treatments were unsuccessful in alleviating symptoms or helping those infected avoid their imminent death.¹¹⁸

In 1903 Roger Casement, a friend and colleague of Shelton, returned to the Belgian Congo to observe conditions and to study the region's ecology. Casement worked for the British Foreign service, was the British Consul to the Belgian Congo from 1901-1904, and also had a personal interest in natural history and diseases. He reported on the labor and sanitary conditions in the Congo and referenced the negative effects of SS on the Congolese.¹¹⁹ He reported that the Belgian colonial enterprise had made drastic changes to the areas topography, and noted appalling labor relations between the Belgian and the Congolese. Casement referenced the slave-like conditions in which the colonial government forced natives to collect rubber and to transport products to local waterways for export. In conjunction with "a diminished and an exhausted population," Casement observed a greater number suffering from SS, emaciation, and lethargy. On the surface, it appeared that the Belgian colonial enterprise had, in less than twenty years, upset a balance which had governed the actions and lives of the natives for centuries. While SS had been present in the region, evident by the awareness of and avoidance of areas where the tsetse fly was most populous, it had not existed in epidemic proportions prior to colonialization. Poor sanitation, lack of healthcare and proper nutrition, forced labor, and the

¹¹⁸ Ibid., 39

¹¹⁹ "Sir Roger Casement 1864-1916," accessed April 2, 2015, <http://www.northantrim.com/rogercasement.htm>.

transport of people and products along waterways made it easy for “people, pests, and diseases to travel.”¹²⁰

While SS had a historical legacy in the Congo region, trade and colonial development facilitated the transfer of disease and resulted in epidemic during the first decade of the twentieth century. The area, covered in thick jungle interspersed by a network of rivers and tributaries, presented a wealth of resources in the form of rubber and gold. In order to access and transport these raw materials, the colonial government established an advanced trade network using the areas abundant waterways. Additionally, harvesting rubber required workers to enter the tsetse habitat, increasing human exposure to the fly and to Trypanosomiasis. “In the Congo Free State, as the intensified Belgian presence caused increased movements of people and their pathogens, SS spread along the rivers,”¹²¹ and quickly grew to epidemic proportions. Outbreaks coincided with the movement of soldiers and laborers, and the disease spread to areas where it was previously relatively rare. The map below shows the central African region controlled by Belgium at the turn of the twentieth century.

¹²⁰ McKelevy, *Man Against the Tsetse: Struggle for Africa*, 40-41.

¹²¹ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940.” *PLOS Neglected Tropical Diseases*,” 5.



MAP 3: The Belgian Congo Region of Central Africa¹²²

Casement’s testimony coincided with recognition by the colonial government that the SS problem heavily conflicted with the colonial incentive to turn a profit from these resources. In response, King Leopold II and the colonial government invested heavily in the research and subsequent treatment for the epidemic. This investment, in many ways retroactive, conflicted with the testimony by Casement that sanitary conditions in hospitals were appalling and the care in European versus African hospitals differed dramatically; “the latter he characterized as an unseemly place” blaming the disease and poor healthcare on the rapid population decline among native groups since colonization.¹²³

Casement’s testimony pertaining to the Belgian government’s poor treatment of the Congolese highlighted the greater issue of colonial domination and imperial gain at the expense of the indigenous populations. King Leopold II and his colonial enterprise were notoriously brutal to the Congolese as imperial gain and economic goals motivated a military takeover of

¹²² “King Leopold’s Ghost,” accessed April 3, 2015, <https://rhapsodyinbooks.wordpress.com/2008/09/10/king-leopolds-ghost-by-adam-hochschild>.

¹²³ McKelevy, Jr. *Man Against the Tsetse: Struggle for Africa*, 41.

lands formerly claimed by the diverse group of tribes and chiefdoms which occupied the Congo region. Leopold, like many other European leaders, was interested in African colonies to promote the power and prestige of his nation, and to secure economic resources to power industrialization.¹²⁴ Secondary to their economic goals, western powers justified African land acquisitions as being part of a philanthropic mission expose Africans to Christianity, education, modern healthcare, and to otherwise ‘improve’ the African landscape and infrastructure.

In the Congo region, the Belgian army forcibly acquired lands and rendered the native population subservient to the European powers that possessed guns and other weaponry which was superior to that of the conquered. Leopold established military bases along rivers and waterways which allowed Belgian soldiers to police local populations. Belgian forces stormed villages, killing inhabitants, forcing the surviving males into slave labor, and kidnapping women for concubines. Prisoners were often mistreated, and many died of injuries or secondary infections from this abuse. Leopold publically stated that his efforts in the region were philanthropic and he made fraudulent claims that he was providing beneficial services to the natives (such as education and healthcare), when in reality facilities were nonexistent or so substandard that they were of little to no value.¹²⁵ Leopold’s notoriously brutal treatment eventually created an uproar within Europe. Casement’s 1904 report highlighted these atrocities and spoke to the egregious mistreatment and horrible conditions in the Congo region. In response, the Belgian parliament eventually forced Leopold to forfeit his control over the colony in 1908.¹²⁶ This period coincided with a major SS epidemic in the region, which was arguably

¹²⁴ Adam Hochschild, *King Leopold’s Ghost*, (New York: Houghton Mifflin Company, 1998), 26-58.

¹²⁵ *Ibid.*, 100-115.

¹²⁶ McKelevy, *Man against the Tsetse*, 40-41.

exacerbated by the conflict, forced labor, and poor sanitary conditions created by the Belgian colonial enterprise.

Prior Leopold's loss of control, the king recognized that SS threatened the native labor force and this presented a tremendous economic threat to his new colony. In September 1903 the Belgian government sponsored a research mission led by the Liverpool School of Tropical Medicine. Drs. J. Everett Dutton, John L. Todd, and Cuthbert Christy led the mission. All three scientists had experience dealing with tropical diseases on the African continent: Todd and Dutton had been working in Senegambia prior to their recall to the Congo Free State for the Liverpool School of Tropical Medicine mission. Dr. Christy had been working in Uganda for the Royal Society's Sleeping Sickness Commission when he was called to join the group in the Congo.¹²⁷ All three had worked together in Uganda as part of the London School of Hygiene and Tropical Medicine research mission, and are the same historical figures whose contributions were discussed in the previous chapter. These scientists were experienced in identifying the trypanosome in blood and spinal fluid, and had observed the clinical symptoms associated with SS in thousands of patients in Uganda and Senegambia. The group's mission was to study SS in the region, to assess sanitary conditions in major economic centers such as Boma and Leopoldville, and to continue to study the association between trypanosomes and the disease as seen in both humans and wildlife.¹²⁸

The expedition made its headquarters in Leopoldville, and was assisted by Dr. Inge Heiberg who was a Belgian doctor already working in the area. Heiberg, a reserve captain in the

¹²⁷ J. Everett Dutton, John L. Todd, and Cuthbert Christy. *Reports of the Trypanosomiasis Expedition to the Congo 1903-1904*, (London: Williams & Norgate for the University Press of Liverpool, 1904), 1-2

¹²⁸ BMJ, "The Liverpool School of Tropical Medicine and Trypanosomiasis," *The British Medical Journal* 2, no. 1 (August 22, 1903): 427.

Norwegian army, had been employed by the Belgian government to study and attempt to treat and control SS in the region. Heiberg's experience in tropical medicine led to a nearly twenty year career in which he became the director of the first SS isolation camp in Ibemo in 1907 and the first *Medican en chef* of the Congo in January of 1911.¹²⁹ The Belgian Colonial Government, whose massive investments in trade and infrastructure were threatened by this disease, spared no expense in facilitating this research mission, and regularly conscripted doctors and researchers from outside of Belgium to assist in solving SS problem. In addition to funding research missions and hiring tropical medicine specialists, the colonial government invested in domestic infrastructure. For example, upon the request of the Liverpool mission, the government erected a new hospital Leopoldville for the study and treatment of SS patients and for the benefit of the scientists studying the disease. Prior to this, available native care facilities were substandard and unsanitary.¹³⁰

Dutton, Christy, and Todd described their experiences and findings working in these hospitals in the first and second progress reports of the Sleeping Sickness research mission. These notes outlined the procedures, findings, and recommendations made to the Belgian Congo based upon the conditions observed and the results gathered through experimentation and observation. The progress reports included references to distribution methods, research strategies and results, policy recommendation, and data analysis. Additionally, the scientists made suggestions for the improvement of sanitary conditions and the management and control of SS in the colony, as well as suggested diagnostic and treatment methods.¹³¹

¹²⁹ Martinez Lyons, *The Colonial Disease: A Social History of Sleeping Sickness in Northern Zaire, 1900 – 1940*. (Cambridge: Cambridge University Press, 2002), 76.

¹³⁰ Dutton, Todd, and Christy, *Reports of the Trypanosomiasis Expedition to the Congo 1903-1904*, 1-2.

¹³¹ BMJ, "The Liverpool School of Tropical Medicine and Trypanosomiasis." *The British Medical Journal* 2, no. 2225 (August 22, 1903): 427.

The progress reports outlining the observations during these first few months at the hospital in Leopoldville indicated that this epidemic was unique in several ways. First, the main symptom of the disease was not lethargy. In fact, many advanced cases did not show this as a major symptom even prior to death. Additionally, observations of cases in the surrounding areas also demonstrated that lethargy was not a marked feature of this epidemic. More common symptoms included weakness, headache, emaciation, enlarged lymphatic glands, and dry/scurfy skin.¹³²

In Leopoldville and surrounding areas, the scientists observed a state of hysteria in communities where SS actively affected the population. The colonial transportation network brought people and products into areas which were previously isolated from outside contact and as a result, many of these communities had not experienced the disease in such a capacity as they existed outside of the natural tsetse habitat. One of the major problems with this lack of experience and cultural understanding with the sickness dealt with patient care. “Children and even adults were, as soon as the slightest symptoms developed, liable to be isolated and shunned by everyone, causing eventually a state of emaciation and filth which ended sooner or later in death.”¹³³ Secondary complications and infections were often the immediate cause of death due to this lack of supportive care. As a result, the mortality rates from SS in some communities may have been distorted.

