

General characteristics

Feeding:

- Phagocytosis, pinocytosis and (endocytosis; cytostome)

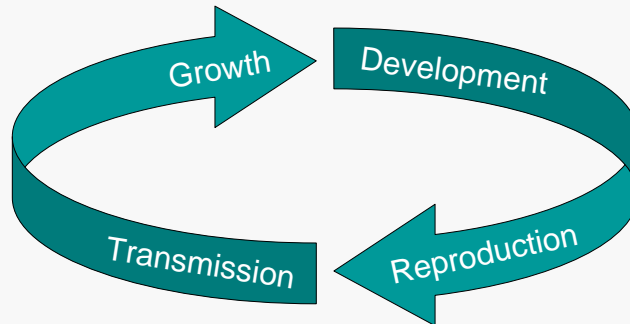
Excretion:

- In general no contractile vacuoles, usually by diffusion
- Insoluble material is dumped or left behind in inclusion bodies

Respiration:

- Both aerobic (Malaria) and anaerobic respiration
- Many protozoa live in reduced oxygen environments (facultative or aerotolerant anaerobes; e.g. Amoeba).

Major components of a parasite life cycle

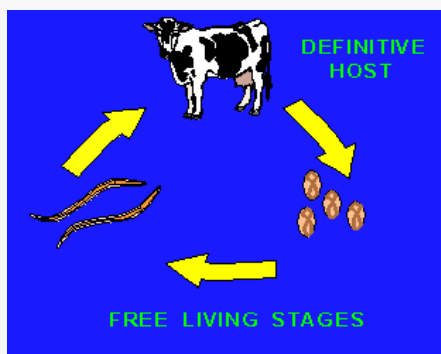


The life of a parasite can be divided into a number of phases:

- Growth and development (maturation)
- Reproductive (sexual and asexual) phases
- Transmission phases

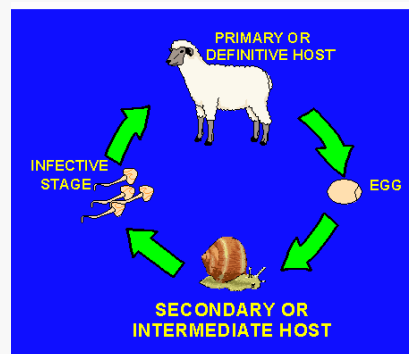
Life Cycle Patterns

Direct or monoxenous life cycle



- Only one host where the parasite spends most of its life as an adult, and where it reproduces

Indirect or heteroxenous life cycle



- Many parasites have more complex cycles which include two or more hosts

Terminology

Transmission

- Important part of any parasite's life cycle
- To continue the cycle, the parasite must produce a stage which is:
 - capable of leaving the host
 - surviving in an exterior environment
 - locating and establishing in a new host

Terminology

The primary or definitive host

- Normally the larger of the hosts (usually a vertebrate)
- A large number of host species can act as an intermediate host,
but only one or a few can act as a definitive host
- Normally where the adult parasites live
- Where sexual reproduction takes place
exception: parasites which only reproduce asexually

Terminology

The secondary or intermediate host

- The entering stage undergoes essential development before transmission
- Frequently a number of developmental stages are involved
- If no development occurs in the intermediate host it is referred to as a transport or paratenic host

Transport or paratenic host

- Often a true intermediate host may be eaten by a predator
- Often the parasite remains dormant until the paratenic host is eaten by the definitive host
- This second intermediate host may not be essential for the parasite to complete its life cycle

Terminology

The reservoir host

- Usually applies to a parasite which normally completes its life cycle in a wild animal host species.
- Host/parasite association is usually well established
- Host is usually well adapted to the parasite and tolerates the infection
- Present a particular problem for protecting the domesticated host species

Example:

Trypanosomes which infect domestic cattle in the African savanna. The wild animal population infected with trypanosomes act as reservoir hosts and the parasite is transmitted to the cattle which contract the disease Trypanosomiasis.

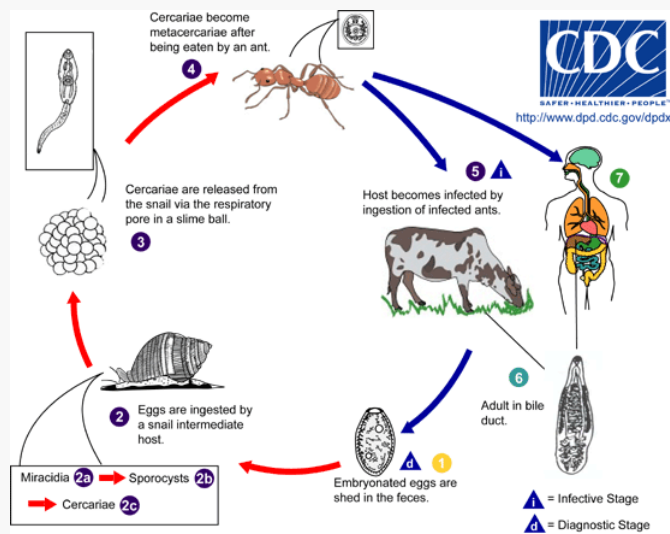
The following descriptions are based on informations given by



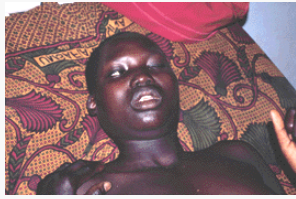
<http://www.cdc.gov>

My favorite parasite

The Lancet liver fluke (*Dicrocoelium dendriticum*)



