- ***Trypanosoma*** is a genus of kinetoplastids (class Kinetoplastida), a monophyletic[1] group of unicellular parasitic flagellate protozoa. The name is derived from the Greek *trypano-* (borer) and *soma* (body) because of their corkscrew-like motion

- All trypanosomes are heteroxenous (requiring more than one obligatory host to complete life cycle) and most are transmitted via a vector. The majority of species are transmitted by blood-feeding invertebrates, but there are different mechanisms among the varying species

Developmental stages of Trypanosomes

A. Amastigote:

Kinetoplast anterior to nucleus, no free flagellum, usually spheroid or subspheroid.

B. Promastigote:

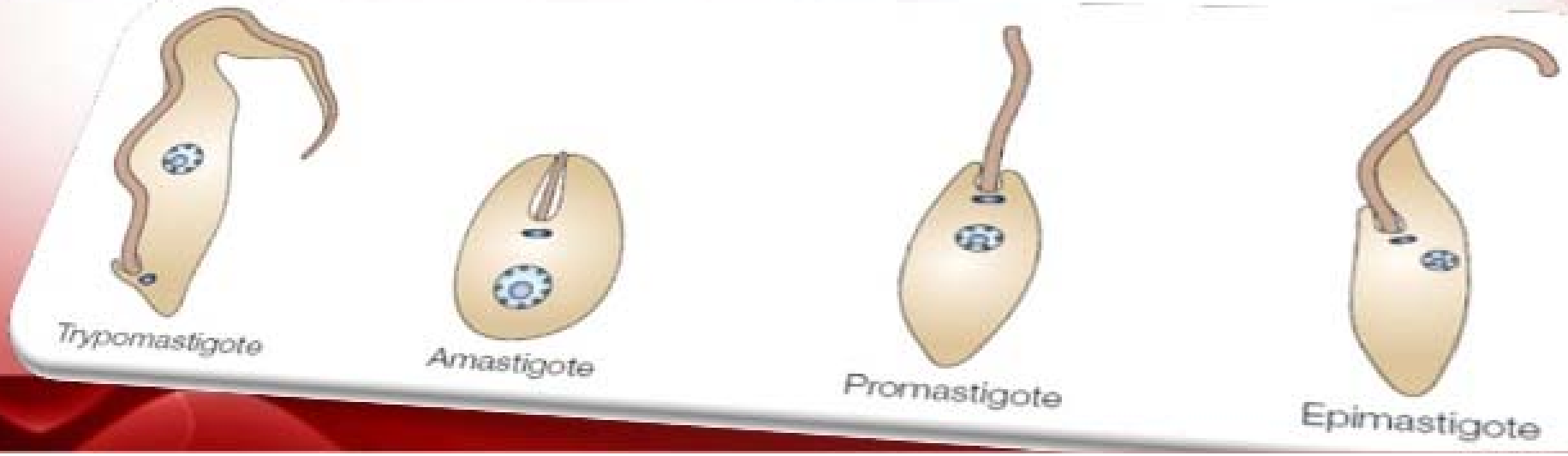
Kinetoplast anterior to nucleus, no undulating membrane.

C. Epimastigote:

Kinetoplast anterior to nucleus, undulating membrane running a portion of the body.

D. Trypomastigote:

Kinetoplast posterior to nucleus, undulating membrane runs length of organism.



TRYPANOSOMA BRUCEI

- Trypanosoma brucei is a protozoan with flagella (protist) species that causes African trypanosomiasis (or sleeping sickness) in humans and nagana in animals in Africa. There are 3 sub-species **of T. brucei: T. b. brucei, T. b. gambiense and T. b. rhodesiense.**

Trypanosoma

Causes

Trypanosomiasis

- West African Trypanosomiasis — *T.brucei gambiense*
- East African Trypanosomiasis — *T.brucei rhodesiense*

→ Sleeping sickness

Transmitted by *Glossina* (tsetse fly)

- American Trypanosomiasis — *T.cruzi*

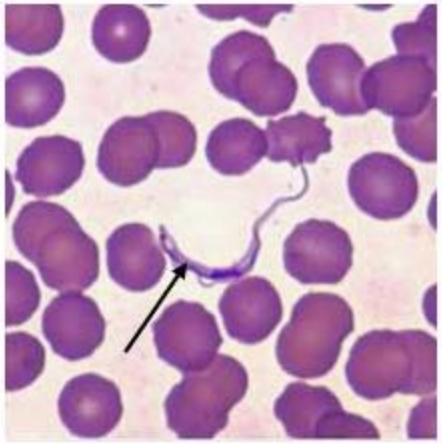
→ Chagas' disease

Transmitted by *Triatoma* (winged bug)

