Parasites are organisms that live in or on another organism and benefits by deriving nutrients at the host's expense. In this case, humans are the definitive host.

Plasmodium Species
Morphology

This family of protozoan parasites are responsible for causing malaria, which is the number one cause of human death of all parasitic species.

There are four species in the plasmodium family: *P. falciparum, p.vivax, p.malarie, and p. ovale*. Humans are the only reservoir. The parasite is transmitted by the female anophele mosquito.

Epidemiology

Endemic to Mexico, South America, Central America, Africa, and Southeast Asia. Responsible for 2 billion infections/year and 3 million death/year.

Plasmodium life cycle

- Sporozoites are transferred from mosquito’s salivary glands to human via bite
- Sporozoites enter the human liver cell within thirty minutes, becoming cryptozoites
- Cryptozoites multiply in the liver into merozoites
- Merozoites infect human red blood cells and asexually reproduce into male and female gametophytes
- The gametophytes are taken up by the mosquito when it takes a meal
- The gametophytes reproduce sexually into the mosquito gut into a zygote and mature into sporozoites
Diagnosis

Blood smears will show merozoites and sporozoites inside the red blood cells.

Clinical Picture

Patients present with fever, anemia and episodes of shaking chills (related to red blood
cell lysis). Prodromes occur about every forty-eight to seventy-two hour intervals. Hepatomegaly, splenomegaly, jaundice, and cerebral malaria (p. falciparum) can also occur.

Treatment

Prevention and vector control are important to protect humans from mosquito bites

- Cover exposed skin in endemic areas
- Use insect repellent, like DEET
- Sleep with mosquito nets

Pharmacology

- Chloroquine is the drug of choice for vivax, p.malarie, and p. ovale
- falciparum has developed resistance to chloroquine. This drug cannot be used in Africa or Southeast Asia where p. falciparum is prevalent.
- Quinine, artemether, atavaquone-proguanil, mefloquine, and pyrimethamin/sulfadoxine can be used as a treatment of prophylaxis in endemic regions for all four species of plasmodium.
- Plasmodium vaccines are currently in development

Toxoplasma Gondii

Image: “Diagram of Toxoplasma gondii” by GrahamColm . License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)
Morphology

Single-celled protozoan that is present in undercooked pork and cat feces. Cats are the definitive hosts and acquire the parasite via consumption of infected animals.

Toxoplasma Life Cycle

- Human consumption of *Toxoplasma* cysts via consumption of undercooked meat or cat feces contamination
- Cysts open in the human duodenum, enter the bloodstream, and release sporozoites
- Sporozoites will infect human macrophages
- Sporozoites can form dormant cysts stay with humans for life once immune system is activated.

Diagnosis

Microscopic cysts containing *Toxoplasma gondii* develop in the tissues of many vertebrates. Here, in mouse brain tissue, thousands of resting parasites (stained red) are enveloped by a thin parasite cyst wall.

Blood test can detect antibodies to *Toxoplasma* to determine previous exposure. Tissue
biopsies can determine if a patient has an active infection.

Clinical Picture

The majority of patients (>99%) with toxoplasmosa are asymptomatic carriers. Some patients will present with fatigue and malaise for and will spontaneously resolve in one month.

Pregnant women, especially in the first trimester are advised to stay away from cats because of risk of congenital toxoplasmosis for their child.

- Parasite can cross the placenta and cause stillbirth, chorioretinitis, intracerebral calcifications, psychomotor disturbances, and hydrocephaly or microcephaly
- Manifestations of this disease can occur after childbirth
- Symptoms not as severe if occurring in the second or third trimester
- Immunocompromised patients can suffer from toxoplasmova encephalitis, pneumonia, or chorioretinitis

Treatment

People can prevent acquiring the parasite by avoiding eating meat that is cooked through and avoiding changing the litter box of cats. Pregnant women are advised to avoid cat feces during pregnancy.

Pharmacological treatment includes pyrimethamine and sulfadiazine.

Leishmania Species

Morphology

Under the acellular culture condition, the protozoa transforms into the form of promastigote, a flagellated and elongated morphology seen in the mid-gut of the vector.

Single-celled protozoan parasite. There are four species (*L. donovani*, *L. tropica*, *L. mexicana*, *L braziliensis*) that can cause visceral leshmaniasis, cutaneous leshmaniasis, and muco-cutaneous leishmaniasis.

