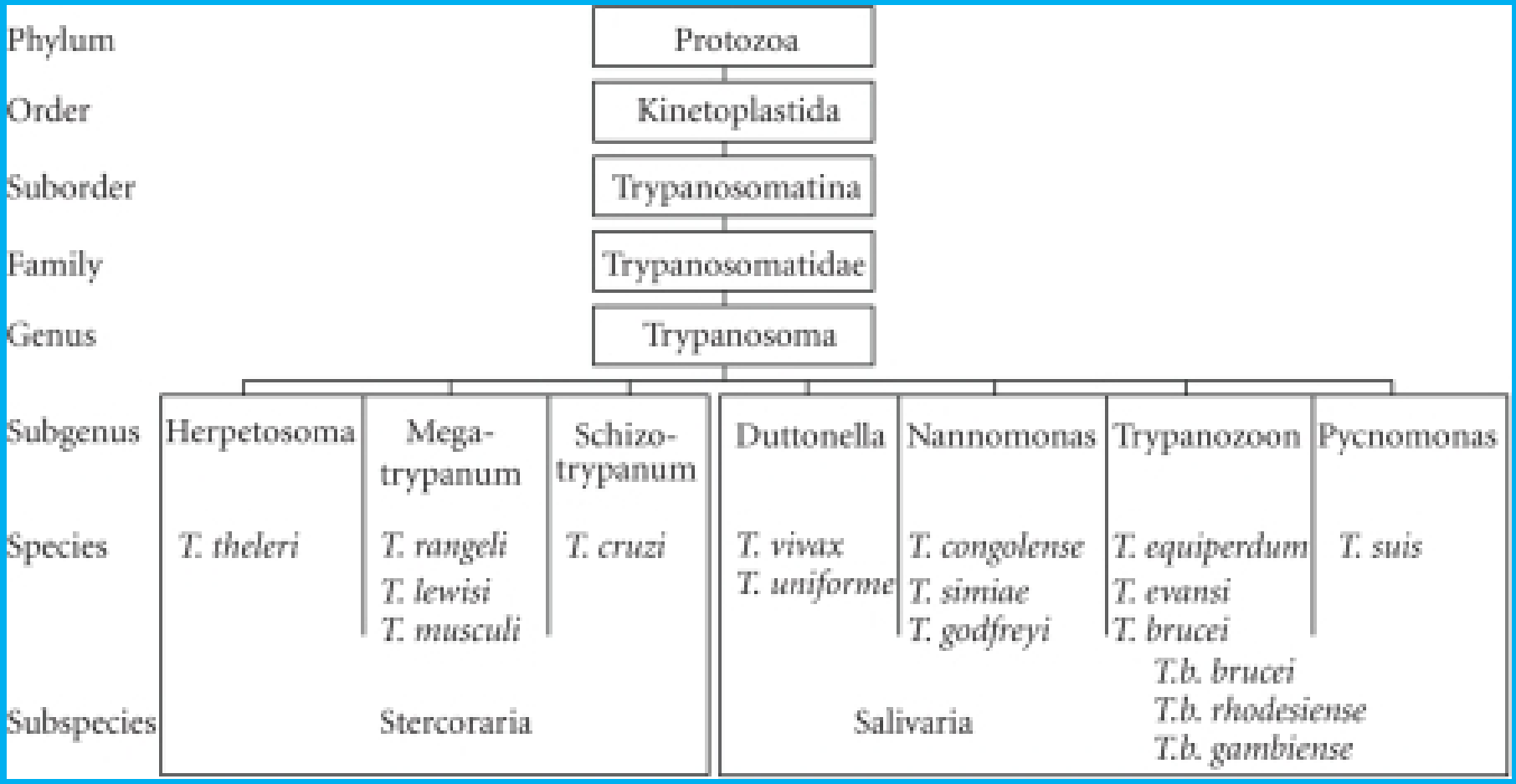
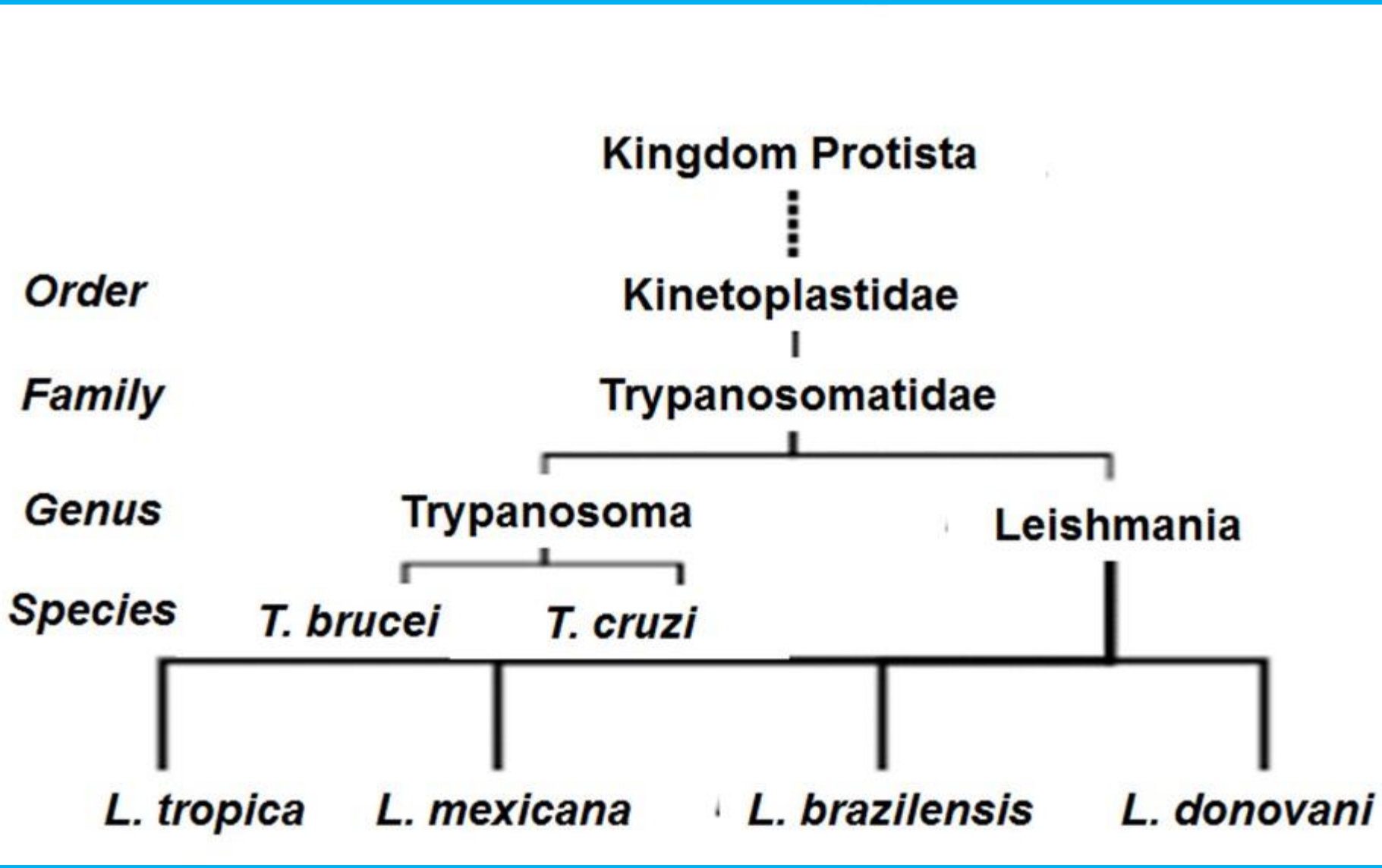
Trypanosma spp.




