International Journal of Science and Technology Research Archive

ISSN: 0799-6632 (Online)

Journal homepage: https://sciresjournals.com/ijstra/

(RESEARCH ARTICLE)

Check for updates

Study of the characteristics of the Glossinidae family (Muscoidea, Oestroidea)

Carlos Henrique Marchiori *

Instituto Federal Goiano, Biological Sciences, Parasitology, Goiânia, Goiás, Brazil.

International Journal of Science and Technology Research Archive, 2021, 01(02), 088–107

Publication history: Received on 08 October 2021; revised on 20 November 2021; accepted on 22 November 2021

Article DOI: https://doi.org/10.53771/ijstra.2021.1.2.0050

Abstract

Oestridae is a family of flies, which includes species whose larvae are internal parasites of several species of mammals. Some species settle in the host's flesh, while others occur in the viscera. This study objective to report the characteristics of the Glossinidae Family. The research was carried out in studies related to quantitative aspects of the Family, Subfamily and Species (taxonomic groups) and conceptual aspects such as: biology, geographical distribution, species, life cycle, damage, laboratory creation, economic importance, medicinal importance, biological aspects, monitoring, control, and reproduction. A literature search was carried out containing articles published from 1950 to 2021. The mini-review was prepared in Goiânia, Goiás, from September to October 2021, through the Online Scientific Library (Scielo), internet, ResearchGate, Academia.edu, Frontiers, Publons, Qeios, Portal of Scientific Journals in Health Sciences, Pubmed, Online Scientific Library (Scielo), internet, ResearchGate, Academia.edu, Frontiers, Biological Abstract, Publons, Qeios, Portal of Scientific Journals in Health Sciences, and Pubmed, Dialnet, World, Wide Science, Springer, RefSeek, Microsoft Academic, Science, ERIC, Science Research.com, SEEK education, Periódicos CAPES, Google Academic, Bioline International and VADLO.

Keywords: Biology; Baites; Goías; Medicinal importance; Scielo

1. Introduction

Glossinidae is a monotypic family of Diptera that includes only the genus *Glossina,* the tsetse flies that transmit the trypanosome that cause sleeping sickness (Figure 1).

Figure 1 Glossinidae: *Glossina (*Source: https://www.sciencedirect.com/science/article/pii/B978012814043700018)

* Corresponding author: Carlos Henrique Marchiori
Instituto Federal Goiano, Biological Sciences, Parasitology, Goiânia, Goiás, Brazil.

Glossina Wiedemann, 1830 is a genus of flies in the Glossinidae family (formerly part of the Muscidae family) which includes the species known by the common name of tsetse flies, a name originating in the mantle languages of equatorial Africa. These species transmit *Trypanosoma brucei*, the trypanosome that causes sleeping sickness (Figure 2).

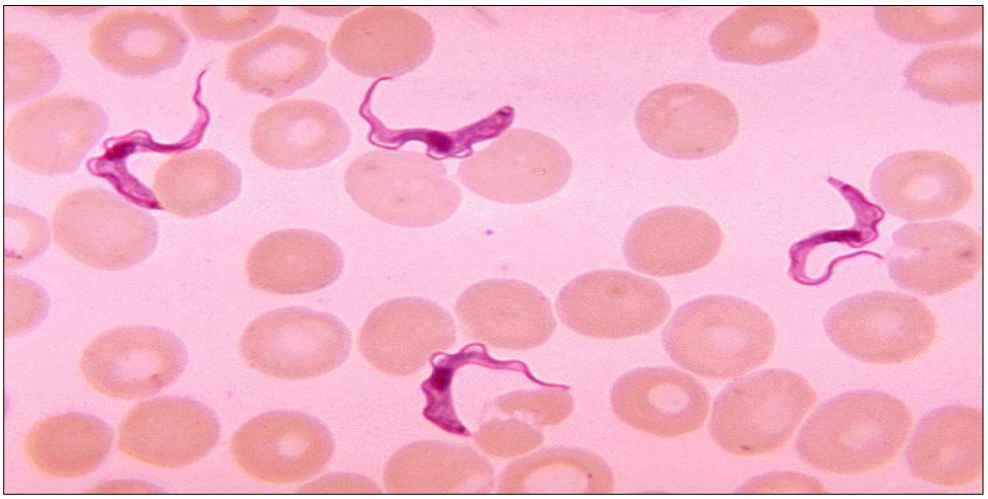

Figure 2 Trypanosoma: Domain Eukaryota, Scientific name: *Trypanosoma,* Higher classification Trypanosomatidae and Order Trypanosomatida. Lower classifications *Trypanosoma brucei, Trypanosoma evansi, Trypanosoma congolense, Trypanosoma rangeli* and *Trypanosoma equiperdum; (*Source: https://alchetron.com/Trypanosoma)

Like many insects, these flies do not lay eggs, but larvae, directly into the ground, where they burrow. A few hours later, they become pupae (intermediate form between the larva and the adult insect) with hard brown cocoons. After six weeks they are adults, ready to carry the trypanosome (Figure 3) [1,2,3].

Figure 3 *Glossina* Wiedemann, 1830; (Source: https://br.pinterest.com/pin/720646377847808791/)

There are three groups of this fly, all hematophiles. It is found from Lake Chad and Senegal in the west to Lake Victoria in the east. This region is bathed by the Congo River and its tributaries, being known as the Green Heart of the African Continent. The local humidity favors the appearance of insects of the most diverse species. The problems caused by the fact that this insect is the transmitter of sleeping sickness have led health authorities to consider extermination Its main (Figure 4) features are:

Size: Up to 1 cm long, Color: amber with striped abdomen (on the back), mouth: In the form of a thin tube, head: Has a groove in front of the head, Wings: transparent and sucking insect (Figures 5, 6 and 7) [1,2,3].

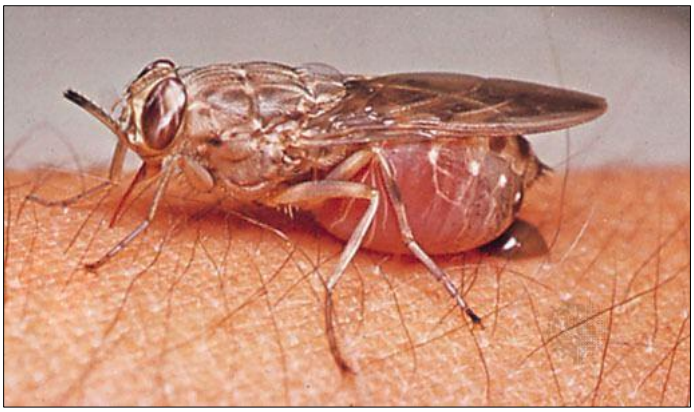

Figure 4 Tsetse have been extensively studied because of their disease transmission. These flies are multivoltine, typically producing about four generations yearly, and up to 31 generations total over their entire lifespans (Source: https://parasitesandvectors.biomedcentral.com/articles/10.1186/s13071-018-3274-x)

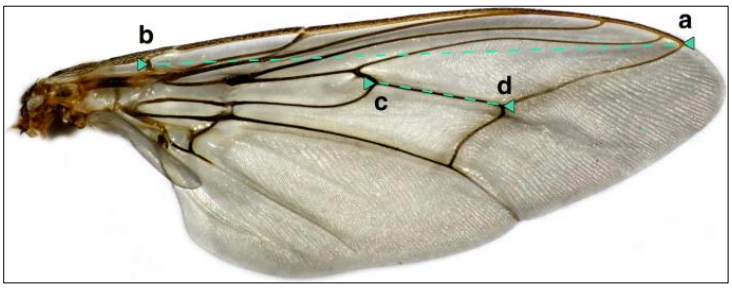

Figure 5 The right wing of a female *G. m. morsitans* showing the endpoints a and b used as a measure of wing length in present studies. The hatchet cell length used in classical studies is measured between points c and d; (Source: https://parasitesandvectors.biomedcentral.com/articles/10.1186/s13071-018-3274-x)

Figure 6 Glossinidae Theobald, 1903 Similar *Trypanosoma,* Fly, *Trypanosoma brucei*, Insect, Glossinidae; (Source: https://alchetron.com/Tsetse-fly)

Figure 7 Tse-tse fly, known as the sleep fly; (Source: Patrick Robert/Sygma/Getty Images)

1.1. Taxonomy

The genus *Glossina* is usually divided into three groups of species, based on their geographic distribution, behavior, and morphology:

1.1.1 Grupo morsitans

(tsetse flies from the savannas): *Glossina austeni* (Newstead, 1912), *Glossina longipalpis* (Wiedemann, 1830), *Glossina orsitans* (Wiedemann, 1850), *Glossina morsitans morsitans* (Wiedemann, 1850), *Glossina morsitans centralis* (Machado, 1970), *Glossina morsitans submorsitans* (Newstead, 1911), *Glossina pallidipes* (Austen, 1903) and *Glossina swynnertoni* (Austen, 1923).