... not a protozoon



T. rhodesiense,
T. gambiense

and

T. cruzi

Trypanosoma

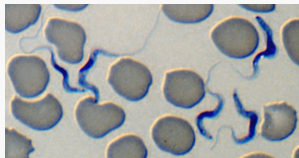
Disease: Sleeping sickness
Chagas disease



Tsetse fly



Kissing bug

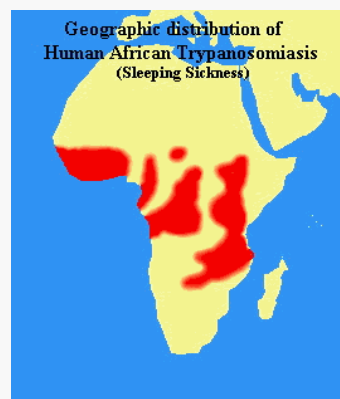


The kissing bug label comes from the insect's ability to steal a blood meal by painlessly piercing the lips, eyelids or ears of a sleeping human victim.

Global distribution of Chagas disease and sleeping sickness



threatens over 120 million people
50,000 deaths per year



threatens 60 million people
~300,000 currently infected
over 60,000 deaths per year

Sleeping sickness

- Often fatal, endemic infectious disease of humans and animals
- Caused by either *Trypanosoma rhodesiense* or *T. gambiense*)
- Transmitted by the tsetse fly
- Infection occurs in 3 stages

1. Trypanosomal chancre

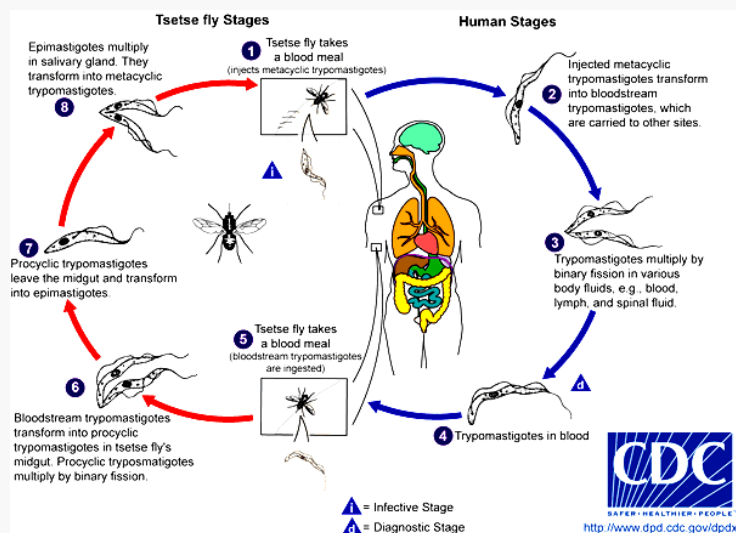
- Can develop on the site of inoculation

2. Hemolymphatic stage

- symptoms include fever, lymphadenopathy (*Erkrankung der Lymphknoten*), and pruritus (*Juckreiz*)

3. Meningoencephalitic stage (invasion of the central nervous system)

- Can cause headaches, somnolence, abnormal behavior, and lead to loss of consciousness and coma



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 entire life cycle of African Trypanosomes is represented by extracellular stages. 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*.

Treatment of Sleeping sickness

Anthelmintics

Parasite biochemical pathways are sufficiently different from the human host to allow selective interference by chemotherapeutic agents

Examples:

- Suramin is trypanocidal and works by inhibiting parasitic enzymes and growth factors
- Trypanocidal is inhibiting parasitic glycolysis
- Eflornithine (Ornidyl) is a selective and irreversible inhibitor of ornithine decarboxylase, a critical enzyme for DNA and RNA synthesis

Chagas disease



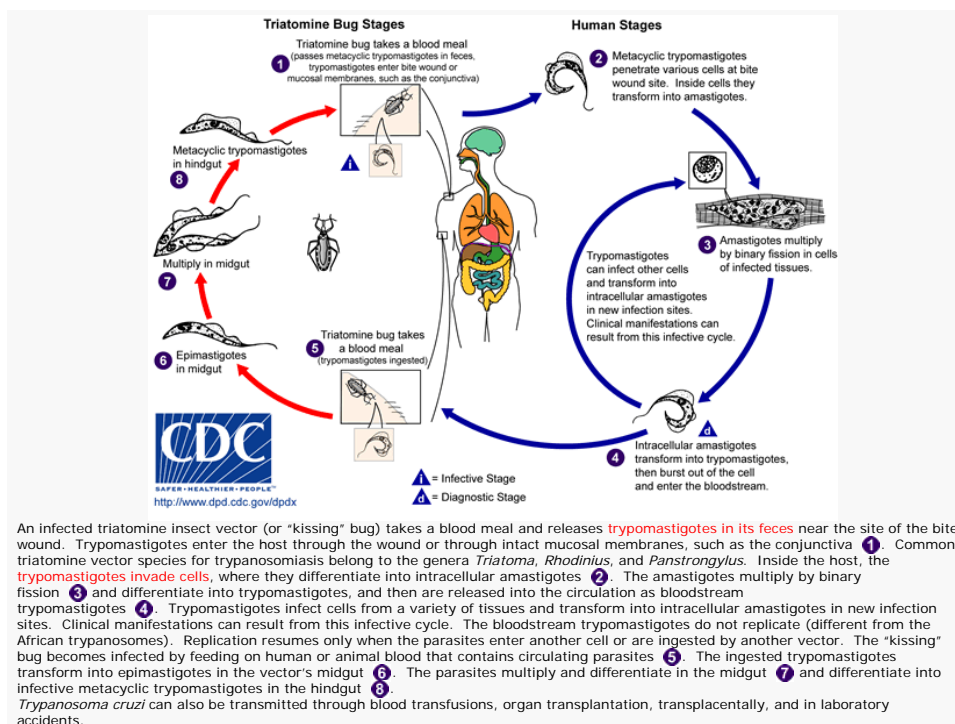
If you believe you have been bitten by this bug, you should contact your personal physician.

