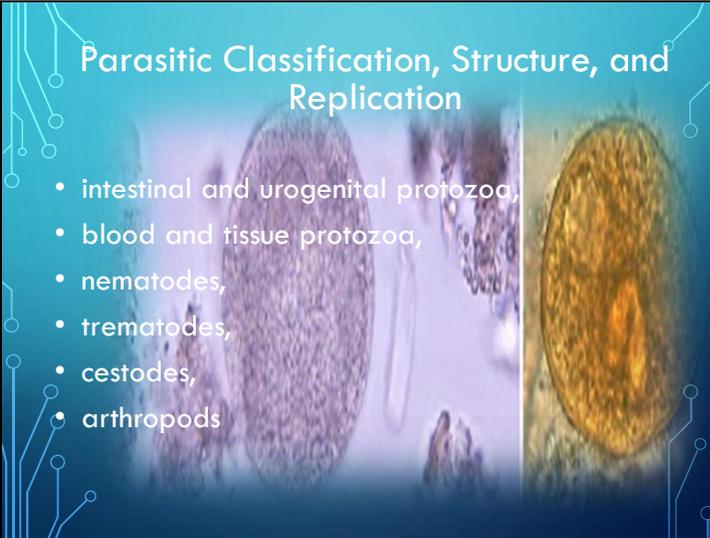
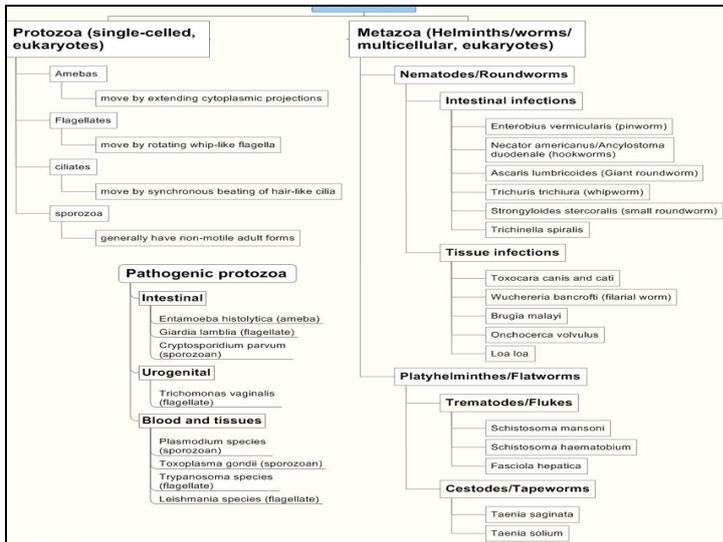


PARASITES

Parasitic Classification, Structure, and Replication

- intestinal and urogenital protozoa,
- blood and tissue protozoa,
- nematodes,
- trematodes,
- cestodes,
- arthropods

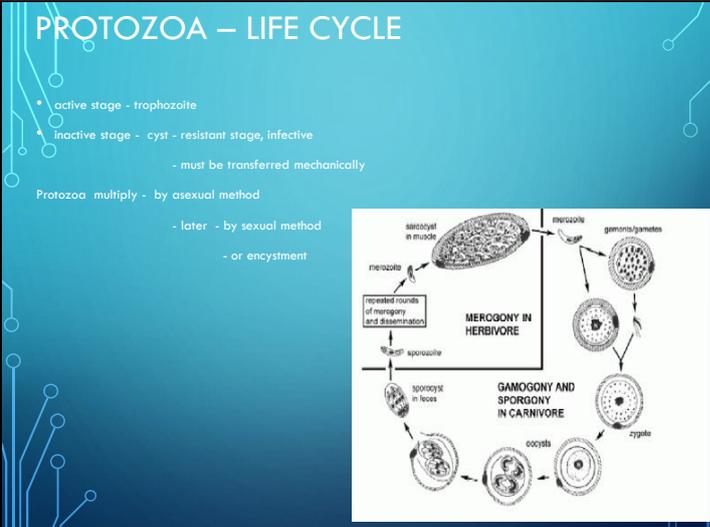



PROTOZOA – LIFE CYCLE

- active stage - trophozoite
- inactive stage - cyst - resistant stage, infective
 - must be transferred mechanically