There are 1.3 million new cases a year with twenty to thirty thousand deaths annually from this parasite.
Parasites are found in Central America, South America, Africa, Middle East, and India.

Zoonotic parasites that are found in dogs, rodents, and foxes. Transmission vector is via the phlebotomous sandfly.

**Leshmaniasis life cycle**

- Sandfly acquires amastigote from zoonotic host
- In the sandfly gut, the amastigote develop into a flagellated promastigote and replicate
- When the sandfly bites a human, the promastigotes exit via the salivary gland into the skin.
- The promastigotes are taken up by macrophages and multiply causing macrophage rupture
- The contents of the macrophage leak into the skin damaging tissue, causing a lesion

Life cycle of the parasites from the genus Leishmania

**Diagnosis**
Leishmaniasis can be diagnosed several ways:

- PCR of a blood sample can identify *Leishmania* DNA
- A histological slide of a biopsy will show the parasite
- ELISA test with blood will show *Leishmania* antibodies confirming infection

**Clinical Picture**

1. Leishmaniasis ulcer on left forearm

*mexicana* and *L. tropicana* cause cutaneous leishmaniasis. When the sandfly bites a human, the parasite infects local macrophages. This will lead to macrophage lysis at the site of the bite causing a dermal lesion and ulceration.

2. *braziliensis* cause mucocutaneous leishmaniasis. In this case, once the sandfly bites a human, the infected macrophages travel to the nasopharynx or the gentials, where the parasite can lay dormant for years. Eventually macrophage destruction will occur and there will be lesions and ulcerations in the infected area.

3. *donovani* causes visceral leishmaniasis, also known as kala-azar. The parasite will
spread from the site of inoculation to the liver and spleen. Here, the parasite will lay dormant for months and eventually multiply resulting in hepatosplanomegaly and anemia. There is also an associated darkening of the skin in the infected region. *L. donovani* is able to cross the placenta and cause a congenital leishmaniasis. In twenty percent of patients, there as an associated dermal inflammation that persists after the disease subsides. Patients will have soft nodules on their face that are asymptomatic and will spontaneously resolve.

**Treatment**

Prevention of leishmaniasis can occur with use of bed nets, long clothing and destruction of the habitat of the sandfly.

Amphotericin B and sodium stibogluconate are effective treatments against leishmaniasis.

**Trypanosoma Species**

**Morphology**

Flagellated, single-celled protozoan parasite. There are three species that have human pathology (*t. brucei gambiense*, *t. brucei rhodesiense*, and *t. cruzi*).

Vector is the tse tse fly in the African species (*t. gambiense* and *t. rhodesiense*). In the American species, the vector is the reduviid bug (*t. cruzi*).

Reservoirs in *t. rhodesiense* are game animals while *t. gambiense* is humans and domesticated animals. There is no animal reservoir in the American species.

**Trypanosoma Life cycle**

The African and American species of the parasite have different life cycles.

In the African Trypanosome:

- The tse tse fly with the *trypanosoma* parasite bites the human and injects the parasite into the bloodstream
- A chancre forms at the site of the bite while the trypomastigotes infiltrate the bloodstream
- The parasite makes its way to the lymph and nervous tissues over the course of months to years
- The body cannot clear the parasite due to antigenic variation
- The tse tse fly will bite an infected human and take up the parasite into its gut with blood
Life cycle of the Trypanosoma brucei parasites

In the American Trypanosome:

- The reduviid bug will bite a human. As they take blood in, the bug will defecate and the parasite is located within the feces.
- As humans scratch after being bitten, the parasite will enter the skin and proceed to the bloodstream.
- The final destination of the parasite will be cardiac muscle and gastrointestinal nerves, where the parasite can lay dormant for years.

Diagnosis

<table>
<thead>
<tr>
<th>The African trypanosomes can be identified histologically via a blood smear or on a CSF sample.</th>
<th>American trypanosomes are identified by blood smear. Another diagnosis method consists of free reduviid bugs are allowed to bite a potential carrier of trypanosomes. One month later, the bugs are analyzed to see if they are infected with the parasite.</th>
</tr>
</thead>
</table>
Clinical Picture

In the African species, patients will first develop a chancre at the site of the infection. As the parasite enters the lymphatic and nervous tissue over the course of months to years, patients will develop intermittent fever and headaches. Eventually the patients develop anorexia, neurological symptoms and apathy. Over the course of the disease, the neurological symptoms worsen and the patient has sleep disturbances. Patients will decompensate further resulting in convulsions, coma, and death.