1.1.2 Grupo fusca

(tsetse flies from forested regions): *Glossina brevipalpis* (Newstead, 1911), *Glossina fusca* Walker, 1849), *Glossina fusca fusca* (Walker, 1849), *Glossina fusca congolensis* (Newstead and Evans, 1921), *Glossina fuscipleuris* (Austen, 1911), *Glossina frezili* (Gouteux, 1987), *Glossina haningtoni* (Newstead and Evans, 1922), *Glossina longipennis* (Corti, 1895), *Glossina medicorum* (Austen, 1911), *Glossina nashi* (Potts,1955), *Glossina igrofusca* (Newstead, 1911), *Glossina nigrofusca nigrofusca* (Newstead, 1911), *Glossina nigrofusca hopkinsi* (Van Emden, 1944), *Glossina severini* (Newstead, 1913), *Glossina schwetzi* (Newstead and Evans, 1921), *Glossina tabaniformis* (Westwood, 1850) and *Glossina vanhoofi* (Henrard, 1952).

1.1.3 Grupo palpalis

(tsetse flies from riparian areas): *Glossina caliginea* (Austen, 1911), *Glossina fuscipes* (Newstead, 1911), *Glossina fuscipes fuscipes* (Newstead, 1911), *Glossina fuscipes martinii* (Zumpt, 1935), *Glossina fuscipes quanzensis* (Pires, 1948), *Glossina pallicera* (Bigot, 1891), *Glossina pallicera pallicera* (Bigot, 1891), *Glossina pallicera newsteadi* (Austen, 1929), *Glossina palpalis* (Robineau-Desvoidy, 1830), *Glossina palpalis palpalis* (Robineau-Desvoidy, 1830), *Glossina palpalis gambiensis* (Vanderplank, 1911) and *Glossina tachinoides* (Westwood, 1850) [2,3].

1.2. Disease Vector Model

Representing a tsetse fly *Glossina morsitans* (Wiedemann, 1850), vector of human African trypanosomosis, commonly referred to as sleeping sickness. Plaster model, consisting of three segments - head, thorax, and abdomen - with complementary structures - wings in wire and Kraft paper, feet in plaster coated wire, antennas in acetate paper, wire proboscis and wax coated with varnish. Brush bristles reproduce the hairy filaments present on the thorax and legs. The model is polychrome and varnished (Figure 8) [4].

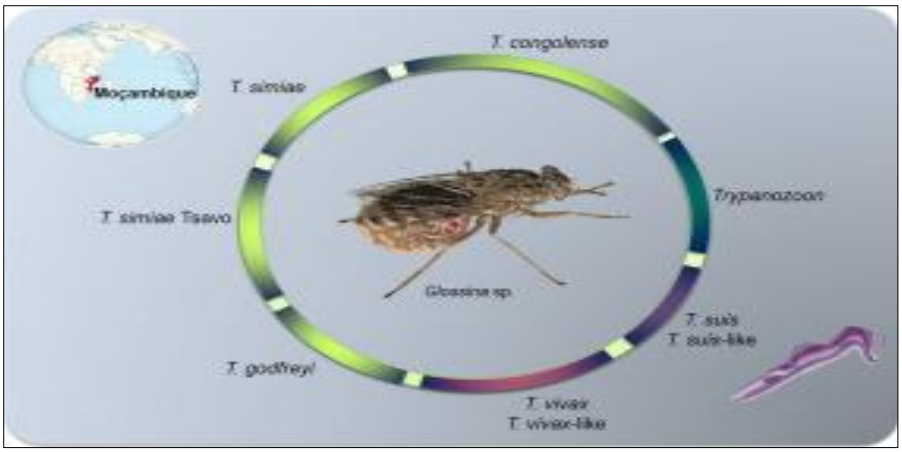

Figure 8 Remarkable richness of trypanosomes in tsetse flies (*Glossina morsitans morsitans* and *Glossina pallidipes*) from the Gorongosa National Park and Niassa National Reserve of Mozambique revealed by fluorescent fragment length barcoding (FFLB); (Source: https://www.sciencedirect.com/science/article/pii/S1567134817302344#s0040)

The model rests on a cylindrical metal foot secured to the base by a hex nut. The base is rectangular and made of wood. In the upper plan cardboard label, held by metal pins, which reads: *Glossina morsitans*. On the front, a metal label with square, fixed by two metal pins, which reads. The object is part of a set of vector models of diseases and pathogens. Glossinas, tsetse flies, or sleeping flies are dipterous insects belonging to the Glossinidae Family, which has a single genus (*Glossina)* and 31 species and subspecies. They are found on the African continent in the sub-Saharan region (Figure 9) [4].

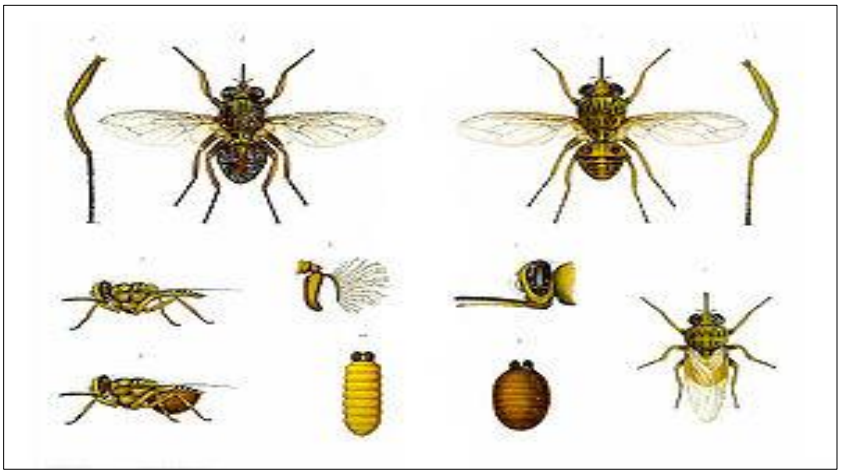

Figure 9 Egg, larvae, pupa, and adult of *Glossina* (tsetse fly); (Source https://en.wikipedia.org/wiki/Tsetse_fly)

The medical and veterinary importance of glossins is because they are vectors of pathogens, both for humans and animals, namely *Trypanosoma brucei gambiense* in West and Central Africa, which gives rise to human African trypanosomosis (THA) *gambiense* type, *Trypanosoma brucei rhodesiense* in East Africa (Rhodesian type THA) and *Trypanosoma brucei brucei* which, in animals, causes Nagana (Figure 10).

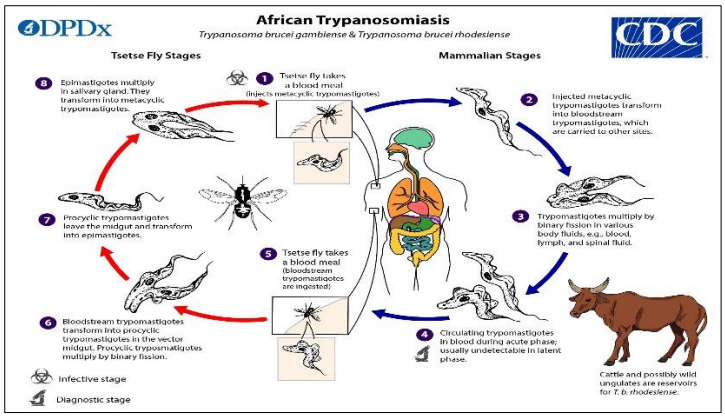

Figure 10 Trypanosomosis (THA)

In these insects both sexes are strictly hematophagous, bite during the day and the three groups (palpalis, morsitans and fusca) have different bioecological and morphological characteristics. Regarding the overall external appearance, all adults or imagos have common characteristics, namely, the central mesh of the wings is in the form of an axe [4].