¹³² J. Everett Dutton and J.L Todd. “Human Trypanosomiasis in the Congo. Being the First Progress Report of the Expedition of the Liverpool School of Tropical Medicine to the Congo 1903.” *The British Medical Journal* 1, no. 2247 (January 23, 1904): 186.

¹³³ *Ibid.*, 186

Within the Leopoldville hospital, Dutton, Todd, and Christy focused on identifying the disease pathogen to confirm that Trypanosomiasis was the culprit in the Congo. The progress report described disease symptom progression in many SS patients. This included an increase in neurological symptoms such as confusion and disorientation, as well as temperature fluctuations. Additionally, the team reported that frequency and concentration of trypanosomes in the blood and cerebro-spinal fluid of patients often fluctuated. Emaciation was another major symptom seen in many victims either initially or as the disease progressed.

Because the epidemic presented different clinical symptoms than previous outbreaks, the team needed to first definitively establish that they were in fact dealing with Trypanosomiasis. Dutton, Todd, and Christy used inoculations to determine if the trypanosomes found in the blood of infected patients were in fact responsible for observed disease symptoms. The team inoculated animals in order to induce disease symptoms which allowed scientists to establish a causal link between the trypanosome and SS. They injected the fluid taken from a diseased victim (either secretions from enlarged glands, blood, or cerebro-spinal fluid containing trypanosomes) either under the skin or in the central body cavity of rats, mice, monkeys, guinea pigs, or other test animals. The scientists found that infection occurred only when they administered fluids directly under the skin, as opposed to into the body cavity. Additionally, some animals did not show any disease symptoms even though parasites were found in their blood and other fluids. Because infection occurred after subcutaneous inoculation, and because trypanosomes appeared in the animals' body fluid, the team was able to determine that they were in fact dealing with another SS epidemic. Furthermore, the physical

characteristics of the trypanosomes when studied under a microscope resembled those of the Uganda and Senegambia epidemics. Due to these findings, the team declared that “We have therefore no reason to suppose that the organisms seen by us in the Congo are other than *Trypanosoma Gambiense*” in these animal experiments.¹³⁴

After establishing that *T.b Gambiense* was causing SS in the region, the team repeated microbiological observations within the human population. For example, a twenty four year old male agricultural worker, observed in late September 1903 after being infected in July, had experienced massive physical deterioration and was extremely thin with poor muscle tone and lack of physical strength and coordination. Poor physical condition increased as the patient approached death, and finally succumbed to the disease months after infection. Researchers found trypanosomes in the blood and spinal fluid of this individual, and the characteristics and physical attributes of the trypanosomes were described and illustrated in the progress report. The team repeated these observations in eighteen patients, and found that the parasites resembled those of other SS infections outside of the Congo region.¹³⁵ This is significant because it reflected the use of germ theory Theory principles and the analysis of empirical data to identify, name, and follow the spread of an epidemic. Despite discrepancies in its presentation in the Congo region, where lethargy was not a major symptom as it was in other regions, this microbiological analysis proved that the scientists were working with the same disease pathogen.

The second progress report illustrated in greater detail the specific symptoms of SS patients, the varying stages of the disease, and the categories and symptoms which

¹³⁴ *Ibid.*, 188

¹³⁵ *Ibid.*, 188

classified each phase. Many of these observations were conducted during the first four months of the expedition when the group was stationed at the hospital in Leopoldville. This hospital, following reports of poor sanitation and lack of patient care, had been recently constructed to house the native sick and to facilitate the Liverpool team's research efforts.¹³⁶ The progress report outlined the cases in the following ways: Type A: cases with no definite symptoms of illness; Type B: cases with few symptoms; Type C: "Fatal cases showing well-marked symptoms, the most notable being fever, lassitude, weakness, and wasting."¹³⁷ This third category was further subdivided to include those cases without sleep symptoms (group one), and those cases with sleep symptoms (group two).

The aforementioned categories, however, were not constructed based on new scientific data so much as shaped by individual's ability to continue working while infected with the disease, and the potential longevity and economic value this diseased individual presented to the colony. For instance, the report contained the following descriptions of Type A patients who were hospitalized during the research mission. "General good body condition, but lymphatic glands enlarged. Patient was able to remain in work for months after initial hospitalization."¹³⁸ Researchers described another patient as being "In generally good condition. Able to work. Glands hard and easily palpable."¹³⁹ More testimonies describing these Type A patients concluded that enlarged glands were the most consistent feature. The patients' ability to continue working until the symptoms began was most relevant because it extended their effective economic

¹³⁶ Dutton, Todd, and Christy, *Reports of the Trypanosomiasis Expedition to the Congo 1903-1904*, 13.

¹³⁷ *Ibid.*, 14

¹³⁸ *Ibid.*, 15

¹³⁹ *Ibid.*, 15

output for the colonial government. Consequently, however, Type A individuals could potentially spread the disease.

Type B patients began to show body deterioration, which included decreased cleanliness, loss of body weight and muscle mass, the characteristic enlarged glands, and decreased economic capacity. This group proved the most difficult to diagnose and as a result, it was difficult to determine the workers' longevity when presenting these symptoms. A Type B patient admitted to the hospital complained of diarrhea and knee and chest pain. The worker had "thin body condition and deteriorating muscles. [The] skin [was] dry and dirty. Lymphatic glands enlarged and hard. Very weak, difficult to walk and stand."¹⁴⁰ Doctors found trypanosomes in blood samples taken on January 5, 1904. However, samples taken the following day showed no trypanosomes. The appearance and subsequent disappearance of trypanosomes in blood samples, as well as a fluctuation in the number of the parasites, is a feature seen in many of those whose blood was collected and studied over a period of time.¹⁴¹ This observation highlighted one of the difficulties in diagnosing the disease in situations where prolonged hospitalization and a series of blood tests or cerebro-spinal taps were not possible. The general deterioration in body condition, muscle weakness, and flu-like symptoms also paralleled the initial onset of malaria. Definitively diagnosing the disease was yet another obstacle in the epidemic's control and eventual eradication.

Type C patients were considered terminal with death occurring in a relatively short period of time. A group one patient, admitted December 29, 1903, presented with

¹⁴⁰ *ibid.*, 17

¹⁴¹ *ibid.*, 18

the characteristic body deterioration and was weak, thin, and had dry skin with little to no muscle tone. The patient was also confused, had tremors, and had hard and enlarged glands. Over the next few weeks the patient lost the ability to walk or get out of bed, even to relieve himself, and lost the ability to speak. He died on January 21, 1904. Other patient examples presented with similar symptoms. Slow loss of mobility coinciding with a lack of awareness and an inability to eat or to control other bodily functions represented the crossing of the trypanosome into the nervous system and thus damaging mental and physical faculties. This advanced disease stage progressed rapidly and death often occurred within weeks of the symptoms onset.¹⁴²

Researchers also noted that when they found trypanosomes in the patients' spinal fluid, neurological symptoms were more common and more debilitating. "On comparing the cases in which parasites were found in the cerebro-spinal fluid with those in which they were not found, we see that most of the few cases in which drowsiness was a feature, together with cases in which head symptoms, e.g., mild mania, epileptic attacks, flexure contractions, and convulsive seizures, were conspicuous are upon the positive side."¹⁴³ Trypanosomes in the spinal fluid indicated they had crossed the blood/brain barrier and entered the late stage of the disease where death was increasingly imminent. Disease symptoms became more severe, and neurological symptoms became more apparent. This finding, in conjunction with the first report's conclusions that the parasite was in fact *T. b. Gambiense*, marked a major advancement in the understanding and life cycle of the parasite within its human host, and confirmed similar theories made during the Ugandan research mission.

¹⁴² Ibid., 20-23

¹⁴³ Ibid., 43

Patients classified as Group Two patients showed advanced neurological symptoms where sleep was a major disease component. A group 2 patient, a young boy who was first observed on December 11, 1903, was weak, emaciated, and slept continuously. He presented with dry and scurfy skin and hard enlarged glands, and showed neurological disease symptoms. This patient succumbed to secondary infection in the form of bronchitis, and eventually died on February 8th of the following year. He again showed the characteristic loss of muscle mass and general emaciation, with hard and palpable glands and a loss of mental and physical coordination.¹⁴⁴ This example illustrated the Type C, group two patient characteristics and outlined how the research team categorized and subsequently studied the infected. Categories related to effective economic output and symptomatic progression from mild to severe.

Christy, Todd, and Dutton compared the hospital study conclusions with observations of other outbreaks of both human African Trypanosomiasis and nanga. In human and animal victims, the similarities to the Congo sickness were extensive. Most cases across Africa presented the following symptoms: listlessness, weakness, lack of energy, emaciation, erratic temperature fluctuation, frequent disappearance of parasite from blood and cerebro-spinal fluid samples, and a chronic disease in which progression can take months or even years.¹⁴⁵

In the Congo, some patients carried the disease for two to five years before the onset of neurological symptoms. Once the patient began to show symptoms, however, their projected lifespan was generally only two to four months. No patients were known

¹⁴⁴ Ibid., 25

¹⁴⁵ Ibid., 33-35

to have recovered from the disease and the confirmed cases had a 100% mortality rate. Finally, the expedition noted that the many of these deaths resulted from secondary infections and complications. Disease hysteria from the epidemic often resulted in patient abandonment when symptoms resembled SS, even if the symptoms were for another disease such as malaria or influenza. This reflects the culture in which the epidemic occurred, and how fear shaped community responses. Scientific diagnosis further exacerbated this problem, resulting in many abandoned sick who were admitted to SS hospitals or left to die in rural villages. The report also highlighted that:

Thirteen out of twenty-two necropsies performed at Leopoldville showed complications or obvious secondary infections. They are as follows: purulent meningitis, purulent pleurisy and pneumonia, pneumonia and localized tubercle of lung, localized gangrene of the lung, enlarged caseating and breaking down glands in thorax and abdomen, dysenteric ulceration of large bowel, universally adherent pericardium, infiltration of pus in femoral, inguinal, and internal iliac glands.¹⁴⁶

Drs. Bruce and Nabarro, working in Uganda during its epidemic, noted similar statistics regarding mortality due to secondary infections, and similar findings during autopsy examinations. Lieutenant Colonel David Bruce and Dr. Davis Nabarro were members of the first team sent by the London School of Hygiene and Tropical Medicine to Uganda in 1902.¹⁴⁷ Both had extensive experience working in a parallel disease epicenter, and their findings supplemented those of the Liverpool School Team in The Congo. In the Uganda epidemic, a majority of

¹⁴⁶ Ibid., 36

¹⁴⁷ Lieutenant Colonel David Bruce and Davis Nabarro, "Progress Report on Sleeping Sickness in Uganda," in *Reports of Sleeping Sickness Commission No 1*. (London: Harrison and Sons, 1903), 11-12.

these secondary complications resulted from meningitis which indicated that the trypanosome had crossed the blood/brain barrier.¹⁴⁸

In Liverpool, scientists continued to study Trypanosomiasis in a lab setting. Using samples sent back by the Congo expedition, Dr. Wolfertain Thomas, Dr. Anton Breinl, and Dr. S. F Lanton (all of whom had worked with Dutton before his departure) experimented with finding a treatment for the disease. The Congo research team sent seven cases to Liverpool for use in inoculation experiments and treatment trials. Six were natives from the Congo region, and one was a European captain. All of these patients eventually succumbed to the disease during the study. The scientists inoculated animals using the trypanosome-containing fluids of these individuals, and, once they confirmed the disease in the test subjects, experimented with different treatment methods to determine if the disease could be controlled or eradicated.¹⁴⁹ Experimental treatments were also used on the patients from the Congo, and the researchers recorded their findings in the report.