Protozoa multiply - by asexual method

- later - by sexual method
- or encystment



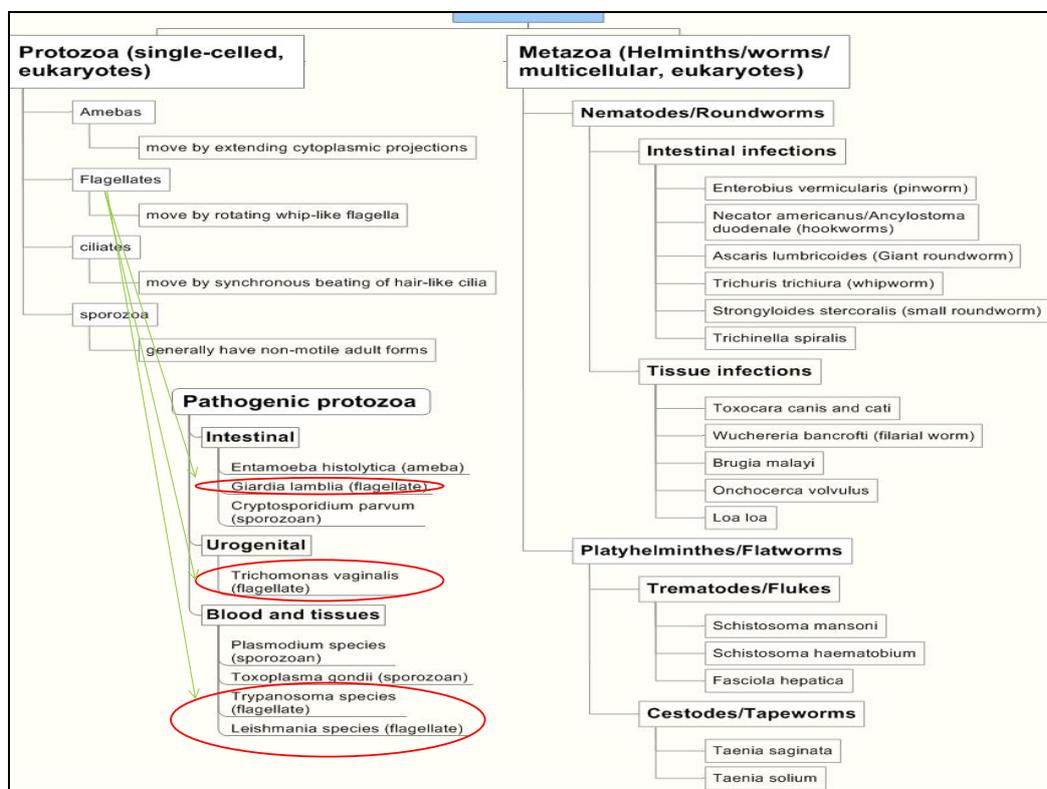
The diagram illustrates the life cycle of a protozoan. It starts with a trophozoite in muscle tissue. This leads to merogony in herbivores, where the parasite forms multiple rounded merogony and disseminates. Sporozoites are then released. In carnivores, the cycle involves gamogony and sporangy, leading to the formation of oocysts and zygotes. The cycle then returns to muscle tissue via feces.

The protozoal parasite possesses the property of being transformed from an active (trophozoite) to an inactive stage, losing its power of motility and enclosing itself within a tough wall. The protoplasmic body thus formed is known as a cyst. At this stage, the parasite loses its power of growth and multiplication.

The cyst is the resistant stage of the parasite and is also infective to its human host. In order to reach a new host, it must be transferred mechanically, either by a carrier or by some intermediaries (insect-house-flies), to food and drink which become contaminated with the cysts of protozoa.

A protozoal parasite may multiply vigorously by asexual method for a long time, and later by a change of process it either has recourse to sexual method of reproduction or undergoes encystment for a change of its host.

The sexual method of reproduction often occurs in a different host other than the one utilized for asexual multiplication; the process is known as alternation of generation accompanied by alternation of host, as seen in Plasmodia.



GIARDIA

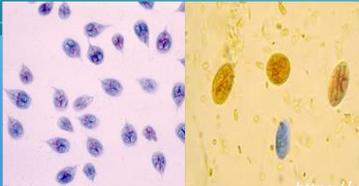
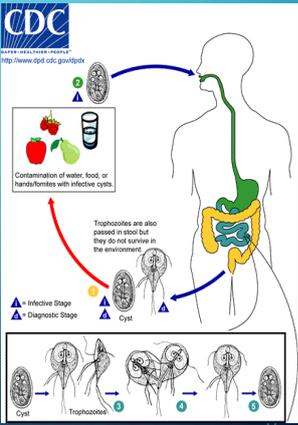
- intestinal flagellates
- *Giardia intestinalis*

cysts - can survive several months in cold water.

- infectious

Trophozoites—multiply - in the lumen of the proximal small bowel

person-to-person transmission is possible.