- Chronic Chagas disease and its complications can be fatal.
- Caused by *Trypanosoma cruzi*
- A local lesion (chagoma) can appear at the site of inoculation
- The acute phase is usually asymptomatic
fever, anorexia (*Appetitlosigkeit*), lymphadenopathy and myocarditis

Most acute cases resolve into a chronic stage (after 2-3 months)

- The symptomatic chronic stage may not occur for years or even decades after initial infection.
- The manifestations include cardiomyopathy, megaesophagus and megacolon and weight loss.





Treatment of Chagas disease

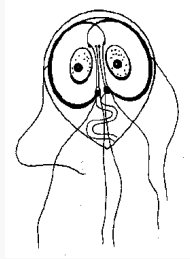
Specific cure for the different symptoms:
e.g. cardiomyopathy, megaesophagus or megacolon

Other medication depending on:
- the infected tissue

Chemotherapeutic agents:
- Anthelmintics



200 million
people
infected each
year

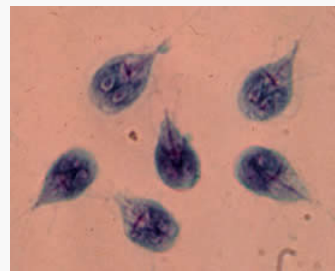


Giardia

Disease: Giardiasis

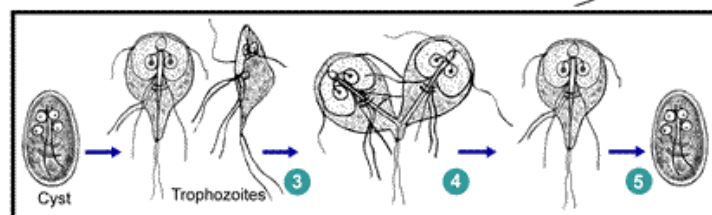


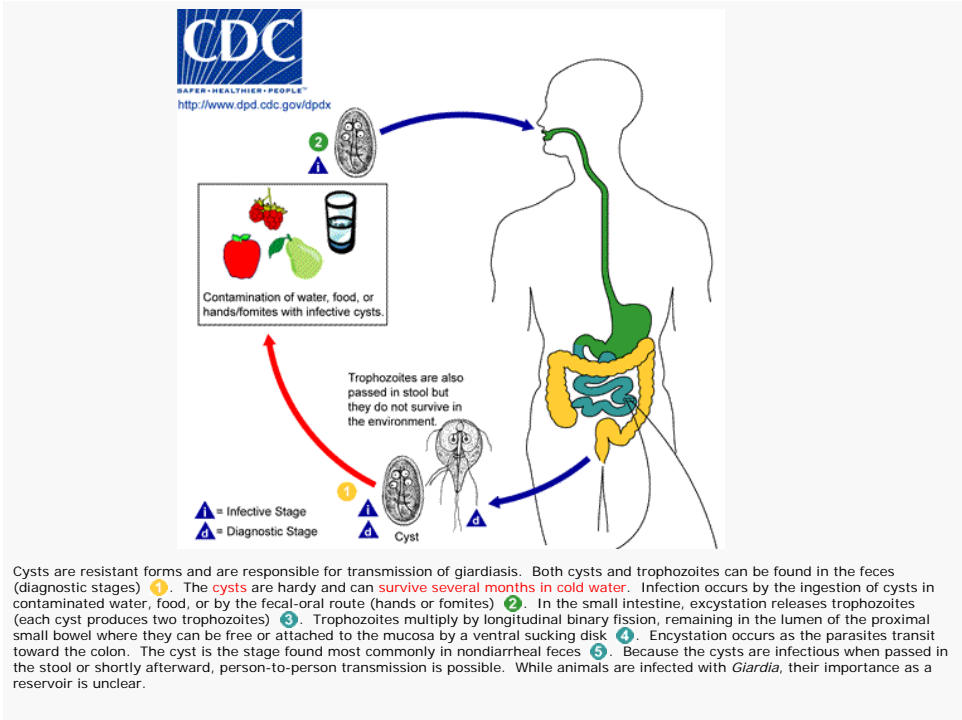
G. intestinalis



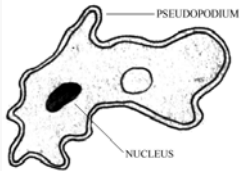
Giardiasis

- The spectrum varies from asymptomatic carriage to severe diarrhea
- Acute giardiasis develops after 1-14 days, usually lasts 1-3 weeks
- Symptoms: diarrhea, abdominal pain, bloating, nausea, vomiting.
- In chronic giardiasis the symptoms are recurrent (*wiederkehrend*) and malabsorption and debilitation (*Schwächung*) may occur.





