TRYPANOSOMIASIS

unranked):	Excavata
Phylum:	Euglenozoa
Class:	Kinetoplastea
Order:	Trypanosomatida
Genus:	*Trypanosoma*
Species:	***T. brucei***

Binomial name

Trypanosoma brucei
Plimmer & Bradford, 1899

Subspecies

T. b. brucei
T. b. gambiense
T. b. rhodesiense

African trypanosomiasis (Sleeping sickness

Trypanosoma

Disease: Sleeping sickness (African trypanosomiasis)

- Trypanosomiasis is a systemic disease caused by the parasite *Trypanosoma brucei.*
- East African trypanosomiasis: *T. rhodesiense*
- West African trypanosomiasis: *T. gambiense*.
- It is transmitted by the bite of the **tsetse fly**, a gray-brown insect about the size of a honeybee.

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Etiology

There are two clinical forms of African trypanosomiasis:

1) a slowly developing disease caused by***Trypanosoma brucei gambiense*** and

2) a rapidly progressing disease caused by ***T. brucei rhodesiense*.**

Epidemiolog

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*. They are transmitted to humans by tsetse fly (*Glossina* genus) bites which have acquired their infection from human beings or from animals harbouring human pathogenic parasites.

Tsetse flies are found just in sub-Saharan Africa though only certain species transmit the disease. For reasons that are so far unexplained, in many regions where tsetse flies are found, sleeping sickness is not.

Rural populations living in regions where transmission occurs and which depend on agriculture, fishing, animal husbandry or hunting are the most exposed to the tsetse fly and therefore to the disease. The disease develops in areas ranging from a single village to an entire region. 6,000 to 10,000 human cases are documented annually. 35 million people and 25 million

cattle are at risk. Regional epidemics of the disease are cause of major health and economicdisasters

Morphology

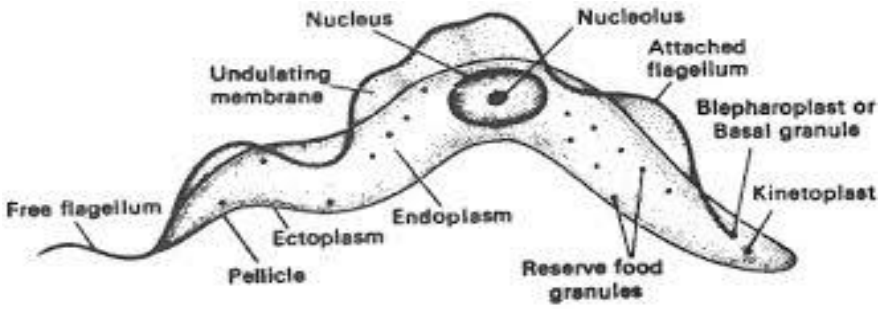
T. b. gambiense and *T. b. rhodesiense* are similar in appearance: The organism

measures 10 - 30 micrometers

x 1-3 micrometers. It has a single central nucleus and a single flagellum originating at the

kinetoplast and joined to the body by an undulating membrane The outer surface of

the organism is densely coated with a layer of glycoprotein,



Trypanosoma gambiense

Life cycle :

During a blood meal on the mammalian host, an infected tsetse fly (genus *Glossina*) injects metacyclic trypomastigotes ito skin tissue.

The parasites enter the **lymphatic system** and pass into the **bloodstream**

❶. Inside the host, they transform into bloodstream**1- trypomastigotes**

❷, are carried to other sites, reach other blood fluids **(e.g., lymph, spinal fluid**), and continue the replication by binary fission

❸. The **tsetse fly** becomes infected with bloodstream **trypomastigotes** when taking a blood meal on an infected mammalian host

