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/ /2020

Medical parasitology

Haemoflagelates

Leishmania Species

Clinical disease

- Veseral leishmaniasis
- Cutaneous leishmaniasis
- Mucocutaneous leishmaniasis

The species of leishmania exist in two forms, amastigote (a flagellar) and promastigote (flagellated) in their life cycle. They are transmitted by certain species of sand flies (Phlebotomus&Lutzomyia).

Leishmania donovani (veseralleishmaniasis)

Important features- the natural habitat of *L.donovaniin* man is the reticuloendothelial system of the viscera, in which the amastigote multiplies by 48 simple binary fission until the host cells are destroyed.

Pathogenesis

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen and bone marrow) are the most severely affected organs, spleen bone marrow .results in anemia, leukopenia. The spleen and liver become markedly enlarged,

Clinical features Symptoms begin with fever, weakness, and diarrhea; chills and sweating that may resemble malaria symptoms are also common early in the infection. As organisms proliferate & invade cells of the liver and spleen, marked enlargement of the organs, weight loss, anemia., (kala-azar) dermal leishmaniasis, occurs.

Laboratory diagnosis

• Examination of tissue biopsy, spleen aspiration, bone marrow aspiration or lymph node aspiration in properly stained smear (e.g. Giemsa stain).

Culture of blood, bone marrow, and other tissue often demonstrates the Old World Cutaneous Leishmaniasis (Oriental sore)

