Reflection essay on microbiology research paper

Science, Biology

African Trypanosomiasis, also known as " sleeping sickness", is a parasitic disease of people and animals caused by microscopic parasites of the species Trypanosoma brucei. It is transmitted by the tsetse fly, which is found only in rural Africa. Although the infection is not found in the United States, historically, it has been a serious publichealthproblem in some regions of sub-Saharan Africa covering about 37 countries and 60 million people (African Trypanosomiasis [also known as Sleeping Sickness] 2010).

The term “ sleeping sickness” was given primarily because when infected, invasion of the cerebrospinal fluid and brain after infection of the blood is often delayed, resulting in symptoms of extreme fatigue that can last for several years before the severe phase of the disease sets in; toxemia, coma and death (Trypanosomiasis, African 2009). With the huge number of people it affects, it is weird that I only discovered it now when faced with a microorganism research paper.

The glaring explanation behind this, is that there are many much larger, deadlier diseases in Africa, that ultimately make diseases like sleeping sickness get overlooked. This may be true, but that is no reason not to pay attention to a disease like this because it has and does affect and kill thousands of people. When researching this topic many questions arose and they were all addressed with further research.

Things addressed in this paper are: how many people the disease effects annually, the cure for humans infected with the organism, where in the world people are affected, whether the number of infections is increasing or decreasing, the side effects when a human is infected, the impact on the

economy if there was to be an outbreak, the kinds of treatment given to those who are infected, the future plans for the infection, the comparison of infections in third world countries to first world, and the long lasting effects the disease can leave after treatment.

Most speculation and hype related to diseases in Africa focus on malaria and AIDS, but while researching Sleeping Sickness it is clear that there are many more large scale, but less known, diseases affecting the population of Africa. I was really hoping to learn more about the issues that come up when trying to treat a large number of people in a country like Africa where disease and ways to treat disease are big problems. I was also interested in the history of the disease, unfortunately because of it being such a confined disease, especiialy in the beginning, it was hard to find information on the early stages of the disease.

Human African trypanosomiasis (HAT) has been present in Africa since the fourteenth century, it started isolated in pockets due to lack of travel of the indigenous people. The earliest recorded account of sleeping sickness comes from upper Niger and the next report came from Guinea in 1734 (Trypanosoma Brucei - Sleeping Sickness). J. E. Dutton was the first to correctly identify the parasite as a trypanosome and subsequently named it Trypanosoma gambiense in 1902. D.

Bruce identified the cause and vector of this disease in 1903, and also the differentiation between the subspecies of the protozoa made in 1910 (Sanger Institute 2011). The first effective treatment, atoxyl, an arsenic-based drug developed by Paul Ehrlich and Kiyoshi Shiga, was introduced in 1910, but blindness was a serious side effect. So next, Suramin was

introduced in 1920 to treat the first stage of the disease. By 1922, Suramin was generally combined with Tryparsamide in the treatment of the second stage of the gambiense form.

It was used during the grand epidemic in West and Central Africa, helping millions of people, and stayed the mainstay of therapy until 1969 (Medical Ecology). Pentamidine, a highly effective drug for the first stage of the disease, has been used since 1939. During the fifties, it was widely used as a prophylactic agent in Western Africa, leading to a sharp decline in infection rates. At the time, it was thought that eradication of the disease was at hand (Kelly, J. M 2012). This disease has been around for thousands of years, and has been mostly confined to Africa.

Of the 36 countries considered endemic for HAT, the seven most affected countries represent 97% of all reported cases. The Democratic Republic of the Congo alone accounts for 2/3 of reported cases. HAT primarily occurs in the poorest, most rural areas in Africa, where difficulty of diagnosis, political instability, and lack of health surveillance make estimates of disease difficult to conduct. It is also a highly focal disease often characterized by distinct outbreaks in a specific area or village (Agriculture and Consumer Protection).

Sleeping sickness endemic areas receive their names from geographical features such as rivers, villages or towns, and administrative divisions, and the size of these areas can range from a single populated place to an entire region. Within a given endemic area, the intensity of the disease can vary from one village to the next. Also, the geographical extent of foci may change significantly over time, as a result of both human mobility and of

environmental dynamics and modifications influencing tsetse fly presence, density, and dispersal.

Furthermore, it was shown that the Rhodesian form of the disease might be introduced into previously unaffected areas by cattle movement. The disease is found all over Africa, and the organism is transferred when bitten by the fly (Odero, R. O 2012). Trypanosoma brucei is the protozoan that causes HAT. There are 3 sub-species of T. brucei: T. b. brucei, T. b. gambiense, and T. b. rhodesiense. T. brucei gambiense causes slow onset chronic trypanosomiasis in humans this is most common in western and central Africa, where humans are thought to be the primary reservoir.