Kingdom Protista
Order
Kinetoplastidae
Family
Trypanosomatidae
Genus
Trypanosoma
Leishmania
Species
T. brucei
T. cruzi
L. tropica
L. mexicana
L. brazilensis
L. donovani

African trypanosomiasis

Causal Agents:

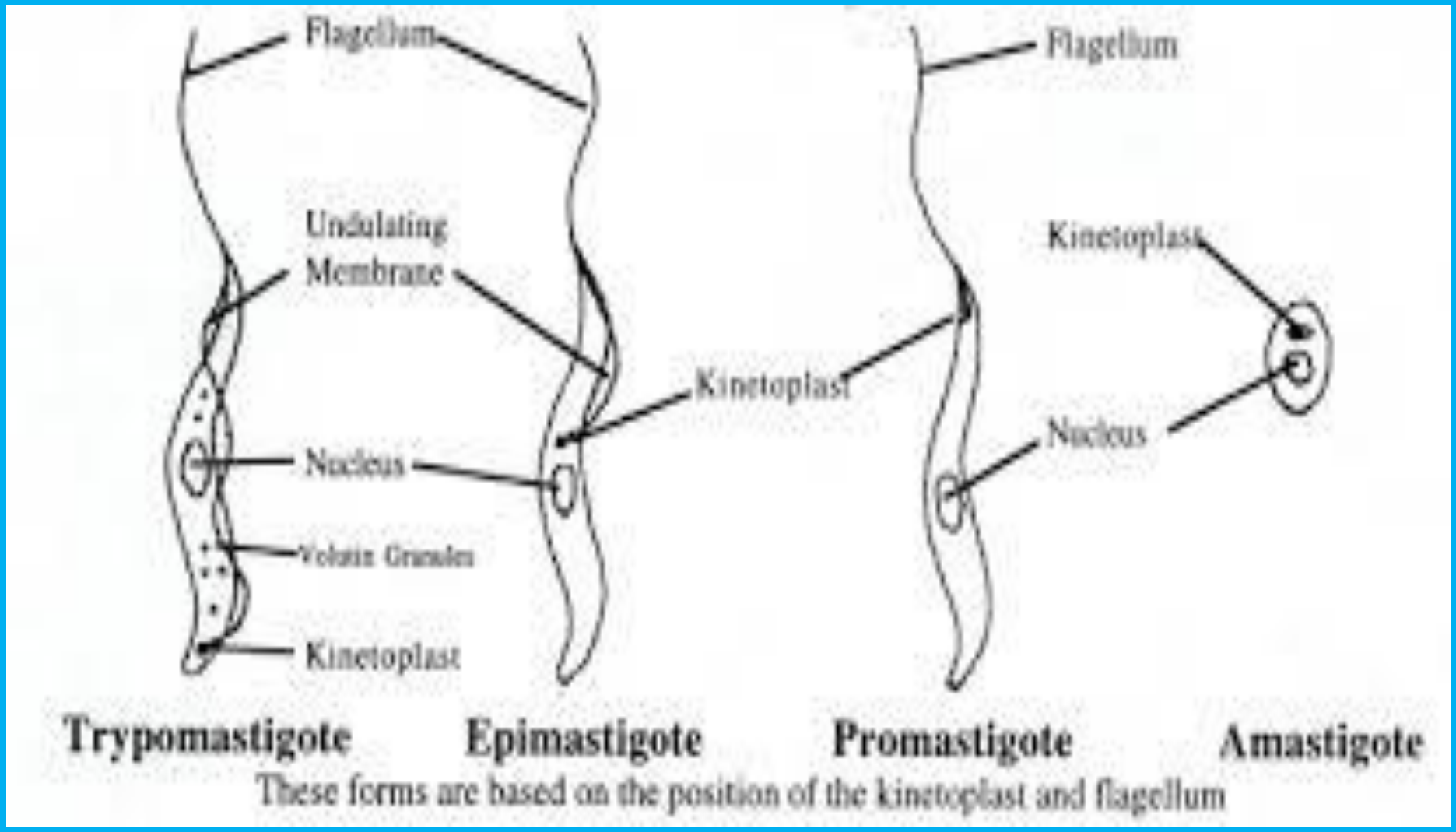
Protozoan hemoflagellates belonging to the complex *Trypanosoma brucei*. Two subspecies that are morphologically indistinguishable cause distinct disease patterns in humans: *T. b. gambiense* causes West African sleeping sickness and *T. b. rhodesiense* causes East African sleeping sickness.