By 1904 the only effective treatment was arsenic, although doctors understood that the side effects of the drug (depending on the methods by which it was prepared) presented a large obstacle in the effective treatment of Trypanosomiasis. “Subcutaneous inoculations of sodium arsenate caused so much pain that this form of medication was abandoned.”¹⁵⁰ In animal trials, sodium arsenate caused ulceration and skin sloughing which often resulted in deadly secondary infections. Some arsenic compounds reduced

¹⁴⁸ Dutton, Todd, and Christy, *Reports of the Trypanosomiasis Expedition to the Congo 1903-1904*, 37.

¹⁴⁹ Wolfertain Thomas and Anton Breinl, *Trypanosomes, Trypanosomiasis, and Sleeping sickness: Pathology and Treatment*, (London: Williams & Norgate for the University Press of Liverpool, 1905), 1-24.

¹⁵⁰ *Ibid.*, 50

the number of trypanosomes in the blood of test animals, along with reduced symptoms and overall physical improvement. However, when treatment stopped, symptoms returned along with trypanosomes in blood and spinal fluid. This indicated that the disease was being managed by the treatment but its effectiveness only lasted so long as it was present in the animals system and it did not fully extinguish the parasite. The group concluded that Atoxyl (a meta-arsenic anilin compound) and trypan red (an aniline dye) compound was the most effective treatment for Trypanosomiasis. Atoxyl was less toxic than other forms of arsenic treatments, but it still caused negative side effects. Despite these side effects, the drug's ability to reduce infection out competed other available treatments. Patients treated with Atoxyl experienced reduced symptoms and reduced infection rates for a time, but the symptoms and parasite frequency in body fluids quickly increased when treatment decreased or ended. By 1904, doctors understood that Atoxyl could prolong a victim's lifespan, but an absolute cure had yet to be created.¹⁵¹ As outlined in the previous chapter, Dr. Koch studied Atoxyl treatments more closely in 1906, and the results from his experiments influenced treatment policies in the Belgian Congo. Despite the known side effects (including blindness), Atoxyl was inexpensive to manufacture, easy to administer, and remained stable in the heat and humidity of equatorial Africa. As a result, it remained in effect long after better treatments with less severe side effects became available.¹⁵² It is also important to note that disease testing and collaboration transcended national boundaries and information passed between scientists working on different epidemics.

¹⁵¹ Dutton, Todd, and Christy. "Reports of the Trypanosomiasis Expedition to the Congo 1903 -1904," preface page 2, and Wolferstan, Thomas, and Breinl, "Trypanosomes, Trypanosomiasis, and Sleeping sickness: Pathology and Treatment," 50-63.

¹⁵² Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900-1940," 4.

In 1905, Dr. Todd published “The Distribution, Spread, and Prophylaxis of Sleeping Sickness in the Congo Free State.” In this document, he summarized the general findings of the 1903-1905 expedition, and made recommendations for diagnosing and managing SS. These recommendations included forced isolation of the infected in SS camps, restricted travel for those infected into uninfected areas, and medical testing and examination and treatment (generally with Atoxyl) for those who presented disease symptoms. The expedition began in September of 1903 and lasted for twenty three months, traveling over two thousand miles. The information gathered included observations made in the Leopoldville hospital, as well as the testimonies of missionaries and local leaders as to the proliferation, frequency, and symptoms of the disease. The information suggested that SS was more prevalent among transient populations (soldiers, migrant laborers, etc), and was more common along waterways.¹⁵³

Dialogue with local groups indicated mixed ideas about the causes of SS. Todd writes that:

We have been told that sleeping-sickness was caused by eating manioc, lacking salt, smoking, drinking palm wine, or by excessive coitus and that it might be contracted through using drinking utensils employed by infected persons, or through coitus with them. All of these statements seem mistaken. In an infected population, all classes, male and female, adult and children, are equally susceptible.¹⁵⁴

This statement is interesting because it conflicted with the testimonies of later historians who claimed that the indigenous populations did in fact understand the connection between the tsetse fly and the nanga and human SS diseases, and made efforts to avoid tsetse infested areas. Todd’s statement demonstrated that there were multiple

¹⁵³ Dr. J. L. Todd. “The Distribution, Spread, and Prophylaxis of Sleeping Sickness in the Congo Free State.” *Transactions of the Epidemiological Society of London* (1905): 1-4.

¹⁵⁴ *Ibid.*, 3

explanations circulating in the area which hypothesized SS transmission methods. These theories reflected the notion that immoral behavior and poor sanitation were at the heart of disease epidemics, and often insinuated that natives were more prone to such behavior do to their inferior status.

Todd himself acknowledged that isolated increases in SS cases coincided with famine or conflict. The expedition also made note that *glossina papalis* (the species of tsetse fly which carried *T. b Gambiense*) lived in abundance along the route traveled, and was particularly numerous during the rainy season and in riverside habitats. The scientists noted that “Apart from this, and expecting always the susceptibility of fishing and river-side people, no peculiarity or tribal custom has been observed to predispose natives to sleeping-sickness.”¹⁵⁵ This suggested that scientific susceptibility superseded any cultural conditions which could have increased disease transmission rates. These testimonies and scientific counter arguments highlighted that social and scientific justifications combined to create a past and present conception of this epidemic. Scientific study and discovery did not eradicate a more traditional justification. Growing evidence to the contrary would slowly influence the traditional assumptions regarding disease transmission, but culture and prejudice strongly influenced popular belief.

The significance of the first passage which refutes the common misconception about SS transmission is important for two reasons. First, a great deal of information gathered by the expedition whilst traveling outside of Leopoldville was done so through the testimonies of missionary leaders and other Europeans who were living among the

¹⁵⁵ Todd, *The Distribution, Spread, and Prophylaxis of Sleeping Sickness in the Congo Free State; from the Expedition to the Congo of the Liverpool School of Tropical Medicine*, 4.

natives in these more remote regions.¹⁵⁶ The association between disease and sinful behaviors such as excessive drinking and coitus reflects the Christian cultural assumptions which predominated nineteenth and twentieth century conceptions of disease. Headrick has argued that a great deal of colonial policy was heavily influenced by the Catholic Church whose missionary presence in the region was extensive. Diseases were often seen as a form of divine punishment for those who violated religious law. Todd's findings demonstrated the impact of science and Germ Theory on the cultural understanding of diseases and their causes. The established link between the tsetse fly and SS and the identification of the *T. b. Gambiense* as the pathogen responsible, helped to refute the religious justification for African lethargy, at least among scientific groups

The disease was more common along waterways, due to increase tsetse fly populations, and greater exposure of humans through fishing and other activities. Some missionary groups reported that SS cases in their localities were actually less during the epidemic than during other times in recent history. The spread of the disease was not universal or random, but it depended on the transfer of goods and people from infected communities to uninfected communities.¹⁵⁷ Todd discussed the ways in which colonization, trade, and conflict increased human travel between communities. For example, he states that “natives came from so far as the neighbourhood of Bandundou to carry loads over the caravan route from Matadia to Leopoldville. Soldiers and labourers employed in the Upper River often came from the infected Lower Congo... In this way large numbers of persons from the infected districts entered this uninfected region.”¹⁵⁸

¹⁵⁶ Ibid., 4

¹⁵⁷ Ibid., 5-7

¹⁵⁸ Ibid., 8-9

Trade and agriculture required that human laborers travel to economic epicenters, and often these groups brought SS from areas of infection to more remote regions where the disease had less of an impact. Todd's report demonstrated that the Liverpool School of Tropical Medicine expedition understood the connection between colonial expansion and infection rates, and considered this a more prominent causal factor than native custom, lifestyle choices, or physical constitution.

Yet another challenge in controlling SS and its spread dealt with definitively diagnosing those infected, and preventing these individuals from traveling to uninfected regions. Identifying trypanosomes in the blood or spinal fluid required a physician, as well as the medical facilities to perform the extractions, and the equipment to view and test the blood and other fluids. As indicated in "The First Progress Report," trypanosomes were not always present in the blood each time it was tested. As a result, scientist needed to preform long-term observation and repeated blood and/or cerebro-spinal tests in order to clinically diagnose the disease. When working in the field away from hospital faculties and doctors, these diagnostic methods were logistically challenging.