Trypanosoma brucei causing Sleeping Sickness

West Africa

T.brucei gambiense



Less plentiful

Cannot live in lab animals

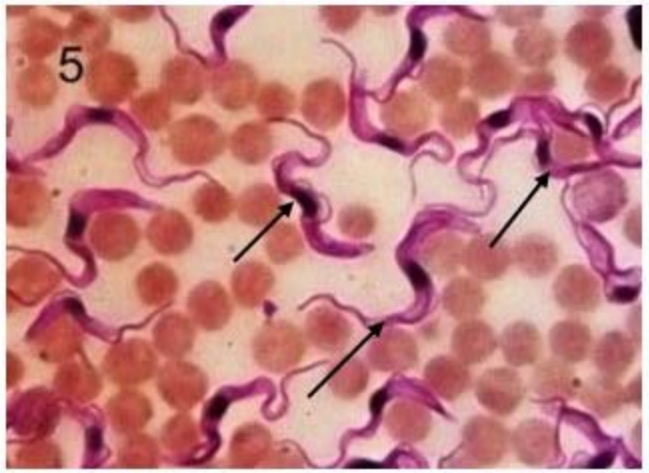
Reservoir host:

goats, cattle & pigs

Transmitted by: *G.palpalis*

East Africa

T.brucei rhodesiense



More plentiful

Can live in lab animals

Nucleus is shifted posteriorly

Reservoir host:

wild game animals

Transmitted by: *G.morsitans*

•There are two subspecies of the parasite that are responsible for initiating the disease in humans. *Trypanosoma brucei gambiense* causes the diseases in west and central Africa whereas,

•, *Trypanosoma brucei rhodesiense* has a limited geographical range and is responsible for causing the disease in east and southern Africa

•In addition, a third subspecies of the parasite known as *Trypanosoma brucei brucei* is responsible for affecting animals but not humans

- These parasites primarily infect individuals in sub-Saharan Africa because that is where the vector (tsetse fly) is located
- *T. b. gambiense* causes a chronic condition that can remain in a passive phase for months or years before symptoms emerge and the infection can last about 3 years before death occurs.

- *T. b. rhodesiense* is the acute form of the disease and death can occur within months since the symptoms emerge within weeks and it is more virulent and faster developing than *T. b. gambiense*

There are two Types of HAT

- *Trypanosoma brucei rhodesiense*
- Acute -- can cause death in weeks or months

- *Trypanosomoa brucei gambiense*
- Chronic --lasts years

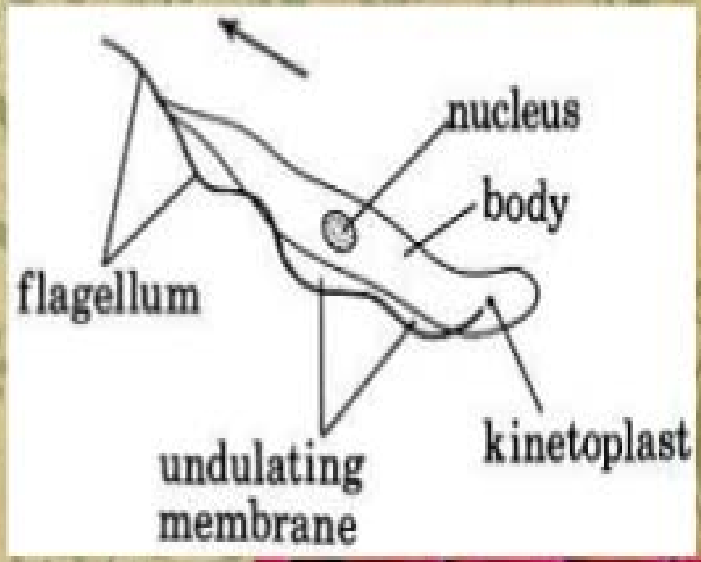
Furthermore

trypanosomes are surrounded by a coat that is composed of variant surface glycoproteins (VSG). These proteins act to protect the parasite from any lytic factors that are present in human plasma. The host's immune system recognizes the glycoproteins present on the coat of the parasite leading to the production of different antibodies (IgM and IgG). These antibodies will then act to destroy the parasites that circulate around the blood. However, from the several parasites present in the plasma, a small number of them will experience changes in their surface coats resulting in the formation of new VSGs. Thus, the antibodies produced by the immune system will no longer recognize the parasite leading to proliferation until new antibodies are created to combat the novel VSGs. Eventually the immune system will no longer be able to fight off the parasite due to the constant changes in VSGs and infection will arise

The Culprit!

