L 2:- Parasitology
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2. Trichomonas vaginalis, Trichomonas hominies and Trichomonas tenax

Trichomonas vaginalis

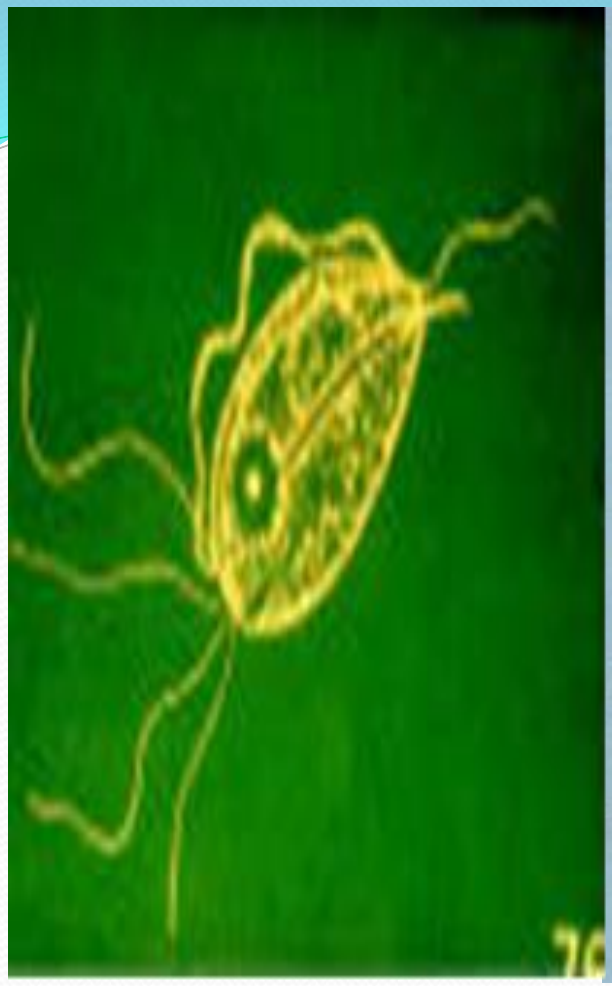
1. Pathogenic to human &causes vaginitis (trichomoniasis).
2. **troph**. Is round or pear like in shape, contains 4-6 flagella, all originating from anterior end & only one extend posteriorly. The motility is rapid & jerky.

The **undulating membrane extending half of the body length**. Prominent axostyle that often curves around the nucleus & granules may be seen along in the axostyle. **The nucleus is oval shape & only one. No cyst is seen**.

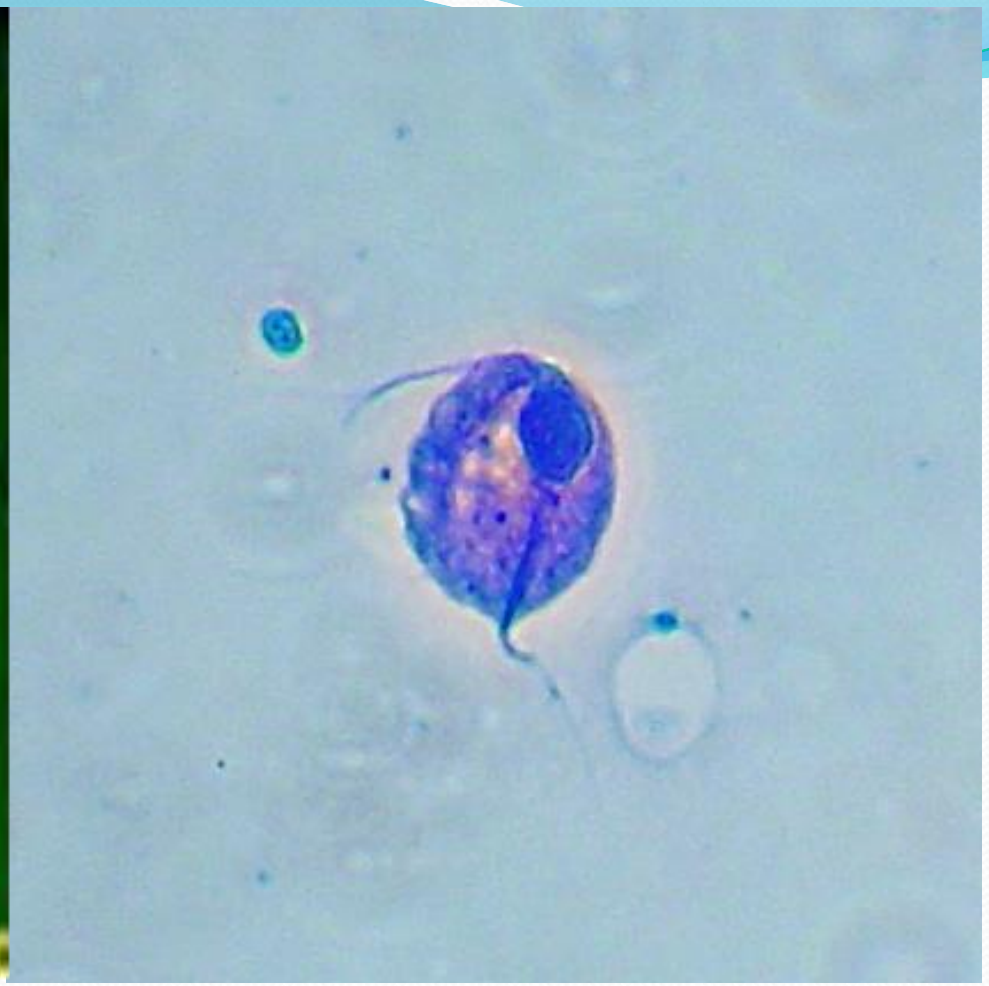
Clinical symptoms

1. *T. vaginalis* reside on the mucosal surface of the vagina in infected women.
2. The most common sites in male is the prostate gland region & the epithelium of the urethra.
3. **Vaginitis** may be found in infected women. It is characterized by foul smelling, greenish-yellow, vaginal discharge, burning & itching may also present. Red lesions may be seen in vaginal mucosa. Urethral involvement, dysuria & increased frequency of urination are among the most commonly symptoms. Cystitis is rare occur.
4. The main mechanism of *T. vaginalis* pathogenicity is cell to cell adherence & hemolysis and secreting soluble proteinases in both males and females human host

- is a urogenital flagellate protozoan parasite.This parasite has medical important because infected women during pregnancy are predisposed to premature due to the rupture of the placental membranes, and low-birth-weight infants. **Transmission:** Infection is transmitted by sexual intercourse