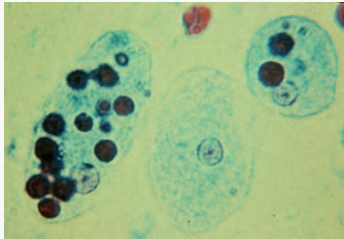
Cysts are resistant forms and are responsible for transmission of giardiasis. Both cysts and trophozoites can be found in the feces (diagnostic stages) ①. The cysts are hardy and can survive several months in cold water. Infection occurs by the ingestion of cysts in contaminated water, food, or by the fecal-oral route (hands or fomites) ②. In the small intestine, excystation releases trophozoites (each cyst produces two trophozoites) ③. Trophozoites multiply by longitudinal binary fission, remaining in the lumen of the proximal small bowel where they can be free or attached to the mucosa by a ventral sucking disk ④. Encystation occurs as the parasites transit toward the colon. The cyst is the stage found most commonly in nondiarrheal feces ⑤. Because the cysts are infectious when passed in the stool or shortly afterward, person-to-person transmission is possible. While animals are infected with *Giardia*, their importance as a reservoir is unclear.



Entamoeba histolytica

Amoeba

Disease: Amebiasis

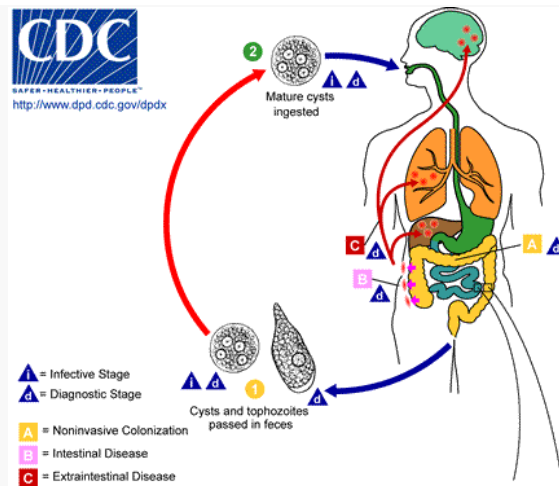
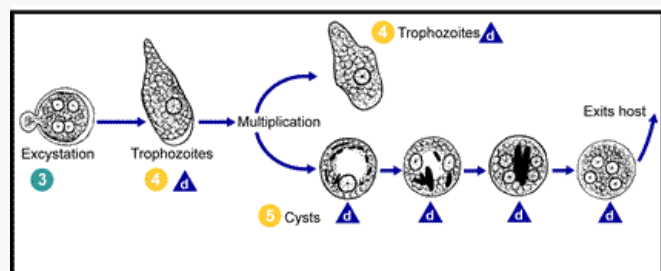


On a global basis, amebiasis affects approximately 50 million persons each year, resulting in nearly 100,000 deaths

Amebiasis

Wide spectrum of infection:

- Asymptomatic ("luminal amebiasis")
- Invasive intestinal amebiasis (dysentery, colitis, appendicitis, toxic megacolon, amebomas)
- Invasive extraintestinal amebiasis (liver abscess, peritonitis (Bauchfellentzündung), pleuropulmonary abscess, cutaneous and genital amebic lesions)



Cysts and trophozoites are passed in feces (1). Cysts are typically found in formed stool, whereas trophozoites are typically found in diarrheal stool. Infection by *Entamoeba histolytica* occurs by ingestion of mature cysts (2) in fecally contaminated food, water, or hands. Excystation (3) occurs in the small intestine and trophozoites (4) are released, which migrate to the large intestine. The trophozoites multiply by binary fission and produce cysts (5), and both stages are passed in the feces (1). Because of the protection conferred by their walls, the cysts can survive days to weeks in the external environment and are responsible for transmission. Trophozoites passed in the stool are rapidly destroyed once outside the body, and if ingested would not survive exposure to the gastric environment. In many cases, the trophozoites remain confined to the intestinal lumen (A: noninvasive infection) of individuals who are asymptomatic carriers, passing cysts in their stool. In some patients the trophozoites invade the intestinal mucosa (B: intestinal disease), or, through the bloodstream, extraintestinal sites such as the liver, brain, and lungs (C: extraintestinal disease), with resultant pathologic manifestations. It has been established that the invasive and noninvasive forms represent two separate species, respectively *E. histolytica* and *E. dispar*. These two species are morphologically indistinguishable unless *E. histolytica* is observed with ingested red blood cells (erythrophagocytosis). Transmission can also occur through exposure to fecal matter during sexual contact (in which case not only cysts, but also trophozoites could prove infective).

Treatment of Amebiasis

The choice of drug depends on:

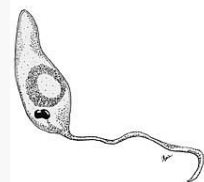
- type of clinical presentation and site of drug action, intestinal wall versus inside the intestine

Drugs e.g. metronidazole:

- antiprotozoal drug with antibacterial effects, often given orally
- may be necessary to drain a liver abscess
- includes stool studies 2 to 4 weeks after treatment



estimated 60,000 who die from the disease each year out of half-million infections worldwide



Leishmania spp.

Leishmania

Disease: Leishmaniasis



Sandfly

Cutaneous and visceral leishmaniasis

The factors determining the form of disease are:

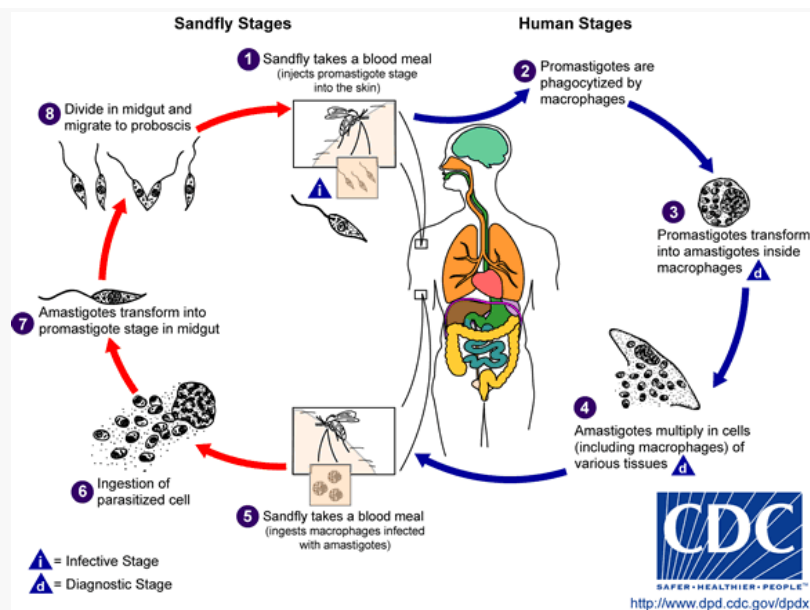
- Leishmanial species, geographic location, host immune response

Cutaneous leishmaniasis

- Characterized by cutaneous lesions where sandflies have fed
- The sores can change in size and appearance over time
- Often end up looking like a volcano, (raised edge and central crater)

Visceral leishmaniasis

- Fever, weight loss, enlarged spleen (Milz) and liver
- Important opportunistic infection with HIV



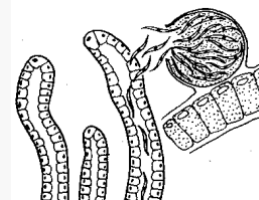
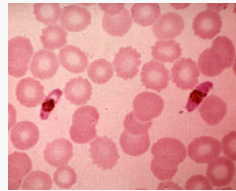
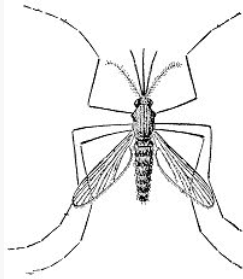
Leishmaniasis is transmitted by the bite of female phlebotomine sandflies. The sandflies inject the infective stage, promastigotes, during blood meals **1**. **Promastigotes** that reach the puncture wound are **phagocytized by macrophages** **2** and transform into amastigotes **3**. Amastigotes multiply in infected cells and **affect different tissues**, depending in part on the *Leishmania* species **4**. This originates the clinical manifestations of leishmaniasis. Sandflies become infected during blood meals on an infected host when they ingest macrophages infected with amastigotes (**5**, **6**). In the sandfly's midgut, the parasites differentiate into promastigotes **7**, which multiply and migrate to the proboscis **8**.

Treatment of Leishmaniasis

Traditionally, an antifungal agent that attacks the cell wall of the Leishmania parasite.

Because Leishmania species are temperature-sensitive, local treatment with heat or cold provides an alternative to pharmaceutical therapy in some cases

Severe leishmaniasis may require surgery!



Plasmodium

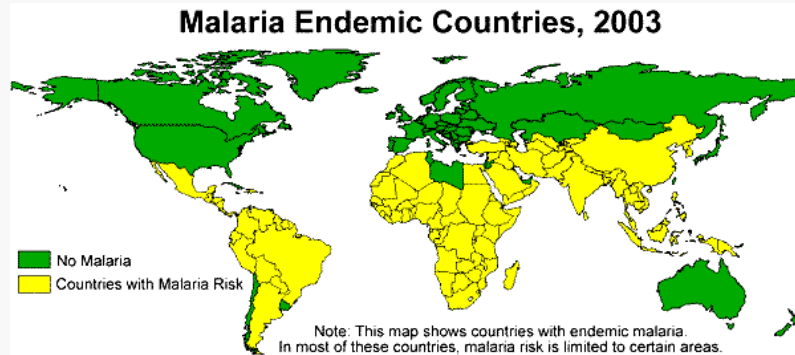
Disease: Malaria

P. falciparum
P. vivax
P. ovale
P. malariae



Anopheles spp.





Malaria kills over one million people annually around the world with over 300 million clinical cases reported yearly

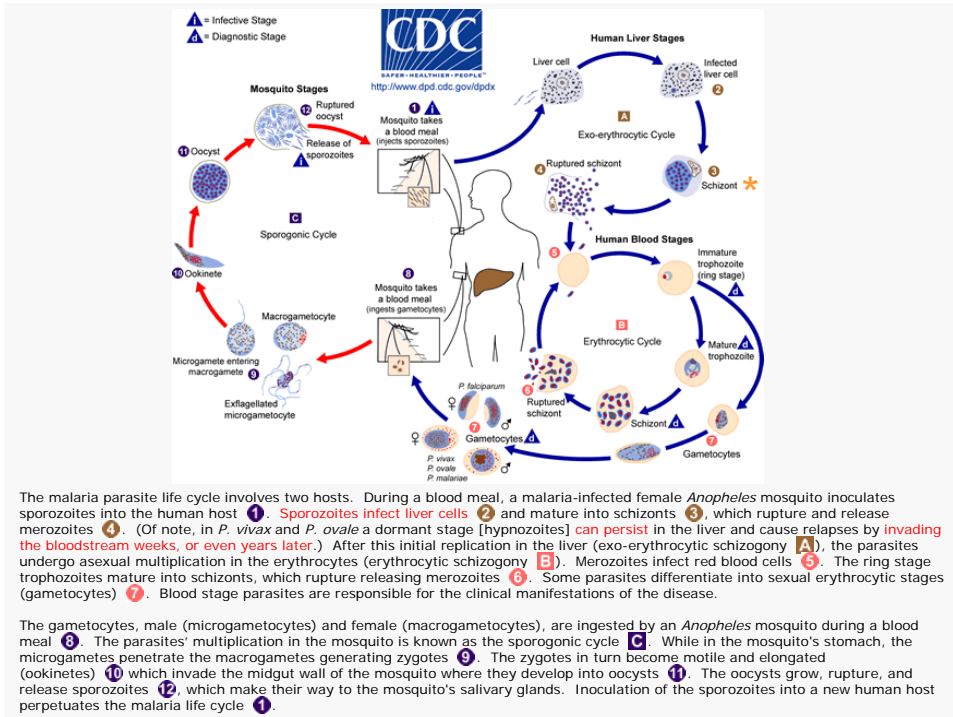
Malaria

Clinical presentation can vary substantially depending on the infecting species, the level of parasitemia, and the immune status of the patient.