Vector

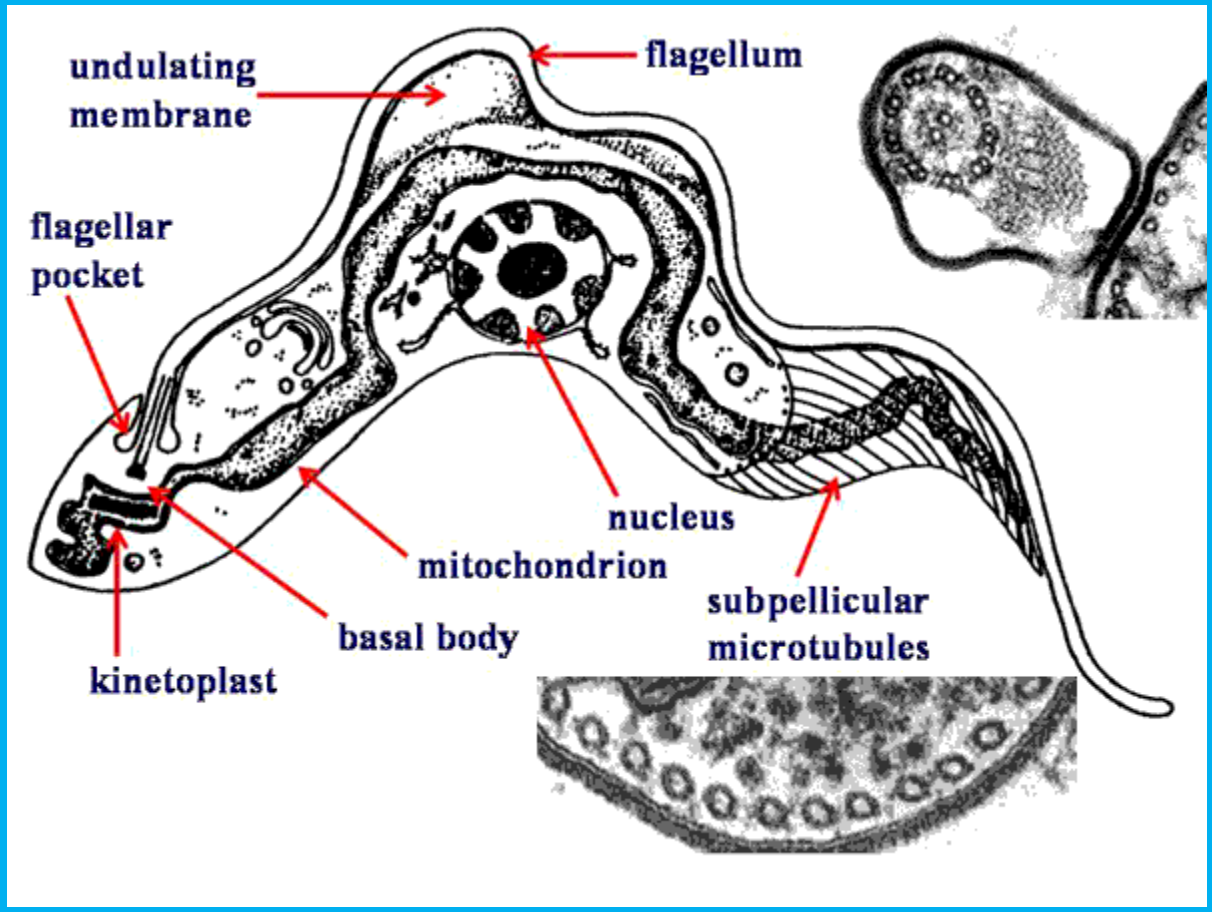
Trasmitted by Tse tse fly (*Glossina morsitans*)

A variety of different forms of trypanosomes, distinguished mainly by the position of the flagellum:

Amastigote	- reduced or absent
Promastigote	- anterior of nucleus
Epimastigote	- anterior of nucleus, connected by a short undulating membrane
Trypomastigote	- posterior of nucleus, connected by a long undulating membrane

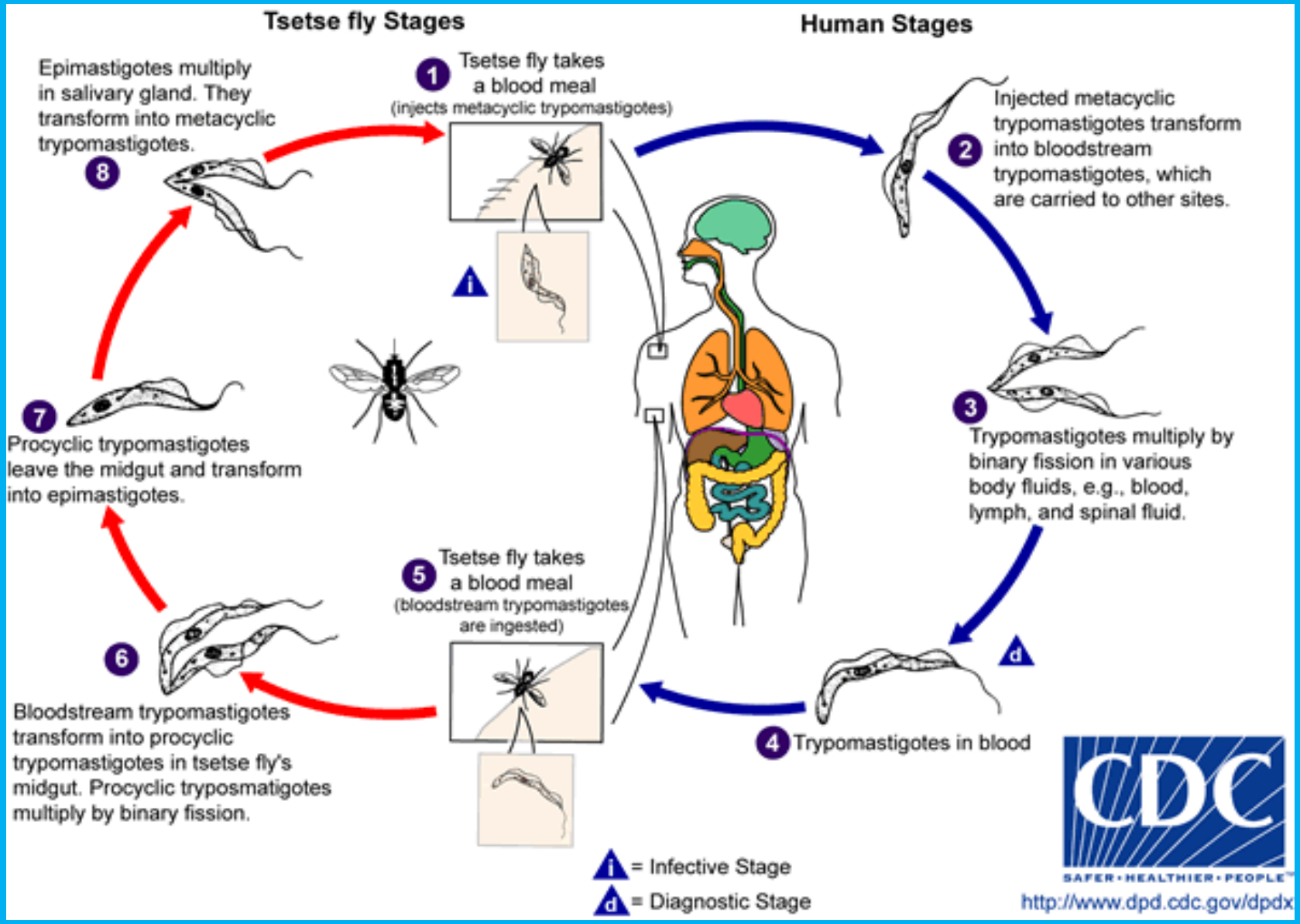


These forms are based on the position of the kinetoplast and flagellum



Trypanosoma brucei

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)
i
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.
d
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.
i = Infective Stage
d = Diagnostic Stage
CDC
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http://www.dpd.cdc.gov/dpdx

During a blood meal on the mammalian host, an infected tsetse fly (genus *Glossina*) injects metacyclic trypomastigotes into skin tissue. The parasites enter the lymphatic system and pass into the bloodstream (1) Inside the host, they transform into bloodstream trypomastigotes (2) are carried to other sites throughout the body, reach other blood fluids (e.g., lymph, spinal fluid), and continue the replication by binary fission (3) .