Clinical disease

L.***tropica*** minor -dry cutaneous leishmaniasis

L.***tropica*** major- wet cutaneous leishmaniasis

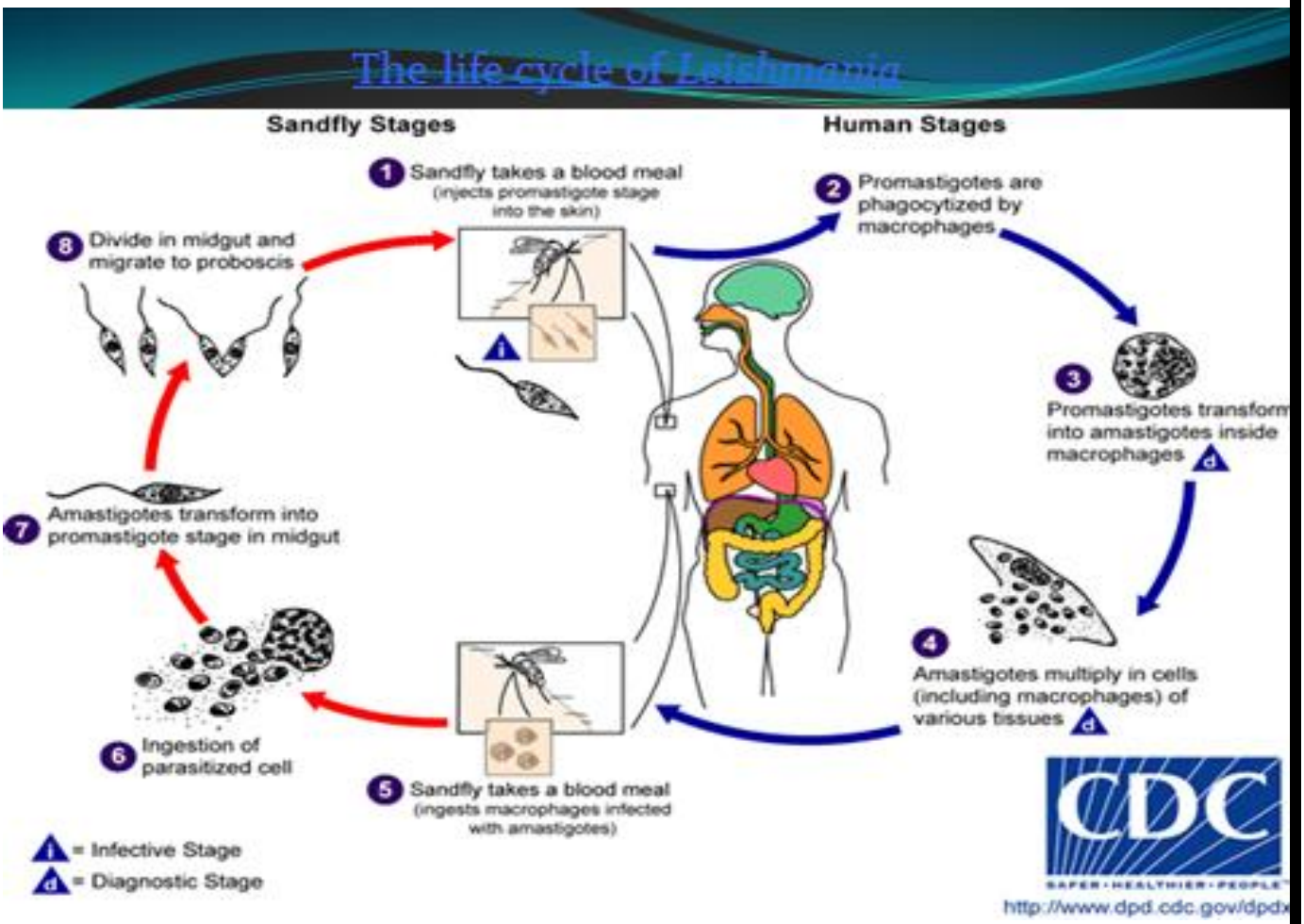
Clinical features

The first sign, a red papule, appears at the site of the fly's bite. This lesion becomes irritated, with intense itching, and begins to enlarge & ulcerate. the ulcer becomes hard,. secondary bacterial infection may complicate the disease. This leads to the formation of disfiguring nodules over the surface of the body.

Prevention

-Prompt treatment & eradication of ulcers

-Control of sand flies & reservoir hosts



Trypanosomiasis

Etiologic agents

Trypanosoma brucei complex– African trypanosomiasis (sleeping sickness)

Trypanosoma cruzi– American trypanosomiasis (Chagas' disease)

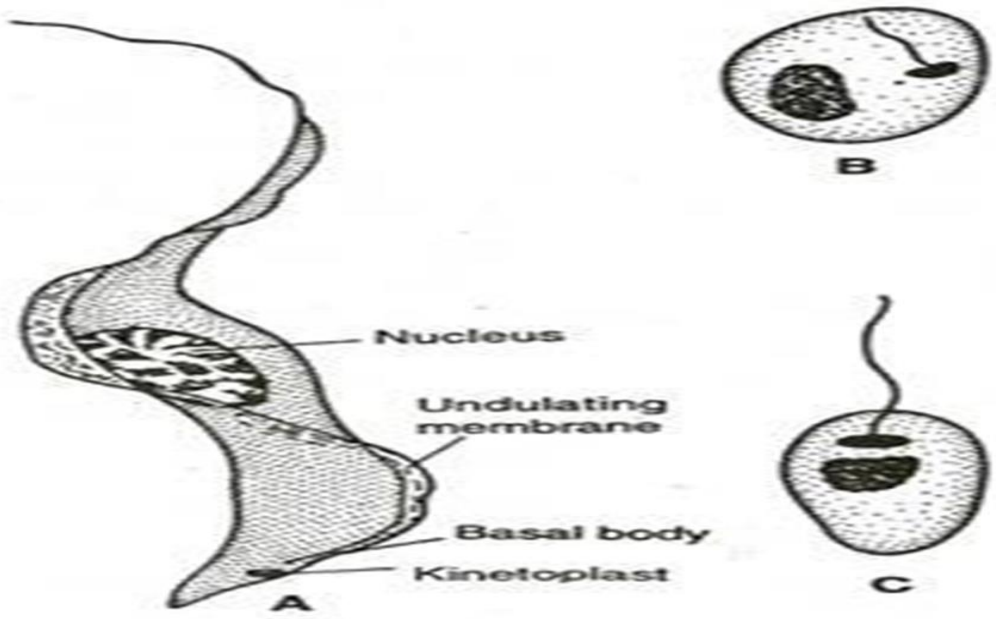


Fig. 185. *Trypansoma cruzi.* A. Trypanosoma from. B. Leishmanial from 3. Crithidial form.