The most frequent symptoms: Fever and chills, accompanied by headache, myalgias, arthralgias (*Gelenkschmerz*), weakness, vomiting, diarrhea.

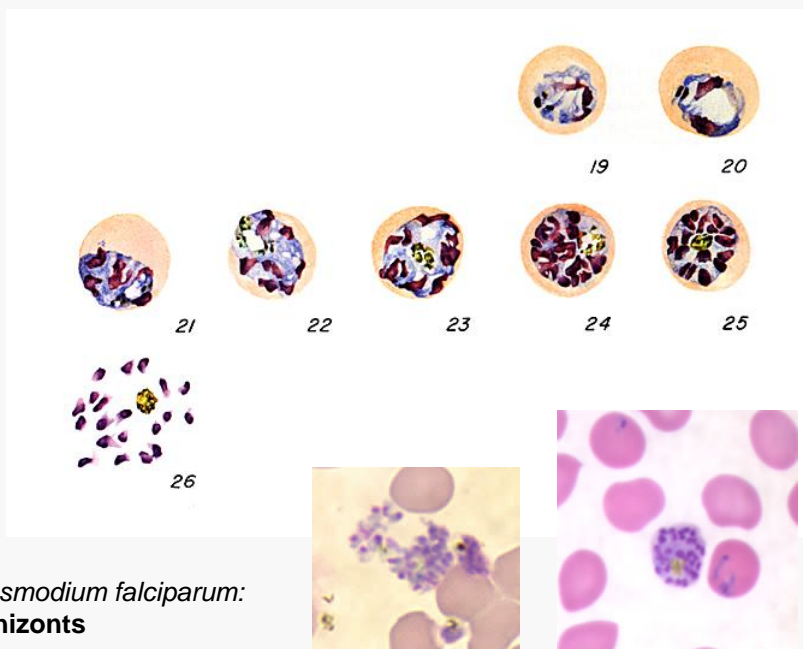
Other symptoms :

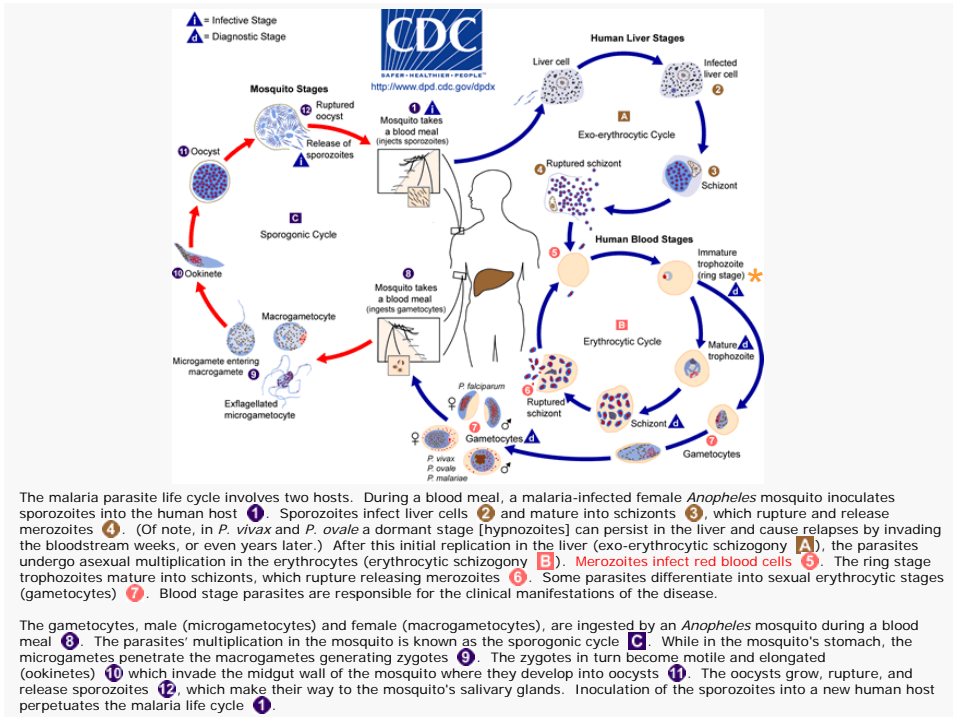
- *P. falciparum*: severe, potentially fatal forms (central nervous system involvement; cerebral malaria), acute renal failure, severe anemia, or adult respiratory distress syndrome.
- *P. vivax*: splenomegaly (with, rarely, splenic rupture (*Milzriss*))
- *P. malariae*: nephrotic syndrome



The malaria parasite life cycle involves two hosts. During a blood meal, a malaria-infected female *Anopheles* mosquito inoculates sporozoites into the human host 1. Sporozoites infect liver cells 2 and mature into schizonts 3, which rupture and release merozoites 4. (Of note, in *P. vivax* and *P. ovale* a dormant stage [hypnozoites] can persist in the liver and cause relapses by invading the bloodstream weeks, or even years later.) After this initial replication in the liver (exo-erythrocytic schizogony A), the parasites undergo asexual multiplication in the erythrocytes (erythrocytic schizogony B). Merozoites infect red blood cells 5. The ring stage trophozoites mature into schizonts, which rupture releasing merozoites 6. Some parasites differentiate into sexual erythrocytic stages (gametocytes) 7. Blood stage parasites are responsible for the clinical manifestations of the disease.