In his report, Todd argues that identifying swollen glands through palpation was a definitive method by which the disease could be identified. Enlarged superficial lymph glands were a common feature among those suspected of carrying SS, even before symptoms became evident. In 1904, Todd identified trypanosomes in the fluid extracted from one of these glands, suggesting that the swelling was in fact a direct result of infection. These tests were also repeated in Uganda with similar results. In two independent studies, Todd and his team tested patients with swollen glands to see if

trypanosomes existed in their bodily fluids. In the first case, 97.2% of the 250 observed patients carried trypanosomes. The second test involved 130 patients, with a 98.5% positive infection rate.¹⁵⁹

Whatever the cause, physicians needed fast field diagnosis in practice and hospital studied helped to determine an effective clinical approach. Todd referenced another test in which the expedition visited an area where SS was not present. They examined 2,414 natives by palpating glands to determine if they were enlarged. Within this healthy population, only 1.4% had enlarged glands, and the rest were considered normal.¹⁶⁰ These experiments suggested that gland palpation (both lymphatic and cervical) could be used by non-physicians in rural populations to determine the infection rates, and thus prevent infected individuals from traveling from the area. Todd concluded that “Gland palpation is a wonderfully simple and very accurate method of detecting especially those cases of Trypanosomiasis in which marked clinical signs are wanting.”¹⁶¹ Todd argued that this valuable test would solve the field diagnosis issue and allow government officials to diagnose and monitor those infected.¹⁶²

After establishing the connection between SS transmission and the growing colonial enterprise, and suggesting a method by which the infected could be identified, Todd made a policy suggestion which involved quarantine and controlled travel in the region, to be enforced by the Belgian soldiers in the Congo. Todd suggested that inspection posts be placed along major roads where colonial authorities examined those

¹⁵⁹ *ibid.*, 11

¹⁶⁰ *ibid.*, 11-12

¹⁶¹ *ibid.*, 12.

¹⁶² John L. Todd, “The Prevention of Sleeping Sickness,” *The British Medical Journal* 2, no. 2493 (October, 1908): 1061.

who passed through and, if infected, prevented these individuals from traveling to certain regions. Also, the government would remove those who were infected and living or working in areas of low infection, to infected areas in order to isolate the healthy groups from the unhealthy groups. Finally, Type A patients could be relocated to work sites within infected zones. In this way, the colonial government could control disease spread while preserving the colony's economic priorities. Some of these measures were already in effect when Todd wrote and published his recommendations. These policies included regulating travel of persons suspected of carrying the disease, and increasing use of gland palpation as a diagnostic tool.¹⁶³

The data outlined in the expedition's progress reports and in other scientific papers describing disease treatments and management were very influential in the subsequent policy decisions made by the colonial government. The Liverpool School of Tropical Medicine confirmed that the pathogen in the Congolese epidemic was in fact *T. b. Gambiense* and, by this time it was understood that the tsetse fly was instrumental in transmitting the disease between human and animal alike. Incentives to control the disease came from a desire to maintain the economic profitability of the colony through the export of raw materials such as rubber and gold. Preservation of a viable work force was instrumental in achieving this goal. Following the completion of the expedition, Dutton, Christy, and Todd recommended that the sick be isolated in SS camps in order to monitor and control the spread of the disease. Additionally, Todd recommended establishing check points along terrestrial and aquatic travel routes to monitor the flow of people from infected to healthy areas and vice versa. As a result, the colonial

¹⁶³ Ibid., 12-14

government established a series of *lazarets* or sick camps for the infected, and cordon sanitaires or quarantine zones around infected regions.¹⁶⁴

The colonial government effectively created a police state where the authorities monitored the movements and actions of the indigenous people. Government and law enforcement officials used gland palpation to diagnose the sick. “Those suspected of being infected were herded into these camps, where they were isolated from outsiders and injected with Atoxyl. These camps proved to be unpopular because of the painful treatments, poor conditions, lack of food, and permanent separation of patients from their families. To prevent the sick from escaping, they had to be guarded by soldiers.”¹⁶⁵ The government employed search and isolate tactics in which local leaders and missionaries were encouraged to report suspected SS victims to local authorities, after which the individuals were forcibly removed to the SS camps and denied visitation from friends and family members.¹⁶⁶

Isolation camp construction and policy enforcement reflected the militaristic approach adopted by the Belgians in the Congo colony. Natives were conscripted to build the camps, and forced relocation for laborers and for the sick was done with military force. “In August 1903 sleeping sickness was added to the official list of infectious and contagious diseases [which] sanctioned the use of army and fines to enforce isolation.”¹⁶⁷ These policies were developed without the consent or consultation of local leaders and were used as a way for the Belgians to tighten control over the

¹⁶⁴ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900 – 1940,” 3.

¹⁶⁵ *ibid.*, 5

¹⁶⁶ Maryinez Lyons, “From ‘Death Camps; to Cordon Sanitaire: The Development of Sleeping Sickness Policy in the Uele District of the Belgian Congo, 1903 – 1914.” *The Journal of African History* 26, no.1 (1985): 77-79.

¹⁶⁷ *Ibid.*, 79

colony. Epidemic severity was often greater in the more economically profitable regions containing such resources as rubber and gold, thus the need to preserve the vitality of the work force combined with a desire to develop a subservient and obedient labor pool were the major goals of the SS policies.

Historian Maryinez Lyons outlined the prison-like conditions inside the camps. After forced removal and isolation from friends and families, the sick were often subject to painful and hazardous treatments. General care, in the form of adequate food and support staff, was however, lacking, and thus the public perception of the camps was extremely negative. Lyons states that “once inside a lazaret, victims received Atoxyl injections and periodic examinations which were carefully recorded by registers. They were kept in varying degrees of isolation, from complete lock-up to relatively unrestrained surveillance. According to Dr. Heiberg, prisons-style confinement was administratively preferable for both Europeans and Africans.”¹⁶⁸ Colonial authorities gave individuals little choice regarding care, and camp policies did not allow for support from other natives. As a result, many did not report cases and avoided colonial officials in order to remain free of these camps. In some cases, family members attempted to hide their loved ones in order to keep them from admittance into what became known as ‘death camps.’

Despite the unpopularity of the lazaret policy, the number of camps steadily increased. In 1910 alone the total number increased from eleven to twenty six within the colony, and the size and complexity of camp structures also evolved. By this time the logistics of total isolation, and the costs for labor and enforcement, made this approach

¹⁶⁸ Ibid., 82

economically unattractive. Rather than strict prison camps, lazaret villages appeared in which patients could live in family groups and receive outpatient treatment (still in the form of compulsory Atoxyl injections) from colonial doctors. Additionally, those in the early stages of the disease who were still able to work were expected to perform their occupational duties until such time that disease symptoms made this impossible. The open lazaret-villages allowed for the transfer of workers from the village to work sites, although these individuals remained confined to epidemic regions as to not spread the disease to healthier districts.¹⁶⁹

While the military-style camps were unpopular, the investment made by the colonial government was extensive and important in the ultimate control of the disease. Todd, Christy, and Dutton's research gave colonial authorities a way to identify the sick and suggested methods by which their travel could be controlled and isolated to areas where the disease was present. Additionally, the increasing complexity of the colonial transportation networks and existence of mandatory health checks led the colony to become relatively sophisticated and organized when it came to identifying and treating SS. By the 1930's nearly 70% of the population or nearly three million natives were examined yearly. The government set up a network of rural clinics, hospitals, and treatment centers for the sick. Overall these treatment methods, coupled with diagnosis and population control, resulted in decreased transmission rates and a decline in new cases by the 1940's. "The Belgian Congo won praise from Europeans for offering the most effective and comprehensive medical care in any European colony.... For Africans, however, it meant living in a police state, with a healthcare system that only overcame an

¹⁶⁹ Ibid., 79-85

epidemic that European colonial rule had exacerbated in the first place.”¹⁷⁰ The SS epidemic gave the Belgian government a vehicle by which it was able to implement and enforce widespread militarized control over a large population. Its forced treatment, labor, and restricted travel proved to be an early example social engineering in Africa. Compulsory western practices, which focused mainly on economic preservation, fundamentally upset the lifestyles of millions living within the Congo region. Despite being touted as a leader in colonial medial policy, the sources suggest that little dignity and value was awarded to those living and working in the jungle. The fact remains that medical research gave the colonial government cause to enact laws which controlled both the disease and people. Europeans spoke little of any social consequences for these methods, and celebrated the scientific advances made with the testing and manipulation of human lives through force. Money, control, and scientific discovery remained distinct priorities in understanding and combatting the Congolese Trypanosomiasis epidemic in the first decade of the twentieth century.

¹⁷⁰ Headrick, “Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900 – 1940,” 5.

Conclusion

The Sleeping Sickness epidemic which ravaged equatorial Africa at the turn of the twentieth century was a multifaceted and extremely complex episode of colonial history. In the Belgian Congo, SS had existed for centuries, but the indigenous populations had developed methods by which they were able to control the disease. This included living in areas which were free of tsetse flies, limiting exposure to tsetse habitats, and traveling routes which avoided tsetse hot spots. When the Belgian government acquired the region and established a colonial trade and transportation network, they fundamentally upset the delicate human/tsetse balance which had been established over centuries. The numerous traversable waterways in the region became the highways by which the disease and its vector were imported to regions previously unaffected by SS. As a result of colonial expansion, the indigenous people suffered catastrophic losses in life and in personal freedom.

Another component to the Congo epidemic lay in the methods by which the colonial government controlled and manipulated the indigenous population. Colonial authorities forced Africans into labor under slave-like conditions, and sent these workers into the dense forests to collect materials such as rubber and gold. This resulted in widespread exposure to the fly habitat and the disease. Additionally, poor sanitary conditions, hard work, and inadequate nutrition left the population weakened and more susceptible to both SS and the secondary infections which caused a large number of casualties. As the economic vitality of the colony diminished under the epidemic, the government hired a research team from the Liverpool School of Tropical Medicine to study the disease and propose a solution. As a result of these recommendations, the colonial government established SS camps where authorities forcibly removed infected people and give them Atoxyl injections which had painful side effects including blindness. The

government also monitored travel and quarantined whole populations who were suspected of harboring the disease. In the Belgian Congo, SS allowed the government to establish a police state in which Europeans regulated the Africans' movements, occupations, exposure, and disease treatment, while denying these individuals human rights and personal dignity. King Leopold's brutal tactics combined with his thirst for power and wealth motivated his colonial policies which reflected Belgium's economic and resource needs.