1.3. Tsetse flies

Are vaguely like other common flies, such as the house fly, but can be distinguished by four characteristics of their anatomy, two of which are easy to observe: The tsetse fly folds its wings fully when at rest so that one rests directly on top of the other on the abdomen. The tsetse fly has a long proboscis, which extends directly forward and is connected by a bulb at the bottom of its head. Other details are the shape of the disc cell of the wing and the edge of the antenna with branching hairs (Figure 11) [5,6].

Figure 11 Tse-Tse Flies Family Glossinidae; (Source: 20886, (c) David Bygott, algunos derechos (CC BY-NC-SA)

1.4. Life cycle

Tsetse flies have an unusual life cycle. The female produces a single egg at each laying, and the larva remains in the uterus during all three stages of its development, a type of viviparity. The female feeds the larvae with the milky secretions of a modified gland in the uterus. The larva emerges after its development is complete, buries itself in the soil, and proceeds to pupate with a sturdy cover. The adult fly emerges after 20 to 30 days [7,8].

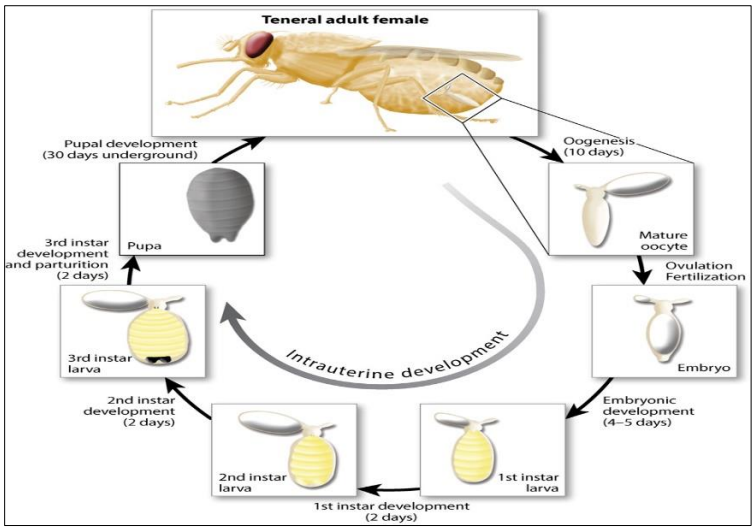

Figure 12 Schematic of the first gonotrophic cycle of a *Glossina morsitans morsitans* (Wiedemann, 1850) female under optimal environmental and nutritional conditions. The different stages of oogenesis, embryogenesis, and larvigenesis within the Glossin reproductive tract (ovaries and uterus) are shown; (Source: https://europepmc.org/article/pmc/pmc4453834)

Objective

This study objective to report the characteristics of the Glossinidae Family.

2. Methods

The research was carried out in studies related to quantitative aspects of the Family, Subfamily and Species (taxonomic groups) and conceptual aspects such as: biology, geographical distribution, species, life cycle, damage, laboratory creation, economic importance, medicinal importance, biological aspects, monitoring and control and reproduction. A literature search was carried out containing articles published from 1950 to 2021. The mini-review was prepared in Goiânia, Goiás, from September to October 2021, through the Online Scientific Library (Scielo), internet, ResearchGate, Academia.edu, Frontiers, Publons, Qeios, Portal of Scientific Journals in Health Sciences, Pubmed, Online Scientific Library (Scielo), internet, ResearchGate, Academia.edu, Frontiers, Biological Abstract, Publons, Qeios, Portal of Scientific Journals in Health Sciences, and Pubmed, Dialnet, World, Wide Science, Springer, RefSeek, Microsoft Academic, Science, ERIC, Science Research.com, SEEK education, Periódicos CAPES, Google Academic, Bioline International and VADLO.

3. Studies performed

3.1. Study 1

We aim to: enhance the contribution of Portuguese researchers who, throughout decades, they dedicated themselves not only to the study of systematic, but also the distribution of called "glossinic stains" in the old colonies, namely in Angola, Guinea, Mozambique and, three times, in the resolution of the glossinic introduction in Príncipe Island, thus how to highlight the historical aspects of the study of sand flies in Portugal; present aspects bioecological of said vectors; spread scientific works that have been carried out, by teams of the IHMT; glossinas or sandflies.

Research was carried out trypanosomal in 4014 people, as well as in domestic animals, 6000 traps were built and distributed for glossinas, captured 166649 flies and identified and dissected 6500 (Figure 13).

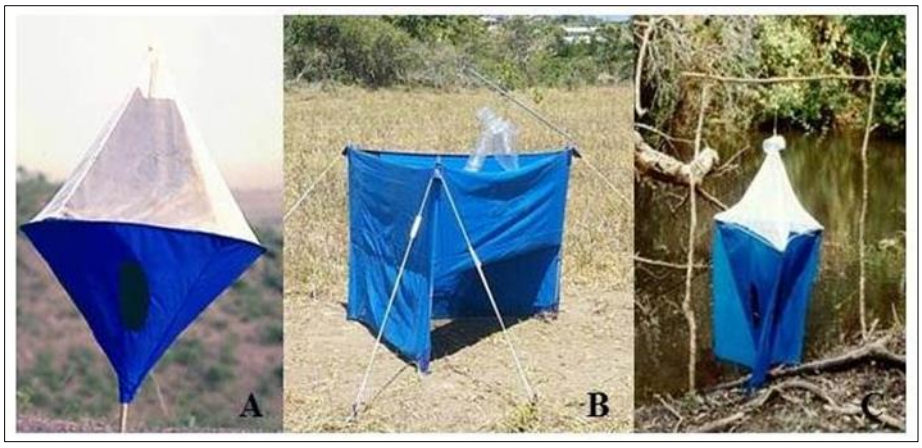

Figure 13 Traps were built and distributed for glossinas; (Source: Carvalho EOA. Genetic structure of populations of *Glossina palpalis gambiensis* (Diptera: Glossinidae) in the Republic of Guinea [Masters Dissertation]. New University of Lisbon. 2011)

Glossinas are flies exclusively African, Sub-Saharan and preferably continental. The Glossinidae family is monogeneric, thus presenting a single genus, the genus *Glossina*, with 31 species and subspecies divided into three groups or subgenres, according to their characteristics ecological and morphological. The distribution limit glossin is due to abiotic and biotic factors. The species and subspecies belonging to the palpalis group or Nemorhina subgenus are distributed mainly by areas of high humidity from West and Central Africa (Figure 5), as well as those belonging to the group Beetle or Austenina subgenus. Those of the group morsitans or *Glossina* subgenus. str. occupies, predominantly savanna and/or woodland areas and open forest areas of East Africa (Figure 14).

Figure 14 Larviposition (*Glossina*); (Source: https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/glossina)

Larviposition is normally diurnal and is carried out in favorable locations for the larva, namely near the usual places of rest of adults, on non-compact soils, by sometimes sandy, under fallen trunks and branches of trees or together with their large roots, in holes in the ground, but always in the shade. In West and Central Africa, during the day, people, in forest, rural and peri-urban areas, as well as their cattle, are subject to being stinged and infected by glossinic species of the palpalis group. In East Africa, hunters, lumberjacks and tourists, in hunting reserves (between other locations), are subject to being chopped an infected by glossinic species of the group morsitans (Figure 14).

Figure 15 Tse-tse pupae: light is young and dark are Order; (Source: Photo courtesy of the DFID Animal Health Program)

African human trypanosomosis (THA), caused by *Trypanosoma brucei gambiense*, occurs in West and Central Africa and can be transmitted, depending on the region, by the following vectors: *Glossina palpalis palpalis, G. p. gambiensis, G. tachinoides, G. fuscipes fuscipes and GF quanzensis*. The THA caused by *T. b.rhodesiense*, which is predominantly seen in East Africa, can be transmitted by *G. morsitans morsitans, G.m. centralis, G. pallidipes* and *G. swynnertoni. Trypanosoma b. brucei, T. congolese* and *T. vivax* are the animal trypanosomes with greater importance in Veterinary Medicine and can be transmitted by glossinas of various species and subspecies.

In susceptible glossins, trypanosomes present morphological transformations, metabolic processes, and multiplications whose place where occur, in the insect, it is variable according to the species trypanosomals. So, *Trypanosoma (Duttonella) vivax*, in the vector, presents the simplest, where almost all of it unrolls in the mouth armor. *T. (Nannomonas)* Congo has an intermediate cycle, where just the proboscis and midgut, or stomach, are involved. *T. (Trypanozoon) brucei* ssp.