Trichomonas tenax



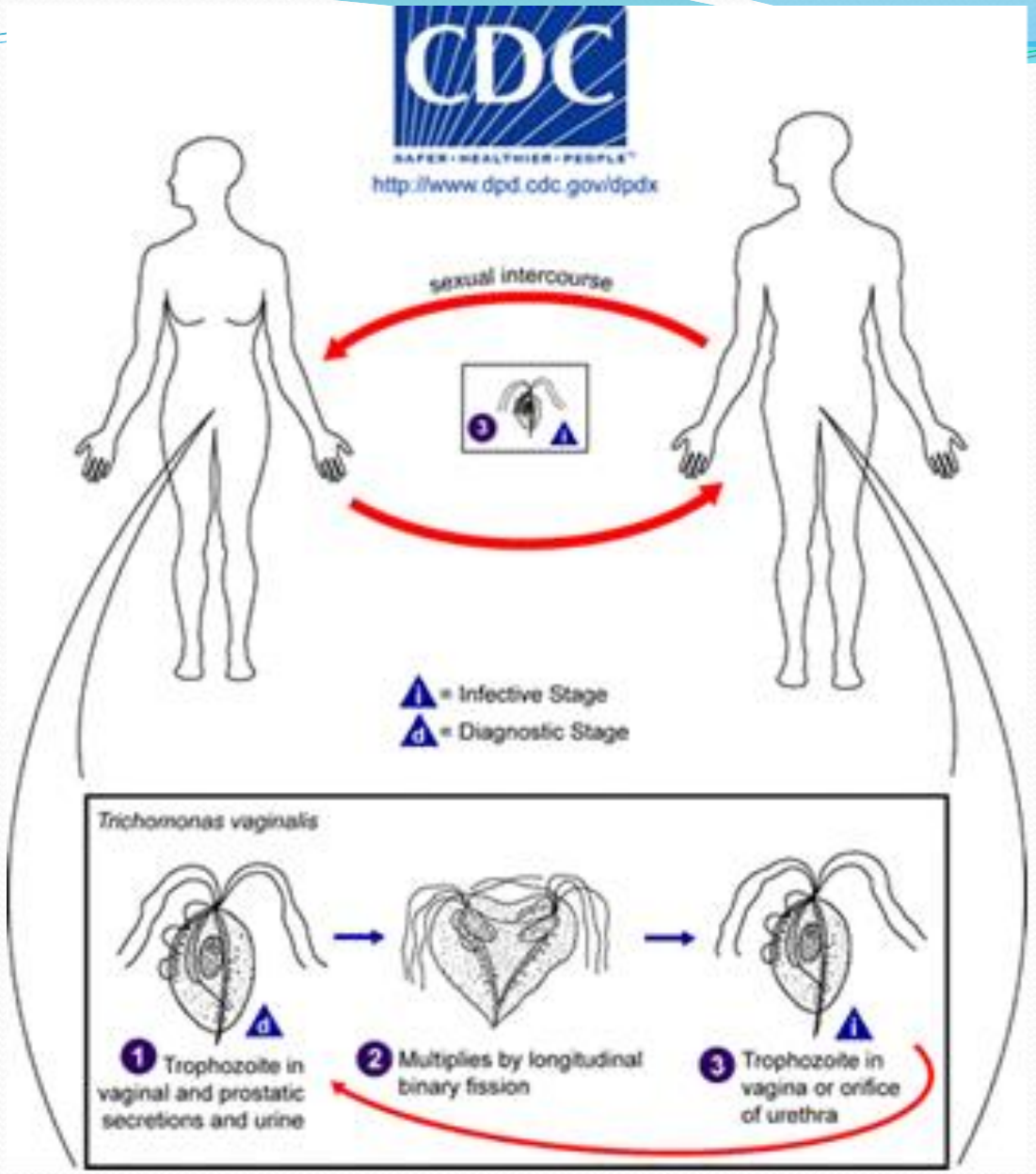
Trichomonas vaginalis

- **Life cycle**

Trichomonas vaginalis trophozoites reside on the mucosal surface of the vaginafeeding on bacteria and leucocytes in infected women. The growing trophozoites multiply by longitudinal binary fission and feed on local bacteria and leukocytes. The *Trichomonas vaginalis* trophozoites thrive in a slightly alkaline or slightly acid PH environment, such as that commonly seen in an unhealthy vagina. The most common infection site of *T. vaginalis* in males is the prostate gland region and the epithelium of the urethra. The detailed life cycle in the male host is unknown.

T.vaginalis is an obligate parasite

Infective and diagnostic stage is trophozoites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sexual intercourse
= Infective Stage
= Diagnostic Stage
Trichomonas vaginalis
1 Trophozoite in vaginal and prostatic secretions and urine
2 Multiplies by longitudinal binary fission
3 Trophozoite in vagina or orifice of urethra

Control and Prevention

- **control of Trichomonas vaginalis** = Condom use remains the best and most reliable protection against STIs. However, due to religious or cultural reasons, condom use may be limited, particularly in some developing countries. Concurrent treatment of sexual partners is recommended to prevent reinfection. However, systemic administration of chemotherapeutics to prevent infection results in increased incidences of nitroimidazole-refractory strains