The story in Uganda proved to be equally detrimental, but with subtle differences. In Uganda, SS was arguably a new disease, and the indigenous groups had little to no experience with the pathogen. Historians speculated that the disease was imported with traveling armies from the west, and as the trypanosomes entered a new virgin population, the destruction was rampant. Millions succumbed to infection in the first decade of the twentieth century. The disease epicenter was located near the shores of Lake Victoria where commerce and transportation resulted in the rapid spread of SS. The London School of Hygiene and Tropical Medicine, which was sent to study the disease in 1901, established a scientific link between the tsetse fly and Trypanosomiasis, and recommended polices which removed humans from the fly habitats, and reduced habitat through brush removal and livestock and wildlife management. The British colonial government mandated forced evacuations of tsetse-infested areas, requiring that people leave their homes and traditional lifestyles in order to reduce disease exposure. Additionally, the government limited access to the lake and curtailed the economic independence of groups who depending on fishing and trade. The government also mandated that natives cut back the woody brush habitat that housed the fly, and even commissioned workers to collect flies in an attempt to decrease their numbers. These policies, although less invasive than the Belgian police state, were highly disruptive to the indigenous population. Additionally, the epidemic,

which resulted from colonial expansion, was far more damaging to the native populations than to the European officials who remained relatively protected from exposure. Ugandan policies differed from those in the Congo due in large part to the landscape and economic capacities of the two different regions. In Belgium, the government forced indigenous peoples into the jungles and to transportation epicenters to collect and transport rubber. In Uganda where trade and agriculture proved to be the more profitable enterprises, forced depopulation was a more viable option. In both colonies, the economic goals motivated the policies which were designed to limit the cost of SS outbreaks while inflicting minimal damage on the colony's economic output.

Sleeping Sickness policies in these two disease epicenters resulted from a combination of climate and commitments to germ theory, which motivated researchers. Thousands in both regions were palpated, had blood drawn, and underwent invasive procedures such as spinal taps and gland surgery, in order to allow the scientists to study and identify the disease. Robert Koch's island research laboratory was designed to test arsenic treatments and their side effects using the native human population, as human testing was perceived to be more valuable than animal experiments. During this time period, scientists were able to identify the pathogen, understand the lifecycle and transmission within the tsetse fly, and track the disease spread and distribution based upon fly habitats. Additionally, while Atoxyl was extremely invasive and dangerous, it was the first in a long series of medications which were relatively effective in treating SS. Any medical advancements were, however, made at the expense of thousands of humans who were used to test these drugs and who suffered from debilitating side effects. Resulting colonial policies reflected the scientific findings and prioritized economic vitality over African dignity. British policies may have been less aggressive than those in the Belgian police state, but they were no less disruptive. The government forced thousands to vacate homes and

economic sources and created a situation in which countless communities were unable to provide for their basic needs.

This case study is also interesting because, despite the competitive climate of scientific discovery and imperial competition, the epidemics' severity motivated collaboration between competing groups in an attempt to find a cure. Scientists published discoveries and cooperated in sharing treatment methods and mitigation strategies across colonial borders. SS affected not only Belgian and British colonies, but other sub-Saharan colonies such as German East Africa, Portuguese Senegambia, and South Africa. Germ theory, made famous by the work of Robert Koch and Louis Pasteur, represented a new revolutionary way of combatting disease epidemics, and attracted scientist from across Europe. New journals and institutions made information available to the western scientific community. The London School of Hygiene and Tropical Medicine, the Liverpool School of Tropical Medicine, the Pasteur Institute, and Berlin's institute for infectious disease represented a few of the leading institutions where germ theory research was ongoing. Many institutes welcomed foreign scientists, creating a cross-cultural exchange in which ideas were exported to other nations. Scientific gatherings, such as the International Sanitary Conference (1851), gave different groups the opportunity to present new findings and collaborate with colleagues. "The competition for credit and success was intense, and it was embedded at the local, national, and international levels, as researchers fought for recognition, financial support and career advancements... Often the competition was the collaboration: the field was furthered when scientists spurred each other on" by combining individual findings with new techniques in a culture of discovery.¹⁷¹ SS, due to the epidemic's severity and its widespread colonial impact, relied on the findings of major researchers who specialized in the

¹⁷¹ Deborah Neill, "Paul Ehrlich's Colonial Connections: Scientific Networks and Sleeping Sickness Drug Therapy Research, 1900-1914," *Social History of Medicine* 22, no. 1 (February 2009): 63-64.

disease and in tropical medicine, even if their national origin differed from that of the colonial authority. The disease fostered a culture of collaboration which was motivated by a tremendous threat to life and to economy.

In this episode of African history, colonialism, tropical medicine, and ecology become major factors in how Europeans approached the SS problem in Africa. Imperial actors imposed western notions of disease and sanitation on a population whose understanding and acceptance of these rules conflicted with traditional lifestyles and cultural practices. Additionally, the Germ theory approach to solving the disease problem within a laboratory setting proved to be less effective than understanding the natural world and limiting human and fly interaction. Epidemics of this magnitude, when human-induced, speak to the importance of ecological study and the power of the natural world, as a failure to understand and respect the rules which governed these habitats can be disastrous. Policies to control the outbreak were (in both Uganda and the Congo) reactive rather than proactive, and the environmental consequences were catastrophic as habitat loss and human casualties spread across the region. Colonial expansion, imperial competition, and a disregard for the African perspective and life were the fundamental catalysis which resulted in the painful and prolonged death of millions.

Human African Trypanosomiasis remains today a formidable human health and economic obstacle in many parts of the African continent. SS is found in the thirty six sub-Saharan African continents where the tsetse fly can survive in the wild. The WHO asserts that the disease mostly affects those living in rural and agricultural settings where the fly habitat is undisturbed by urban development. In these regions, the ability to manage the fly, treat the victims, and spread awareness of the disease is also limited due to the remote locations of some towns and villages. Human African Trypanosomiasis, therefore, remains a disease of the

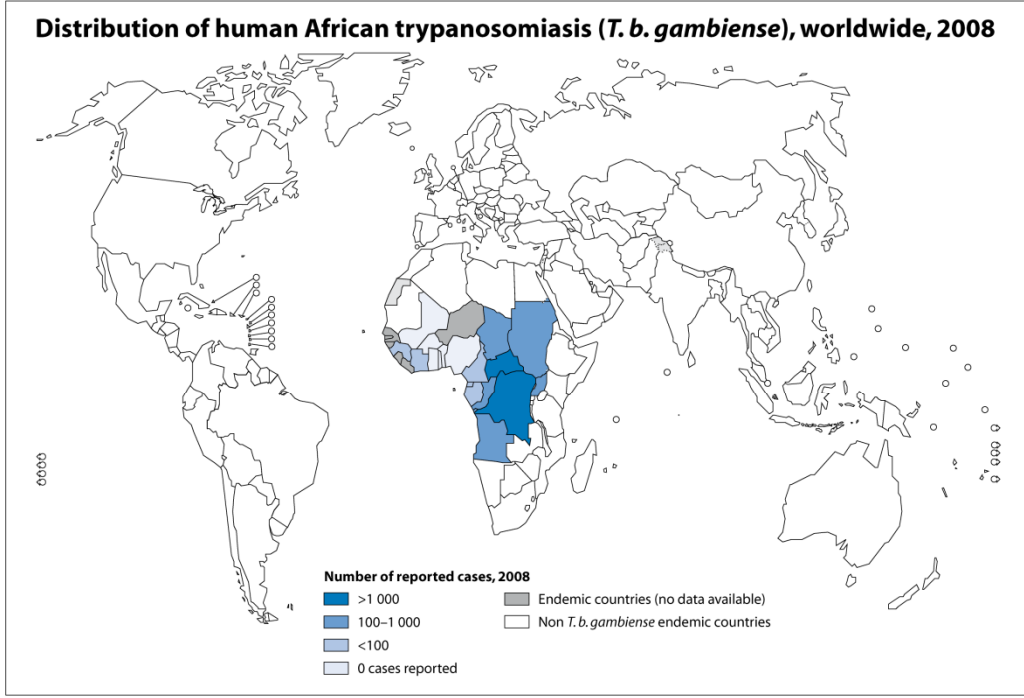
impoverished and affects those groups who are often least able to afford treatment or travel to healthcare facilities.¹⁷²

WHO statistics show a decrease in new SS cases during the first and second decade of the twenty first century. Attempts to limit the fly populations with pesticides, and attempts to eliminate tsetse habitats have proven to be the most effective control methods. “In 2009, the number of cases reported dropped below 10,000 (9878) for the first time in 50 years, and in 2012 there were 7216 cases recorded.”¹⁷³ A majority of SS cases (more than 98%) are caused by *T.b. Gambiense*, which is endemic in twenty four countries. This strain of the disease affects western and central Africa and takes years to cause death in its victims. Conversely, the acute strain, *T.b. Rhodiense*, is found in eastern and southern Africa and is responsible for less than 2% of newly reported cases. The WHO reports that chronic cases (*T.b. Gambiense*) have fallen by 78% from 1999 to 2013, and acute cases by 86% in the same time period.¹⁷⁴ These statistics may be distorted due to nonrepeating of some cases, but the trend shows an overall decrease in the disease’s impact on rural populations. The following images show the distribution of *T.b. Gambiense* and *T. b Rhodiense* respectively:

¹⁷² World Health Organization, “Trypanosomiasis, human African (Sleeping Sickness), updated March 2014, <http://www.who.int/mediacentre/factsheets/fs259/en/>.

¹⁷³ World Health Organization, “Trypanosomiasis, human African (Sleeping Sickness), updated 2015, accessed March 21, 2015. <http://www.who.int/mediacentre/factsheets/fs259/en/>.

¹⁷⁴ World Health Organization, “Human African Trypanosomiasis, Situations and Trends,” updated March 2014, accessed March 21, 2015. <http://www.who.int/mediacentre/factsheets/fs259/en/>.