The tsetse fly becomes infected with bloodstream trypomastigotes when taking a blood meal on an infected mammalian host (4,5). In the fly's midgut, the parasites transform into procyclic trypomastigotes, multiply by binary fission (6) , leave the midgut, and transform into epimastigotes(7).

The epimastigotes reach the fly's salivary glands and continue multiplication 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 *T. b. rhodesiense*.

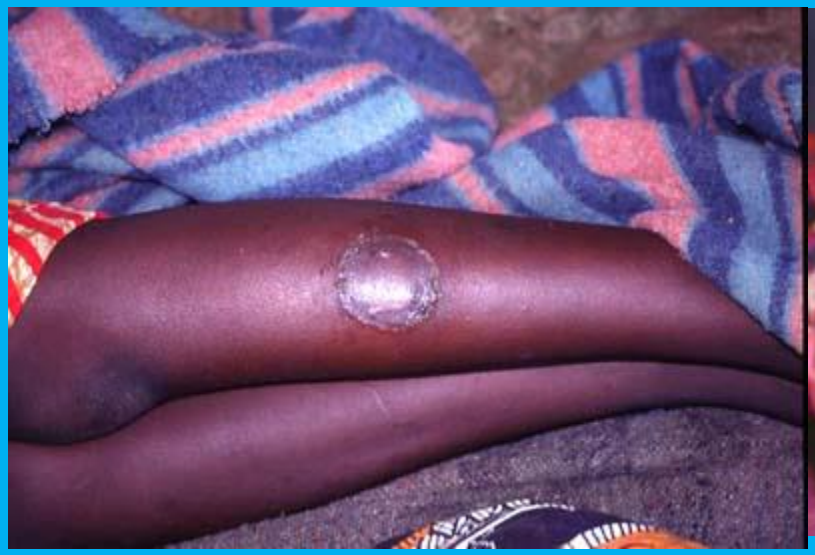
Geographic Distribution:

T. b. gambiense is found in foci in large areas of West Africa. The distribution of *T. b. rhodesiense* is much more limited, with the species found in East Africa.

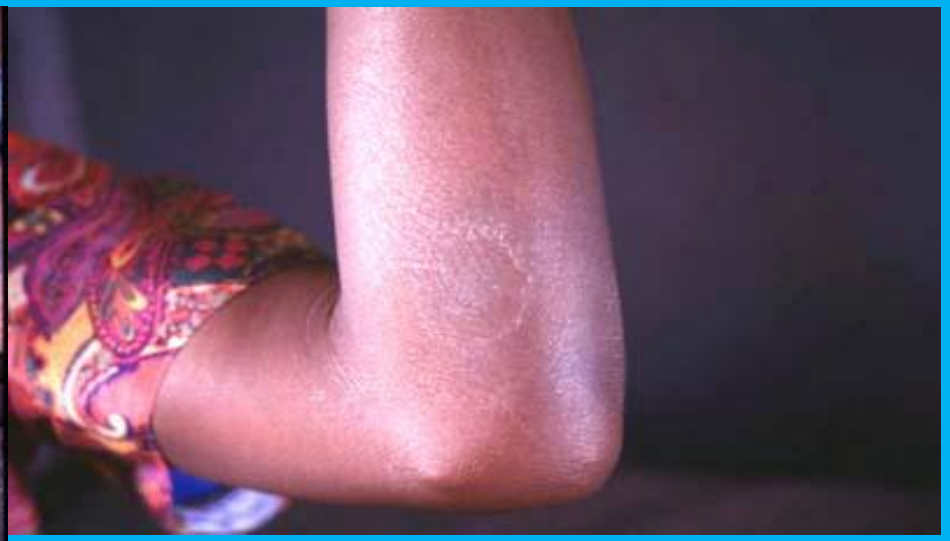
Clinical Features:

Infection occurs in 3 stages.

1- Bite reaction: A non-pustular, painful, itchy chancre (painless ulceration) (as shown in the Figures) forms 1-3 weeks after the bite and lasts 1-2 weeks. It leaves no scar.



The leg of girl who has sleeping sickness, showing the chancre at the site of the tsetse fly bite



The partially healed chancre on the arm of a female

This is followed by

2- Haemolymphatic stage: with symptoms that include fever, lymphadenopathy, and pruritus (itch).