- Treatment of **Trichomonas vaginalis** = Metronidazole

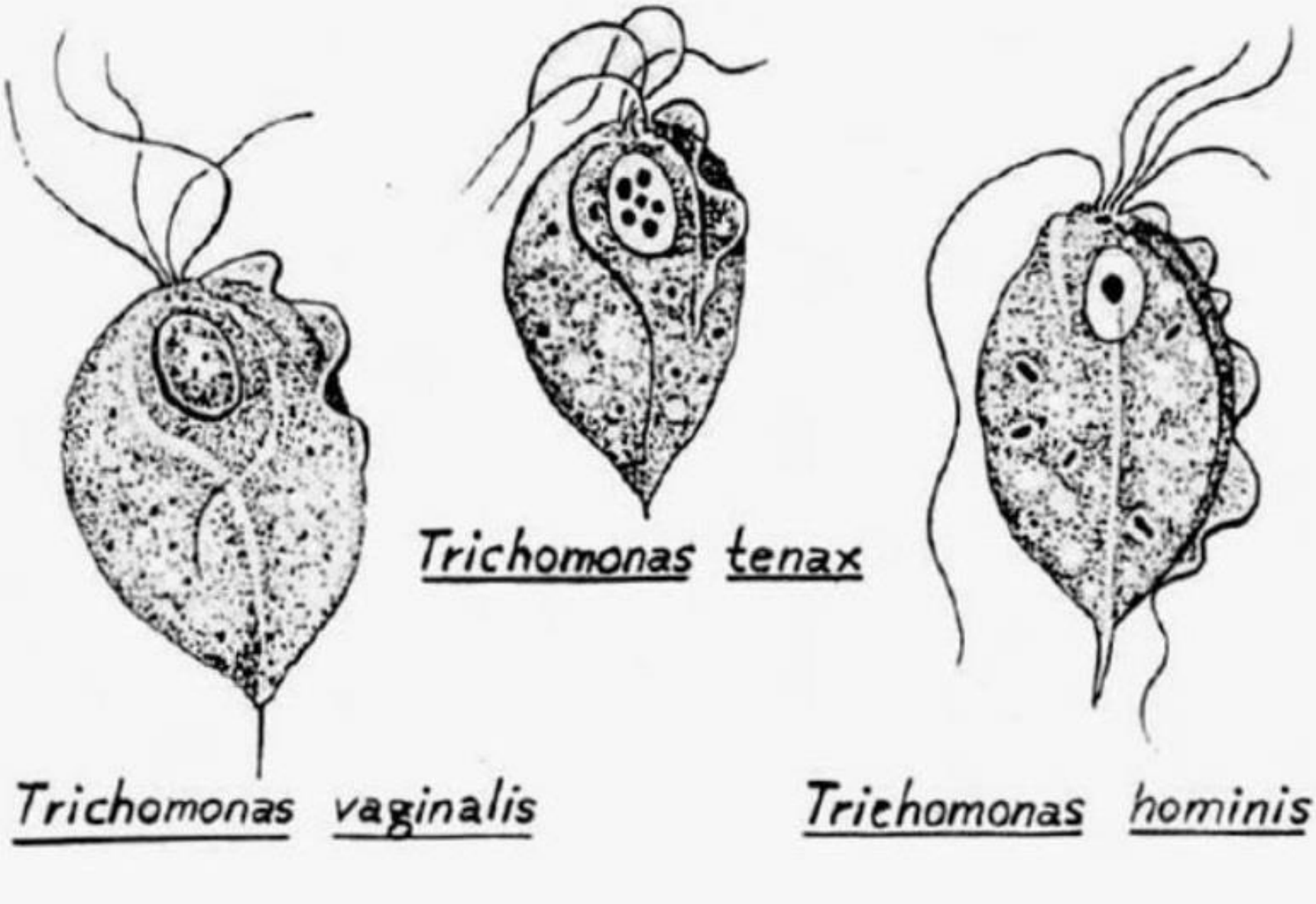
3.Trichomonas hominis

It is pyriform, considered a nonpathogenic inhabitant of the large intestine. Trophozoites of this may be observed in fresh smears of both normal and diarrheic fecal samples.

It inhabits the caecum of man and several other primate species and feeds on enteric bacteria. It does not invade the intestinal mucosa. Though it has occasionally been found in the diarrhoeic stools, its pathogenicity is yet to be established. In freshly passed specimens, particularly in unformed stools, the motility may be visible. In wet preparation, look for the flagellar movement, Undulating membrane which extends the entire length of the body and projects from the body like a free flagellum

4.Trichomonas tenax

Trophzoite: Oval to pear in Shape. Have one nuclei, vesicular filled with chromatin granules. Have five flagella, all originating anteriorly, four extends anteriorly, one extends posteriorly. **Undulating membrane extending 2/3** of body length. Thick axostyle and Small anterior cytosome opposite undulating


Trichomonas tenax
Trichomonas vaginalis
Trichomonas hominis

- **Life cycle**
- *Trichomonas tenax* trophozoites survive in the body as mouth scavengers that feed primarily on local microorganisms. Located in the tartar between the teeth, tonsillar crypts pyorrheal pockets, and gingival margin around the gums, *T. tenax* trophozoites multiply by longitudinal binary fission. These trophozoites are unable to survive the digestive process.

- **Clinical symptoms**
- The typical *Trichomonas tenax* infection does not produce any notable symptoms. On a rare occasion, *T. tenax* has been known to invade the respiratory tract, but this appears to have mainly occurred in patients with underlying thoracic or lung abscesses of pleural exudates.

5.Chilomastix mesnili

is a common flagellate living as a harmless commensal in the caecum and colon of humans. It has well-defined trophozoite and cystic stages.

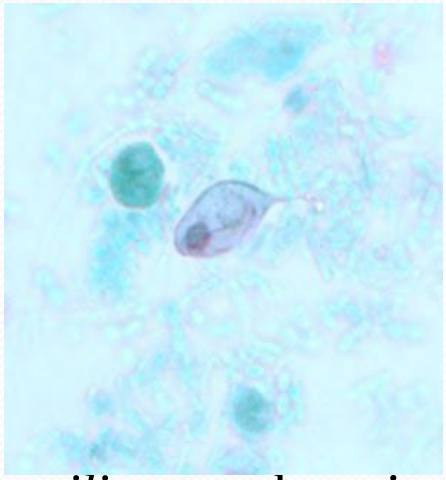
Transmission: ingestion of cysts in contaminated water and food.

Clinical Presentation: Diarrhea

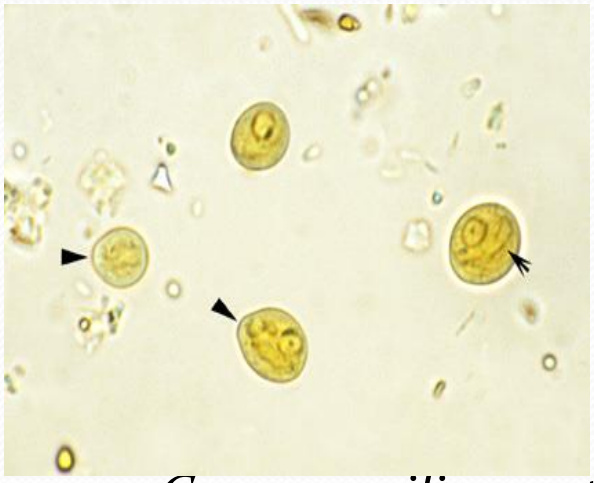
Morphology: The pear –shaped trophozoites has three flagella and a spiral groove . A slit-like cytosome (mouth) enclosing a fourth flagellum , has one nucleus . The cyst has single nucleus. Trophozoites reproduces by binary fission