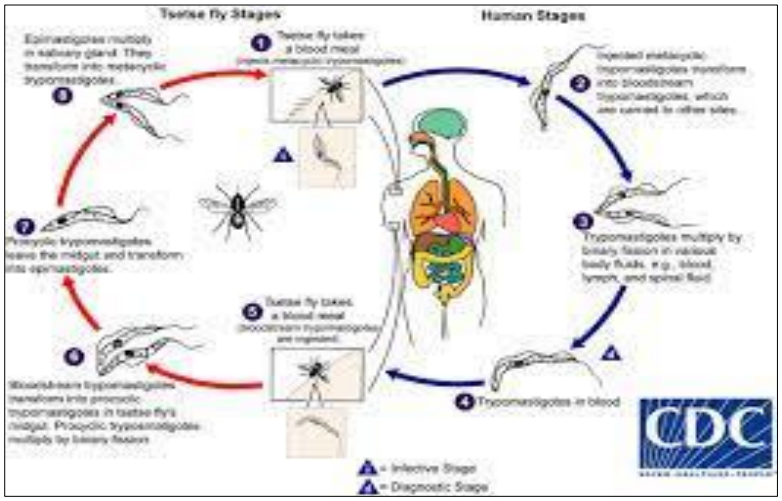

Figure 16 Cycle: *Trypanosoma brucei*, tsetse fly and man. During a blood meal in the mammalian host, an infected tsetse fly (genus *Glossina*) injects metacyclic trypomastigotes into the skin tissue. The parasites enter the lymphatic system and pass into the bloodstream 1. Within the host, they transform into bloodstream trypomastigotes 2. are transported to other locations throughout the body, reach other blood fluids (e.g., lymph, spinal fluid) and continue replication by binary fission 3. The entire life cycle of African trypanosomes is represented by extracellular stages. The tsetse fly is infected with bloodstream trypomastigotes when it takes a blood meal from an infected mammalian host (4,5). In the midgut of the fly, the parasites transform into procyclic trypomastigotes, multiply by binary fission 6, leave the midgut, 7. And transform into epimastigotes. The epimastigotes reach the fly's salivary glands and continue to multiply by binary fission 8. The cycle in the fly takes approximately 3 weeks. Humans are the main reservoir for *Trypanosoma brucei gambiense,* but this species can also be found in animals. Wild game animals are the main reservoir of *Trypanosoma brucei rhodesian; (*Source: lagoasreal.blogspot.com/2017/03/mosca-glossina-palpalis-tse-tse-vetor-doenca-sono-Trypanosoma-brucei.html)

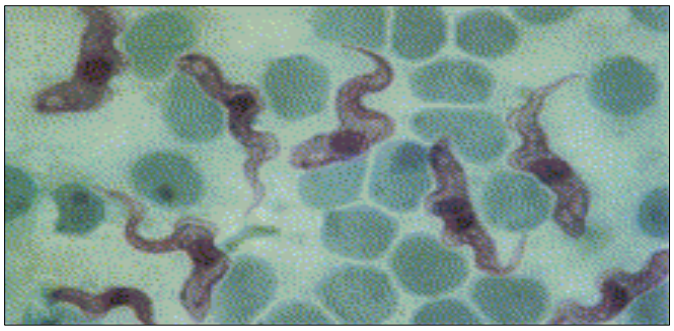

Figure 17 *Trypanosoma brucei gambiense; (*Source: Dorn PL, Noireau F, Krafsur SE, Gregory C, Lanzaro CA Cornel J. Genetics of major insect vectors. Genetics and Evolution of Infectious Disease. 2011; 7: 411-472)

Figure 18 Biology of *Glossina* spp; (Source**:** https://slideplayer.com/slide/13368509/)

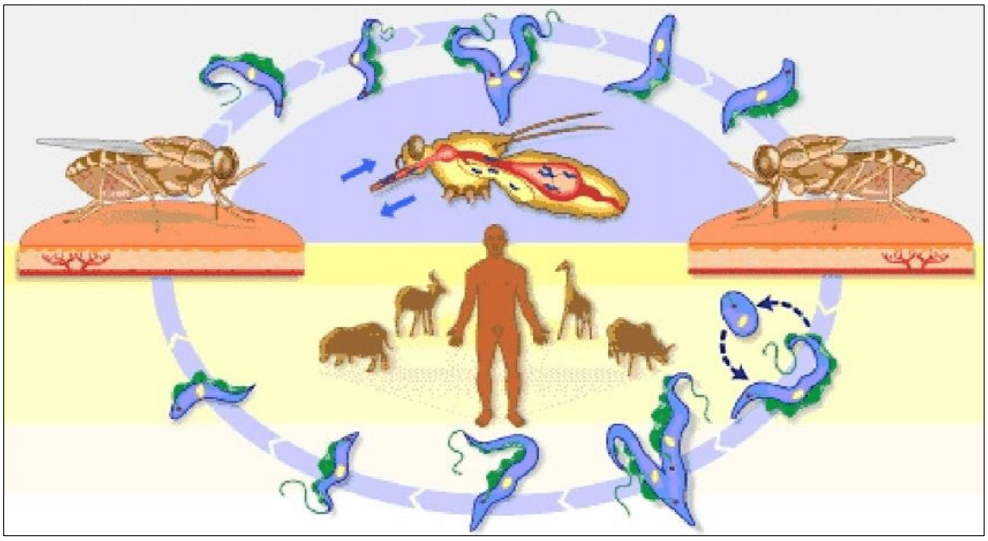

Figure 19 Interaction between *Trypanosoma brucei* (Wiedemann, 1830) and *Glossina morsitans*. During the life cycle of *T. brucei brucei*, the parasites migrate between various organs of the insect. After the blood meal, blood trypomastigotes in their long and short forms are ingested by the insect. Due to the action of proteases, the long forms are eliminated, while the short forms manage to bypass this first defense mechanism. These forms quickly differentiate into procyclic forms, stimulated by factors such as heat shock, citrate, cis-aconitate and proteases. In addition, there are other forms of defense such as reactive oxygen species (ROS), lectins and antimicrobial peptides (atacin, defensin, cecropin and

others not identified). From 3-5 days after infection, the parasites migrate to the ectoperitrophic space, where there is a differentiation from the procyclic to the mesocyclic form. Procyclic trypomastigotes migrate to the proventriculus, a space in which they differentiate into long trypomastigotes, followed by asymmetric division of the trypomastigote into long epimastigote and short epimastigote. Salivary gland infection occurs by short epimastigotes, which are believed to attach to the salivary gland epithelium through the interdigitation of their membranes. After replication, the epimastigote form differentiates into the metacyclic form, which is a free and infective form for vertebrate hosts, transmitted through the saliva of the vector; (Source: https://www.researchgate.net/figure/Life-cycle-of-Trypanosoma-brucei-source-http-wwwwhoint-tdr-diseases_fig2_40447689)

Presents the most complex cycle, implying the stomach (procyclical forms of the parasite), the proventriculus (forms proventricular), the proboscis and, finally, the salivary glands where forms proventriculates transform into epimastigotes, multiply and give rise to forms infective metacyclics that, through saliva, will be inoculated into the vertebrate host. THE duration of cycles, depending on the species of trypanosomes, and environmental conditions, can vary, in the vectors, on average from 6 to 30 days (Figure15 and 16) [9,10,11,12,13,14,15].

3.2. Study 2

Have you ever thought about going into a deep sleep from a fly's sting? This is one of the symptoms of Human African Trypanosomiasis or sleeping sickness. Transmitted by the bite of the tsetse fly, it can lead to death if not treated correctly. Understand what the disease is and how it works.

Sleeping sickness is more common in sub-Saharan Africa, particularly inland. The fly feeds on blood, with the ox as its main host. Even so, it also has human and wild and domestic animals as “victims”, which can end up suffering attacks. The evolution of the disease until it reaches a more aggravated stage, however, is not as fast as it seems. According to research, in all, there are three phases, however, until reaching the third one, it can take months or even years to act in the human body (Figure 18).