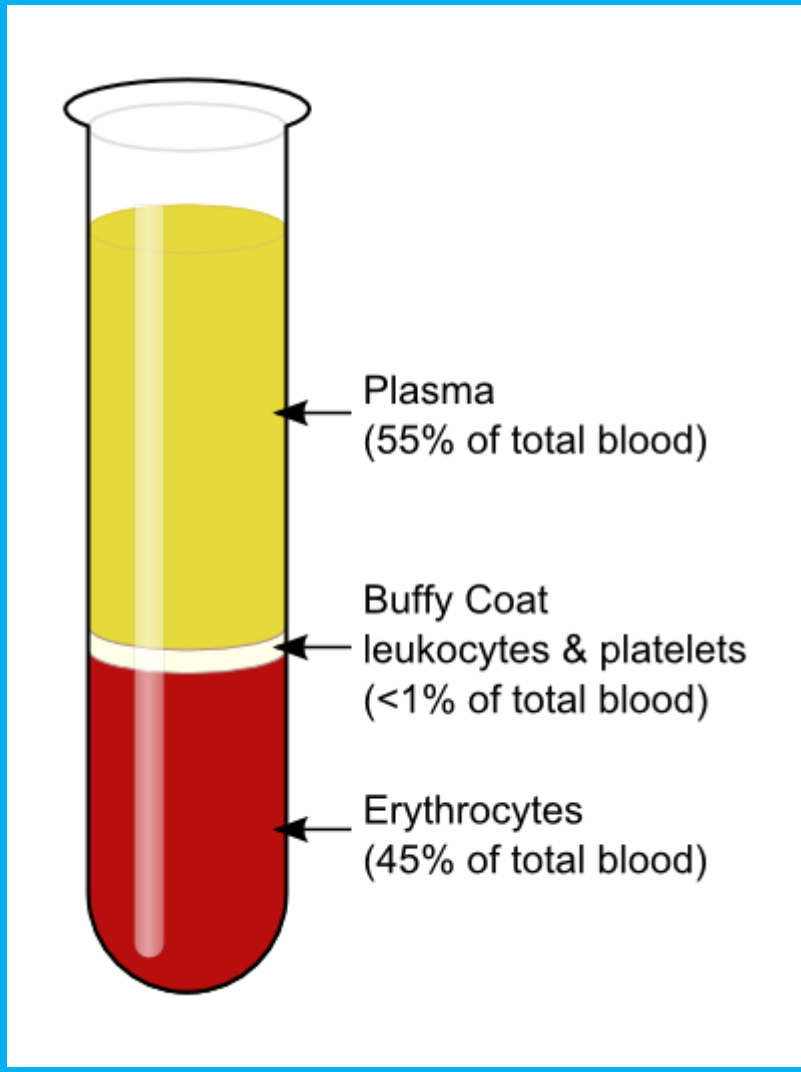
3- Meningoencephalitic stage: invasion of the central nervous system can cause headaches, somnolence (strong desire for sleep), abnormal behavior, and lead to loss of consciousness and coma.

Laboratory Diagnosis:

The diagnosis rests upon demonstrating trypanosomes by microscopic examination of chancre fluid, lymph node aspirates, blood, bone marrow, or, in the late stages of infection, cerebrospinal fluid. Concentration techniques can be used prior to microscopic examination.

For blood samples, these include centrifugation followed by examination of the buffy coat. For other samples such as spinal fluid, concentration techniques include centrifugation followed by examination of the sediment.

The **buffy coat** is the fraction of an anticoagulated blood sample after centrifugation that contains most of the white blood cells and platelets.



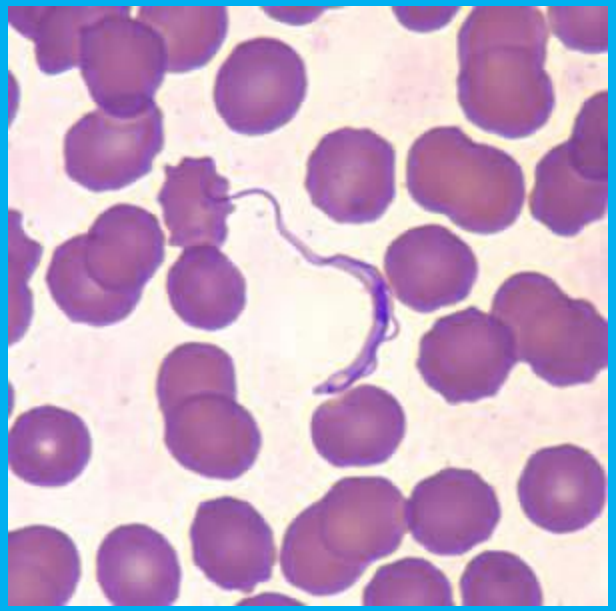
Blood components after centrifugation

Antibody detection has sensitivity and specificity that are too variable for clinical decisions. In addition, in infections with *T. b. rhodesiense*, seroconversion (antibody arise) occurs after the onset of clinical symptoms and thus is of limited use.

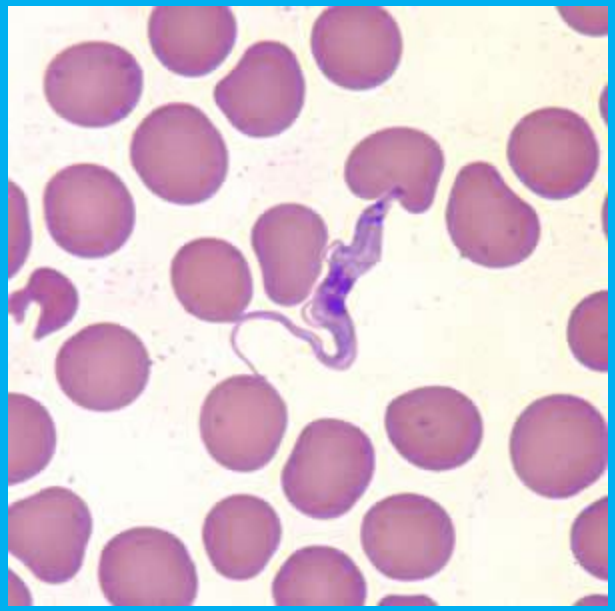
Microscopy:

T. b. gambiense and *T. b. rhodesiense*, are indistinguishable morphologically. Trypomastigotes are the only stage found in patients. Trypanosomes range in length from 14 to 33 µm

C



D



C: *Trypanosoma brucei* sp. in a thick blood smears stained with Wright-Giemsa.
D: *Trypanosoma brucei* sp. in a thin blood smear stained with Wright-Giemsa