The diagnosis of C. mesnili

C. mesnili in the faecal of trophozoites and cysts of can be made by detection smear



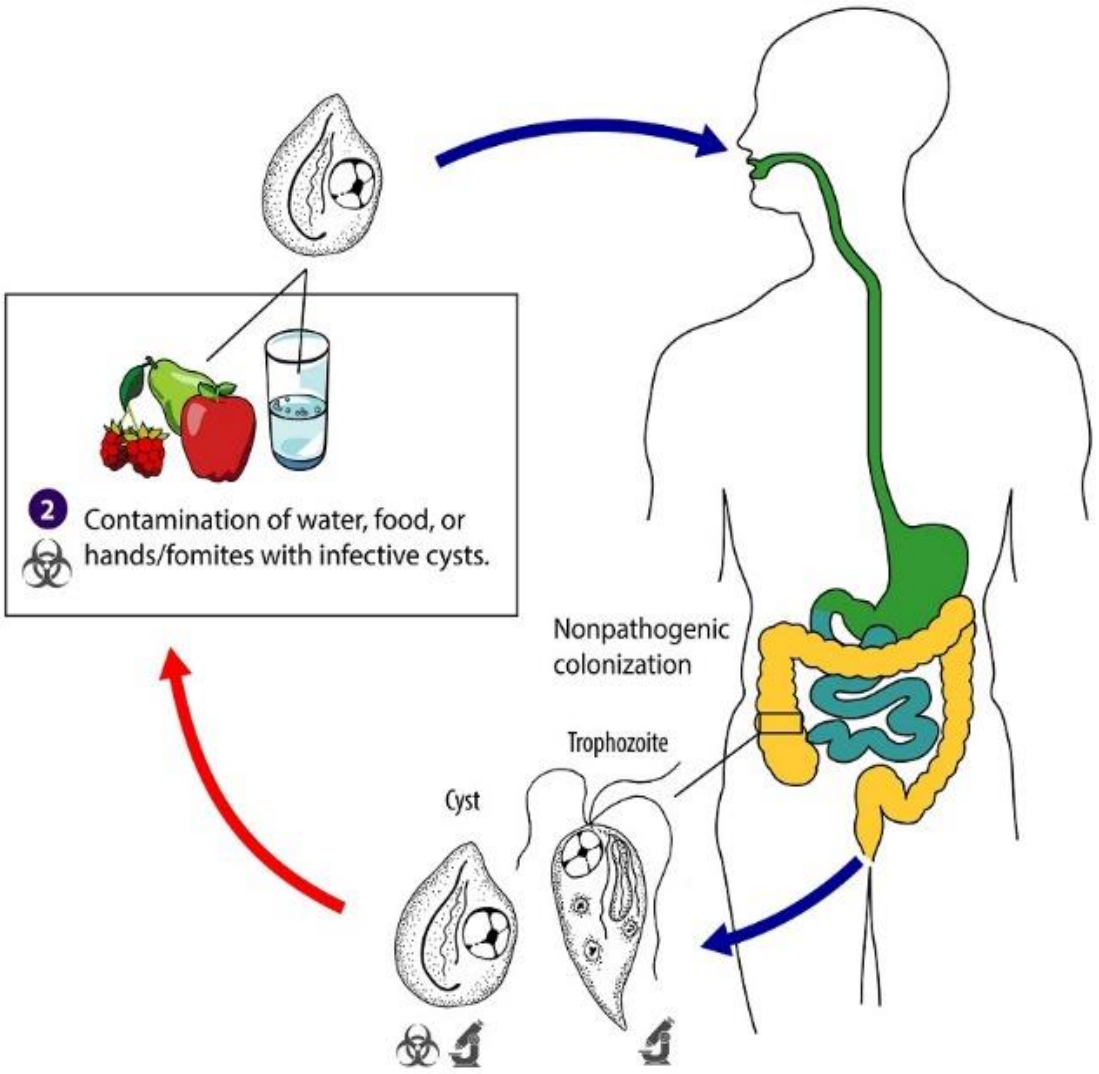
C . mesenili trophozoite



C . mesenili cyst

DPDx

Chilomastix mesnili



CDC

Class Haemoflagellates; have two genera ***Leishmania & Trypanosoma***

Common features of these parasites are:

1.All members of the family (Trypanosomatidea) have similar life cycles. the live cycle completed between two host: vertebrate host (terminal or definitive host like human) and arthropod host (intermediated host like the fly) in many stages with different shapes

Leishmania = mediated host= Sand fly

Trypanosoma**=** mediated host= Tse-Tse fly

2. They live in the blood, tissues of skin and endothelial layer of organs in the host, and in the gut of the insect vector.

3. Multiplication in both the vertebrate and invertebrate host is by binary fission. No sexual cycle is known.

4. Heamoflagellate has a nucleus, kinetoplast and a single flagellum.

5. Haemoflagellates exist in two or more of four morphological stages, which depend on: the shape of the body, presence of flagellate or absent, the shape and locate of motile generator and the presence of waved (undulating) membrane or absent.

Leishmania= have **Amastigotes, Promastigote**

Trypanosoma=have **Amastigotes, Promastigote, Epimastigotes, Trypomastigotes**

Morphological stages of heamoflagellates

1- **Amastigotes (Leishmanial stage): The roundish to oval, have kinetoplast (motile generator of flagella) and the large single nucleus is typically located in the center, sometimes present more toward the edge of the organism. Amastigote is a stage that does not have a visible external flagella. The form lives in the human macrophages**

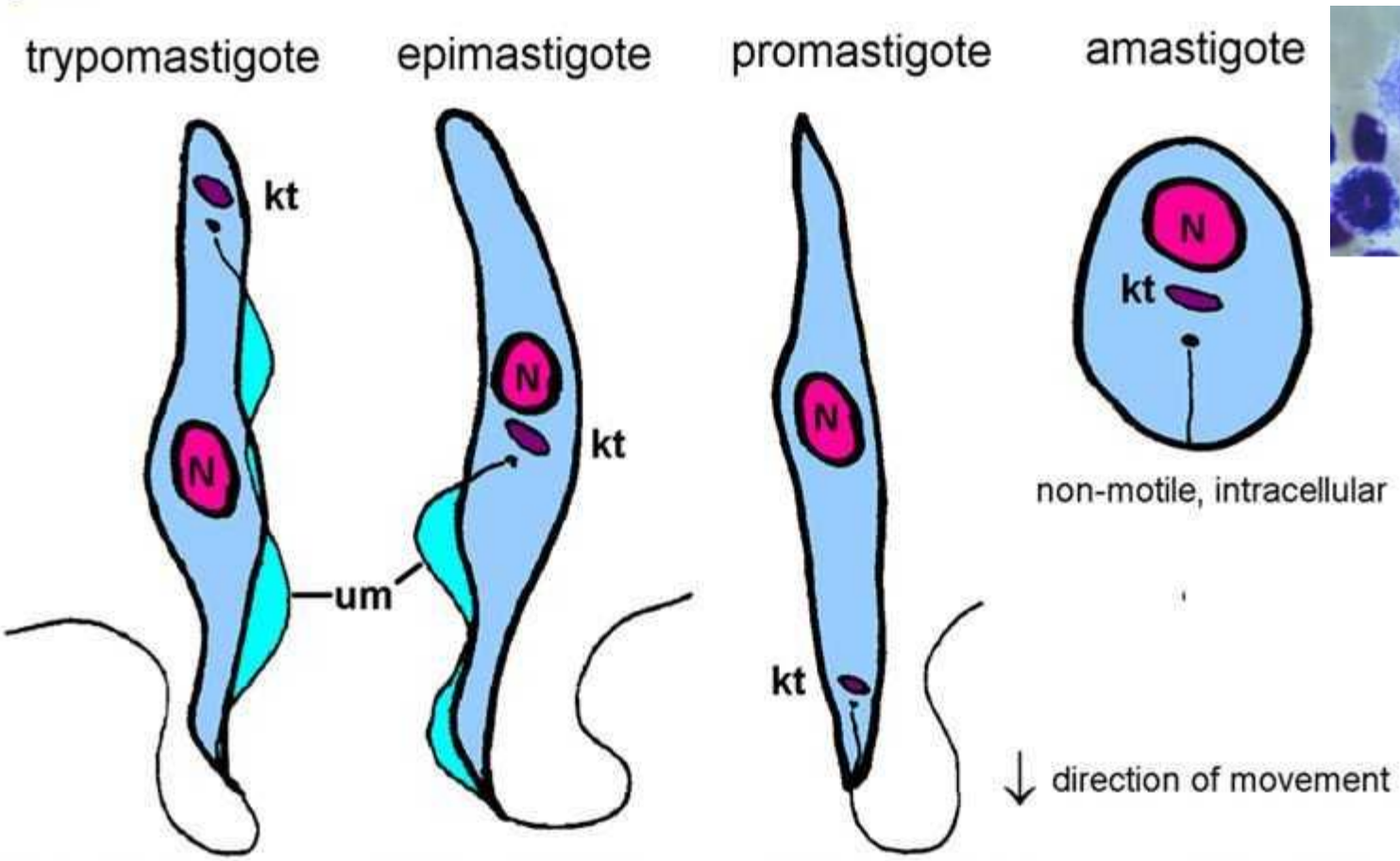
2- **Promastigotes (Leptomonad stage): The body is the spindle, the large single nucleus is located in or near the center of the body. The kinetoplast is located in the anterior end of the organism. A single free flagellum extends anteriorly. It is the form the parasite lives in the vector sand fly gut.**

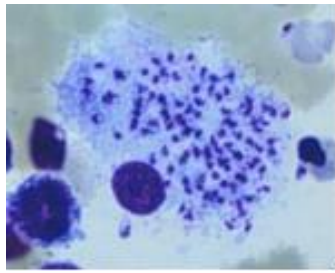
3- **Epimastigotes (Crithidial stage). The body is a spindle, and the large single nucleus is located in the center of the organism. The kinetoplast is located anterior to the nucleus. An undulating membrane measuring half the body length forms into a free flagellum at the anterior end of the epimastigote.**

4- **Trypomastigotes (Trypanosomal stage): The body is the spindle and has a single nucleus located in the center of the organism. The kinetoplast is located in the last of the body. Have anterior free flagellum and length undulating membrane covering all the body.**

Trypanosomatidae

Stages:





Trypomastigote:
blood stream form;
infective form
replicative

Epimastigote:
replicative stage
in insect

Promastigote:
infective stage of
Leishmania spp.

