BHARATHIDASAN UNIVERSITY

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Programme: M.Sc., Biomedical Science

Course Title : Clinical Microbiology

Course Code: BM36C9

Unit-V

Trypanosoma

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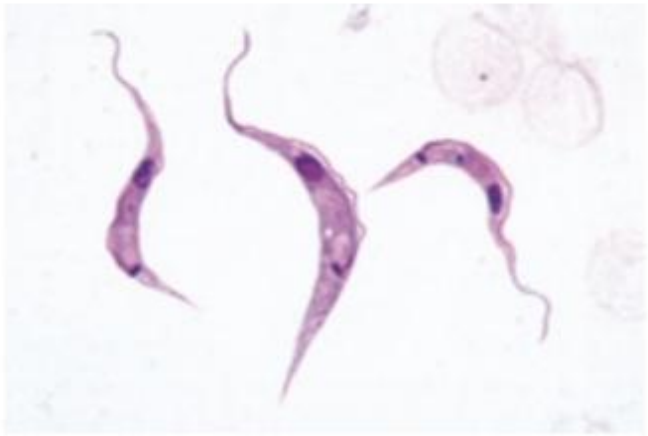
TRYPANOSOMA

African Trypanosomiasis: Introduction

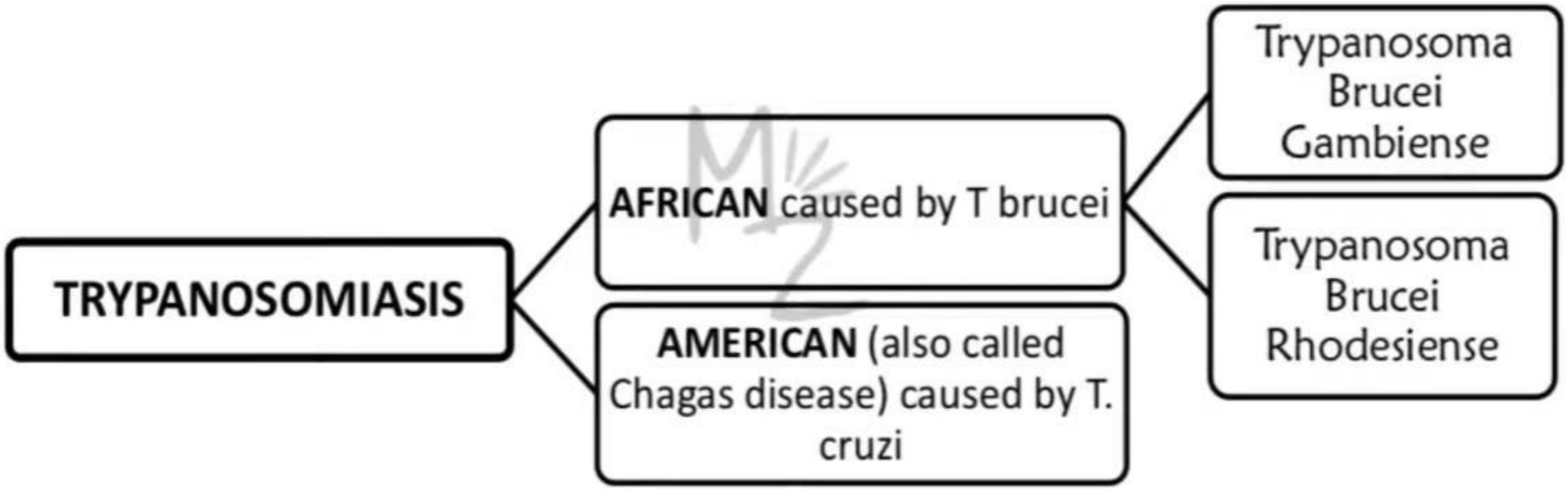
- What is it?
  - *Disease caused by an infection with parasitic protozoa (Trypanosoma spp.)*
  - AKA "African Sleeping Sickness"
  - Transmitted by ***tsetse flies*** of the genus *Glossina*
  - Estimated 100,000 deaths occur each year
  - Sub-Saharan Africa distribution