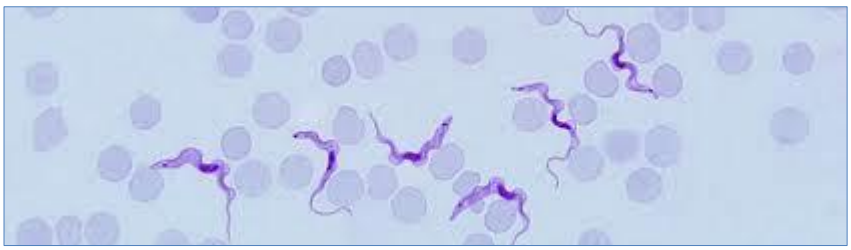

Figure 20 African Trypanosomiasis, also known as “sleeping sickness”, is caused by microscopic parasites of the species *Trypanosoma brucei*. It is transmitted by the tsetse fly (*Glossina* species), which is found only in sub-Saharan Africa. Two morphologically indistinguishable subspecies of the parasite cause distinct disease patterns in humans: *T. b. gambiense* causes a slowly progressing African trypanosomiasis in western and central Africa and *T. b. rhodesiense* causes a more acute African trypanosomiasis in eastern and southern Africa. Control efforts have reduced the number of annual cases and for the first time in 50 years, the number of reported cases fell under 10,000 in 2009. In 2017–2018, fewer than 2000 cases were reported to WHO; (Source: https://www.who.int/gho/neglected_diseases/human_african_trypanosomiasis/en/)

Therefore, it is common for symptoms to be confused with other diseases, or even with a good quality of sleep, as the patient starts to sleep more over time. Main symptoms of sleeping sickness after being infected, the patient has the protozoan in the bloodstream. As a result, it enters the central nervous system and causes symptoms in the body. Check out some of them: High fever; Headaches; Muscle aches; Continuous state of sleep; Deep sleep (Figure 19).

Though far less deadly than years ago, sleeping sickness is still a tough opponent for science to beat. This is because although there is a treatment made with a protein, which can kill the parasite and resolve the issue, it is always one step ahead of the body's immunity. The reason that sleeping sickness still has a high level of mortality is that it worsens its symptoms. At this stage, it is common to experience seizures, deep sleep, and coordination difficulties. This combination increases the patient's chances of death (Table 1) (Figure 19).

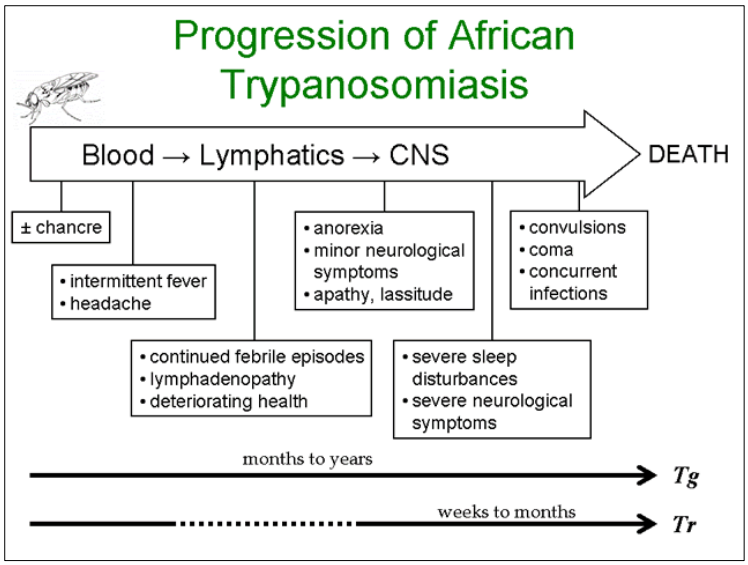

Figure 21 The evolution of the disease until it reaches a more aggravated stage, however, is not as fast as it seems. According to research, in all, there are three phases, however, until reaching the third one, it can take months or even years to act in the human body; (Source: https://www.tulane.edu/~wiser/protozoology/notes/kinet.html)

Table 1 Symptoms present in trypanosomiasis

First (haematolymphatic stage)	Second (meningoencephalitic stage)
Chancre	Sleep disturbances
Lymphadenopathy	Alteration of mental state
Fever	Abnormal reflexes
Headache	Tone disorders
Pruritus (scratch marks)	Abnormal movements
Skin rash	Sensory disorders
Hepatomegaly	Coordination disorders
Splenomegaly	Neurological disorders : convulsions,
Musculoskeletal pains	hemiplegia, neurovegetative disorders,
Anemia	archaic reflexes, deterioration of
Edema (arms & legs)	consciousness
Ascites	Cachexia
Cardiovascular disorders	Coma
Endocrinological disorders	
Renal involvement (albuminuria)	
Intercurrent infections (lung infections)	

Source: https://www.researchgate.net/figure/Major-signs-and-symptoms-in-sleeping-sickness_tbl12_277557185

Treatment difficulties: Another factor that makes proper treatment difficult, in addition to late diagnosis, is that the parasite has more than a thousand versions of a protein. Thus, every time the body tries to fight sleeping sickness by producing antibodies, the parasites transform a new layer of protein. Therefore, sleeping sickness when it is already in the body, dominates the central nervous system. Always changing, she dribbles the human body, staying active even when the organism tries to kill her. The practice contributes to the difficulty of treatment and can worsen the patient's health condition, who is at risk of reaching deep sleep (Figure 20).

Figure 22 *Trypanosoma brucei*; (Source: chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/viewer.html?pdfurl=https%3A%2F%2Fwww.philadelphia.edu)

Attempts to eradicate sleeping sickness One of the main attempts to stop sleeping sickness once and for all is the creation of species of oxen resistant to the parasite. The hypothesis has been tested by scientists since animals are the main hosts of the disease. However, other attempts are also being studied, such as the creation of a protein capable of fighting the disease in the body. Having already been produced, it undergoes improvements in the laboratory to make it work in humans.

However, the attempts come up against the few ongoing research on the subject and the lack of investment by the pharmaceutical industry. Until recently, not many resources were invested to develop drugs aimed at sleeping sickness. As a result, the possibilities for treating infected patients were not expanded. The most recent information indicates that the scenario is changing. Although slowly, the pharmaceutical industry is already forming partnerships and seeking to invest more in research aimed at sleeping sickness. Still, there is a long way to go for healing [16].

3.3. Study 3

3.3.1 Kinetoplastid DNA

Kinetoplastid DNA is relatively abundant and consists of mini-circles and maxi-circles. The two types of ktDNA occur in a concatenated mass within the mitochondria. Maxi-circles encode several mitochondrial genes and are more-or-less equivalent to the mtDNA. Mini circles are heterogeneous and rapidly evolving and their function is less clear. Both mini-circles and maxi-circles encode guide RNA genes. Some genes on the maxi-circles have 'errors' which need to be edited. The guide RNAs are important for this RNA editing that takes place in the mitochondria of kinetoplastids. The editing of these 'cryptogenes' is believed to occur in a hypothetical 'editosome' particle. The extent of editing seems to correlate with different parasite life cycle stages and the corresponding changes in metabolism (i.e., aerobic vs. anaerobic) that are associated with the different life cycle stages. Mini-circle DNA is also used for parasite detection and distinguishing different isolates (Figure 21).

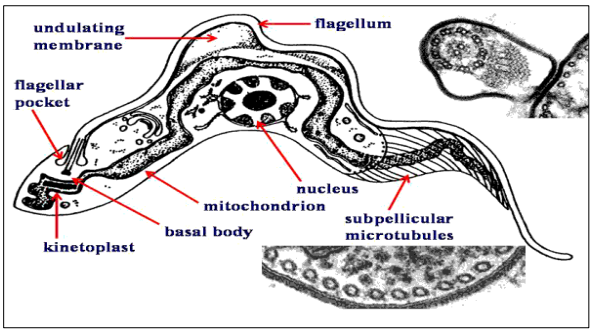

Figure 23 Single flagellum present in many of the morphological forms. A paraxial rod runs along beside the axoneme. The flagellum is sometimes attached to cell body to form undulating membrane; (Source: https://www.tulane.edu/~wiser/protozoology/notes/kinet.html)

- The flagellum emerges from a flagellar pocket. Endo- and exocytosis is limited to this flagellar pocket.

- A single and often branched mitochondria with discoid (rarely flattened or tubular) cristae characterized by the ktDNA discussed above.
- The presence of a peroxisome-like organelle called the glycosome in which glycolysis occurs.
- Organism.