Amastigote:
non-motile;
intracellular,
stage in vertebrate

Leishmania

Parasite in human present in amastigote (diagnostic stage for human), while in the insect (Sand fly) promastigote (infective stage for human).

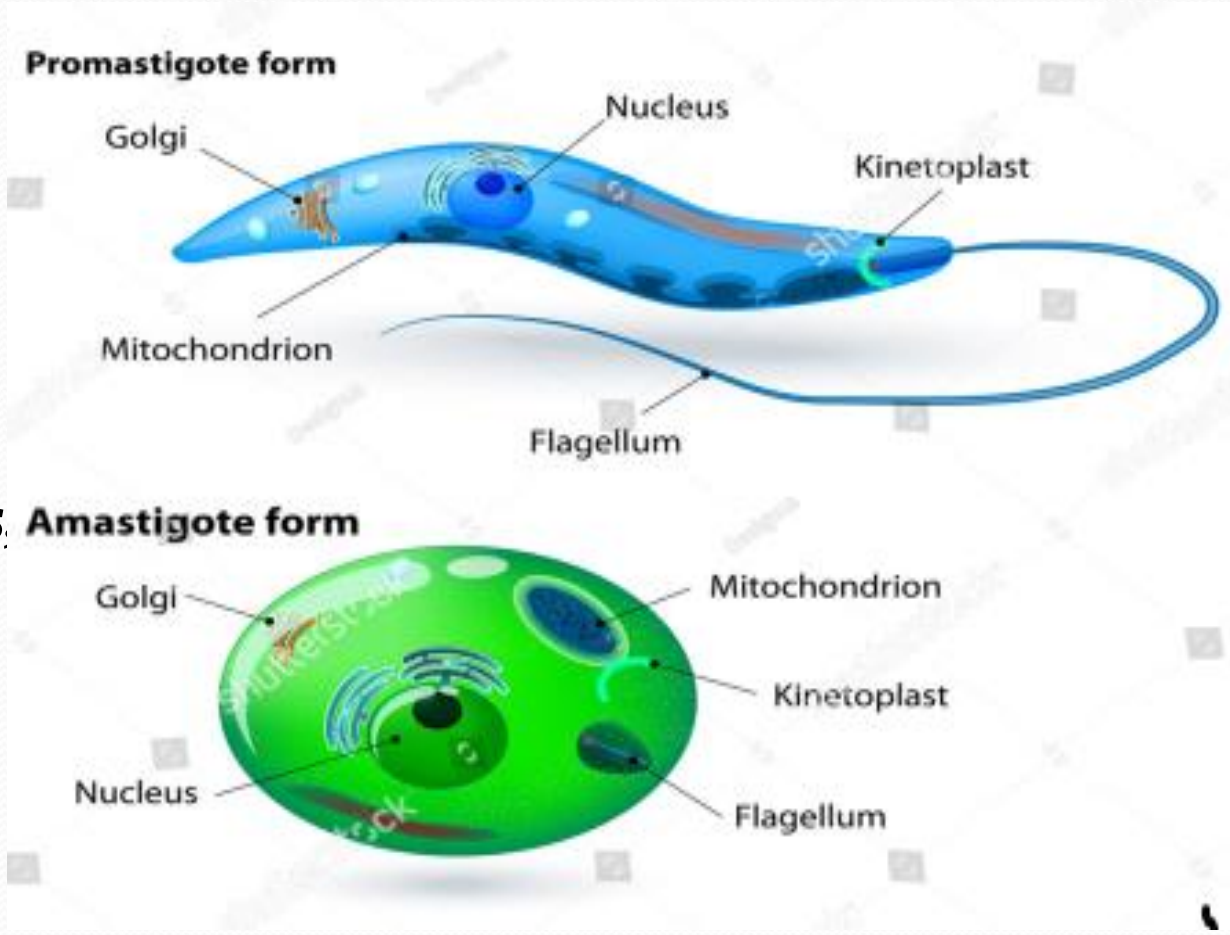
Include 4 major species:

Leishmania donovani,

Leishmania tropica,

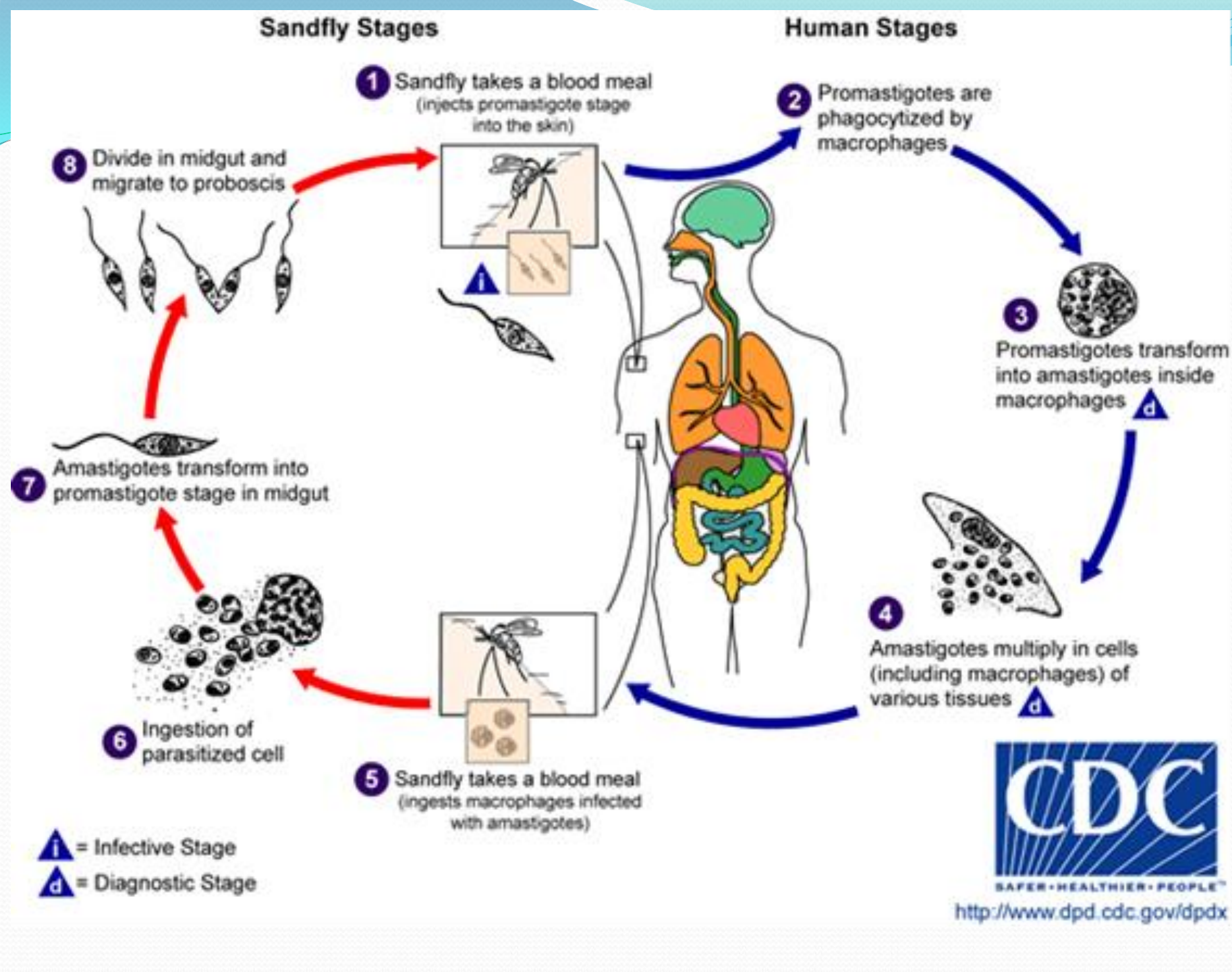
Leishmania braziliensis,

Leishmania Mexicana



life cycle

The life cycle involves the sand fly as the vector and a variety of mammals such as dogs, foxes, and rodents as reservoirs. Only female flies are vectors because only they take blood meals (a requirement for egg maturation). Shortly after an infected sand fly bites a human, the promastigotes are engulfed by macrophages, where they transform into amastigotes. The infected cells die and release amastigotes that infect other macrophages and reticuloendothelial cells. When the female sandfly sucks blood from an infected host, it ingests macrophages containing amastigotes. After dissolution of the macrophages, the freed amastigotes differentiate into promastigotes in the gut. They multiply and then migrate to the pharynx, where they can be transmitted during the next bite. The cycle in the sandfly takes approximately 10 days.


Sandfly Stages
Human Stages
1 Sandfly takes a blood meal
(injects promastigote stage
into the skin)
2 Promastigotes are
phagocytized by
macrophages
3 Promastigotes transform
into amastigotes inside
macrophages
d
4 Amastigotes multiply in cells
(including macrophages) of
various tissues
d
5 Sandfly takes a blood meal
(ingests macrophages infected
with amastigotes)
6 Ingestion of
parasitized cell
7 Amastigotes transform into
promastigote stage in midgut
8 Divide in midgut and
migrate to proboscis
i = Infective Stage
d = Diagnostic Stage
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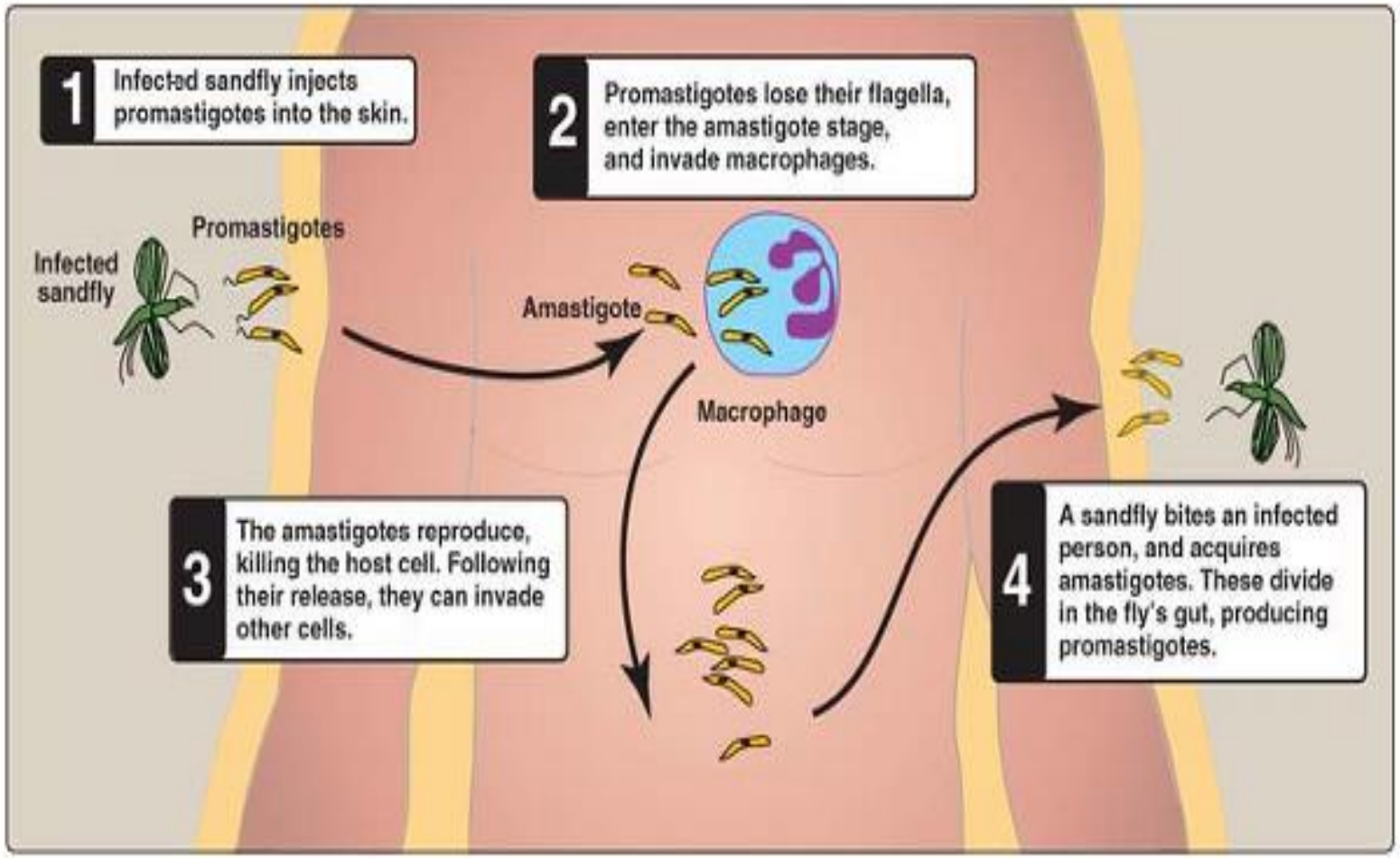


Figure 21.13 Life cycle of *Leishmania*.

L.donovani: **causes visceral Leishmianiasis, Post kala-azar dermal leishmaniasis (PKDL), Kalaazar and Dum Dum fever. Spleenomegaly & hepatomegaly. And black fever**

Visceral leishmaniasis (local name**, kala-azar**): This disease is caused by Leishmania donovani in India, East Africa, and China. In the visceral disease, the parasite initially infects macrophages, which, in turn, migrate to the spleen, liver, and bone marrow, where the parasite rapidly multiplies. The spleen and liver enlarge, and jaundice may develop. Most individuals have only minor symptoms, and the disease may resolve spontaneously. However, in some cases, complications resulting from secondary infection and emaciation result in death. Untreated severe disease is nearly always fatal as a result of secondary infection.

Clinical symptoms

Intermittent fever, weakness, and weight loss.

Massive enlargement of the spleen is characteristic.

Hyperpigmentation of the skin.