Trypanosoma

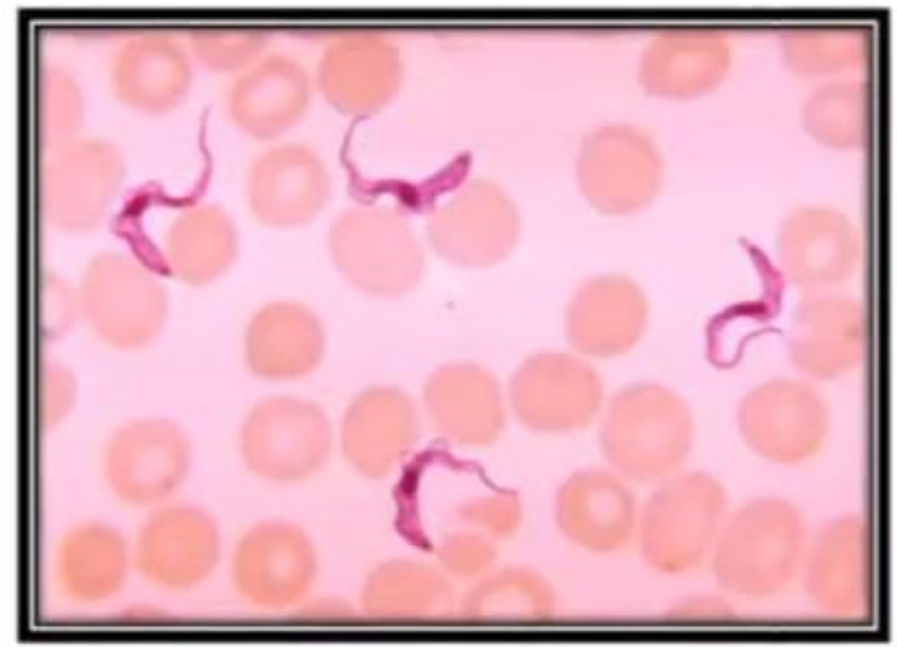
The genus Trypanosoma has three major pathogens

- Trypanosoma cruzi
- Trypanosoma gambiense
- Trypanosoma rhodesiense


TRYPANOSOMIASIS
AFRICAN caused by T brucei
AMERICAN (also called Chagas disease) caused by T. cruzi
Trypanosoma Brucei Gambiense
Trypanosoma Brucei Rhodesiense

Trypanosoma Gambiense & Rhodesiense

- Causes **African sleeping sickness**
- It is called **nagana** in livestock and game animals



Habitat

- **Humans** are considered the main reservoir for Trypanosoma brucei gambiense, but this species can also be found in animals, including primates and ungulates
- **Domestic cattle** are thought to be the most epidemiologically-relevant animal reservoir of Trypanosoma brucei rhodesiense. Bushbuck and other antelopes may serve as a reservoirs

Transmission

- Transmitted to humans by the bite of tsetse flies
- Transplacental transmission also occurs