3.3.2 Morphological Forms

Several different morphological forms of kinetoplastids are observed. These various morphological forms are associated with different life cycle stages in the various species. The different forms are distinguished by the position of the kinetoplastid in relation to the nucleus and the presence or absence of an undulating membrane. The four major morphological forms found in kinetoplastids which cause human disease are:

3.3.2.1 Trypomastigote

The kinetoplast (kt) is located on the posterior end of the parasite. The flagellum emerges from the posterior end and folds back along the parasite's body. This attachment of the flagellum to the body forms an undulating membrane (um) that spans the entire length of the parasite, and the free flagellum emerges from the anterior end. This is considered the anterior end since the flagellum pulls the organism and the end with the free flagellum is the front in reference to the direction of movement. The undulating membrane functions like a fin and increases the motility of the organism (Figure 22).

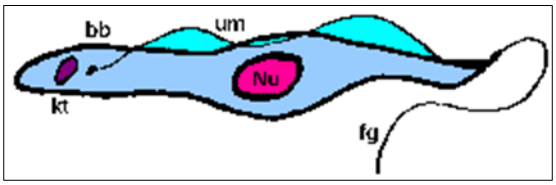

Figure 24 Trypomastigote (Source: https://www.who.int/gho/neglected_diseases/human_african_trypanosomiasis/)

3.3.2.2 Epimastigote

The kinetoplast (kt) is more centrally located, usually just anterior to nucleus (Nu). The flagellum (fg) emerges from the middle of the parasite and forms a shorter undulating membrane (um) than observed in trypomastigotes. Epimastigotes are noticeably less motile than trypomastigotes (Figure 23).

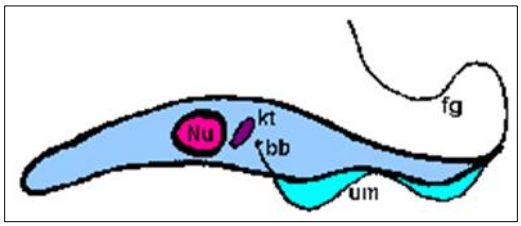

Figure 25 Epimastigote (Source: https://www.who.int/gho/neglected_diseases/human_african_trypanosomiasis/)

3.3.2.3 Promastigote

The kinetoplast (kt) is towards the anterior end and a free flagellum (fg) with no undulating membrane emerges. The end that the free flagellum emerges from in all three motile forms is designated as the anterior end because they swim in that direction. In other words, the flagellum pulls the organism (Figure 24).

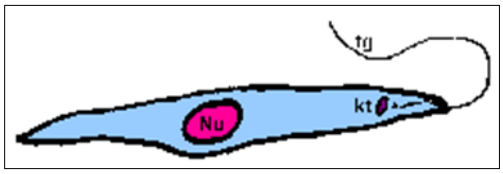

Figure 26 Promastigote (Source: https://www.who.int/gho/neglected_diseases/human_african_trypanosomiasis/)

3.3.2.3 Amastigote

The parasite is more spherical in shape and has no free flagellum. A basal body (bb) and the base of the flagellum is still present. The kinetoplast (kt) is usually detectable as a darkly staining body near the nucleus (Nu). This form is a non-motile intracellular stage (Figure 25) [17,18].

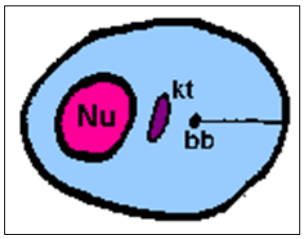

Figure 27 **A**mastigote (Source: https://www.who.int/gho/neglected_diseases/human_african_trypanosomiasis/en/)

3.4. Study 4

African sleeping sickness is an infection caused by the protozoa *Trypanosoma brucei gambiense* or *Trypanosoma brucei rhodesiense*. It is transmitted by the bite of a tsetse fly. Sleeping sickness occurs only in equatorial Africa (Figure 26).

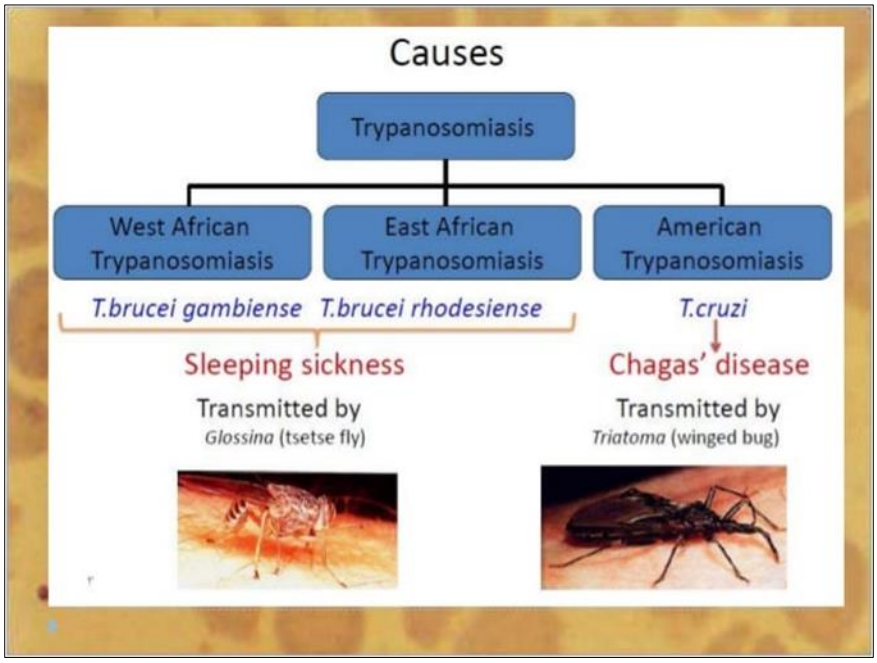

Figure 28 Causes of trypanosomes in Africa and the Americas; (Source: https://pt.slideshare.net/NoeMendez9/trypanosoma-68686921)

A painful lump or ulceration may form at the site of the fly sting, followed by fevers, chills, headaches, swollen lymph nodes, sometimes a rash, and finally drowsiness, trouble walking and, if left untreated, coma and death. Doctors usually confirm the diagnosis by identifying the protozoa in a blood sample, fluid collected from a lymph node, or cerebrospinal fluid. All infected people should be treated with one of several drugs that are effective against *Trypanosoma*.

Sleeping sickness occurs only in parts of equatorial Africa where tsetse flies live. There are two forms of sleeping sickness. Each is caused by a different species of *Trypanosoma*. One form (caused by Trypanosoma brucei gambiense) occurs in west and central Africa. The other form (caused by *Trypanosoma brucei rhodesiense*) occurs in eastern Africa. Both take place in Uganda (Figure 27).

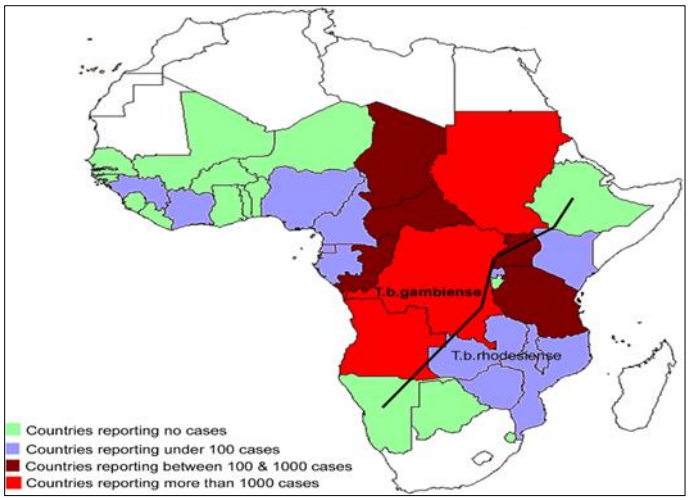

Figure 29 Geographical distribution: The illness of sueno eases millions of people in 36 countries of sub-Saharan Africa. Human African trypanosomiasis, also known as sleeping sickness, is a vector-borne parasitic disease. It is caused by infection with protozoan parasites belonging to the genus *Trypanosoma*. These organisms are morphologically indistinguishable and belong to the *T. brucei* complex: *T. brucei brucei* (animal pathogen), *T. brucei rhodesiense* (Dutton 1902 (East African trypanosomiasis) and *T. brucei gambiense* Dutton, 1902 (West African Trypanosomiasis); (Source: https://old.com.fundacionio.es/salud-io/enfermedades/parasitos/trypanosomiasis-africana-enfermedad-del-sueno/)

There has been a dramatic decrease (>95%, with less than 1,000 cases in 2018) in the number of African sleeping sickness cases reported over the past twenty years because of control efforts. Eradication of the disease has been one of the goals of the World Health Organization. On average, one case is diagnosed in the United States each year, always in travelers returning to the United States or immigrants from endemic regions.