Trypanosoma brucei gambiense

Kingdom: **Protista**
Phylum: **Sarcomastigophora**
Class: **Zoomastigophora**
Order: **Kinetplastida**
Family: **Trypanosomatidae**
Genus: ***Trypanosoma***
Species: ***T. brucei***
Subspecies: ***T. b. gambiense***



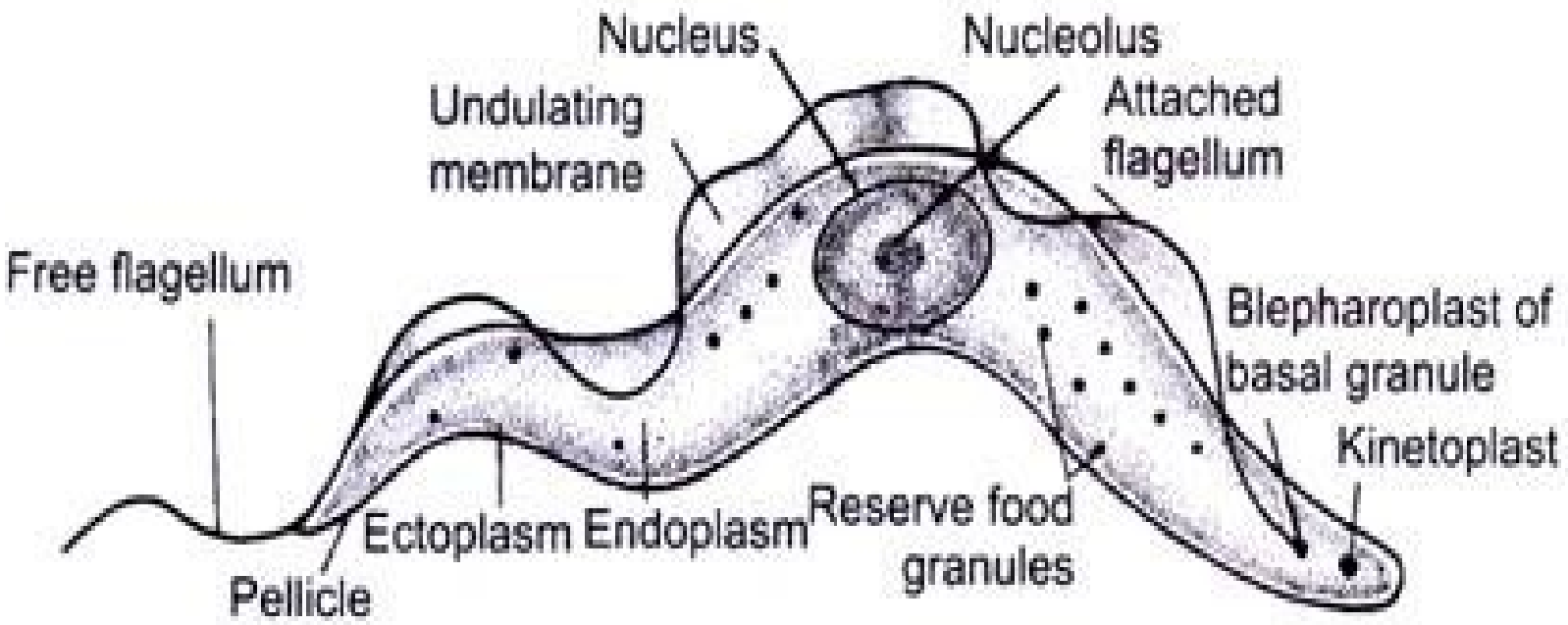
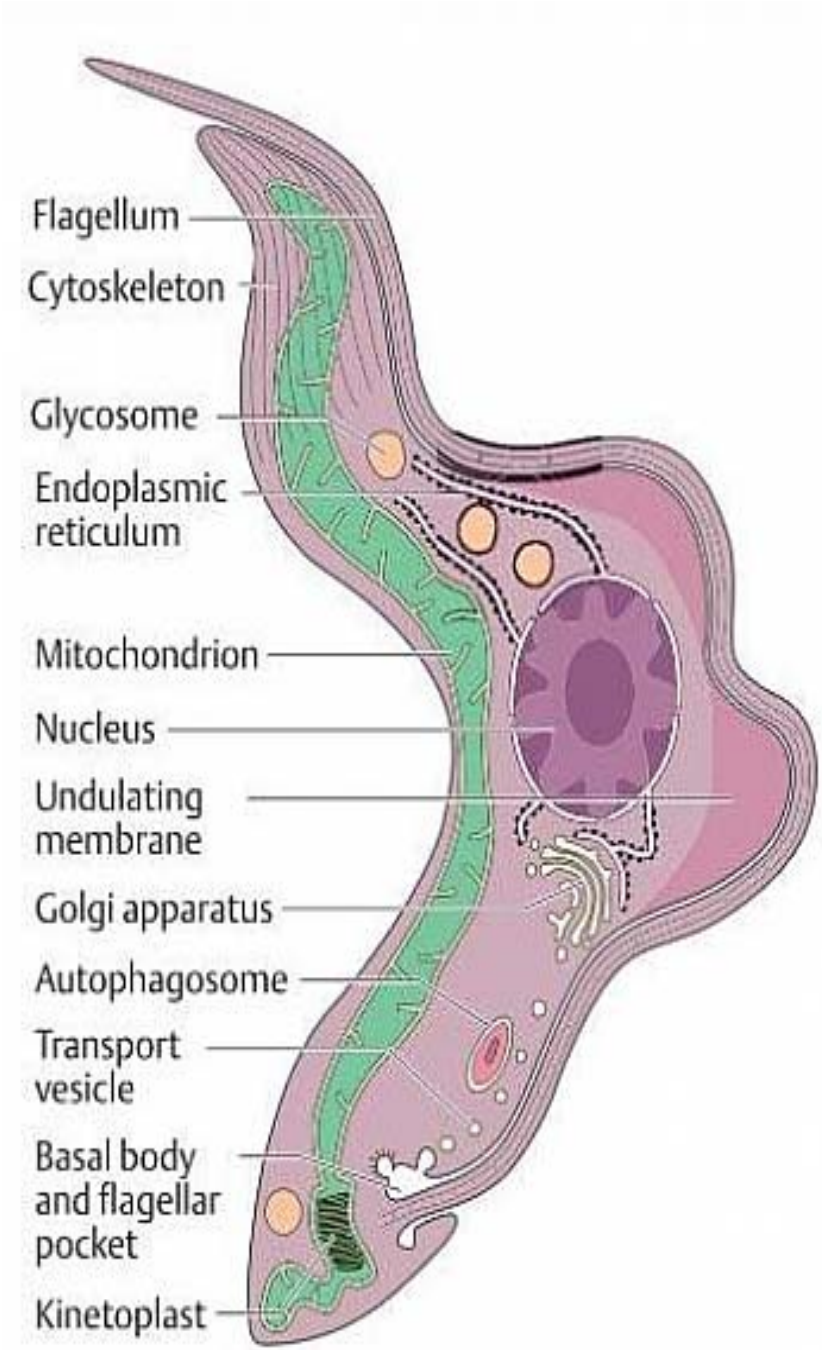
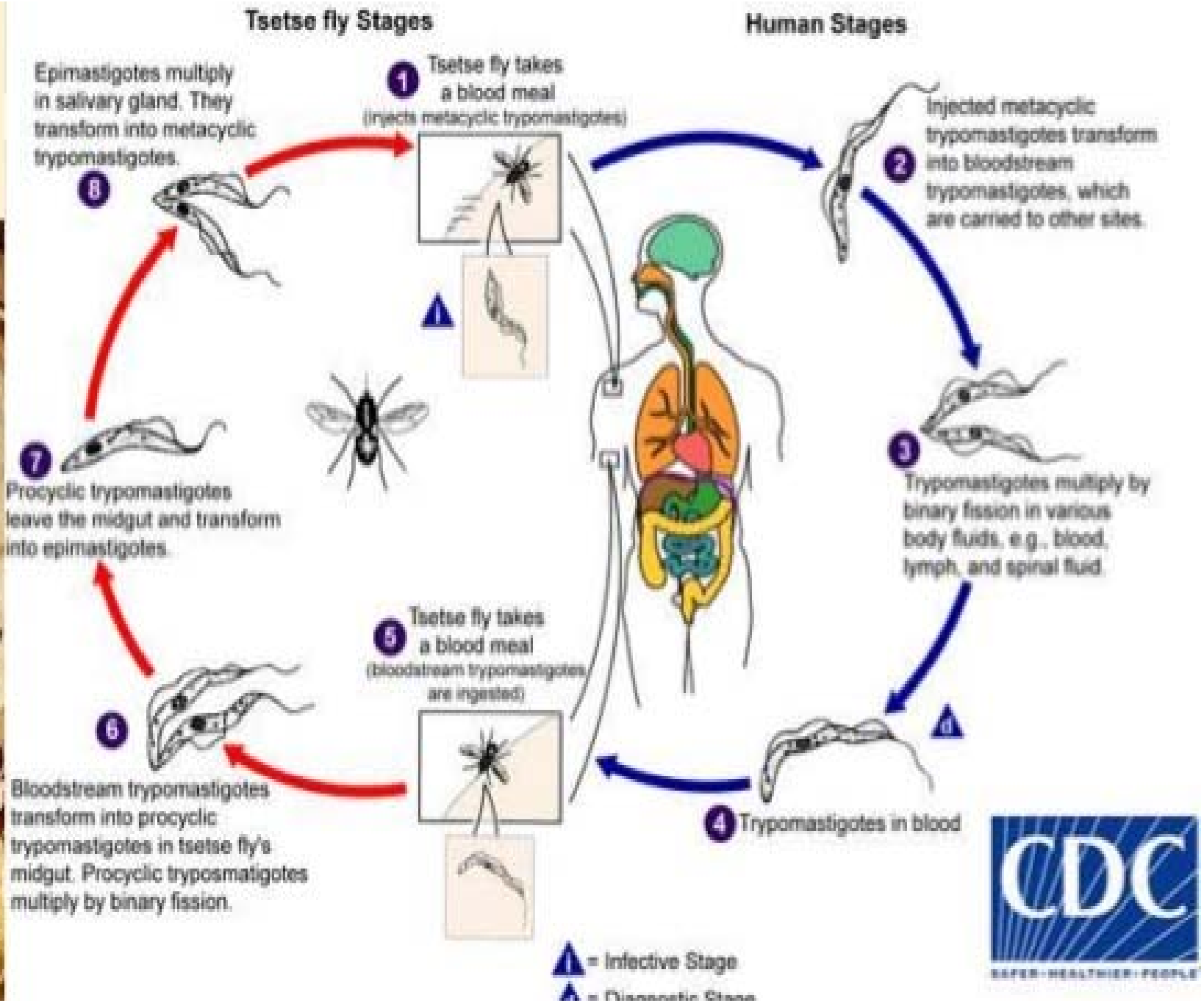