Morphology of Trypanosome

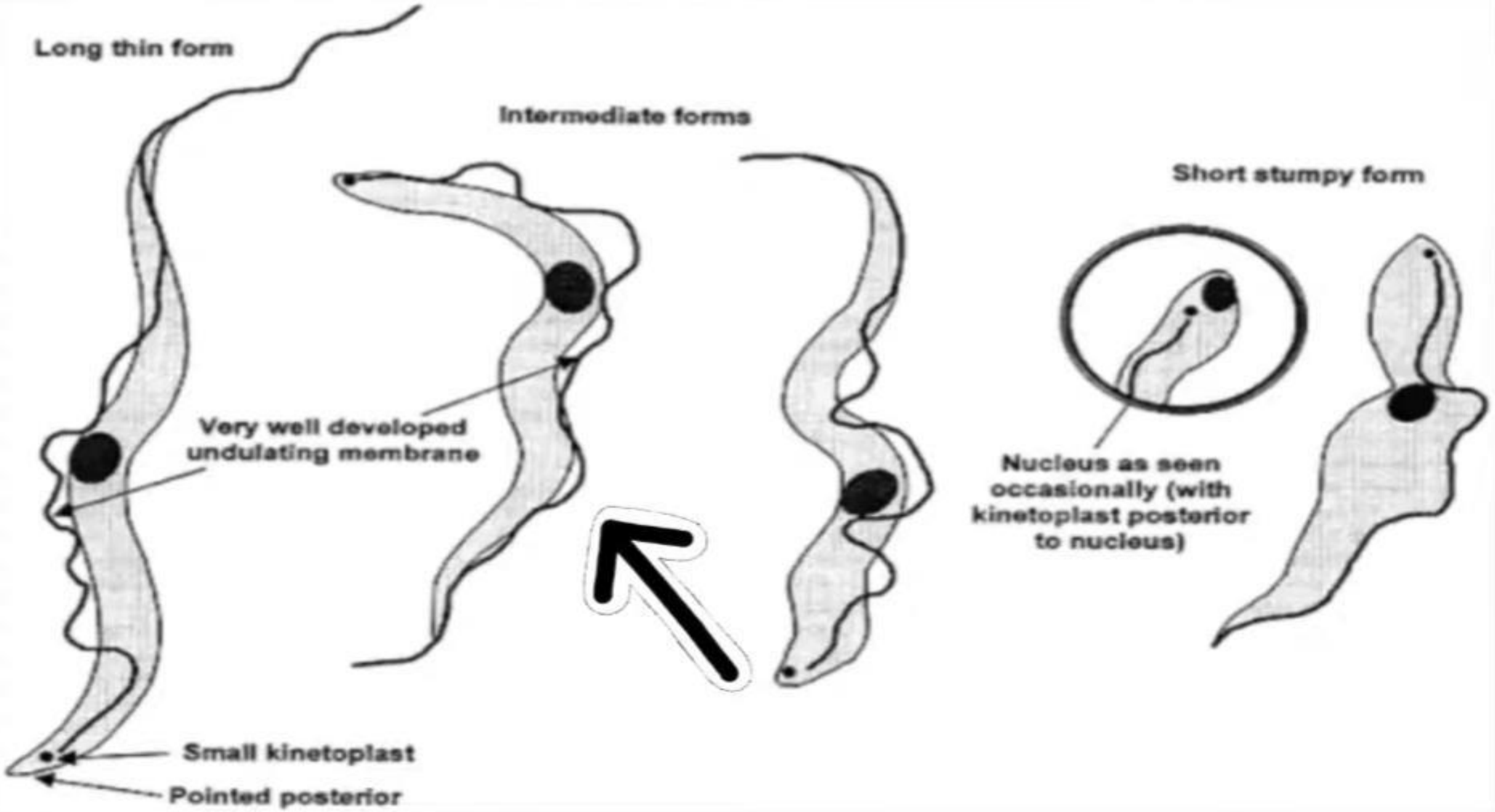
It exists in 2 interchangeable forms

1. **Trypomastigote**
   In blood, lymph, tissue space of various organs & CNS
2. **Epimastigote**
   In salivary glands of vector & culture media

Trypomastigotes (polymorphic trypanosomes)

Spindle shaped, central nucleus, free flagellum & an undulating membrane is present

i. **Long slender form:** active, motile, with free flagellum
ii. **Short stumpy form:** sluggish, without free flagellum
iii. **Intermediate form:** with a short free flagellum


Long thin form
Intermediate forms
Short stumpy form
Very well developed undulating membrane
Nucleus as seen occasionally (with kinetoplast posterior to nucleus)
Small kinetoplast
Pointed posterior

Life Cycle

It involves two stages

1. Tsetse fly stage
2. Mammalian stage

Life Cycle

Tsetse Fly Cycle:

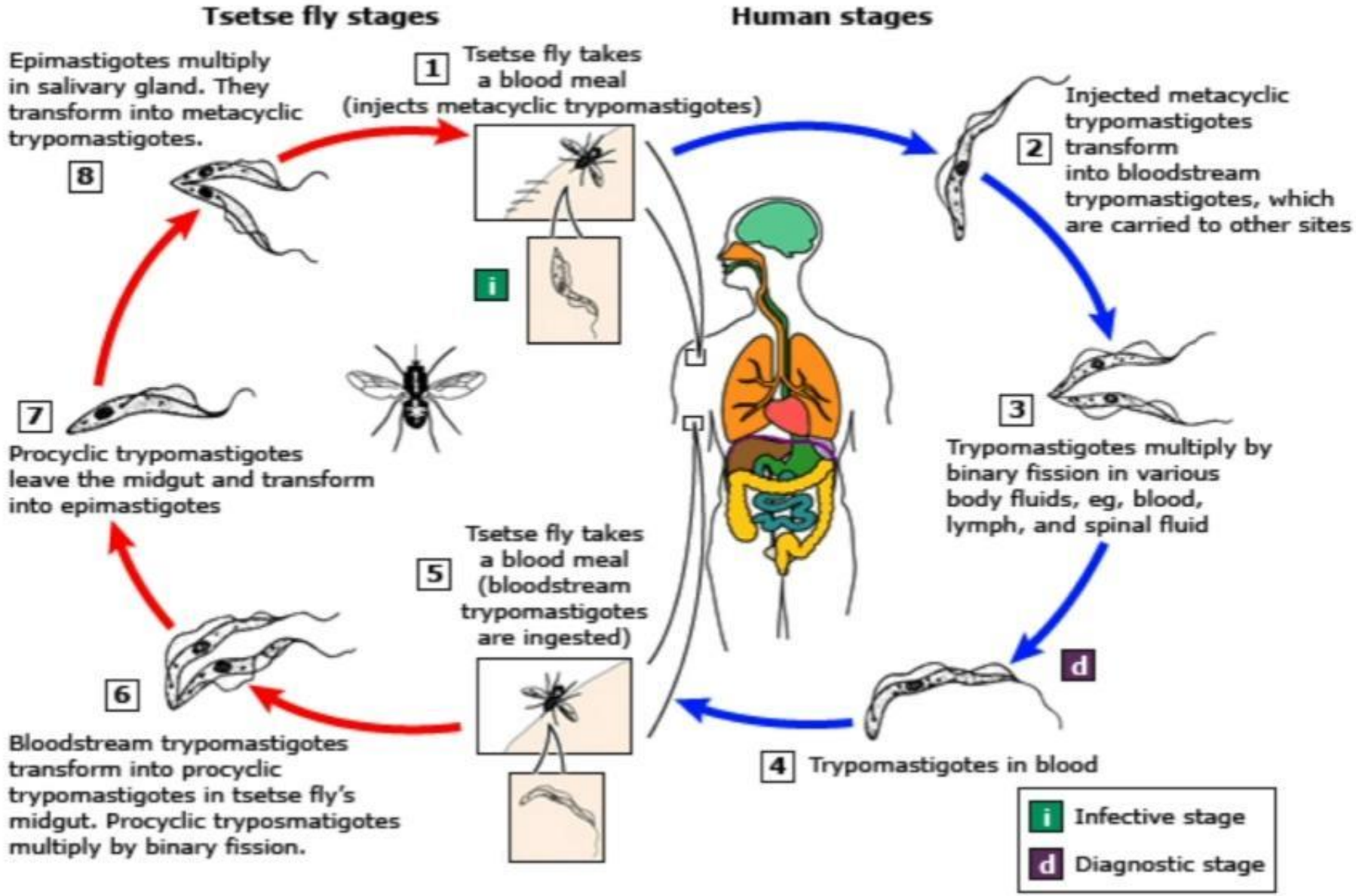
- Tsetse fly takes blood meal along with trypomastigotes
- In the midgut of the vector fly, these are converted into procyclic trypomastigotes and then these multiply by binary fission
- The procyclic trypomastigotes leave the midgut and transform into epimastigotes
- Epimatigotes multiply in salivary gland and gets converted into metacyclic trypomastigotes
- Tsetse fly takes blood meal and injects the metacyclic trypomastigotes into the human body