As anemia, leukopenia, and thrombocytopenia become more profound, and gastrointestinal bleeding occur.

***L.tropica* : the insect transport *L.tropica* is sand fly, causes tropic sore or Baghdad boil, oriental sore,cutenaeous Leishmianiasis**

Cutaneous leishmaniasis (local name, oriental sore): This disease is caused by Leishmania tropica in north and west Africa, Iran, and Iraq. The cutaneous form of the disease is characterized by ulcerating single or multiple skin sores. Most cases spontaneously heal, but the ulcers leave unsightly scars. In Mexico and Guatemala, the cutaneous form is due to Leishmania mexicana, which produces single lesions that rapidly heal.

L. braziliensis: causes **Mucocutaneous leishmaniasis** (local name, **espundia)**:

This disease is caused by Leishmania brasiliensis in Central and South America, especially the Amazon regions. In this form of the disease, the parasite attacks tissue at the mucosal-dermal junctions of the nose and mouth, producing multiple lesions. Extensive spreading into mucosal tissue can obliterate the nasal septum and the buccal cavity, ending in death from secondary infection.

Diagnosis of *L.donovani*

1.thick blood film (amastigot).

2. skin test: is used to measure delayed hypersensitivity.

3. detection of antibody by ELISA.

4. can be cultured on NNN media (Novy Macneel Nicolle)

Prevention and Control

control of **Leishmania donovani** = the vector control, and avoidance sand fly

control of **Leishmania tropica** = the vector control, and avoidance sand fly

Treatment of **Leishmania donovani** = Pentostam +Sodium Stibogluconate

- Treatment of **Leishmania tropica** = Paromomycin + Sodium Stibogluconate

Trypanosoma:

1-Trypanosoma brucei : This genus has two subspecies:(T.gambiense & T.rhodesiense). **the mediated host is Tse_Tse fly**

T. gambiense: **causing African Trypanosomiasis or sleeping sickness (sleeping disease) to human.** The disease is endemic in sub-Saharan Africa, the natural habitat of the tse-tse fly (temperature & humidity). The trypomastigotes spread from the skin through the blood to the lymph nodes and the brain. The typical sleeping sickness progresses to coma as a result of a demyelinating encephalitis. **Clinical symptoms**: Irregular heartbeat ,Congestive heart failure Sudden cardiac arrest, difficulty swallowing due to enlarged esophagus, enlarged colon .Abdominal pain , constipation , Swollen glands , eyelid swelling.

2-T. cruzi: **cause chagas' disease, American trypanosomiasis**. Chagas' disease is transmitted to humans by **bugs.** Chagas' disease occurs primarily in rural Central and South America (temperature & humidity).

Trypomastigotes which enter the blood and converted amastigotes within host cells. When the amastigotes can cause inflammation, consisting mainly of mononuclear cells. Cardiac muscle is the most frequently and severely affected tissue.

Diagnosis : Microscopic detection by blood smear stained with Giemsa stain, aspirate lymph node fluid and cerebrospinal fluid.

Treatment: Pentamidine, for first stage of *Trypanosomes* , Suramin, melarsoprol and nifurtimox can be used , serial examinations done for two years to prevent relapse.

Control: There is no vaccine against Trypanosomiasis. Preventive contact with tsetse flies ,use insect repellent and reducing the reservoir and vector of T .