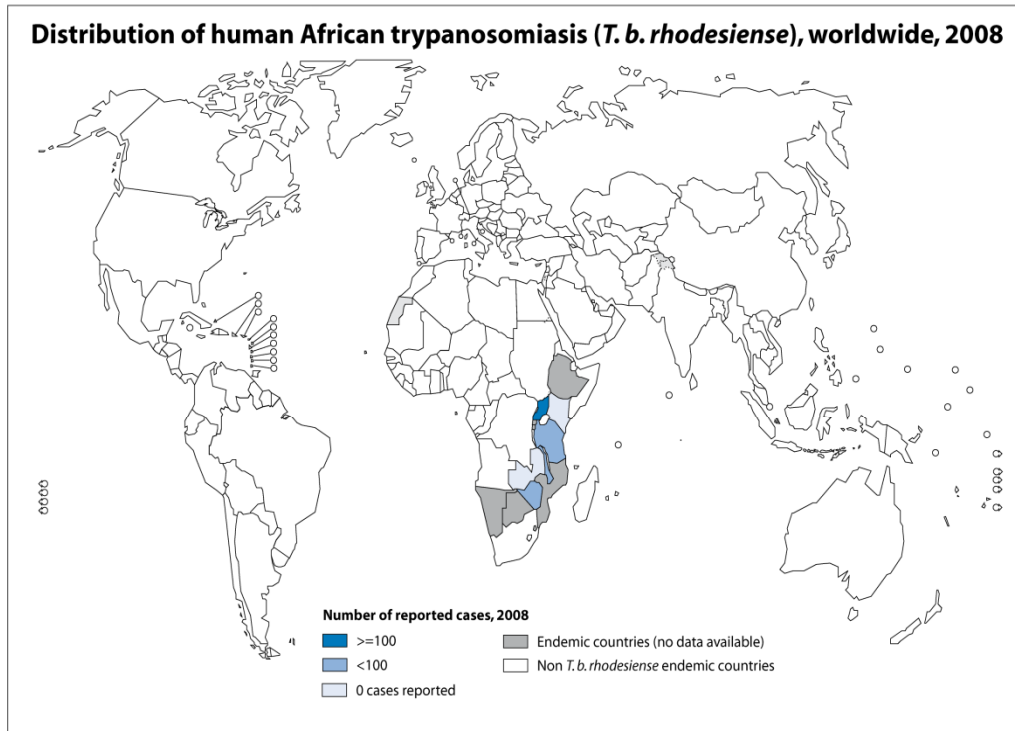


The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement. © WHO 2010. All rights reserved

Data Source: World Health Organization
Map Production: Control of Neglected Tropical Diseases (NTD)
World Health Organization



Map 4: Distribution of *T. b. Gambiense* in 2008



The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement. © WHO 2010. All rights reserved

Data Source: World Health Organization
Map Production: Control of Neglected Tropical Diseases (NTD)
World Health Organization



175

Map 5: Distribution of *T. b. Rhodiense* in 2008

Due to the lack of widespread healthcare and public awareness of disease risk factors, one of the major problems with control of SS still lies in early not achieving diagnosis. Early stage SS is far easier and less expensive to treat. However, diagnostic tests are expensive and in many places inaccessible. Additionally, many early stage symptoms resemble those of malaria or influenza, and as a result the disease is often misdiagnosed.¹⁷⁶ Consequently, modern African communities face many of the the same disease challenges as those living during the Ugandan

¹⁷⁵ WHO, "Distribution of human African Trypanosomiasis, worldwide, 2008," World Health Organization, accessed March 12, 2015, http://gamapserver.who.int/mapLibrary/Files/Maps/Global_trypanosomiasis_gambiense_2008.png.

¹⁷⁶ Geoff Hide, "History of Sleeping Sickness in Africa," *Clinical Microbiological Reviews* 12, no. 1 (January 1999): 113.

and Congolese epidemic in the first decade of the twentieth century. Disease stage diagnosis is based upon the analysis of patient cerebral spinal fluid. Patients who are infected with the disease but have yet to progress into the later stages will show trypanosomes in the blood, but not in the cerebral spinal fluid. When the parasite has crossed the blood/brain barrier trypanosomes can be detected in spinal fluid as well as patient blood. Proper diagnosis using spinal taps requires trained medical professionals, anesthesia, sterile equipment, and laboratory equipment. This medical infrastructure is not always readily available in rural Africa, and as a result, it is difficult to distinguish between early stage and other diseases, resulting in thousands of cases which are misdiagnosed and subsequently progress to the neurological stage.¹⁷⁷

Disease treatment has not made significant progress in the last five decades. This comes from mutation in the parasitic protein coat which makes vaccination development extremely difficult, as well as a lack of ‘disease celebrity’ as seen in other diseases such as HIV/AIDS and Malaria. Research and treatment funding is far less available, despite the fact that SS still kills tens of thousands of Africans every year. The WHO statistics indicate a decrease in newly diagnosed cases, but the chronic form of the disease can often take years to kill its victims and as a result, thousands succumb to the disease annually. “Pentamidine and suramin, developed before the 1920’s, remain the drugs of choice for treatment of early-phase disease, while the arsenic-based melarsoprol, developed in 1949, remains the primary drug treatment of late-phase sleeping sickness. Unfortunately, melarsoprol induces reactive encephalopathy in 10% of patients treated and is fatal in half of those instances.”¹⁷⁸ Encephalopathy is described as abnormal brain function or brain damage, and symptom severity can be debilitating or deadly in

¹⁷⁷ Michael Dumas, Bernard Bouteille, and Alan Buguet, *Progress in Human African Trypanosomiasis, Sleeping Sickness*, (New York: Springer, 1999), 253-254.

¹⁷⁸ Hide, “History of Sleeping Sickness in Africa,” 113.

a small percentage of cases.¹⁷⁹ Suramin and Pentamidine are both arsenicals and, once administered, remain within the lymphatic system and are only effective in treating the parasite during the first stage. Melarsoprol is the only treatment available for second stage *T.b Rhodiense* infections. The drug is also used to treat advanced *T.b Gambiense* infections, and is recommended by the WHO as a stable and relatively safe treatment. Melarsoprol contains arsenoxide which affects the drug's lipid solubility and allows it to pass through the blood/brain barrier to kill trypanosomes in the nervous system.¹⁸⁰ Eflornithine (or α -difluoromethylornithine) is another drug used in second stage *T.b Gambiense* infections, but it is not effective for *T.b Rhodiense* infections.¹⁸¹ α -difluoromethylornithine, is as effective as melarsoprol but is considered safer with fewer deadly side effects. These treatments are often expensive, relatively scarce, and require supportive healthcare staff and facilities in order to monitor patients for these potentially serious side effects.

Because *T.b Gambiense* and *T.b Rhodiense* are two different parasites, it is essential that the specific strain be identified in order to cultivate the proper treatment regimen. Generally, geographic distribution makes this easy to distinguish. However, in Uganda, the disease exists in both forms, as the foci for *T.b Gambiense* in the north, and *T.b Rhodiense* in the south east are only 200k apart, making contamination by both strains possible. Medical professionals and scientists are generally able to diagnose *T.b Rhodiense* through straightforward microscopic analysis of blood or lymph node fluid.¹⁸² The Serological Card Agglutination Test (CATT) is

¹⁷⁹ Ibid., 113

¹⁸⁰ Andrew Jonathan Nok, "Arsenicals (melarsoprol), Pentamidine and suramin in the treatment of Human African Trypanosomiasis," *Parasitology Research* 90, no. 1 (2003):71.

¹⁸¹ WHO, "Human African Trypanosomiasis, Symptoms, Diagnosis, & Treatment," *World Health Organization*, accessed April 2, 2015, http://www.who.int/trypanosomiasis_african/diagnosis/en/.

¹⁸² CDC, "Parasites – African Trypanosomiasis (also known as Sleeping Sickness) Diagnosis," *Centers for Disease Control*, Last Updated August 29, 2012. <http://www.cdc.gov/parasites/sleepingsickness/diagnosis.html>.

used to diagnose *T. b Gambiense*, but is not effective in identifying *T.b Rhodiense*. Both testing methods required trained healthcare professionals and the availability of the necessary equipment and infrastructure. Again, the problem of healthcare availability and treatment facilities hinders infection diagnosis, control, and treatment in African populations. If a patient is treated with the wrong medication, this can lead to the establishment of resistant strains and the exposure of the patient to the medication side effects which are potentially life threatening.¹⁸³

In addition to the inconsistency in diagnosis and the risks associated with available treatments, epidemics since the initial outbreaks in the early 20th century have coincided with periods of political and social unrest within these regions. “Historically, large sleeping sickness epidemics have resulted from ecosystem disruption [such as] periods of political and civil instability,” and are human induced.¹⁸⁴ Lea Berrang Ford makes a compelling argument for this case in “Civil Conflict and Sleeping Sickness in Africa in General and Uganda in Particular.” The historian claims that conflict has disrupted environmental equilibriums bringing SS to unaffected regions through human travel. Additionally, the negative externalities of war such as famine and the destruction of key infrastructure including healthcare facilities, exacerbated existing epidemics and created situations where the disease thrived unabated.¹⁸⁵

After the initial epidemics in the Congo and Uganda were brought under control by the 1960’s, the diseases reemerged in epidemic proportions in the 1970’s alongside post-independence political turbulence. In Uganda, the nation gained independence from Britain in

¹⁸³ Lea Berrang Ford, “Civil Conflict and Sleeping Sickness in Africa in general and Uganda in Particular,” *Conflict and Health* (2007), 2.

¹⁸⁴ Peter M. Atkinson, Eric M. Fevre, Nicola A. Wardrop, and Susan C. Welburn, “The Dispersal of Ecology of Rhodesian Sleeping Sickness Following its Introduction to a New Area,” *PLoS Neglected Tropical Diseases* 7, no. 10 (October 2013): 1.

¹⁸⁵ Ford, “Civil Conflict and Sleeping Sickness in Africa in general and Uganda in Particular,” 1-10.