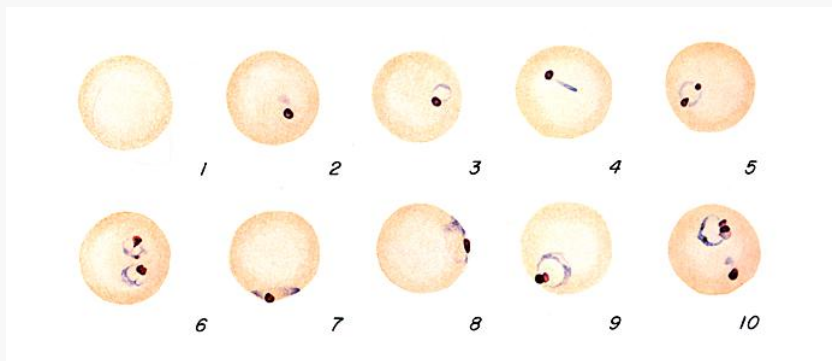
The gametocytes, male (microgametocytes) and female (macrogametocytes), are ingested by an *Anopheles* mosquito during a blood meal 8. The parasites' multiplication in the mosquito is known as the sporogonic cycle C. While in the mosquito's stomach, the microgametes penetrate the macrogametes generating zygotes 9. The zygotes in turn become motile and elongated (ookinets) 10 which invade the midgut wall of the mosquito where they develop into oocysts 11. The oocysts grow, rupture, and release sporozoites 12, which make their way to the mosquito's salivary glands. Inoculation of the sporozoites into a new human host perpetuates the malaria life cycle 1.



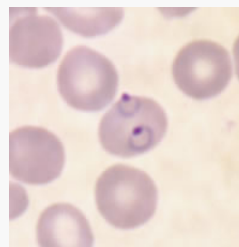


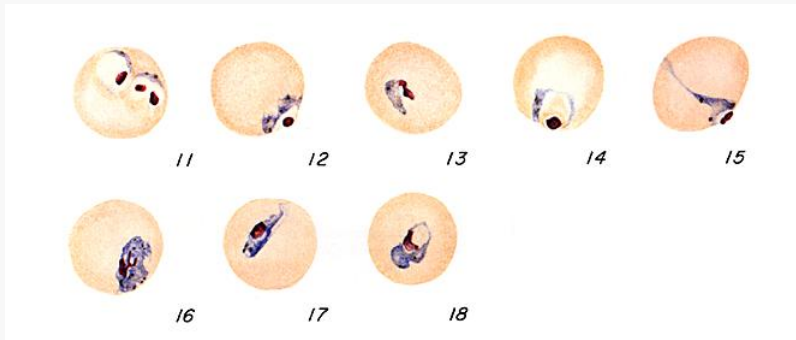
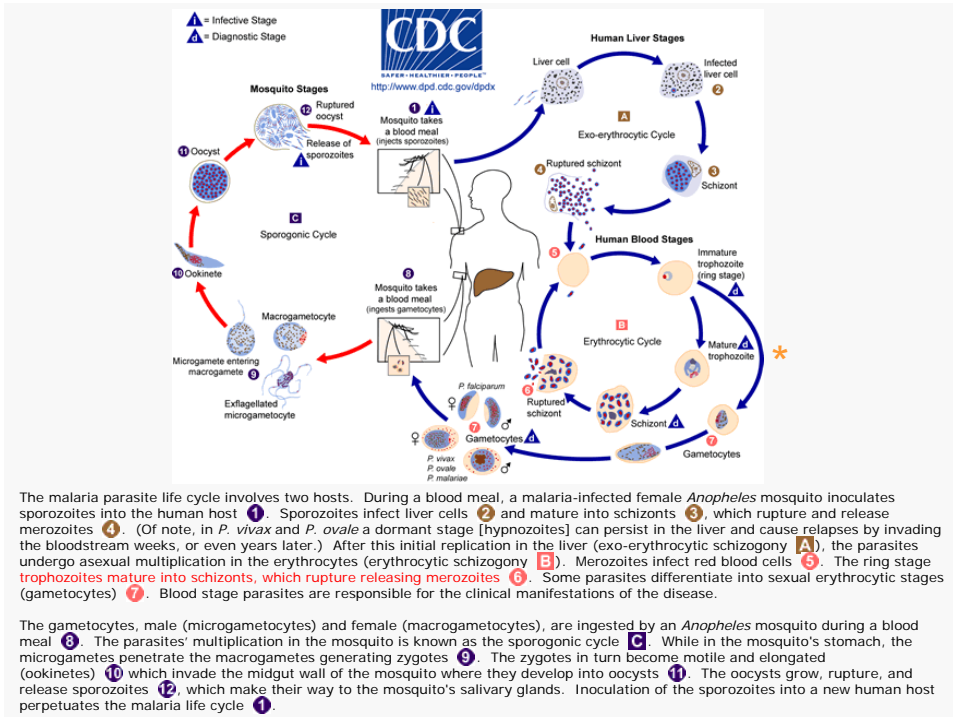
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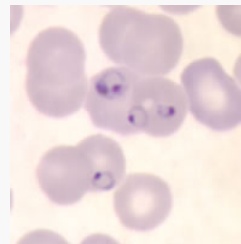