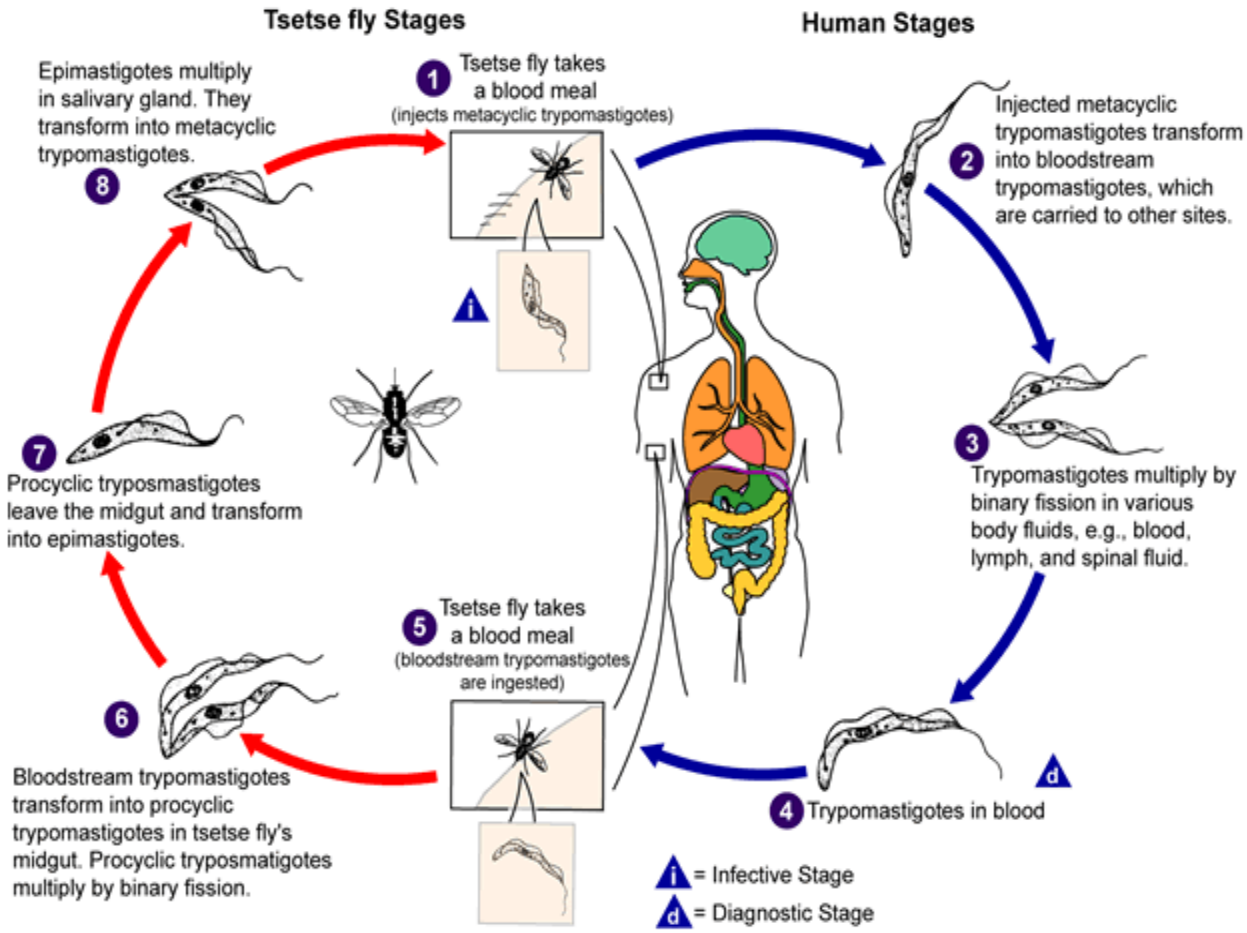
(❹, In the fly's **midgut,** the parasites transform into procyclic trypomastigotes, multiply by binary fission

❻, leave the midgut, and transform into **epimastigotes**

❼. The epimastigotes reach the fly's **salivary glands** and continue multiplication by binary fission transforms to metacyclic trypomastigotes

❽. 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. at a later stage, cross the choroid plexus into the brain and cerebrospinal fluid



The clinical features of Gambian and Rhodesian disease are the same,

. Classically, the progression of African trypanosomiasis can be divided into three stages:

1- **the bite reaction (chancre),**
2- **parasitemia (blood and lymphoid tissues),**
3- **and CNS stage.1**

Bite reaction: A non-pustular, painful, itchy chancre forms 1-3 weeks after the bite and lasts 1-2 weeks. It leaves no scar.