Treatment and Control

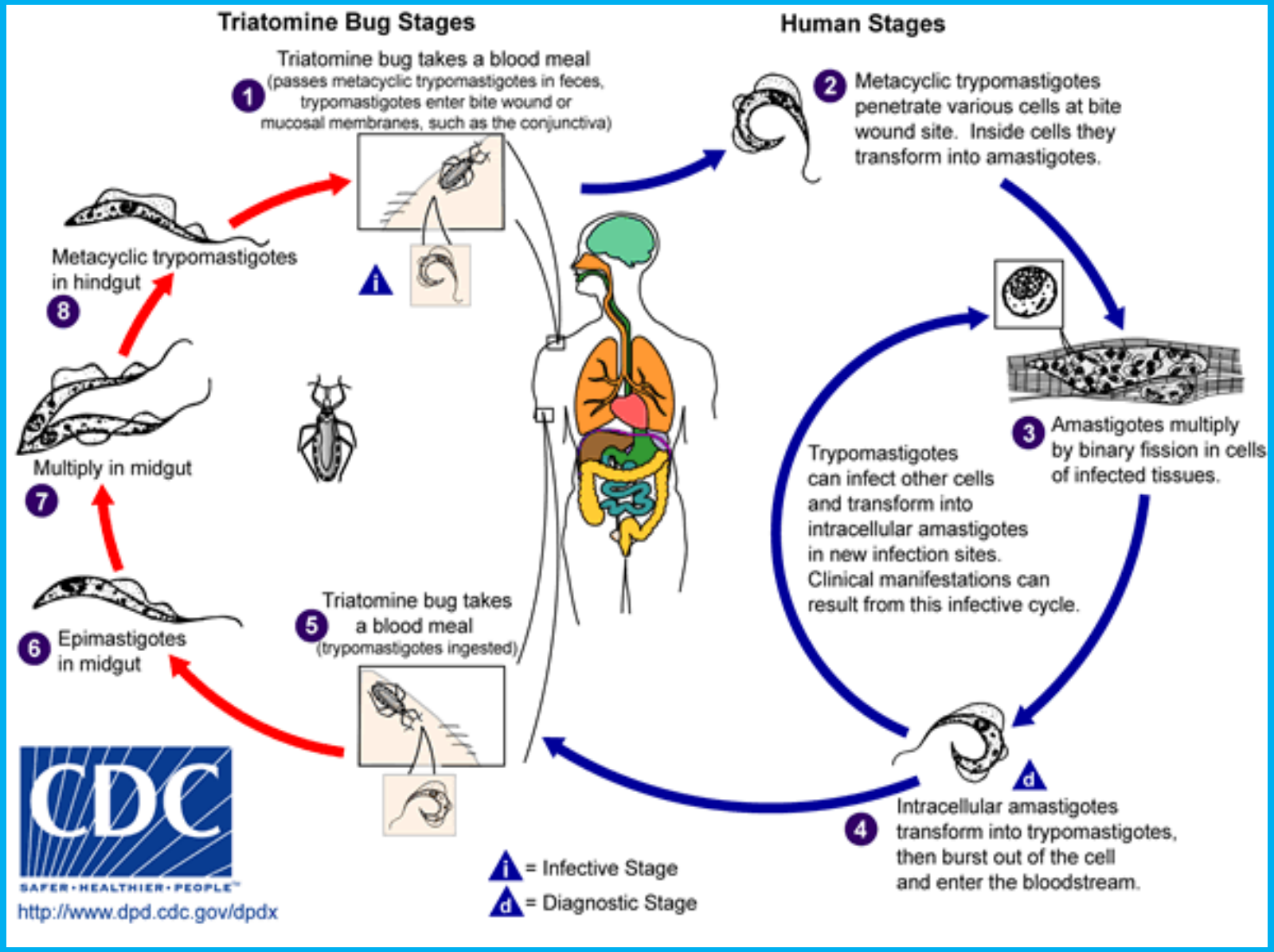
The blood stage of African trypanosomiasis can be treated with reasonable success with Pentamidine isethionate or Suramin. These drugs have been reported also to be effective in prophylaxis although they may mask early infection and thus increase the risk of CNS disease.

American trypanosomiasis

Causal Agent:

The protozoan parasite, *Trypanosoma cruzi*, causes Chagas disease, a zoonotic disease that can be transmitted to humans by blood-sucking triatomine bugs.

Life Cycle:


Triatomine Bug Stages
Human Stages
Triatomine bug takes a blood meal
(passes metacyclic trypomastigotes in feces, trypomastigotes enter bite wound or mucosal membranes, such as the conjunctiva)
1
2
Metacyclic trypomastigotes penetrate various cells at bite wound site. Inside cells they transform into amastigotes.
Metacyclic trypomastigotes in hindgut
8
Multiply in midgut
7
3
Amastigotes multiply by binary fission in cells of infected tissues.
Trypomastigotes can infect other cells and transform into intracellular amastigotes in new infection sites. Clinical manifestations can result from this infective cycle.
Epimastigotes in midgut
6
Triatomine bug takes a blood meal
(trypomastigotes ingested)
5
4
Intracellular amastigotes transform into trypomastigotes, then burst out of the cell and enter the bloodstream.
i = Infective Stage
d = Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE™
http://www.dpd.cdc.gov/dpdx

An infected triatomine 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 .

Inside the host, the trypomastigotes invade cells near the site of inoculation, where they differentiate into intracellular amastigotes .

The amastigotes multiply by binary fission and differentiate into trypomastigotes, and then are released into the circulation as bloodstream trypomastigotes. Trypomastigotes infect cells from a variety of tissues and transform into intracellular amastigotes in new infection sites.

Clinical manifestations can result from this infective cycle. The bloodstream trypomastigotes do not replicate (different from the African trypanosomes). Replication resumes only when the parasites enter another cell or are ingested by another vector. The “kissing or assassin” bug becomes infected by feeding on human or animal blood that contains circulating parasites .

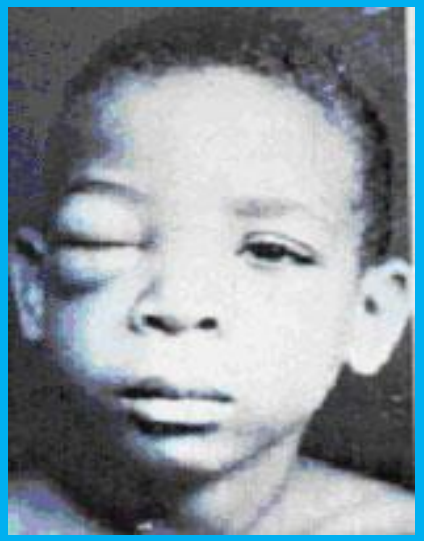
The ingested trypomastigotes transform into epimastigotes in the vector's midgut . The parasites multiply in the midgut and differentiate into infective metacyclic trypomastigotes in the hindgut . *Trypanosoma cruzi* can also be transmitted through blood transfusions, organ transplantation, transplacentally, and in laboratory accidents.

Geographic Distribution:

Mostly in poor, rural areas of Mexico, Central America, and South America. Chronic Chagas disease is a major health problem in many Latin American countries.

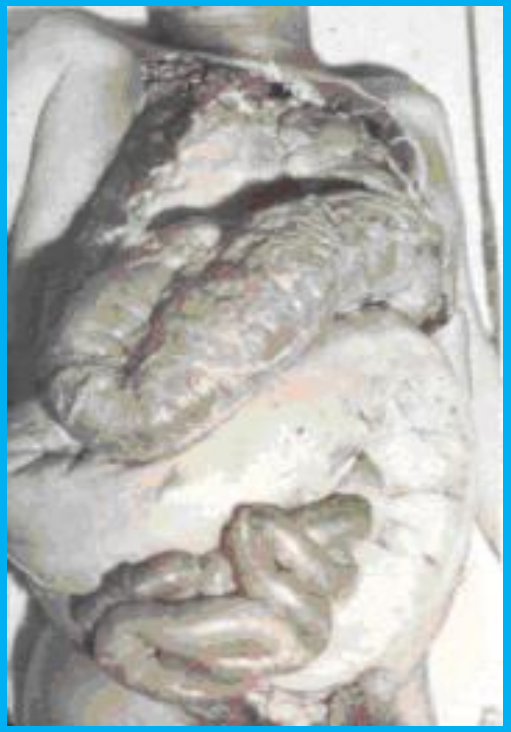
Clinical Features:

The acute phase is usually asymptomatic, but can present with manifestations that include fever, anorexia (decreased appetite), lymphadenopathy, mild hepatosplenomegaly, and myocarditis. Romana's sign (unilateral and periocular swelling) may appear as a result of conjunctival contamination with the vector's feces. A nodular lesion, usually called chagoma, can appear at the site of inoculation.