Important features

Typical trypanosome structure is an elongated spindle-shaped body that , a centrally situated nucleus , an undulating membrane proceeding forward along the margin of the cell membrane and a single free flagellum at the anterior end

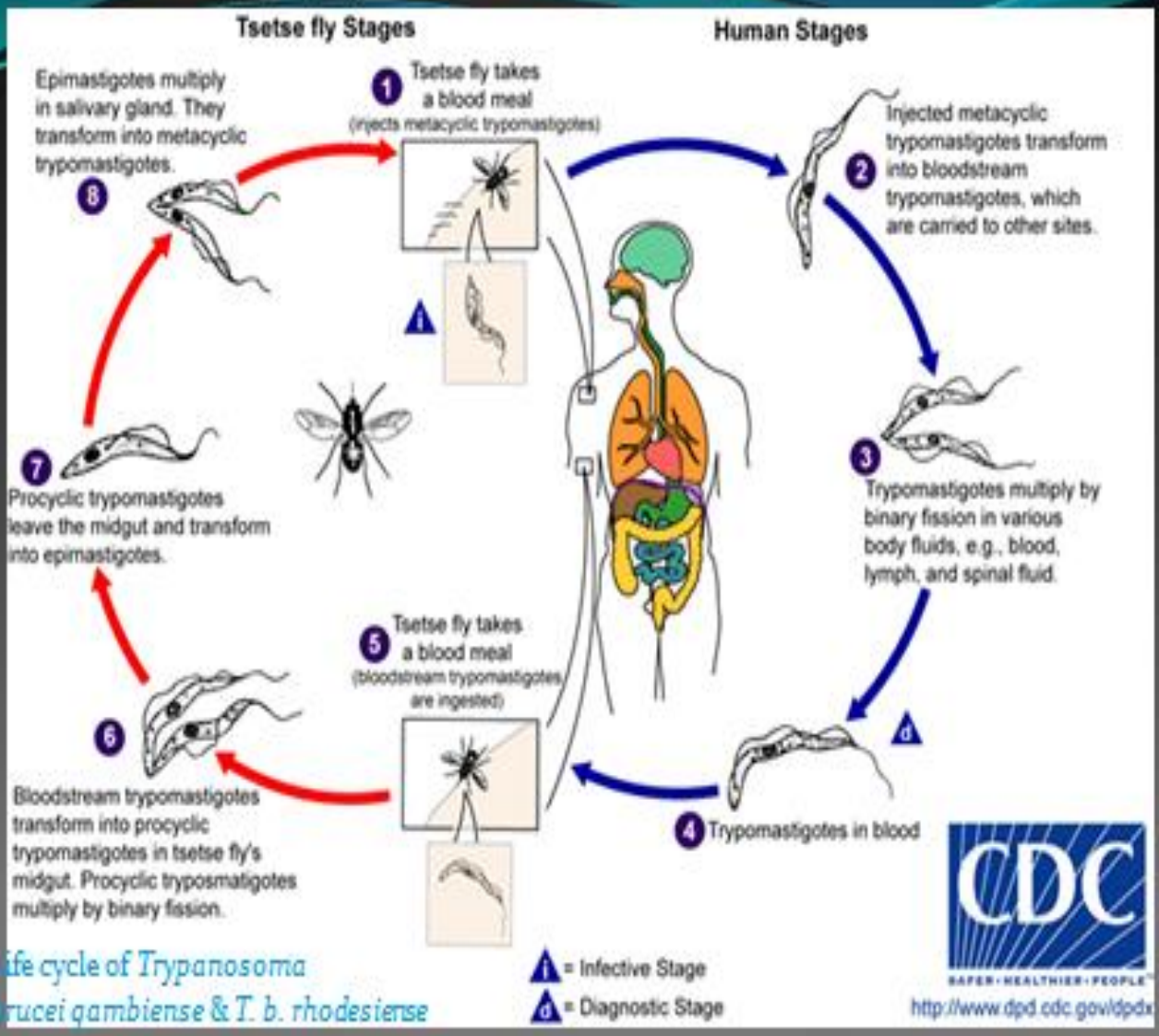
1. **African trypanosomiasis**

causative agents of the African typanosomiasis, transmitted by insect bites. The vector for both is the tsetse fly.

Pathogenesis

The trypomastigotes(infected stage)spread from the skin through the blood to the lymph node and the brain. The (sleeping sickness) usually progresses to coma , cyclical fever spike (approximately every 2 weeks).