<https://www.cdc.gov/parasites/giardia/pathogen.html>

Cysts are resistant forms and are responsible for transmission of giardiasis. Both cysts and trophozoites can be found in the feces (diagnostic stages)

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)

In the small intestine, excystation releases trophozoites (each cyst produces two trophozoites)

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.

Encystation occurs as the parasites transit toward the colon. The cyst is the stage found most commonly in nondiarrheal feces.

Because the cysts are infectious when passed in the stool or shortly afterward, person-to-person transmission is possible.

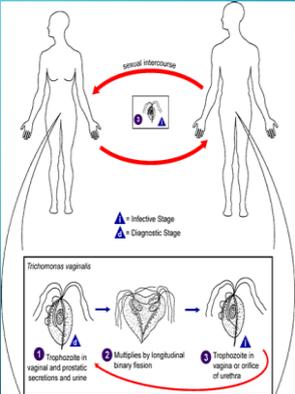
While animals are infected with *Giardia*, their importance as a reservoir is unclear.

TRICHOMONAS VAGINALIS

- genitourinary flagellates
- Incubation Period : 5 to 28 days
- No known cystic form
- May survive in a host for 2+ years

Morphology

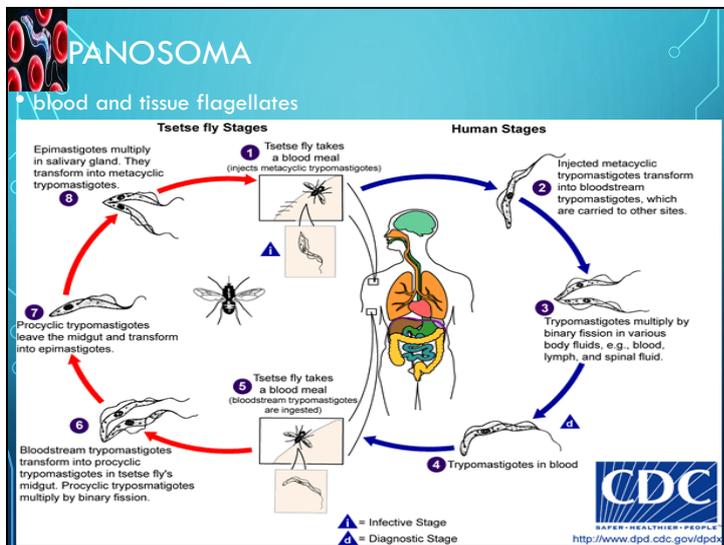
- *T. vaginalis* : pear-shaped protozoan
- Four flagella – movement, fifth - help with direction
- High motility - pathogenicity
- Reproduces through binary fission

<https://www.cdc.gov/dpdx/trichomoniasis/index.htm>

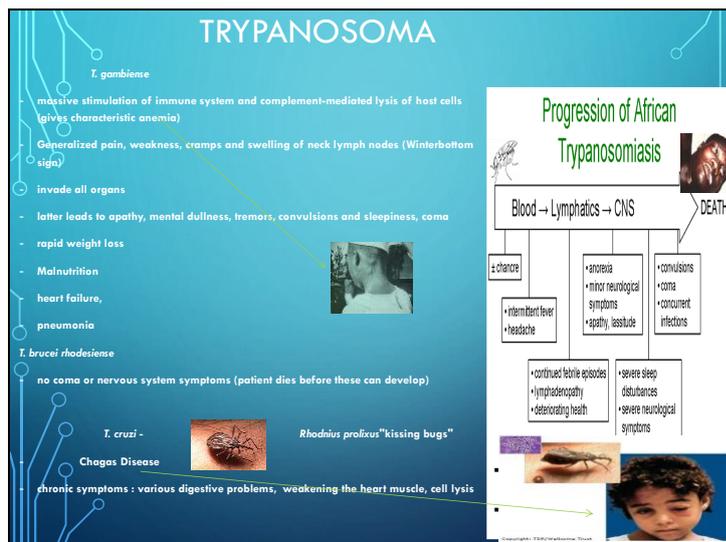
Trichomonas vaginalis resides in the female lower genital tract and the male urethra and prostate, where it replicates by binary fission. The parasite does not appear to have a cyst form, and does not survive well in the external environment. *Trichomonas vaginalis* is transmitted among humans, its only known host, primarily by sexual intercourse.

Trichomonas vaginalis infection in women is frequently symptomatic. Vaginitis with a purulent discharge is the prominent symptom, and can be accompanied by vulvar and cervical lesions, abdominal pain, dysuria and dyspareunia. The incubation period is 5 to 28 days. In men, the infection is frequently asymptomatic; occasionally, urethritis, epididymitis, and prostatitis can occur.