1962, and in the following decades, numerous episodes of civil war resulted as political factions battled for control. The resulting unstable governments caused healthcare inconsistencies, and forced thousands to move into the rural landscape to avoid fighting. Famine also ensued as food sources became scarce in some regions. As a result, humans were travelling into the tsetse habitat in greater numbers, and the conflict in the region prevented many from receiving available SS treatments.¹⁸⁶

Ford outlines other instances where SS outbreaks correlated with internal conflict. In Angola, the disease had been under control until 1975 (the year of Angola's independence). Cases reemerged during the subsequent civil war. She cited a similar instance in the Sudan where civil war began in the 1980's. "By 1997, sleeping sickness had reemerged in Sudan with prevalence rates as high as 19% in south-western communities bordering the Democratic Republic of the Congo." The DRC currently has one of the highest rates of disease prevalence, and is also one of the most politically unstable regions in Africa. In 1994 a village reported an infection rate of 72%, and disease and malnutrition as a result of unrest and famine were among the leading causes of death, more so than the actual fighting.¹⁸⁷

In Uganda, disease epidemics coincided with major political events. In the first decade of the 20th century, the first outbreak occurred during colonization. The second outbreak, which lasted from 1940-1946, coincided with WWII, and the final outbreak from 1976-1990 resulted from the political instability in the region following independence. "Uganda's civil war influenced the transmission potential of sleeping sickness in a number of ways, including: breakdown of veterinary and public health services, collapse of vector control, regrowth of bushy

¹⁸⁶ *Ibid.*, 2

¹⁸⁷ *Ibid.*, 3-4

tsetse habitat in abandoned agricultural fiends, increase displacement of human an animal population into... areas where they are more likely to be bitten.”¹⁸⁸ This disease was, therefore, another casualty of war and arguably a major contributor to loss of life during these times of conflict in sub-Saharan Africa.

The sleeping sickness epidemics in Uganda and the Belgian Congo during the first decade of the twentieth century showed modern historians the social consequences of environmental decline and disruption, and the resulting externalities of reactive medical control polices. Environmental degradation and political unrest continue to exacerbate sleeping sickness epidemics across the continent, speaking to the need for both ecological understanding and political stability. One of the largest “contributors to human health problems is food insecurity and malnutrition due to human (polices, prices, etc) and environmental (drought, soil infertility, etc) factors that deprive peoples’ entitlement to goods.”¹⁸⁹ Population growth combined with an agricultural and healthcare system which cannot meet demand increases the populations’ susceptibility and exposure to SS. The lack of available healthcare and medical infrastructure has made diagnosing and treating this disease both challenging and in some cases, logically impossible. SS reflects a state of decline where conflict and social collapse are inextricably intertwined with environmental preservation and ecological understanding. To neglect one element is to neglect another, and understanding this symbiotic relationship is the first and very necessary step to disease eradication and control.

¹⁸⁸ Ibid., 5

¹⁸⁹ Mohamed Silah M.A., E.d, *Local Environmental Change and Society in Africa*. London: Kluwer Academic Publishers, 2001, 1-5

In Africa, the historical legacy of these epidemics and colonialism still affect modern ecologies and methods for disease control. Colonialism in the late nineteenth and early twentieth century fundamentally destabilized an evolved equilibrium which governed the actions of humans, animals, and natural fauna on the African continent. The imperial actors who imposed these changes unraveled the old system and modern medicine must acknowledge the consequences of western interference and respond accordingly. This case study contextualizes debates in both ecology and history which argue that disrupting a natural order leads to catastrophic events. In the case of sleeping sickness, colonial expansion proved to be the catalyst. Today, conflict and social unrest continue to destabilize the new system, and solving the medical dilemma involves, as Humphrey argued, addressing the natural ecology and controlling human behavior in addition to identifying a medical solution. A new medical system should satisfy the needs of a newer, more fluid equilibrium in which modern medicine must combine with the current social context to address the rules of post-colonial Africa; science needs to respect local knowledge as medicine and social conceptions are inextricably intertwined. History teaches us that epidemics of this magnitude are a combination of multiple factors and to address only one aspect of the larger network is insufficient in solving the problem. Because Human African *Trypanosomiasis* is still a considerable health threat in affected regions, understanding its history can provide modern health officials with considerable knowledge that can help prevent future epidemics and suffering.

Bibliography

- Anderson, Warwick. *Colonial Pathologies: American Tropical Medicine, Race, and Hygiene in the Philippines*. Durham: Duke University Press, 2006.
- Atkinson, Peter M., Fevre, Eric M., Wardrop, Nicola A., and Susan C. Welburn. "The Dispersal of Ecology of Rhodesian Sleeping Sickness Following its Introduction to a New Area." *PLoS Neglected Tropical Diseases* 7, no. 10 (October 2013): 1-11.
- Beinart, William. "African History and Environmental History," *African Affairs* 99, no. 395 (April 2000): 269 – 302.
- British Medical Journal. "Reports of the Royal Society's Commission on Sleeping Sickness." *The British Medical Journal* 2, no. 2233 (Oct. 17, 1903): 1.
- BMJ. "African Sleeping Sickness." *The British Medical Journal* 336, no. 7646 (March 29, 2008), 679
- BMJ. "African Sleeping Sickness." *The British Medical Journal* 1, no. 3362 (June 6, 1925): 1048 – 1049.
- BMJ. "The African Sleeping Sickness." *The British Medical Journal* 2, no. 3651 (December, 1930): 1094 – 1095.
- BMJ. "The Liverpool School of Tropical Medicine and Trypanosomiasis." *The British Medical Journal* 2, no. 2225 (August 22, 1903): 427.
- Barrett, Michael P., and Francesco Checchi. "African Sleeping Sickness." *British Medical Journal* 33, no. 7646 (March 29, 2008): 679-80.
- Bouteille, Bernard, Buguet, Alain, and Michel Dumas. *Progress in Human African Trypanosomiasis, Sleeping Sickness*. New York: Springer, 1999.
- Browning, Carl H. "Experimental Chemotherapy in Trypanosomiasis Infections." *The British Medical Journal* 2, no. 2446 (Nov 16, 1907): 1405-1409.
- Bruce, David. "Weekly Evening Meetings: Sleeping Sickness in Uganda." Paper presented at the Weekly Evening Meetings for the Ugandan Colonial Government, Uganda, April 22, 1904.

- Bruce, David and Colleagues. *Reports of the Sleeping Sickness Commission of the Royal Society*. London: Darling and Son LTD, 1914.
- Bruce, David, Hamerton, A.E, Bateman, H.R, and F.P Mackie, "Sleeping Sickness in Uganda – Duration of the infectivity of the *Glossina Palpalis* after the Removal of Lake Shore Population." *Proceedings of the Royal Society of London* 82, no. 552 (December 20, 1909): 56-63.
- Bruce, David and Davis Nabarro, "Progress Report on Sleeping Sickness in Uganda," in *Reports of the Sleeping Sickness Commission of the Royal Society*, 11-88. London: Harrison and Sons, 1903.
- Castellani, Aldo, M.D. "Reports of the Sleeping Sickness Commission of the Royal Society." in *Reports of the Sleeping Sickness Commission of the Royal Society*, 1-10. London: Harrison and Sons, 1903.
- CDC. "Parasites – African Trypanosomiasis (also known as Sleeping Sickness) Diagnosis." *Centers for Disease Control*, Last Updated August 29, 2012. <http://www.cdc.gov/parasites/sleepingsickness/diagnosis.html>.
- Chappuis, Francois, Stivanello, Elisa, Adams, Karen, Kidane, Solon, Pittet, Anne and Patrick A. Bovier. "Card Agglutination Test for Trypanosomiasis (CATT) end dilution titter and cerebrospinal fluid cell count as predictors of Human African Trypanosomiasis among Serologically Suspected Individual in Southern Sudan." *The American Journal of Tropical Medicine and Hygiene*. Accessed March 30, 2015, <http://www.ajtmh.org/content/71/3/313.short>.
- Christy, Cuthbert. "Reports of the Sleeping Sickness Commission of the Royal Society, no III, in *Reports of the Sleeping Sickness Commission*. London: Harrison and Sons, 1903.
- "Dr. Cuthbert Christy." *Journal of the Royal African Society* 31, no 124 (July, 1932), 339.
- Dumas, Michael, Bouteille, Bernard, and Alan Buguet. *Progress in Human African Trypanosomiasis, Sleeping Sickness*. New York: Springer, 1999.
- Dutton, J Everett, and Todd, J.L, "Human Trypanosomiasis on the Congo. Being the First Progress Report of the Expedition of the Liverpool School of Tropical Medicine to the Congo. 1903." *The British Medical Journal* 1, no. 2247 (January 23, 1904): 186-188.
- Dutton, J. Everett, Todd, J.L, and Cuthbert Christy. *Reports of the Trypanosomiasis Expedition to the Congo 1903 – 1904*. London: Williams and Norgate for the University Press of Liverpool, 1904.
- Eckart, Wolfgang U. "The Colony as Laboratory: German Sleeping Sickness Campaigns in German East Africa and in Togo, 1900 – 1914," *History and Philosophy of Life Sciences* 24, no. 1 (2002): 69-89.
- Ferguson, J.A.E. "The Sleeping Sickness." *The British Medical Journal* 1, no. 1988 (Feb 4, 1899): 315.
- Ford, Lea Berrang. "Civil conflict and sleeping sickness in Africa in general and Uganda in particular." *Conflict and Health* (2007): 1-10.