Patients with American trypanosomes, the first clinical sign of infection is a chagoma, which is an inflammatory nodule present at the site of the bite of the reduviid bug. If the bite is near the eye, unilateral swelling of the eye can occur. This sign is also known as Romana’s sign. Primary infection is characterized by fever and lymphadenitis. These symptoms usually resolve within one month.

Patients with chronic Chagas disease, the parasite will enter cardiac muscle and can create arrhythmias and a dilated cardiomyopathy. The parasite can also enter gastric nervous tissue, destroy nerve plexuses and create a megacolon and megaesophagus.

Treatment

Limiting contact with the tse tse fly and the reduviid bug vector by wearing long clothing and using bug nests while sleeping, preventing inoculation.

Pharmocologically, efloirthine, pentamidine, and suramin can be given to treat the African parasite, however, once neurological symptoms are present, then the drugs lose their effectiveness.

Regarding the American trypanosome, benznidazole and nifurtimox can be used for treatment.

Helminths

Morphology

Multi-celled organisms that can reproduce sexually or are hermaphroditic. They have the ability to develop into dormant cysts. Helminths can be transmitted via fecal-oral, fecal-skin, or ingestion. Disease burden is directly related to the amount of worms that are in the host.

Ascaris Lumbricoides
An adult Ascaris lumbricoides worm. Diagnostic characteristics: tapered ends; length 15 to 35 cm (the females tend to be the larger ones).

This is a roundworm that lives in the human intestinal tract. It can reach a length of thirty centimeters and up to twenty-five percent of the world’s population are infected with this helminth.

The lifecycle of this worm begins by human ingestion of eggs through contaminated food. The eggs will hatch in the intestine and make their way into the bloodstream and into the alveoli of the lungs. The worms will become lodged in these air sacs. They pierce the alveoli and migrate up the bronchus into the trachea and enter the GI system where they mature and lay eggs that leave the body through feces.

Infection of these helminthes cause malabsorption of nutrients for their host. They also have the potential to obstruct the biliary tract and cause peritonitis.

Diagnosis involves analysis of the patients stool for worms and eggs.

Treatment consists of an antihelmithic drug: mebenazole, albendazole, ivermectin, or nitazonide.

Patients should also avoid eating food contaminated with feces.
**Necator americanus**

This helminth is a hookworm, which exist in human feces. The hookworm larvae live in the soil and can infect humans when they step on infected soil without shoes. These helminthes will then enter the bloodstream and travel directly to the alveoli, where they can escape and enter the GI tract via the bronchus and trachea.
Once in the intestinal system, the helminths will attach to the lumen of the GI tract and feed off the human blood. These organisms will lay eggs that will leave the human host via their feces.

Patients will present with iron deficiency anemia and failure to thrive. Diagnosis involves inspection of human feces for larvae and eggs. Treatment is with mebendazole.

*Image: “Necator americanus Life Cycle” by SuSanA Secretariat. License: CC BY 2.0*

**Trichinella Spiralis**
Is a nematode that is present in undercooked pork and other wild types of meat. The larvae are consumed and enter the body and mature in the intestines where helminths can enter muscles cells. At this point, the parasites become encapsulated in the nurse cells in the muscle.

Patients present with diarrhea, abdominal pain, muscle pain, and headache

Treatment is with albendazole or mebendazole. However, once the helminth enters muscle cells, pharmacological treatment will not work.

**Schistosoma**

These are a family of helminths that include four species (*S. manconii*, *S. japonicum*, *S. haematobium*, and *S. mekongi*). Globally, there are between two and three hundred infections. The snail is an intermediate host to this trematode.

*Schistosoma* larvae enter the snail where they mature into cercaria. Once mature, they will penetrate human flesh and enter the blood stream and infect the liver. The shistosoma organisms will mate in the liver and the female will lay eggs that leave the body via
feces.

Humans infected with schistosomes, suffer from portal hypertension because the eggs laid by the female will impede the blood flow to the liver. The eggs also result in granuloma formation as a result of the body mounting an immune response. Esophageal varices can form as a result of the portal hypertension.

Treatment is with praziquantel which will prevent egg production by the females.

References


Legal Note: Unless otherwise stated, all rights reserved by Lecturio GmbH. For further legal regulations see our legal information page.