T. brucei rhodesiense causes fast onset acute trypanosomiasis in humans, it is most common in eastern and southern Africa, where game animals and livestock are thought to be the main reservoir. Lastly, T. brucei causes animal African trypanosomiasis, along with several other species of trypanosoma. These are all obligate parasites, parasitic organisms that cannot complete their life cycle without exploiting suitable hosts, which means they live within an insect vector and a mammal host (World Health Organization 2012).

The trypanosome goes through complex changes throughout its life due to the large differences between its two hosts. The cell is a fairly typical eukaryotic cell. It features flagella, the cytoskeleton that is made up mostly of microtubules, and the only unusual feature it has is a single, large mitochondria (Van Den Abbeele, Caljon, De Ridder, De Baetselier, Coosemans 2010). African trypanosomes are extracellular organisms, both in

the mammalian and insect host. The morphologically is indistinguishable, measuring 25-40 µ m in length.

Infection in the human host begins when the infective stage, known as the metacyclic stage, is injected intradermally by the tsetse fly. The organisms rapidly transform into blood-stage trypomastigotes, and divide by binary fission in the interstitial spaces at the site of the bite wound. The buildup of metabolic wastes and cell debris leads to the formation of a chancre. One of its unusual features is that the entire DNA of the mitochondrion, is localized in the kinetoplast, adjacent to the flagellar pocket. Kinetoplast DNA exists in two forms: mini-circles and maxi-circles.

Mini-circle DNA encodes guide RNAs that direct extensive editing of RNA transcripts post-transcriptionally. Maxi-circle DNA contains sequences that, when edited, direct translation of typically mitochondrially-encoded proteins. In the vertebrate host, trypanosomes depend entirely upon glucose for energy and are highly aerobic, despite the fact that the kinetoplast-mitochondrion completely lacks cytochromes. Instead, mitochondrial oxygen consumption is based on an alternative oxidase that does not produce ATP. When in the insect vector, the parasite develops a conventional cytochrome chain and TCA cycle.

The surface of the trypanosome has numerous membrane-associated transport proteins for obtaining nucleic acid bases, glucose, and other small molecular weight nutrients. None of these proteins react well with antibodies, because although they lie in exposed regions of membrane, they are shielded by allosteric interference provided by the variant surface glycoprotein coat proteins. The number of parasites in the blood is generally

so low that diagnosis by microscopic examination is often negative. At some point, trypanosomes enter the central nervous system, with serious pathological consequences for humans.

Some parasites transform into the non-dividing short, stumpy form, which has biochemistry similar to those of the long, slender form and the form found in the insect vector (Jackson, Sanders, Berry, McQuillan, Aslett, et al 2010). African Sleeping Sickness is mostly transmitted through the bite of a tsetse fly, but there are also other ways people can be infected. It can be transferred from a mother to child through crossing the placenta and infecting the fetus. Mechanical transmission can happen through other blood-sucking insects, this happens on a much smaller scale than the tsetse fly.

Although this number is very small, accidental infections have happened in labs where people are pricked with contaminated needles (Trypanosoma Brucei - Sleeping Sickness). In the first stage, the trypanosomes multiply in subcutaneous tissues, blood and lymph. This is known as a haemolymphatic phase, which entails bouts of fever, headaches, joint pains and itching. In the second stage the parasites cross the blood-brain barrier to infect the central nervous system. This is known as the neurological phase (Kelly 2012). In general this is when more obvious signs and symptoms of the disease appear: changes

of behaviour, confusion, sensory disturbances and poor coordination. The symptoms and signs of sleeping sickness are usually quite different, but can be easily confused because of the variability of symptoms and length of time until onset depends heavily on host characteristics. The chancre, a leathery

swelling at the site of the bite, is usually the first symptom of the disease, primarily for T. b. rhodensiense. Within weeks, those with opportunistic levels of infection with T. b. rhodensiense start to experience irregular intermittent fevers associated with the waves of parasitaemia that are characteristic of T.

b. rhodensiense infections. For T. b. gambiense, lymphoadenopathy occurs more frequently. Oedema of the face is another frequent sign of infection, and anemia may be present, particularly in T. b. rhodensiense (News Medical 2007) Disturbance of the sleep cycle, which gives the disease its name, is an important feature of the second stage of the disease. Since the parasite constantly changes its surface, it can avoid the immune defense of humans and invade the central nervous system, which leads topersonalitydisturbances, sleep disruptions, and ultimately death (Medical Ecology).