African Trypanosomiasis



Life cycle of *Trypanosoma brucei gambiense* & *T. b. rhodesiense*

MEDICALLY IMPORTANT CILIATES

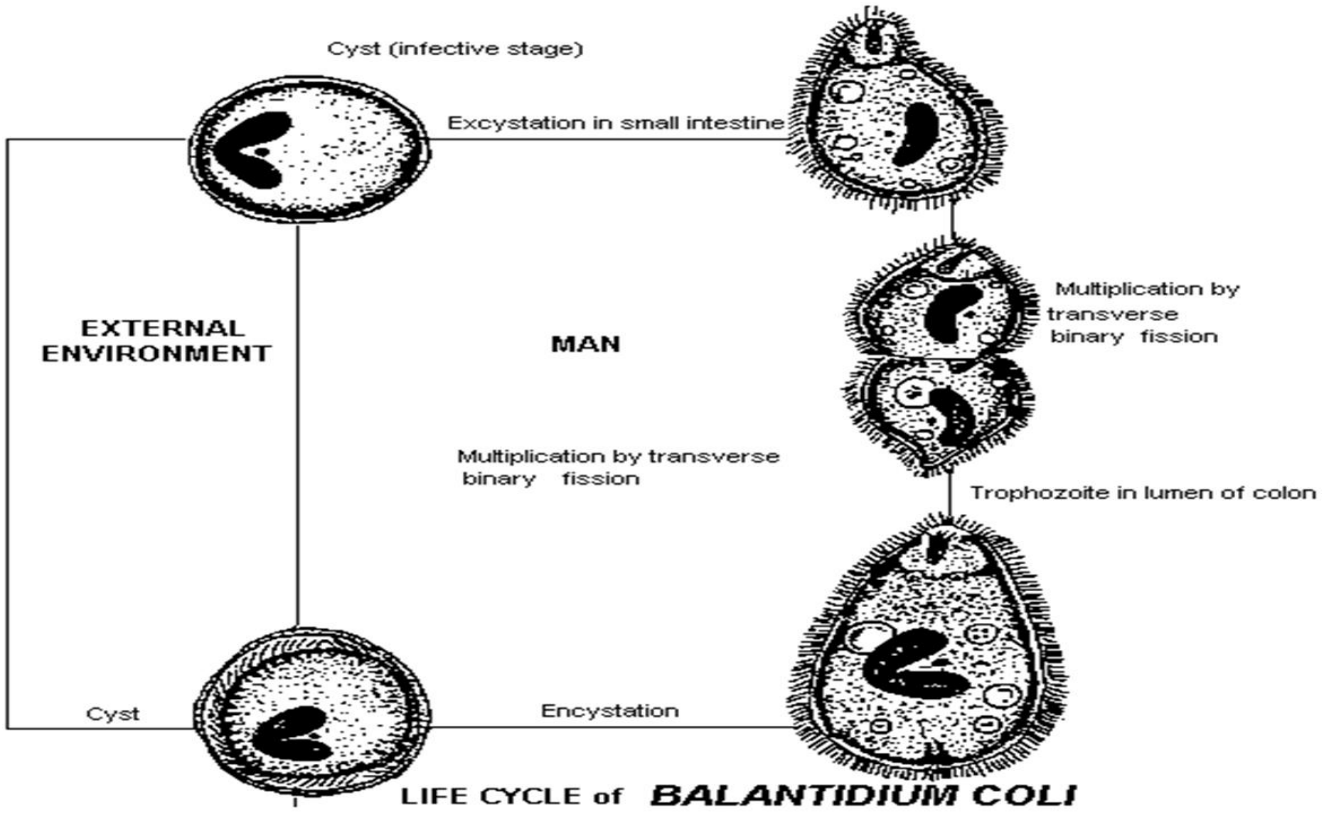
Balantidiasis

The intestinal protozoan ***Balantidium*** ***coli*** the only member of the ciliate group

that is pathogenic for humans. Disease produced by ***B. coli*** similar to amebiasis, because the organisms elaborate proteolytic and cytotoxic substances that mediate tissue invasion and intestinal ulceration.