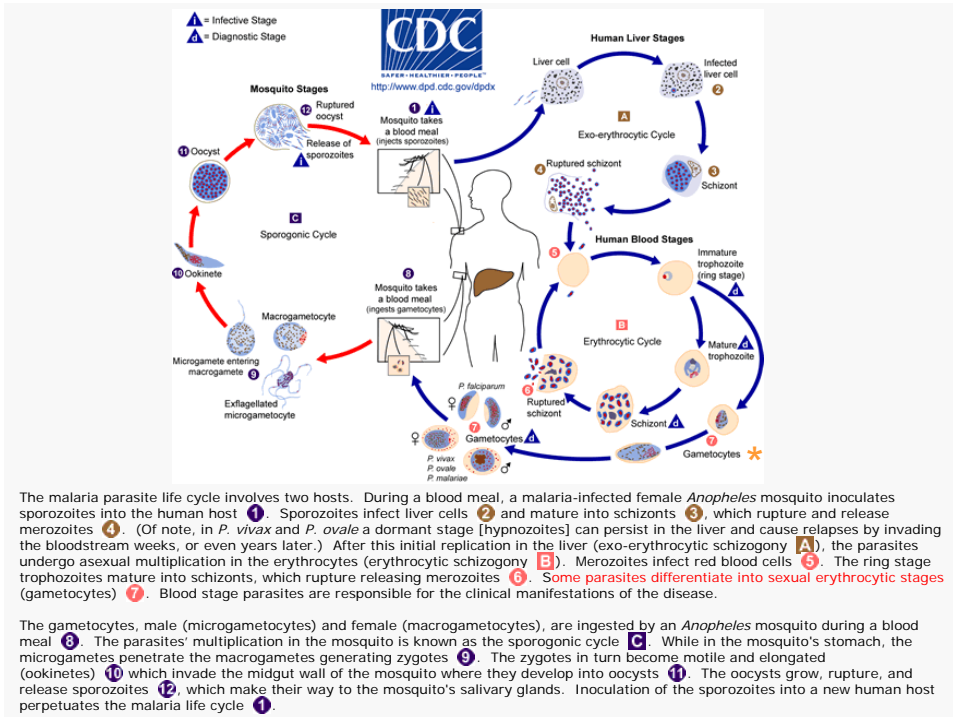
Plasmodium falciparum:
 Ring Stage Parasites





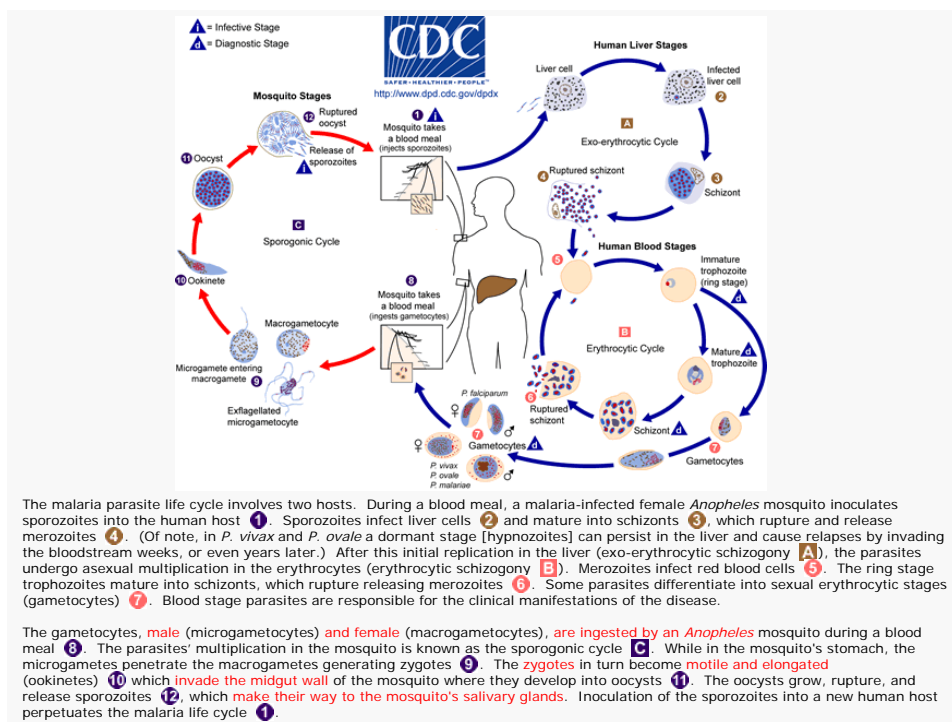
Plasmodium falciparum:
Trophozoites





Plasmodium falciparum:
Gametocytes





Malaria prophylaxis

Chloroquine phosphate (Aralen)

Effective for *P. vivax*, *P. ovale*, *P. malariae*, and drug-sensitive *P. falciparum*. Can be used for prophylaxis or treatment.

Atovaquone/proguanil (Malarone)

For both prophylaxis and treatment of chloroquine-resistant malaria. May be a good prophylactic option for patients who are visiting areas with chloroquine-resistant malaria

Mefloquine (Lariam)

Acts as a blood schizonticide. May act by raising intravesicular pH within parasite acid vesicles. Structurally similar to quinine. For prophylaxis treatment of drug-resistant malaria.

Gin Tonic



Don't strip tease
for Anopheles



The anopheles mosquito
is a carrier of

MALARIA

KEEP COVERED



World War II Poster
(1941-1945)