For patients affected by a severe T brucei infection in the central nervous system, there are no medicines that can treat both subspecies without incurring extremely serious side effects. Although symptoms and signs associated with nervous system involvement are varied for African sleeping sickness, advanced disease epileptic seizures, maniacal behavior, somnolence, and coma are some typical late stage symptoms (African Trypanosomiasis [also known as Sleeping Sickness] 2010). Unfortunately, survival rates are drastically reduced once the trypanosomes infect the central nervous system.

The disease management is made up of three steps: the first is the screening for potential infection by serological tests and checking for clinical

symptoms. The second step is to diagnose whether the parasite is present in the body or not, and the final step is to determine the progression of the disease, usually by examining cerebro-spinal fluid. Today, there are only a handful of active drugs available for treatment of human African Trypanosomiasis, and no significant development has been made over the last 2 decades.

The current line of treatment is problematic for many reasons: firstly, the drugs are harmfully toxic requiring extensive hospitalization. Secondly, regular follow-ups to check for relapse is essential but difficult in many of the areas where sleeping sickness is endemic (News Medical 2007). Many individuals die before they can ever be treated for the disease because the first stage is usually asymptomatic, and in Africa, an extensive testing of a large number of people who may or may not be infected, is to costly of an investment.

The type of treatment depends on the progression of the disease. As it is in most diseases the earlier it is identified, the easier it is to treat and cure. The drugs used in the first stage of the disease are of lower toxicity and easier to administer. Treatment success in the second stage depends on a drug that can cross the blood-brain barrier to reach the parasite. Such drugs are toxic and complicated to administer (African Trypanosomiasis [also known as Sleeping Sickness] 2010).

Over the past years, public awareness of the dangers associated with insecticides is increasingly changing the way we treat ourenvironment. Efforts to introduce more environmentally friendly methods of insect control provide the world with challenges to understand more about the insects that

transmit HAP. We live in a modern world where various means of control are available to stop the spreading of multiple diseases, however, sleeping sickness is a disease of the developing world. In Africa, despite the multitude of control strategies, issues have been widely neglected and abandoned.

One of the main components needed to bring effective change, is to consider a control strategy that will last and can be carried through by local communities (Jackson, Sanders, Berry, McQuillan, Aslett, et al 2010). Apart from efforts to reduce the spread of disease through environmental controls, there is also need to improve current tracking anddiagnosticprocedures. Chances of death can be drastically reduced when cases can be diagnosed early enough to prevent onset of late-stage sleeping sickness. Training and resources are desperately needed in affected areas for safe and proper diagnostics.

With this disease there is an issue with treatment options, and the availability of drugs in Africa. Drug and vaccine development for diseases in developing countries have always been lagging, and trypanocidal drugs are no exception. An estimated 300, 000 to 500, 000 people are currently infected and suffering from HAT with no hope for treatment. In 2000, the USFDA approved the use of eflornithine. Hopefully some of the profits from the sale of this drug are used to help the situation in the poorest parts of Africa infected with this disease (Jackson, Sanders, Berry,

McQuillan, Aslett, et al 2010). The biggest issue with this disease is that it is confined to people in Africa. Because of this most of the world is not aware of the seriousness of it, and most people do not know how they can help. Also, with all the diseases in Africa, many smaller scale sicknesses can be

overlooked. Third world countries often have trouble being able to control all the different infections that affect so many people in them. Researching this opened my eyes to the multiplicity and severity of the diseases that affect so many people.

It was shocking to learn that this disease has been around for a few thousand years and people are still dying of it daily. And furthermore, that this was the first time I had ever even heard of the disease. It was really interesting to learn about the tsetse fly also, the only other sickness I had heard of before that was transferred by a blood sucking insect, was malaria. The hardest thing to understand was how although there is a cure for this disease, people are still dying of it every day.

I was not aware of the complete lack oftechnologyand awareness of the illness in the places affected. If I were to research further on HAT, I would like to research about foundations that donate and help the people and countries affected by this disease. I am very interested in charities and companies that help the less fortunate. Overall, the research I did on this subject has been very informative and thought provoking, it has made me a more knowledgeable person on not only this specific disease, but also many diseases killing people in Africa and other third world countries.

I learned a lot about the aspects of microbiology, I got to see it applied to the real world and the affects it can have on people. This has better informed me about the course I am taking and the application of it to something other than a lab station. I learned so much more than I needed to know, but it has made me a better person in the long run, I am a better, smarter person after writing this paper.