Ramana's sign: unilateral conjunctivitis and orbital swelling

The chronic phase include manifestations of cardiomyopathy , pathologies of the digestive tract such as megaesophagus and megacolon; and weight loss. Chronic Chagas disease can be fatal.



Megacolon in Chaga's disease

Laboratory Diagnosis:

Demonstration of the causal agent is the diagnostic procedure in acute Chagas disease. It almost always yields positive results, and can be achieved by:

Microscopic examination of blood

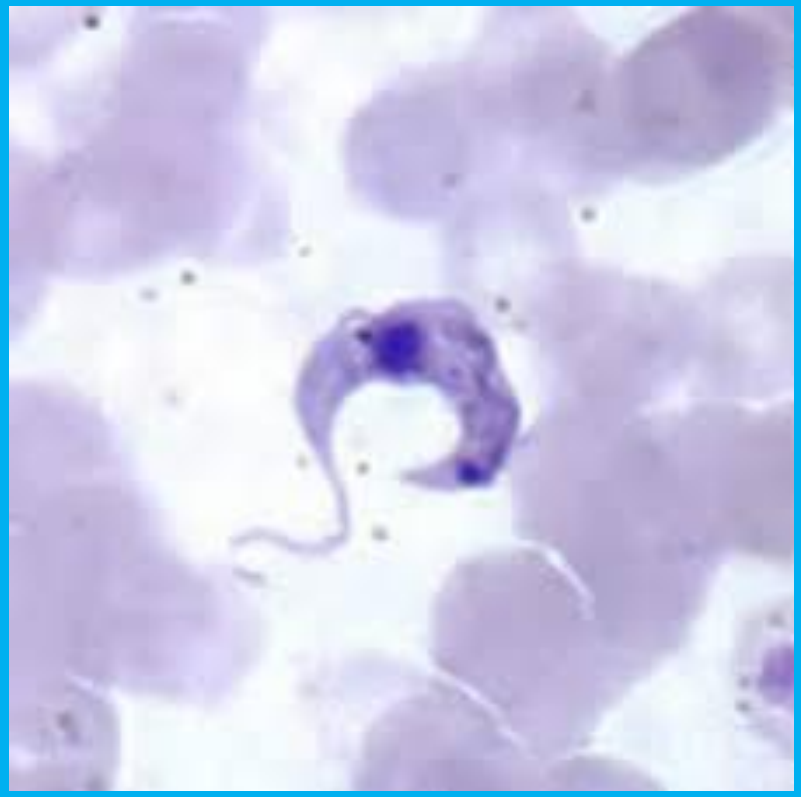
.

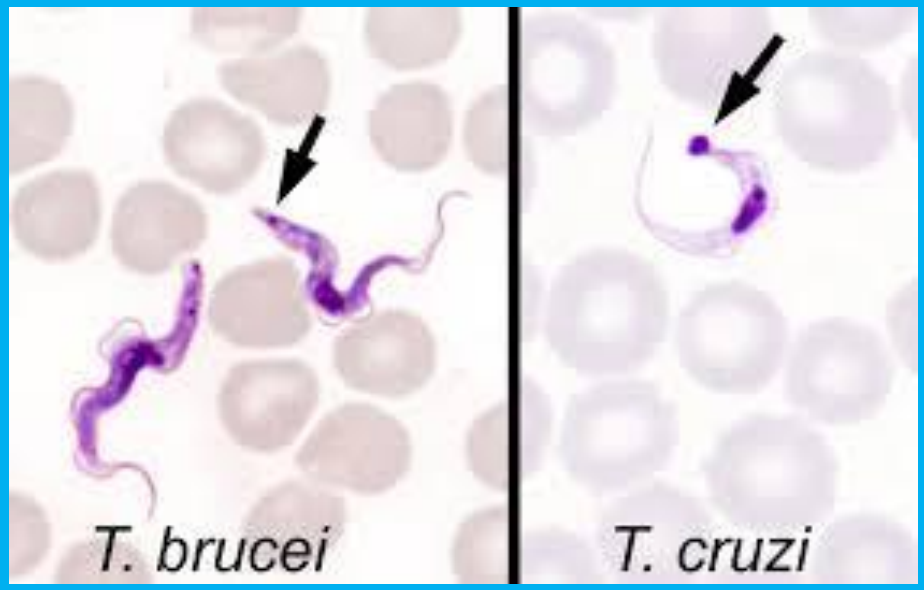
Clinical diagnosis is usually easy in endemic areas. Cardiac dilation, megacolon and megaesophagus in individuals from endemic areas indicate present or former infection.

Definitive diagnosis requires the demonstration of trypanosomes

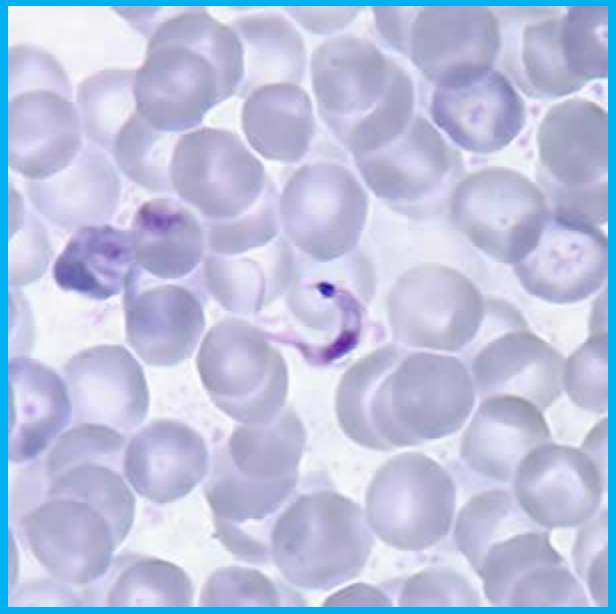
-Detection of Antibodies : Using serological tests