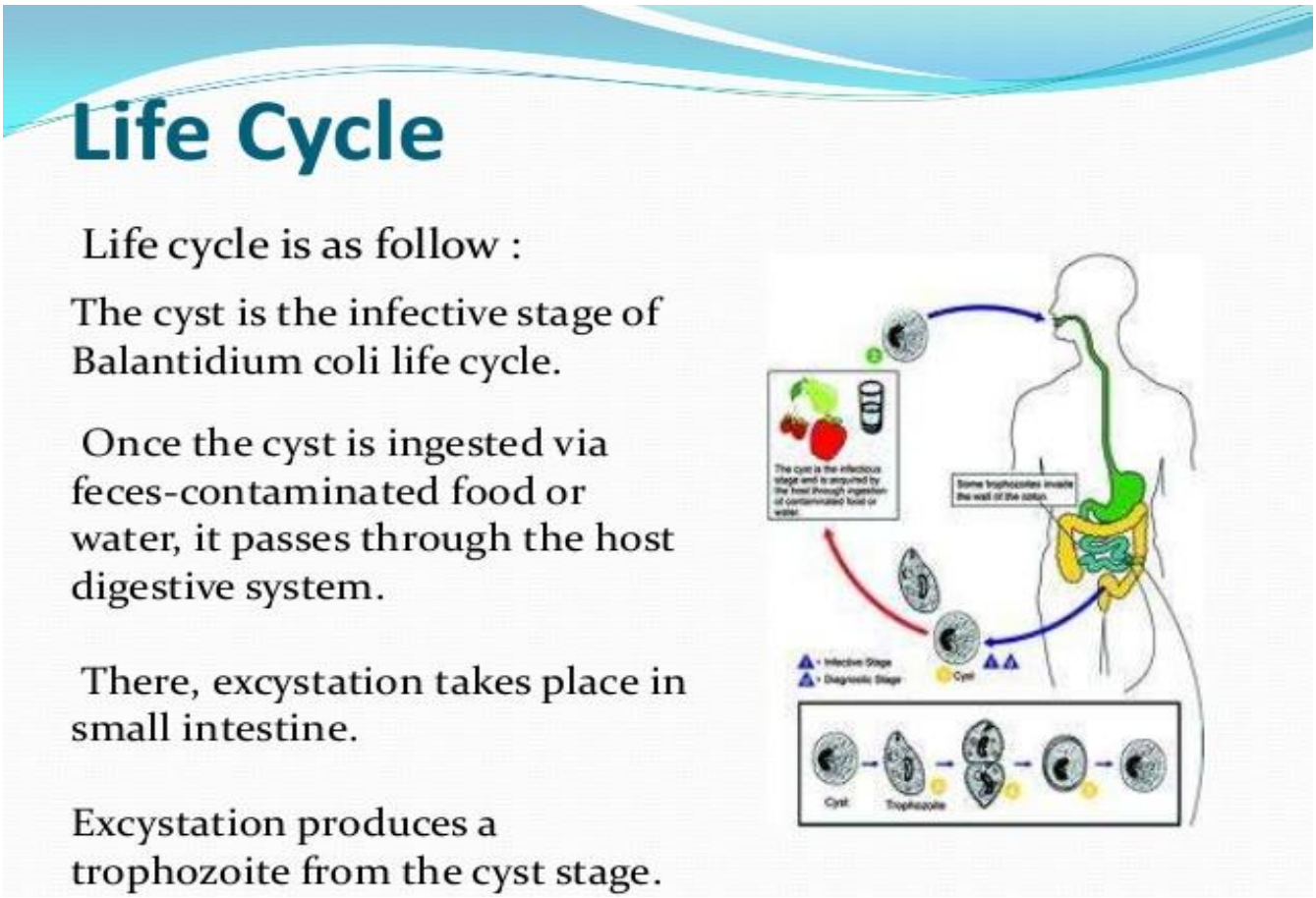
Life cycle

The life cycle of ***B. coli*** simple, involving ingestion of infectious cysts, excystation, and invasion of trophozoites into the mucosal lining of the large intestine. The trophozoite is covered with rows of hair like cilia that aid in motility. Morphologically more complex than amebae, *B.coli* has a funnel-like primitive mouth called a cytostome, a large (macro) nucleus and a small (micro) nucleus involved in reproduction.



LIFE CYCLE of ***BALANTIDIUM COLI***

Adapted and redrawn from NCDC



Clinical features

Symptomatic disease is characterized by abdominal pain, tenderness, nausea, anorexia, and watery stools with blood and pus. Ulceration of the intestinal mucosa, as with amebiasis, can be seen; a secondary complication caused by bacterial invasion into the eroded intestinal mucosa can occur.