Life Cycle

Mammalian Cycle

- Starts after the introduction of metacyclic trypomastigotes
- These metacyclic trypomastigotes are transformed into trypomastigotes in bloodstream and are taken to other sites
- Trypomastigotes multiply by binary fission in various body fluids; blood, lymph and CSF
- In acute phase, trypomastigotes are circulating in blood
- In latent phase, trypomastigotes are undetectable

Life Cycle of Trypanosoma



Epidemiology

- African trypanosomiasis is restricted to recognized tsetse fly belts
- It causes a small number of cases but is more virulent
- Congenital infection occurs in hyperendemic areas

Pathogenesis

- After the introduction of metacyclic trypomastigotes, these multiply at the site of inoculation to cause variable induration and swelling (the primary lesion) which may progress to form a trypanosomal chancre
- The African forms multiply extracellularly as trypomastigotes in blood and lymphoid tissues
- They spread to bloodstream, lymph nodes and in terminal stages, to the CNS where they produce the typical sleeping sickness syndrome: lassitude, inability to eat, tissue wasting, unconsciousness, and death

Pathogenesis

- CNS involvement is characteristic feature of African Trypanosomiasis
- T b rhodesiense appears in CSF in about 1 month and T b gambiense in several months, but in small numbers
- T b gambiense infection is chronic and leads to progressive diffuse meningoencephalitis, with death usually in 1-2 years
- The fatal T b rhodesiense rapidly produces somnolence and comma only during the final weeks of the terminal infection

Pathogenesis

- The African trypanosomes of T b complex are remarkable in that they undergo antigenic variation through a series of genetically controlled glycoproteins, that coat the surface of the organism (variant surface glycoproteins, or VSGs)
- This process is due to genetically induced changes of the surface glycoprotein
- By producing different antigenic surface membranes, the parasite able to evade the host's antibody response

Pathogenesis

- Each population is reduced but is promptly replaced with another antigenic type before the preceding one is eliminated
- Each trypanosome is thought to possess about 1000 VSG genes, an example of mosaic gene formation

Clinical Findings

Characterized by:

1. Lymphadenopathy
2. Fever
3. Excessive sleepiness due to encephalopathy or encephalitis
4. Painful skin chancre hat appears about 5-15 days after the bite
5. Headaches
6. Muscular and joints pain
7. Rash or itchy skin
8. Weight loss

Treatment

- **Early-stage** of African Trypanosomiasis: Suramin or pentamidine
- **Late stage** of African Trypanosomiasis when CNS is involved: Melarosoprol
- **American** trypanosomiasis: Nifurtimox

Prevention

- Controlling movement of people in and out of fly belts
- Using insecticides
- Instituting fly control, principally with aerial insecticides and by altering habitats

References:

- Taxonomy of African Trypanosoma species.,msu.edu. Retrieved 2019-03-28.
- https://www.who.int/news-room/fact-sheets/detail/trypanosomiasis-human-african-(sleeping-sickness)

THANK YOU