Hemoflagellate (Trypanosoma, leishmania -- move by the mean of single flagella, asexual only)

General developmental stages: amastigote [contains kinetoplast which is the origin of the flagellum] → promastigote → epimastigote → trypomastigote [contains flagellum + undulating membrane on the same axis of the protozoa]

Trypanosoma

Causes trypanosomiasis = African trypanosomiasis [sleeping sickness] caused by Trypanosoma brucei complex + American trypanosomiasis [Chagas disease] caused by Trypanosoma cruzi ➔ both are vector borne, can be transmitted through blood transfusion and transplacental

Morphology

Inside humans: trypomastigote + amastigote (round intracellular form presents only in American trypanosomiasis)

Inside vectors: promastigote + epimastigote

Antigen variation

A unique feature of African trypanosomes is their ability to change the antigenic surface coat of the outer membrane of the trypomastigote (VSG), helping to evade the host immune response.

1- African trypanosomiasis

Caused by: T. brucei gambiense -> west African trypanosomiasis [chronic, slowly, reservoir: humans], T. brucei rhodesiense -> east African trypanosomiasis [acute, faster, reservoir: animals, less frequent]

Vector: Tsetse fly (Glossina spp.)

Infective stage: metacyclic trypomastigotes

Diagnostic stage: trypomastigotes

Clinical features: after the host has been bitten by an infected tsetse fly, a painless nodule (chancre) at the site may develop – stage 1: the patient have systemic trypanosomiasis without CNS involvement + The trypomastigotes enter the bloodstream and invade the lymph nodes + irregular fever with night sweats, enlargement of liver and spleen, Winterbottom's sign – stage 2: organisms invade the CNS, the sleeping sickness stage of the infection is initiated, coma, death

Laboratory diagnosis: trypomastigotes inside the blood, antigen detection, antibody detection, molecular diagnostics
Therapy: if the CNS isn’t affected → Suramin, if the CNS is affected (bad prognosis) → Melarsoprol

Prevention: preventing flies from biting, screening of people at risk, treatment cases

2- **American trypanosomiasis:**

Caused by Trypanosoma cruzi (Chagas’ disease)

Vector: reduviid bugs (kissing bugs) / defecate while taking a blood meal / enter through wounds or mucosal membranes

Epidemiology: throughout central and south America

**Infective stage:** metacyclic trypomastigotes

**Diagnostic stage:** amastigote (in tissues)

trypomastigote (in blood)

Clinical features: nodule chagoma – **acute phase:** start after 1 week of infection, fever, lymph node enlargement, enlarge liver and spleen, unilateral swelling of eyelids **Romana's sign**, acute myocarditis – **chronic phase:** involve the heart, where enlargement of the heart, including cardiac changes (can affect any organ – enlargement of the colon)

Therapy: Nifurtimox

Prevention: vector control, transfusion control, testing of organ, tissue or cell donors and receivers

**Leishmania:**

Vector: female sand fly

Obligate intracellular organism

Infects primarily phagocytic cells and macrophages

**Infective stage:** promastigotes

**Diagnostic stage:** amastigotes

Transmission: bites of sand fly, transfusion blood and transplantation, mother to baby, direct contact from man to man through nasal secretion
A. **Cutaneous leishmaniasis**: Leishmania tropica, L major, L infantum / lesions / **Leishmania major** is the major species of Leishmania parasite in Jordan.

B. **Mucocutaneous leishmaniasis (naso-pharyngeal)**: L. braziliensis

C. **Visceral leishmaniasis (kala-azar / black fever)**: L. donovani / liver, spleen, bone marrow / enlarged liver and spleen / India + Sudan

**Laboratory diagnosis**: amastigotes inside the macrophages / **intradermal Montenegro test [type 4 / delayed hypersensitivity reaction]**

**Therapy**: cutaneous leishmaniasis -> lesions usually heal spontaneously / mucocutaneous + visceral leishmaniasis → **sodium stibogluconate**

**Prevention**: vector control