Laboratory Diagnosis

Microscopic examination of faeces for trophozoite and cysts is performed.

COCCIDIA (SPOROZOA)

INTRODUCTION

Coccidia are members of the class sporozoa, Phylum Apicomplexa. Apical complex is present at some stage and consists of elements visible with electron microscope. The life cycle is characterized by an alternation of generations, i.e. sexual (gametogony) and asexual (schizogony) reproduction and most members of the group also share alternative hosts. The locomotion of a mature organism is by body flexion, gliding, or undulation of longitudinal ridges. The genus Plasmodium that are the causes of malaria is the prototype of this class.

Malaria

There are four species normallyi nfecting humans, namely, *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, and *Plasmodium malariae*.

Life cycle

The life cycle of malaria is passed in two hosts (alternation of hosts) and has sexual and asexual stage (alternation of generations).

1- Vertebrate host - man (intermediate host), where the asexual cycle takes place. The parasite multiplies by schizogony and there is formation of male and female gametocytes (gametogony).

2- Invertebrate host - mosquito (definitive host) where the sexual cycle takes place. Union of male and female gametes ends in the formation of

sporozoites (sporogony).

The life cycle passes in four stages:

Three in man:- Pre – erythrocytic schizogony

Erythrocytic schizogony

- Exo- erythrocytic schizogony

One in mosquito - Sporogony

Introduction into humans- when an infective female Anopheles mosquito bites

man, it inoculates saliva containing sporozoites (infective stage).

Pre- Erythrocyticschizogony- sporozoites reach the blood stream and the liver, initiating a cycle of schizogony. Multiplication occurs in tissue schizonts, to form thousands of tiny merozoites. Merozoites are then liberated on rupture of schizonts about 7th – 9th day of the bites and enter into the blood stream. These merozoites either invade the RBC's or other liver cells. In case of P. falciparumand possibly *P. malariae,* all merozoites invade RBC's without re-invading liver cells. However,

For *P*. *vivax* and *P*. *ovale*, some merozoites invade RBC's and some re-invade

liver cells initiating further Exo-erythrocyticschizogony, which is responsible for

relapses.

Erythrocytic schizogony (blood phase) The merozoites reinvade fresh RBC's repeating the schizogonic cycles Erythrocytic merozoites do not reinvade the liver cells. So malaria transmitted by blood transfusion reproduces only erythrocytic cycle .

Gametogony

Some merozoites that invade RBC's develop into sexual stages (male and female gametocytes). These undergo no further development until taken by the mosquito.

Sporogony (extrinsic cycle in mosquito)

When a female Anopheles mosquito vector bites an infected person, it sucks

blood containing the different stages of malaria parasite. All stages other than

gametocytes are digested in the stomach.