Parasitemia: Parasitemia and lymph node invasion is marked by fever which starts 2-3 weeks after the bite

CNS Stage: The late or CNS stage is marked by changes in character and personality.

.. Death results from coma, intercurrent infection or cardiac failure

-the clinical feature of **Rhodesies disease** are similar but briefer and more acute.

Death is due to cardiac failure within 6-9 months.

Diagnosis

-Detection of parasite in the bloodstream

- Cerebrospinal fluid must be examined for organisms

. Immuno-serology may be indicative.

Treatment and Control

The blood stage of African trypanosomiasis can be treated with reasonable success with

Pentamidine isethionate or Suramin

CNS disease. Cases with CNS involvement should be treated with Melarsoprol, an

organic arsenic compound.

The most effective means of prevention is to avoid contact with tsetse flies.

Vector eradication is impractical due to the vast area involved

American trypanosomiasis (Chagas' disease)

Trypanosoma cruzi

- **Chagas disease**
- Endemic to Central and South America
- Reduviid bug (kissing bug) is the vector
  - Bug feces is inoculated into a cutaneous portal
- Local lesion, fever, and swelling of lymph nodes, spleen, and liver
- Heart muscle and large intestine harbor masses of amastigotes
- Chronic inflammation occurs in the organs (especially heart and brain)
- Treatment nifurtimox and benzonidazole

Etiology :Chagas' disease is caused by the protozoan hemoflagellate, *Trypanosoma cruzi*.

Epidemiology
American trypanosomiasis, also known as Chagas' disease, is scattered in

Central and South America

,. It is estimated that 16-18 million people are infected by the parasite

About 50,000 people die each year from the disease

Morphology
Depending on its host environment, the organism occurs in **three different** forms

1-The trypanosomal **(trypomastigote)** found inmammalian blood, is 15 to 20 microns long

and morphologically similar to African trypanosomes.

2-The crithidial **(epimastigote))** is found in the insect intestine.

3-The leishmanial **(amastigote)** found intracellularly or in in mammalian viscera

(particularly in myocardium and brain),

is round or oval in shape, measures 2-4 microns and lacks a prominent flagellum.

Life cycle

An infected insect vector (or "kissing" bug) takes a blood meal and releases **trypomastigotes**

in its feces near the site of the bite wound.

Trypomastigotes enter the host through the wound or through intact mucosal membranes,

such as the conjunctiva

1. Inside the host, the trypomastigotes invade cells, where they differentiate into intracellular amastigotes

2. The amastigotes multiply by binary fission and differentiate int trypomastigotes, and then are released into the bloodstreamas trypomastigotes

4. Trypomastigotes infect cells from a variety of tissues and transform into **intracellular amastigotes** in new infection sites. Replication resumes only when the parasites enter anothercell or are ingested by another vector.