- Ford, Lea Berrang. "Civil Conflict and Sleeping Sickness in Africa in General and Uganda in Particular." Accessed March 1, 2015, http://openi.nlm.nih.gov/detailedresult.php?img=1851948_1752-1505-1-6-1&req=4.
- Gradmann, Christoph. *Laboratory Disease: Robert Koch's Medical Bacteriology*. Baltimore: Johns Hopkins University Press, 2009.
- Gore, Albert A, M.D., F.R.C.S.I., Surgeon-major. "The Sleeping Sickness in Western Africa." *The British Medical Journal*, (January 2, 1875): 5-7.
- Headrick, Daniel R. "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900 – 1940." *PLOS Neglected Tropical Diseases* 8, no. 4 (April 2014): 1-8.
- Hide, Geoff. "History of Sleeping Sickness in East Africa." *Clinical Microbiology Reviews* 12, no. 1 (January 1999): 112-125.
- Hoppe, Kirk Arden. "Lords of the Fly: Colonial Visions and Revisions of African Sleeping-Sickness Environments on Ugandan Lake Victoria, 1906-61." *Journal of the International African Institute* 67, no. 1 (1997): 86-105.
- Hochschild, Adam. *King Leopold's Ghost*. New York: Houghton Mifflin Company, 1998.
- Humphrey, Margaret. *Yellow Fever in the South*. New Brunswick: Rutgers University Press, 1992.
- "King Leopold's Ghost." Accessed April 3, 2015. <https://rhapsodyinbooks.wordpress.com/2008/09/10/king-leopolds-ghost-by-adam-hochschild>.
- Helge Kjekshus. *Ecology control and Economic Development in East African History*. London: Heinemann Educational Books Ltd, 1977.
- Knight, C. Gregory. "The Ecology of African Sleeping Sickness." *Annals of the Association of American Geographers* 1, no. 1 (March 1971): 23-44.
- Koch, Robert. "The Aetiology of Tuberculosis." In *Source Book of Medical History*, Compiled by Logan Clendening M.D, 398-399. New York: Dover Publications, 1942.
- Koerner, T., Raadt, P. de, and I. Maudlin. "The 1901 Uganda Sleeping Sickness Epidemic Revisited," *Parasitology Today* 11, no 8 (1999): 1-4.
- Lawson, Barbara, Miller, Pamela and Kristen Dobbin. *Dr. John Lancelot Todd*. Montreal: McGill University, 2011. Accessed April 1, 2015, <https://www.mcgill.ca/library/files/library/osler-toddbibliography.pdf>.
- Livingstone, David. "Arsenic as a Remedy for the Tsetse Bite." *The British Medical Journal* 1, no. 70 (May 1, 1858): 360-361.
- Loustaunau, Martha O., and Elisa J. Sobo. *The Cultural Contest of Health, Illness, and Medicine*. London: Bergin & Garvey, 1997.

- Low, George C and Aldo Castellani, "Report on Sleeping Sickness from its Clinical Aspects." In *Reports of the Sleeping Sickness Commission of the Royal Society*. London: Harrison and Sons, 1903.
- Lyons, Martinez. *The Colonial Disease: A Social History of Sleeping Sickness in Northern Zaire, 1900-1940*. Cambridge: Cambridge University Press, 2002.
- Lyons, Maryinez. "From 'Death Camps; to Cordon Sanitaire: The Development of Sleeping Sickness Policy in the Uele District of the Belgian Congo, 1903 – 1914." *The Journal of African History* 26, no. 1 (1985): 69-91.
- Mac Queen, Peter,. "Sleeping Sickness in Uganda." *Bulletin of the American Geographical Society* 43, no. 3 (1911), 181-184.
- Mackenzie, John M. *Empire of Nature: Hunting, Conservation, and British Imperialism*. Manchester: Manchester University Press, 1988.
- Maddox, Gregory H. *Sub-Saharan Africa: an environmental history*. Santa Barbara: ABC-CLIO, 2006.
- Manson, Sir Patrick. "Sleeping Sickness and Trypanosomiasis in a European: Death: Preliminary Note." *The British Medical Journal* 2, no. 2240 (December 5, 1903): 1461-1462.
- Manson, Sir Patrick. "Remarks on a Case of Trypanosomiasis." *The British Medical Journal* 1, no. 2213 (May 30, 1930), pp. 1249-1252.
- McKelevy, John J. Jr. *Man against the Tsetse: Struggle for Africa*. Ithaca: Cornell University Press, 1973.
- Neill, Deborah. "Paul Ehrlich's Colonial Connections: Scientific Networks and Sleeping Sickness Drug Therapy Research, 1900-1914." *Social History of Medicine* 22, no. 1 (February 2009): 61-77.
- Nok, Andrew Jonathan. "Arsenicals (melarsoprol), Pentamidine and suramin in the treatment of Human African Trypanosomiasis." *Parasitology Research* 90, no. 1 (2003):71-73.
- Planet Earth Online. "Seeing Great Sorrows: Climate and Disease Linked by Missionary Medical Records." Planet Earth Online. Last modified November 29, 2007, <http://planetearth.nerc.ac.uk/features/story.aspx?id=149&cookieConsent=A>.
- Rosenberg, Charles E. *Framing Disease: Studies in Cultural History*. New Brunswick: Rutgers University Press, 1992.
- Russell, E. *War and Nature: Fighting Humans and Insects with Chemicals from World War I to Silent Spring*. Cambridge: Cambridge University Press, 2001.
- Salih, Mohamed M.A., Ed. *Local Environmental Change and Society in Africa*. London: Kluwer Academic Publishers, 2001.
- "Sir Roger Casement 1864-1916." Accessed April 2, 2015. <http://www.northantrim.com/rogercasement.htm>.

Stephens, J.W.W, Fantham, H.B, Kleine, F.K, Mesnil, F., Kinghorn, Allan, Yorke, Warrington, Wolbach, S.B. and C. A. L Binger, "Papers Dealing with Trypanosomiasis," *The British Medical Journal* 2, no. 2705, (Nov 2, 1912): 1182-1188.

The Royal Society. *Reports of the Sleeping Sickness Commission*. London: Harrison and Sons, 1903.

Thomas, Wolferstan H, and Anton Breinl. *Trypanosomes, Trypanosomiasis, and Sleeping Sickness: Pathology and Treatment*. London: Williams and Norgate for the University Press of Liverpool, 1905.

Todd, Dr. J. L. *The Distribution, Spread, and Prophylaxis of Sleeping Sickness in the Congo Free State; from the Expedition to the Congo of the Liverpool School of Tropical Medicine*. London: The Bedford Press, 1905.

Todd, John L. "The Prevention of Sleeping Sickness." *The British Medical Journal* 2, no. 2493 (October, 1908): 1061 – 1063.

WHO. "Distribution of human African Trypanosomiasis, worldwide, 2008." World Health Organization. Accessed March 12, 2015. http://gamapserver.who.int/mapLibrary/Files/Maps/Global_trypanosomiasis_gambiense_2008.png.

WHO. "Trypanosomiasis, human African (Sleeping Sickness)." World Health Organization. Last modified March 2014, <http://www.who.int/mediacentre/factsheets/fs259/en/>.

WHO, "Human African Trypanosomiasis, Symptoms, Diagnosis, & Treatment," *World Health Organization*, accessed April 2, 2015, http://www.who.int/trypanosomiasis_african/diagnosis/en/.

WHO. "Human African Trypanosomiasis, Situations and Trends." *World Health Organization*, updated March 2014, accessed March 21, 2015. <http://www.who.int/mediacentre/factsheets/fs259/en/>.

Wolferstan, Thomas and Anton Breinl. *Trypanosomes, Trypanosomiasis, and Sleeping Sickness: Pathology and Treatment*. London: Williams and Norgate for the University Press of Liverpool, 1905.

Appendix:

- **Dr. David Bruce (1955-1931):** Lieutenant Colonel in the Royal Army. Sent to Uganda in 1902 as part of the first research group commissioned by the Royal Society (Part of the London School of Hygiene and Tropical Medicine). instrumental in establishing the link between the tsetse fly and Trypanosomiasis along with Castellani.
- **Roger Casement (1864-1916):** worked for the British Foreign service, was the British Consul to the Belgian Congo from 1901-1904, and also had a personal interest in natural history and disease study. He reported on the labor and sanitary conditions in the Congo and references the negative effects of sleeping sickness on the Congolese.
- **Dr. Aldo Castellani (1877-1971):** instrumental in establishing the link between the tsetse fly and Trypanosomiasis along with David Bruce
- **Dr. Cuthbert Christy (1863-1932):** Worked with London School of Hygiene and Tropical Medicine, travelled to Uganda in 1901 and later to the Belgian Congo with Todd and Dutton to study sleeping sickness epidemics in each region.
- **Dr. Joseph Everett Dutton (1874-1905):** 1902 identified the “worms” as the protozoa later named *T. b. Gambiense*. Dutton was a member of the southern and northern Nigeria expedition of the Liverpool School, 1899-1900, the leader of the Gambia expedition 1901-1902, and of the Senegambia expedition, 1902-1903, and worked in Uganda and the Belgian Congo studying Trypanosomiasis. Dutton died in 1905 in the field from tick fever.
- **Dr. Paul Ehrlich (1854-1915):** immunologist working under Robert Koch. Began work in his lab in 1891. Was instrumental in developing staining techniques to identify and later treat specific disease. Also instrumental in developing chemotherapeutic techniques for disease treatment. Became the head of the Royal Institute for Experimental Therapy in Frankfurt. Earned a Nobel Prize for discovering salvarsan, the cure for Syphilis.
- **Dr. Robert Forde (1861-1948):** British Colonial Surgeon. Was the first to observe “worms” in the blood of a SS victim. These worms would later be identified as the disease pathogen.
- **Dr. E.D.W Greig (1869):** Led field research team in Uganda in 1903. Was sent to study the disease on behalf of the Indian government.

- **Dr. Albert A. Gore, M.D., F.R.C.S.I.** Surgeon Major: Studied Sleeping Sickness in Western Africa while serving in the Military. Reported early cases and documented early treatment methods, symptoms, and other disease observations
- **Dr. Robert Koch (1843-1910):** Instrumental in developing modern germ theory. Discovered the pathogens for Cholera, Anthrax, and Tuberculosis. Koch also studied arsenic-based treatments on the Sese Islands in Lake Victoria to determine the best medication and dosage for treating sleeping sickness.
- **Dr. Patrick Manson (1844-1922):** Director of the London School of Tropical Medicine (1899), instrumental in germ theory development.
- **Dr. Davis Nabarro:** Along with Dr. Bruce, Nabarro led the second Uganda expedition sent by the London School of Hygiene and Tropical medicine.
- **Dr. Ronald Ross (1857-1932):** Director of the Liverpool School of Tropical Medicine (1898), prominent germ theorist, and an expert in tropical medicine..
- **Dr. John L. Todd (1876-1949):** Canadian educated scientist who received a fellowship to work with the Liverpool School of Tropical medicine in 1900. In 1901 he travelled Dutton to Senegambia to study Trypanosomiasis. He would also study the disease in Uganda and the Belgian Congo. Todd's work in diagnostics and his recommendations would influence the Belgian colonial government to establish sleeping sickness quarantine camps and to monitor travel between infected and uninfected regions.