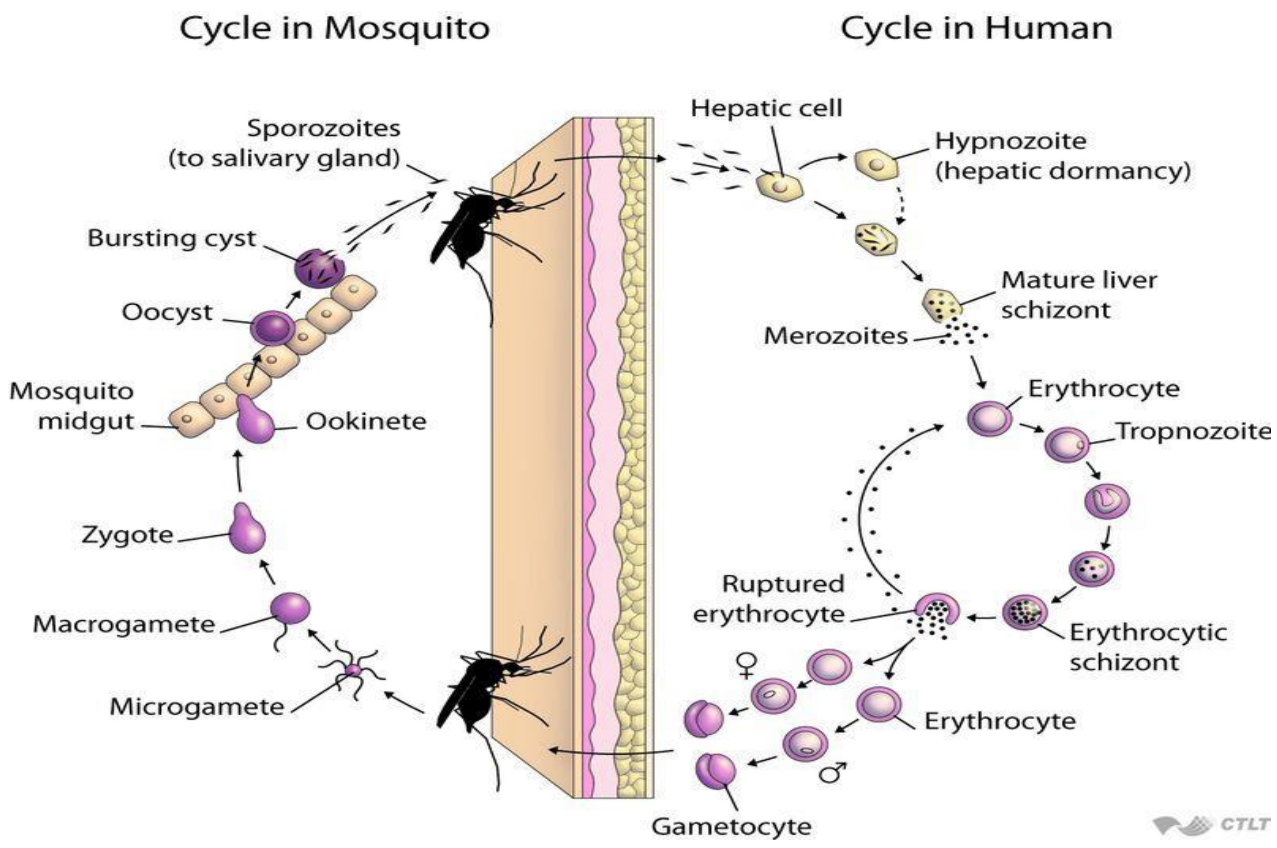
The microgametocyte.

The microgametocyte ex-flagellation.divides by reduction division into 6-8 pieces, which migrate to the periphery., microgametes, are actively motile and separate from the gametocyte.

The macrogametocyte by reduction division becomes a macrogamete.

Fertilization occurs by entry of a micro gamete into the macro gamete forming a zygote. The zygote changes into a worm like form, the ookinete, which penetrates the wall of the stomach to develop into a spherical oocyst. The oocystes increase in size. Thousands of sporozoites develop inside the oocysts. Oocysts rupture and sporozoites are liberated in the body cavity and migrate everywhere particularly to the salivary glands. Now the mosquito is infective