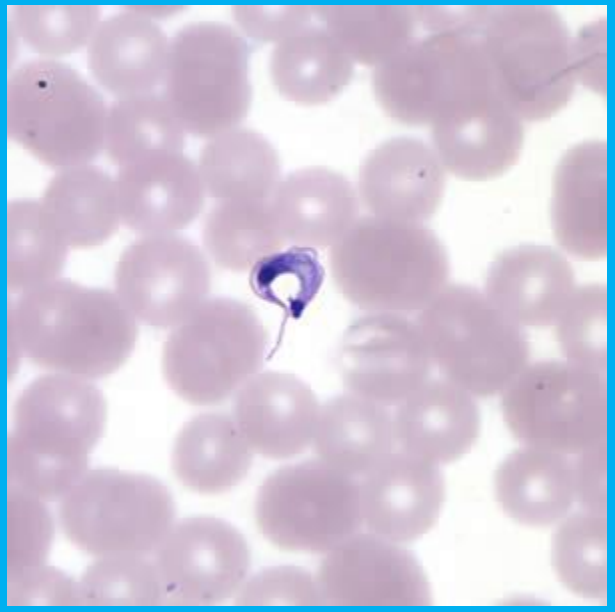
-PCR test





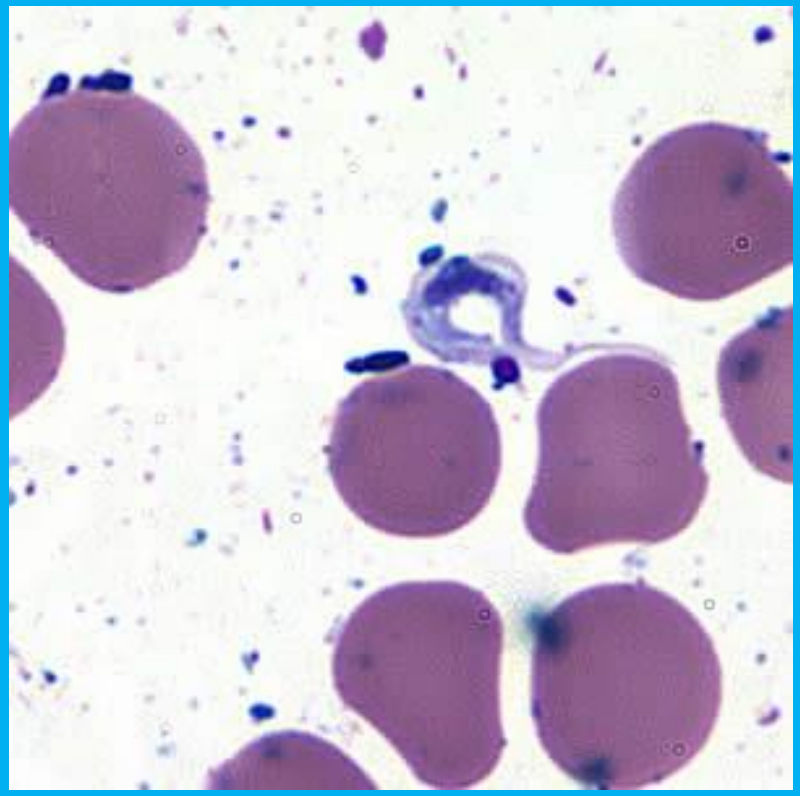
T. cruzi trypomastigotes in a thick blood smear stained with Giemsa.



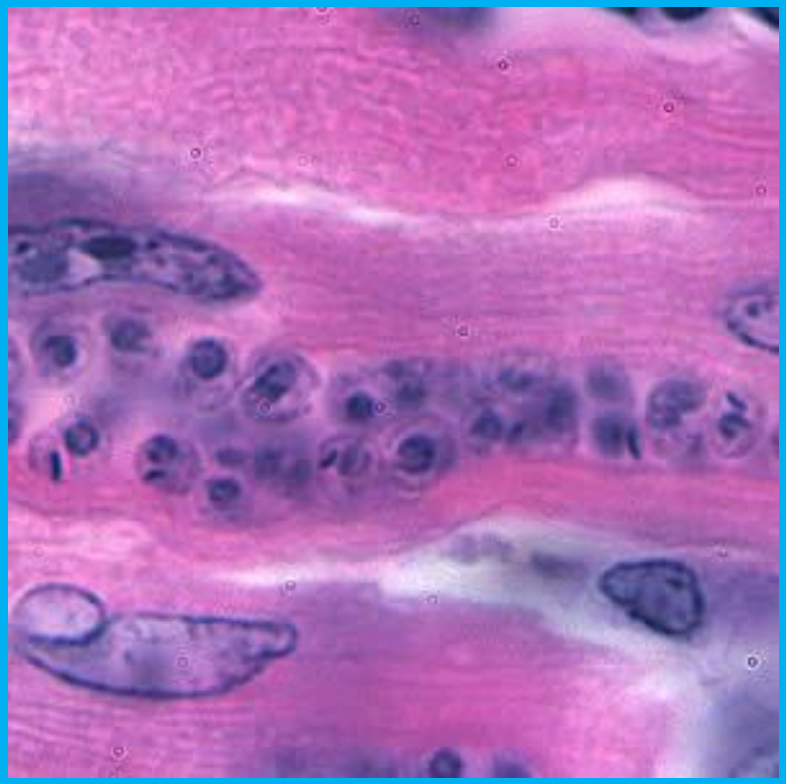


T. cruzi trypomastigotes in thin blood smears stained with Giemsa. Note the typical C-shape of the trypomastigote that characterizes *T. cruzi* in fixed blood smears

E



F



E: *T. cruzi* trypomastigote in cerebrospinal fluid (CSF) stained with Giemsa.
F: *T. cruzi* amastigotes in heart tissue. The section is stained with hematoxylin and eosin (H & E).

Treatment:

Medication for Chagas disease is usually effective when given during the acute phase of infection and may be indicated for patients in the chronic phase as well. The drugs of choice are benznidazole or nifurtimox

Tsetse → (Inject) Metacyclic trypo → (blood) Blood trypo → (multiply) tsetse → Procy trypo,mid gut

Leve mid gut trans into epimast → Salivary glands trans into metacyclic trypo

African Trypanosome, life cycle

Triatomine bug → (feces) Metacyclic trypo → (Wound or m mem) Amastigote intra cellular → (multiply) trypo

Enter blood → trypo → (bug) Epimast, mid gut → Metacyclic trpo, hind gut

Infect other cells → Amastigote , intracellular

American trypanosome, life cycle