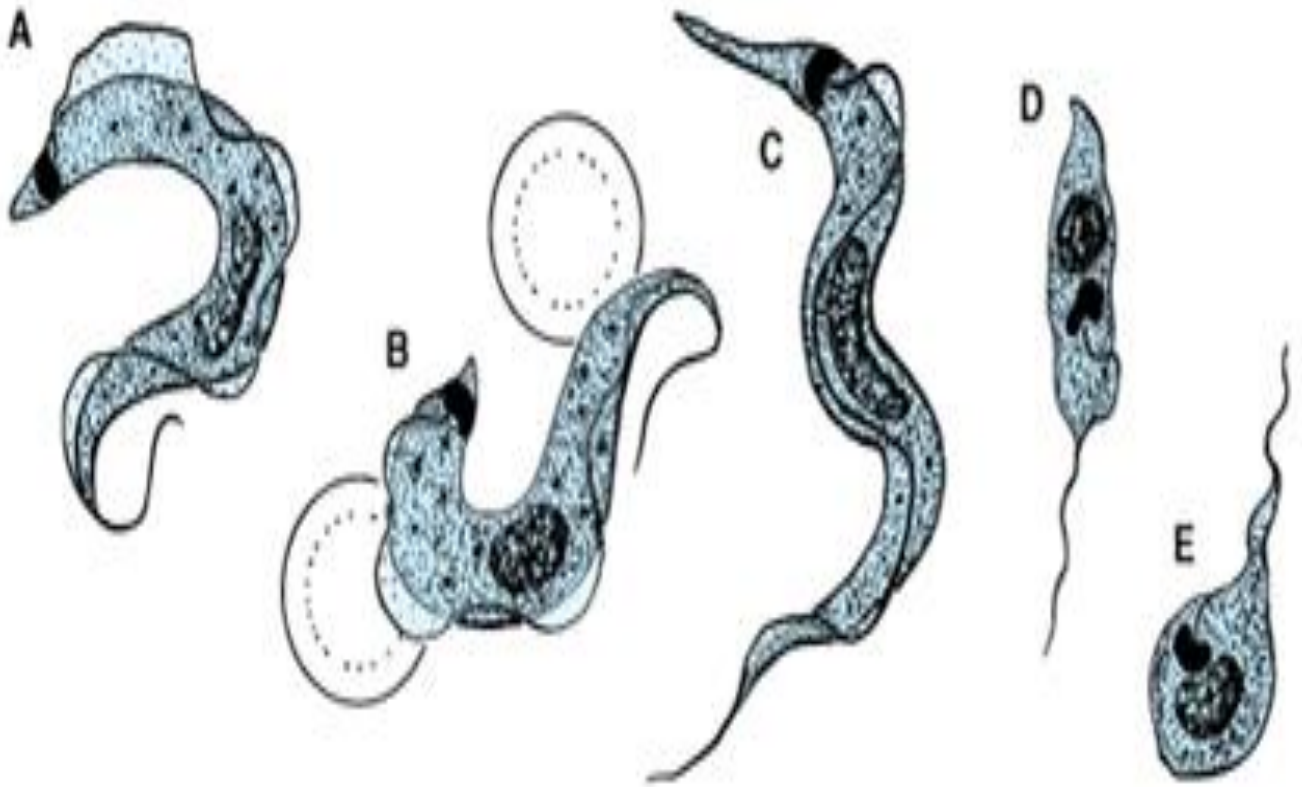
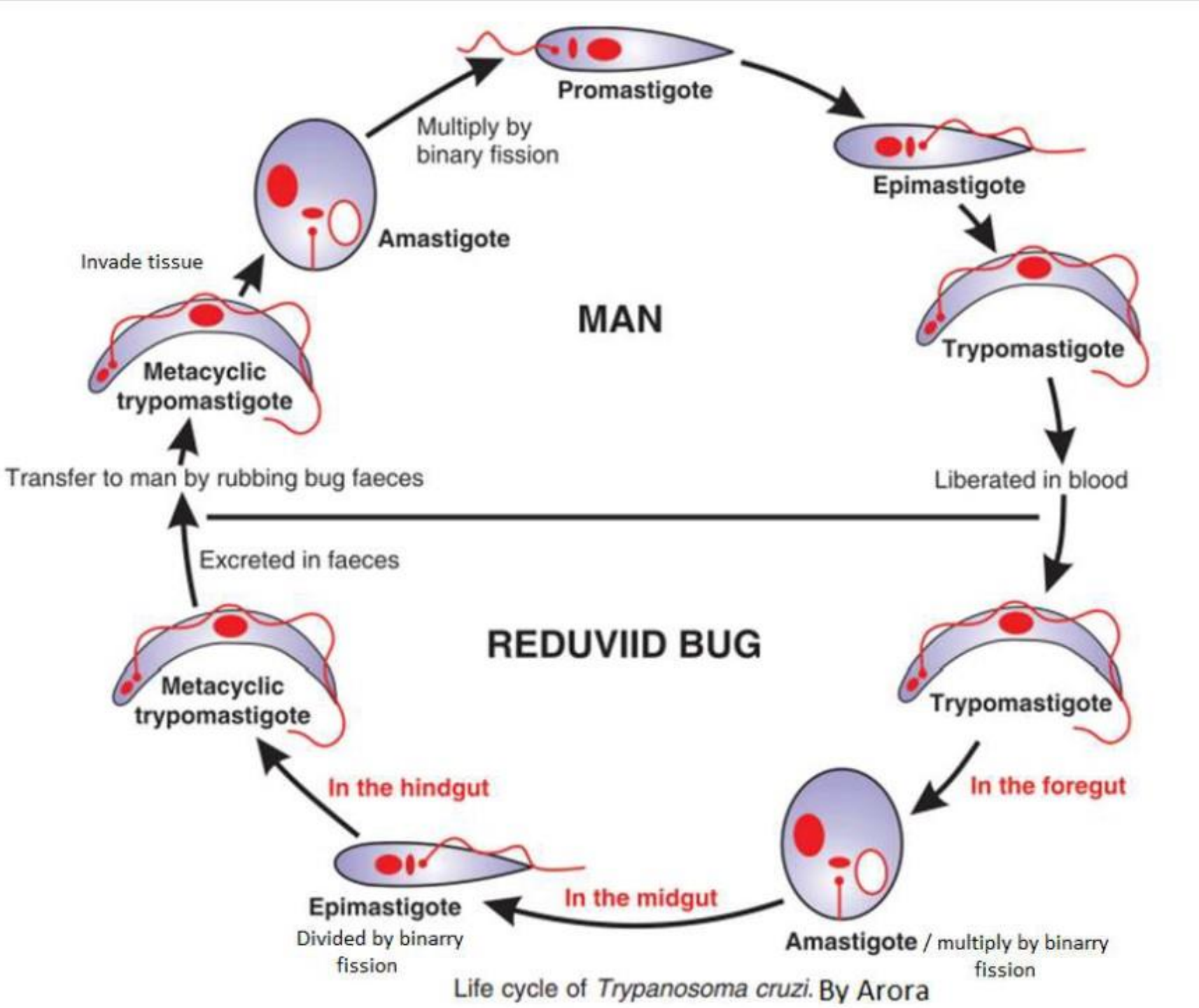
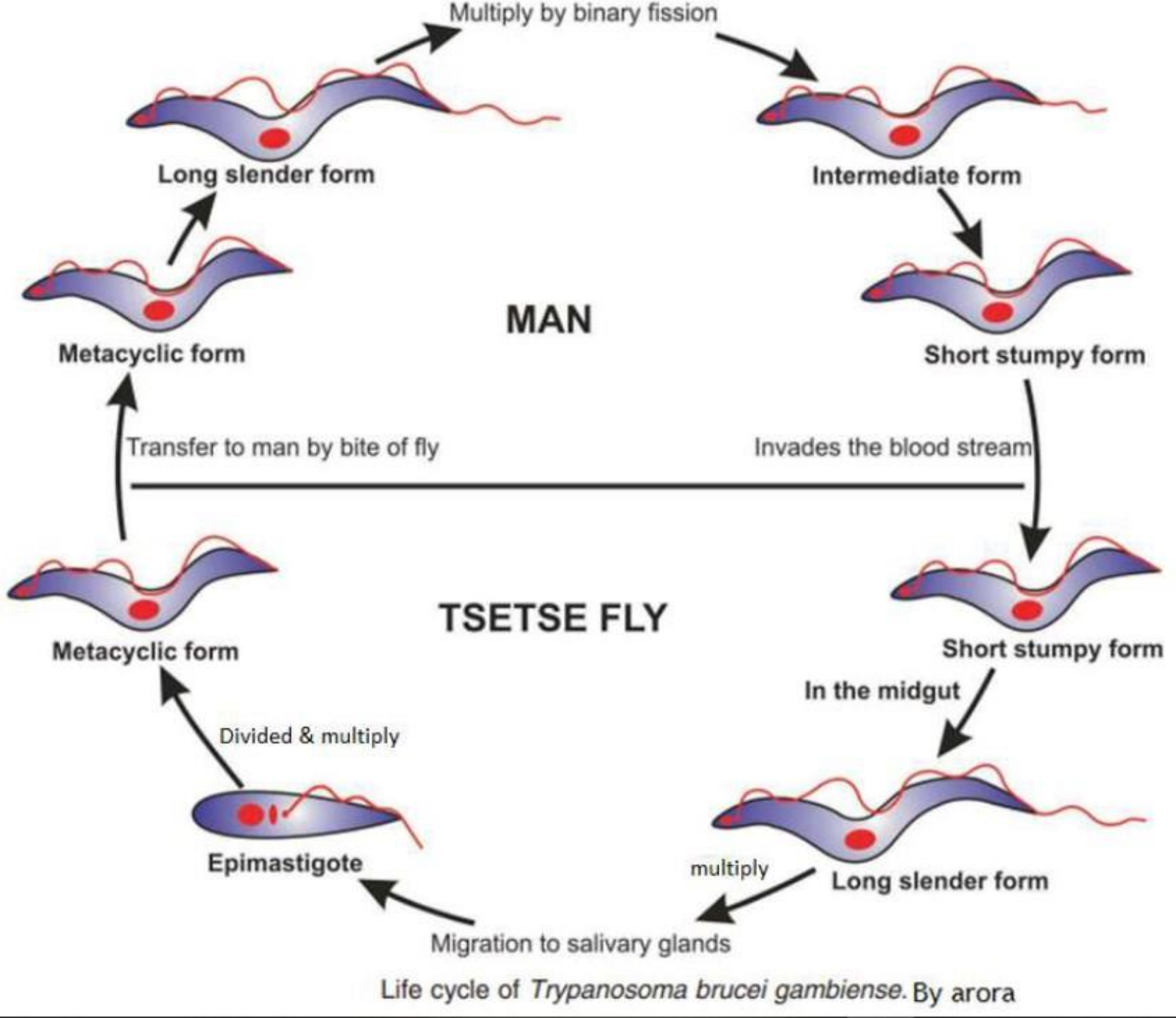


Figure:

A, B, C: Trypomastigotes in blood;
D: epimastigote,
E: promastigote,
F: amastigote colony in heart muscle



Life cycle of *Trypanosoma cruzi*. By Arora



Life cycle of *Trypanosoma brucei gambiense*. By arora