The "kissing" bug becomes infected by feeding on human or animal blood that

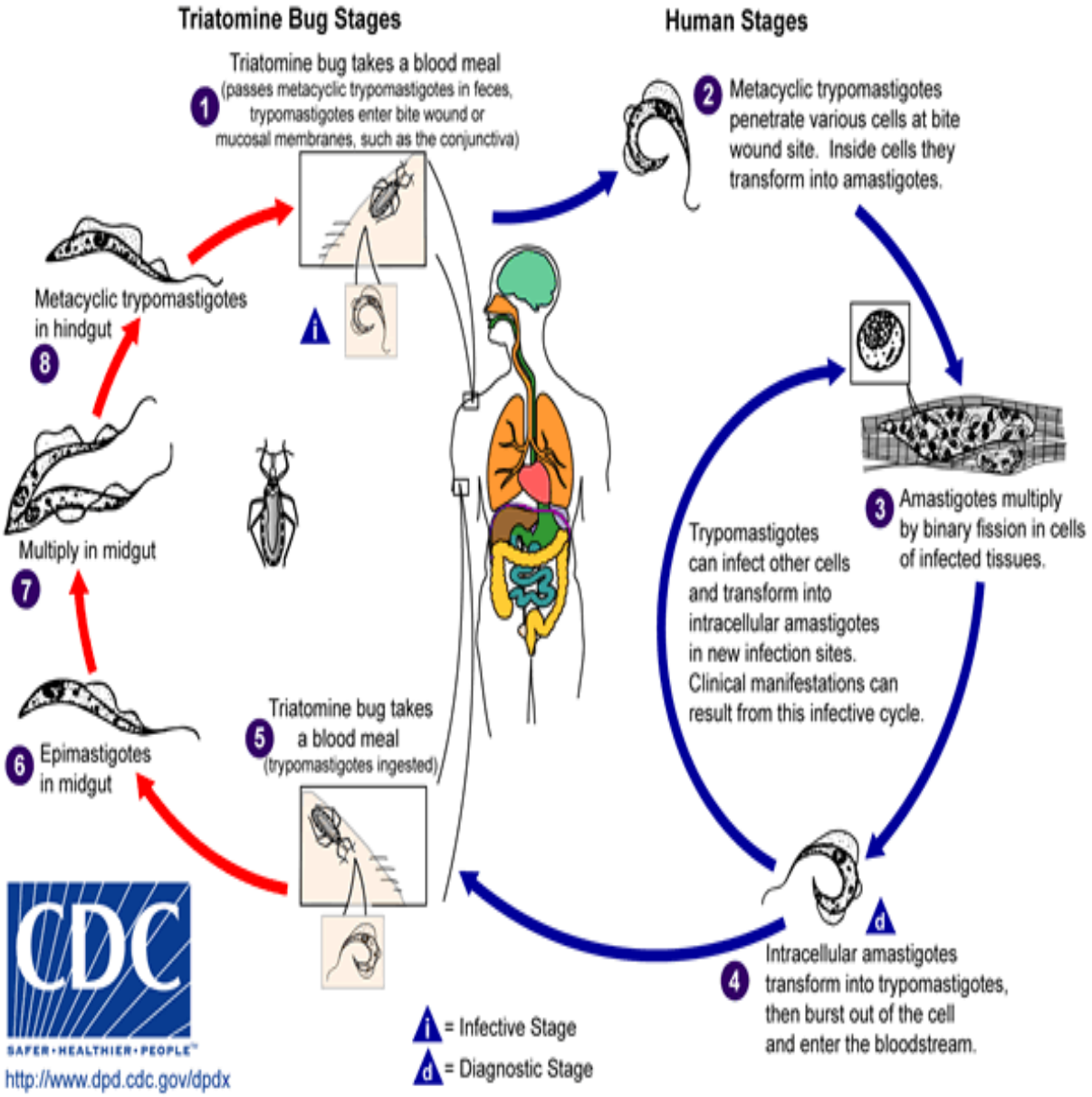
contains circulating parasites

❺. The ingested **trypomastigotes** transform into **epimastigotes** in the **midgut**

❻. The parasites multiply and differentiate in the midgut into infective

metacyclic trypomastigotein the hindgut ❽.Trypanosoma cruzi can also be transmitted

through blood transfusions, organ transplantation, , and in laboratory accidents



symptoms
Chagas' disease can be divided into three stages:

1-The **primary lesion**, chagoma, appearing at the site of infection,

.. It is usually found on the face, eyelids, cheek, lips or the conjunctiva, but may occur on the abdomen or limbs

2-Acute Stage: It is characterized by restlessness, sleeplessness, malaise, increasing, chills, fever and bone and muscle pains. Chagas' disease may cause meningo-encephalitis andcoma.

3-Chronic Stage. The chronic disease results in an abnormal function of the hollow organs, particularly the heart, esophagus and colon

Diagnosis
-Clinical diagnosis is usually easy among children in endemic areas.

. Definitive diagnosis requires the demonstration of trypanosomes by microscopy

-Serological test provide presumptive diagnosis.

Treatment and Control.

-Benzonidazol used in acute stage of the disease,

-Control measures are limited to those that reduce contact between the vectors and man.