Another species, *Trypanosoma cruzi* Chagas 1909, is endemic to South and Central America and causes Chagas disease (American trypanosomiasis) (Table 2).

Table 2 Features of *T.b. gambiense* and *T.b. rhodesiense* HAT

		T.b. gambiense	*T.b. rhodesiense*
Geographical spread		West Africa	East/South Africa
% of all HAT cases		90	10
Disease specificity		Mainly humans	Wild/domestic animals and humans
Time frame		Chronic	Acute
Acute symptoms (weeks to months)		Few	Swelling at bite site; occasional headaches; irregular fevers; pruritis; adenopathies
Chronic symptoms (months to years)		Severe headaches; sensory and motor disturbance (hyperaesthesia, paralysis); sleep disorders; psychiatric disorders (confusion, agitation)	n/a
Drug treatment	Stage 1	Pentamidine	Suramin
	Stage 2	Melarsoprol; eflornithine; *eflornithine-nifurtimox*	Melarsoprol

Source: https://www.researchgate.net/figure/Features-of-Tb-gambiense-and-Tb-rhodesiense-HAT_tbl1_47533527

Trypanosoma brucei gambiense Dutton, 1902 and *Trypanosoma brucei rhodesiense* Dutton, 1902 are usually transmitted to people when they are bitten by an infected tsetse fly, and it injects the protozoa into their skin. Protozoa migrate to the lymphatic system and bloodstream, where they multiply. Then they travel to organs and tissues throughout the body and eventually reach the brain. Infection is spread when a fly bites an infected person or animal and then bites another person. An infected mother can transmit protozoa to her baby during pregnancy or delivery. In rare cases,

people are infected by blood transfusions. Theoretically, the infection could be transmitted through an organ transplant from an infected donor [19,20,21].

3.5. Study 5

The human infectivity of *T. b. rhodesiense* is imparted by a serum-resistance-associated gene (RAS) product that makes the parasite resistant to lytic activity in human serum. Also, gambiense is genetically and biochemically different from the other two species (Figure 28) [22,23,24,25,26].

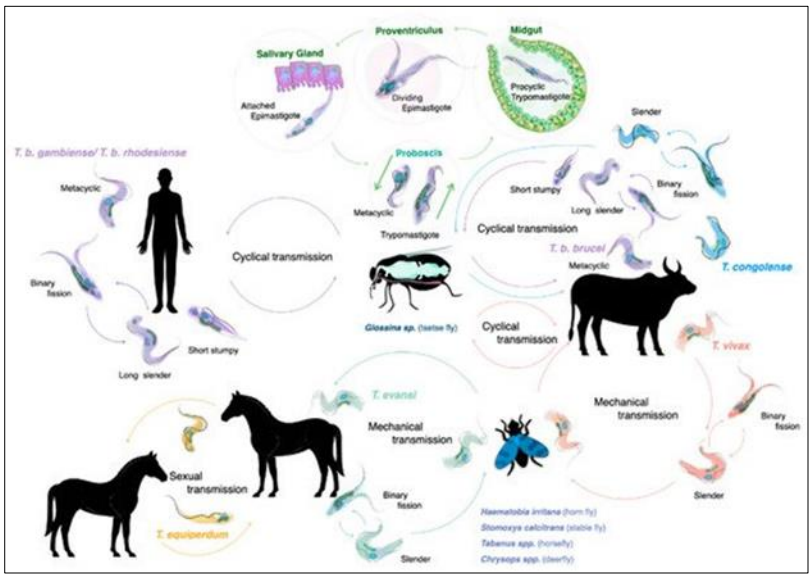

Figure 30 The life cycles of salivarian trypanosomes. For most HAT- and AT-associated trypanosomes, the tsetse (fly) serves as a central transmission vector. Most of the developmental stages of the trypanosome, as well as occasional sexual reproduction, take place inside the fly, making it the definitive host for the trypanosome. This is the case for all *T. brucei* sub-species, *T. congolense* and *T. vivax*. While *T. vivax* is also passed through mechanical transmission, involving mostly non-tsetse biting flies, such transmission is much less effective in the case of *T. congolense*. *T. evansi* is mainly transmitted by mechanical transmission through a wide host reservoir, while the closely related *T. equiperdum* is a sexually remitted parasite of equines; (Source: https://www.mdpi.com/2076-0817/10/6/679/htm)

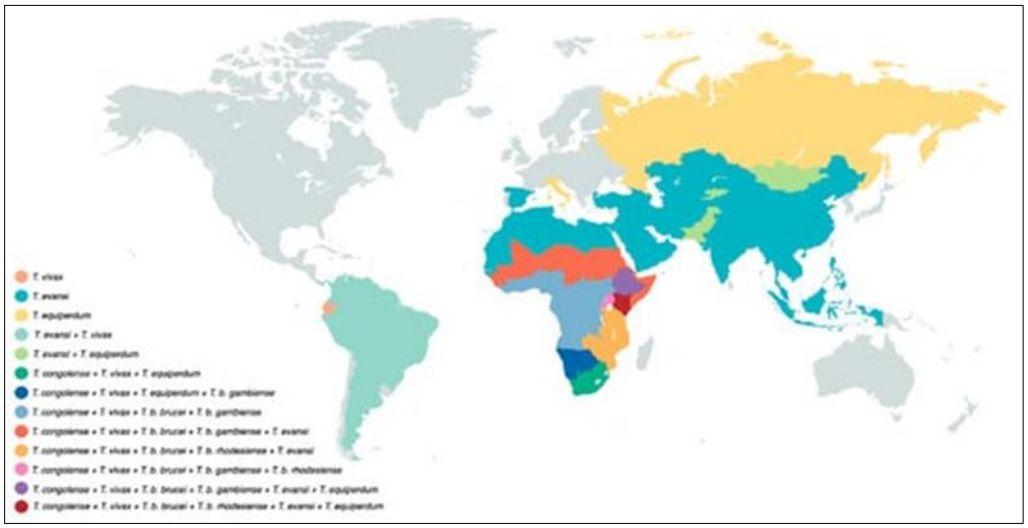

Figure 31 Salivarian trypanosomes have a vast near-worldwide distribution. Tsetse-transmitted *T. brucei* parasites occur only in sub-Saharan Africa, with the human-infective *T. b. gambiense* being present in West and Central Africa, while *T. b. rhodesiense* is restricted to East Africa. *T. congolense* has a similar sub-Saharan Africa distribution. Due to the possibility of mechanical transmission, *T. vivax* has a wider distribution and occurs in sub-Saharan Africa as well as South America. *T. evansi* has an even wider geographic distribution, including locations on four different continents. *T.*

equiperdum has a rather unique distribution pattern as it does not use insect vector transmission as a means of propagation; (Source: https://www.mdpi.com/2076-0817/10/6/679/htm)

Infected feral or domestic animals, such as cattle, act as reservoirs for human disease. The trypanosome is absorbed by a blood meal of the female tsetse fly Glossina from a human or animal and undergoes a complex cycle of morphological and biochemical development. Flies become infectious about 21 days after feeding on an infected host. The tsetse flies again feeds on blood and injects trypanosomes through its saliva into the human's skin. The parasite is taken up by local lymph nodes and enters the bloodstream (Figures 29 and 30) (Table 3).