Fig. 9.22 *A Trypanosoma*


Flagellum
Cytoskeleton
Glycosome
Endoplasmic reticulum
Mitochondrion
Nucleus
Undulating membrane
Golgi apparatus
Autophagosome
Transport vesicle
Basal body and flagellar pocket
Kinetoplast

Life Cycle

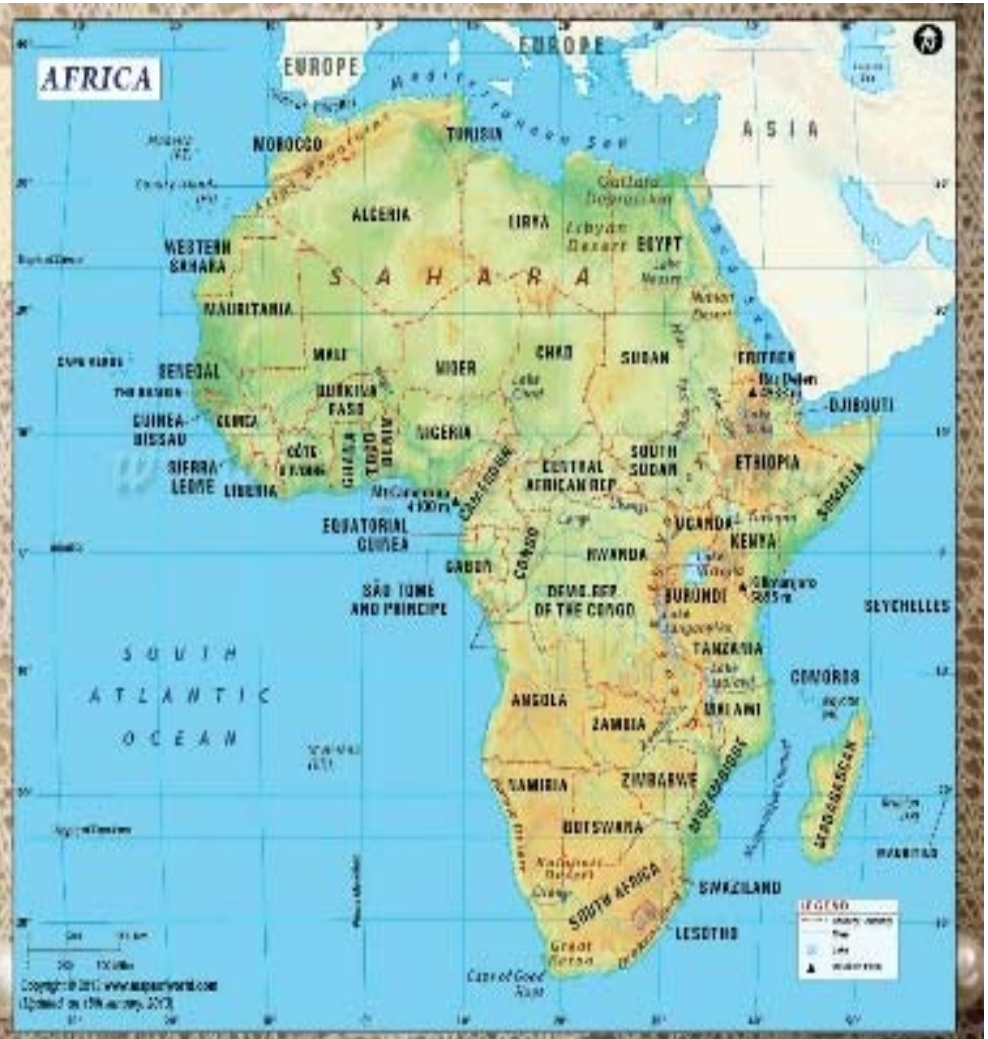

Tsetse fly Stages
Human Stages
Epimastigotes multiply in salivary gland. They transform into metacyclic trypomastigotes.
8
1 Tsetse fly takes a blood meal (injects metacyclic trypomastigotes)
2 Injected metacyclic trypomastigotes transform into bloodstream trypomastigotes, which are carried to other sites.
3 Trypomastigotes multiply by binary fission in various body fluids, e.g., blood, lymph, and spinal fluid.
4 Trypomastigotes in blood
5 Tsetse fly takes a blood meal (bloodstream trypomastigotes are ingested)
6 Bloodstream trypomastigotes transform into procyclic trypomastigotes in tsetse fly's midgut. Procyclic tryposmatigotes multiply by binary fission.
7 Procyclic trypomastigotes leave the midgut and transform into epimastigotes.
= Infective Stage
= Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE

The Vector

Glossina palpalis, commonly known as Tsetse fly, is the carrier of *Trypanosoma brucei gambiense*

Epidemiology

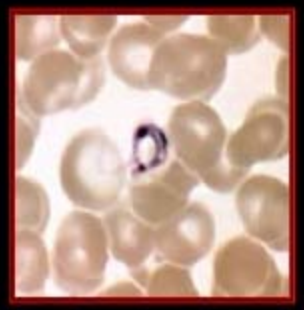
- Limited/distributed only to Central and West Africa



TRYPANOSOMA CRUZI AND CHAGAS' DISEASE

- The etiological agent of Chagas' disease is the intracellular protozoan parasite *Trypanosoma cruzi (T. cruzi)*, which is transmitted by the insect vector *Triatoma infestans* (reduviid bug)
- Reduviid bugs live in mud filled walls of huts in rural areas
- The bug bites human hosts and transmits the parasite

Triatoma infestans
(Reduviid bug)



Trypanosoma cruzi
with human erythrocytes