The sporogonous cycle in the mosquito takes 8-12 days depending on temperature



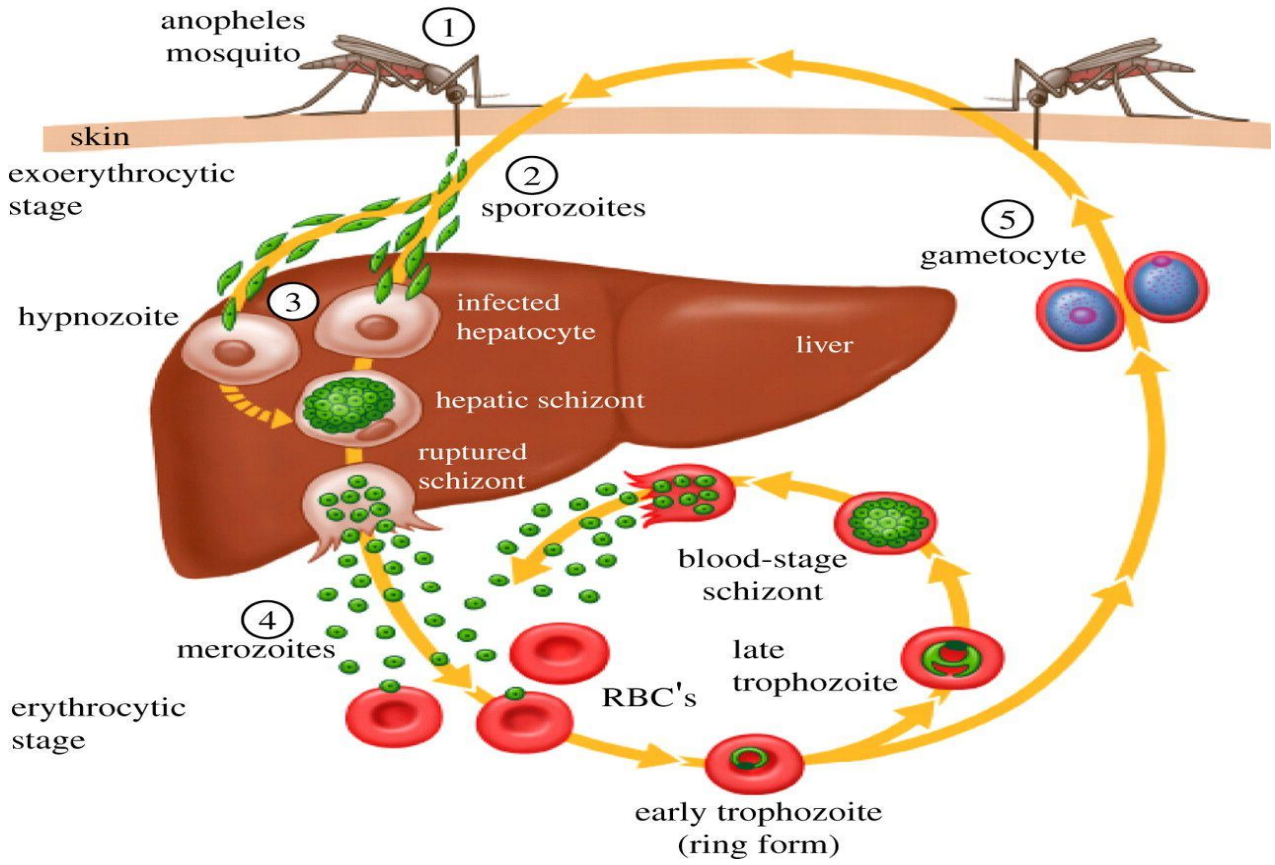


Table 2. Characteristics of infection with the five species of Plasmodia infecting humans

Characteristics	*Plasmodium falciparum*	*P.knowlesi*	*P.malariae*	*P.ovale*	*P.vivax*
Pre-erythrocytic stage (days)	5-7	8-9	14-16	9	6-8
Pre-patent period (days)	9-10	9-12	15-16	10-14	11-13
Erythrocytic cycle (days)	48	24	72	50	48
Red cells affected	All	All	Mature erythrocytes	Reticulocytes	Reticulocytes
Parasitaemia per µL • Average • Maximum	 20,000-500,000 2,000,000	 600-10,000 236,000	 6000 20,000	 9000 30,000	 20,000 100,000
Febrile paroxysm (hours)	16-36 or longer	8-12	8-10	8-12	8-12
Severe malaria	Yes	Yes	No	No	Yes
Relapses from liver forms	No	No	No	Yes	Yes
Recurrences	Yes (treatment failure)	Yes	Yes (as long as 30-50 years after primary attack)	No	Yes (treatment failure)

Other coccidian parasites Toxoplasma gondii– causes toxoplasmosis.

Toxoplasma gondii – causes toxoplasmosis. The definitive host is the

domestic cat and other felines. Humans and other mammals are intermediate hosts. T.gondii is usually acquired by ingestion and transplacental transmission from an infected mother to the fetus can occur. Human–to–human transmission, other than transplacental transmission, does not occur. After infection of the intestinal epithelium, the organisms spread to other organs, especially the brain, lungs, liver, and eyes. Most primary infections in immunocompetent adults are asymptomatic. Congenital infection can result in abortion, stillbirth, or neonatal disease with encephalitis, chorioretinitis and hepatosplenomegaly. Fever, jaundice, and intracranial calcifications are also seen. For the diagnosis of acute and congenital infections, an immunofluorescence assay for detection of antibody is used. Microscopic examination of Giemsa–stained preparations shows crescent–shaped trophozoite. Cysts may be seen in the tissue. Treatment is with a combination of sulfadiazine and pyrimethamine.

References

1-Diagnostic medical parasitology .2007 ,5th edition.

2-Human parasitology .2012 , 4th edition.