Table 3 Features of *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense* HAT (Human African trypanosomiasis)

		T.b. gambiense	*T.b. rhodesiense*
Geographical spread		West Africa	East/South Africa
% of all HAT cases		90	10
Disease specificity		Mainly humans	Wild/domestic animals and humans
Time frame		Chronic	Acute
Acute symptoms (weeks to months)		Few	Swelling at bite site; occasional headaches; irregular fevers; pruritis; adenopathies
Chronic symptoms (months to years)		Severe headaches; sensory and motor disturbance (hyperaesthesia, paralysis); sleep disorders; psychiatric disorders (confusion, agitation)	n/a
Drug treatment	Stage 1	Pentamidine	Suramin
	Stage 2	Melarsoprol; eflornithine; *eflornithine-nifurtimox*	Melarsoprol

Source: https://www.researchgate.net/figure/Features-of-Tb-gambiense-and-Tb-rhodesiense-HAT_tbl1_47533527

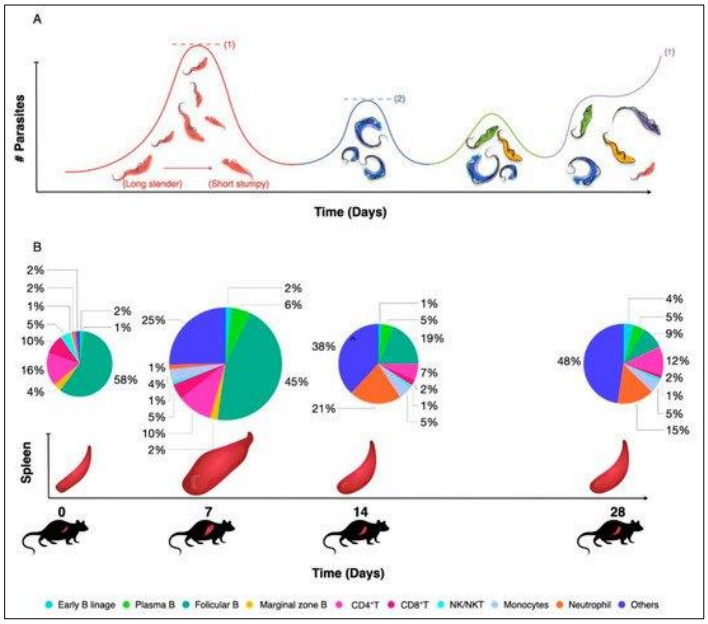

Figure 32 Salivarian trypanosomes use antigenic variation of their surface coat as a first line of defense against host antibody attack. During early infection, quorum sensing ensures that peak parasitemia does not reach lethal levels (1). After clearance of the first variant, parasitemia is characterized by the presence of parasites expressing a novel VSG coat, usually giving rise to several low-peak infections (2). Improved peak control results from a combination of antibody activity, innate inflammatory responses, and intrinsic quorum sensing. Subsequent parasitemia waves start to be comprised of multiple VSG variants that occur at the same time, indicating a loss of proper antibody-mediated parasite population control. In experimental models, infection will most often result in late-stage uncontrolled parasitemia and death. (B) As early parasitemia progresses in mice, infection-associated splenomegaly results in an

initial increase in organ size and cellularity (7 dpi). By 14 dpi, spleen cell numbers usually drop and important populations such as Marginal Zone B cells start to disappear. Organ structure is also destroyed. As infection progresses, most adaptive immune cell populations collapse, while the spleen is being filled with non-immune cells such as pre-erythrocytes. This stage of spleen dysfunction coincides with the loss of parasitemia control. The diameter of the pie-charts is representative of the total spleen numbers during infection. Percentages of all major immune cell populations are indicated in the color-coded pie charts; (Source: https://www.mdpi.com/2076-0817/10/6/679/htm)

4. Conclusion

Currently, due to the seriousness of the African trypanosomoses, several researchers of the IHMT, in collaboration with other institutions, have carried out studies on glossins, although, like sandflies, tsetse flies are considered of "less importance" in relation to other vectors. However, this prejudice is, luckily, to disappear.

References

[1] Van VJA. The Tsetse Fly Glossina fuscipes Newstead, 1911, in East Africa: some aspects of its biology and its role in the epidemiology of human and animal trypanosomiasis. [Doctoral Thesis]. University of Amsterdam. 1971.

[2] Ahmed AB. A peridomestic population of the tsetse fly *Glossina palpalis palpalis* Robineau-Desvoidy, 1830 (Diptera: Glossinidae) at Kontagora town, Niger state, Nigeria. Entomología y Vectores. 2014; 11(4): 599-610, 2004

[3] Gouteux JP. Une nouvelle glossine du Congo: Glossina (Austenina) frezili sp. nov. (Diptera: Glossinidae). Tropical Medicine and Parasitology. 1987; 38(2): 97–100.

[4] Tsetse fly [Internet] Lisboa: Museu da Saúde © 2021 [cited 2021 Nov 01].

[5] Farrar JHP, Junghanss T, Kang G, Lalloo D, White NJ. Manson's tropical diseases. 23rd ed. Philadelphia: Saunders. 2013.

[6] Liverpool school of tropical medicine. encyclopedia of arthropod-transmitted infections of man and domesticated animals. 1st ed. New York: Centre for Agriculture and Biosciences International. 2001.

[7] Jordan AM. Tsetse-flies (Glossinidae). Tropical Medicine and Parasitology. 1987; 38(2): 97-100-143.

[8] Savini G, Scolari F, Ometto L, Rota-Stabelli O, Carraretto D, Gomulski LM, Gasperi G, Abd-Alla AMM, Aksoy S, Attardo GM, Malacrida AR. Viviparity and habitat restrictions may influence the evolution of male reproductive genes in tsetse fly (*Glossina*) species. BMC Biology. 2021: 19(1): 211-223.

[9] Alfonso MO. Bioecology and medical importance of Glossiins (Diptera, Glossinidae) and of Phlbotomes (Diptera, Psychodidae) as trypanosomatid vectors. 2012; 11: 85-89.

[10] Carvalho EOA. Estrutura genética de populações de *Glossina palpalis gambiensis* (Diptera: Glossinidae) na República da Guiné [Masters dissertation]. Universidade Nova de Lisboa. 2011.

[11] Mosca Glossina palpalis (tsé-tsé), vetor da doença do sono. [Internet] Alagoas: Alagoas Reals Bloh © 2021 [cited 2021 Nov 01].

[12] Dorn PL, Noireau F, Krafsur SE, Gregory C, Lanzaro CA, Cornel J. Genetics of major insect vectors. Genetics and Evolution of Infectious Disease. 2011; 7: 411-472.

[13] van Vesten AJ. The Tsetse Fly *Glossina fuscipes fuscipes* Newstead, 1911, in East Africa: some aspects of its biology and its role in the epidemiology of human and animal trypanosomiasis. [Doctoral Thesis]: Amsterda: University of Amsterdam. 1971.

[14] Jordan AM. Tsetse-flies (Glossinidae). 1th ed. London: Chapman and Hall. 1993.

[15] More A, Richer, M. Re-emergence of epidemic sleeping sickness in south-Sudan. Tropical Medicine & International Health. 2012; 6:342-347..

[16] Parasites - African trypanosomiasis (also known as sleeping sickness) [Internet] New York: The Centers for Disease Control and Prevention (CDC) © 2020 [cited 2021 Nov 01].

[17] Gibson W. Sex and evolution in trypanosomes. International Journal of Parasitology. 2001; 31(5-6): 643-637.

[18] Tait A, MacLeod A, Tweedie D, Masiga C, Turner CMR. Genetic exchange in *Trypanosoma brucei*: evidence for mating prior to metacyclic stage development. Molecular and Biochemical Parasitology. 2007; 151: 133.

[19] Pearson RD. Doença do sono africana (Tripanossomíase africana) [Internet] Kenilworth: Merck Sharp & Dohme Corp © 2020 [cited 2021 Nov 01].

[20] Beaver PC, Jung RC. Clinical Parasitology. 1st ed. Philadelphia: Lea and febiger Publishers. 1984.

[21] Neva FA, Brown HW. Basic Clinical parasitology. 1st ed. Connecticut: Appleton and Lange Publishers. Norwalk, Connecticut. 1994.

[22] Despommier GH. Parasitc Diseases. Protista Phylum: Protozoa Class: Zoomastogophora Order: Kinetoplastida Family: Trypanosomatidae. 1st ed, New York Kingdom: Apple Trees productions. 1990.

[23] Magez S, Pinto TJE, Oh S, Radwanska M. Salivarian Trypanosomes have adopted intricate host-pathogen interaction mechanisms that ensure survival in plain sight of the adaptive immune system. Pathogens 2021; 31;10(6):679.

[24] Wardrop NA, Fèvre EM, Atkinson PM, Welburn SC. The dispersal ecology of rhodesian sleeping sickness following its introduction to a new area. PLOS Neglected Tropical Diseases. 2013; 7(10): e2485.

[25] Neuberger A, Meltzer E, Leshem E, Dickstein Y, Stienlauf S, Schwartz E. The Changing Epidemiology of Human African Trypanosomiasis among Patients from Nonendemic Countries –1902–2012. PLoS ONE. 2014; 9(2): e88647.

[26] Chappuis F, Loutan L, Simarro P, Lejon V, Büscher P. Options for field diagnosis of human African trypanosomiasis. Clinical Microbiology Reviews. 2005; 18(1): 133-46.