“African trypanosomes” or “Old World trypanosomes” are protozoan hemoflagellates of the genus *Trypanosoma*, in the subgenus *Trypanozoon*. Two subspecies that are morphologically indistinguishable cause distinct disease patterns in humans: *T. brucei gambiense*, causing chronic African trypanosomiasis (“West African sleeping sickness”) and *T. brucei rhodesiense*, causing acute African trypanosomiasis (“East African sleeping sickness”).

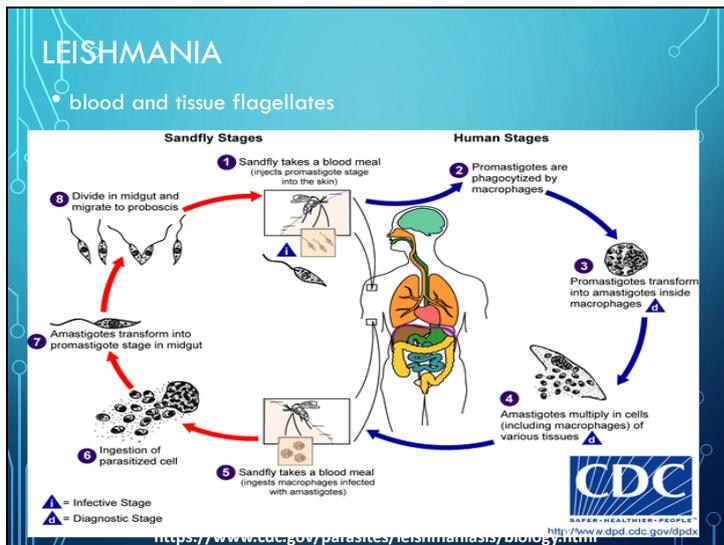
During a blood meal on the mammalian host, an infected tsetse fly (genus *Glossina*) injects metacyclic trypomastigotes into skin tissue. The parasites enter the lymphatic system and pass into the bloodstream. Inside the host, they transform into bloodstream trypomastigotes, are carried to other sites throughout the body, reach other blood fluids (e.g., lymph, spinal fluid), and continue the replication by binary fission. 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 (,). In the fly’s midgut, the parasites transform into procytic trypomastigotes, multiply by binary fission, leave the midgut, and transform into epimastigotes. The epimastigotes reach the fly’s salivary glands and continue multiplication by binary fission. 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 animals are the main reservoir of *T. brucei rhodesiense*.



Infection occurs in two stages, an initial haemolymphatic stage followed by a meningoencephalitic stage after the trypanosomes invade the central nervous system (CNS). However, many of the signs and symptoms are common to both stages, making it difficult to distinguish between the two stages by clinical features alone.

First-stage symptoms may be preceded by the development of a trypanosomal chancre at the site of inoculation within two days to two weeks of being bitten by an infected fly (occurs most commonly with *T. b. rhodesiense*, rarely with *T. b. gambiense* although chancres are observed with *T. b. gambiense* in travelers from non-endemic countries). The first stage involves nonspecific, generalized symptoms occurring 1–3 weeks after the tsetse fly bite with *T. b. rhodesiense*; the incubation period for *T. b. gambiense* is less well characterized but disease progresses more slowly than that caused by *T. b. rhodesiense*. First-stage symptoms for both types of sleeping sickness include headache, malaise, weakness, fatigue, pruritis, and arthralgia. First-stage signs can include hepatosplenomegaly, weight loss and intermittent fevers lasting one day to one week. The intervals between fevers can last days or months. Lymphadenopathy, mainly posterior cervical but in some cases axillary, inguinal or epitrochlear, may also occur. Posterior triangle cervical lymphadenopathy, or “Winterbottom’s sign” is commonly seen in *T. b. gambiense* infections.

T. b. gambiense infection progresses to the second stage after an average of 300–500 days, whereas *T. b. rhodesiense* infection progresses to the second stage after an estimated 21–60 days. In second-stage disease, invasion of the central nervous system causes a variety of neuropsychiatric manifestations to appear in addition to the first-stage signs and symptoms, with fever occurring less frequently over time. The sleep/wake cycle becomes reversed, hence the common name “African sleeping sickness”, with daytime somnolence, nocturnal insomnia, and sudden urges to sleep. Compared to *T. b. gambiense*, *T. b. rhodesiense* is more likely to result in endocrine abnormalities such as adrenal insufficiency, thyroid dysfunction and hypogonadism; and cardiac involvement, such as myocarditis, is more severe.



Leishmaniasis is transmitted by the bite of infected female phlebotomine sandflies. The sandflies inject the infective stage (i.e., promastigotes) from their proboscis during blood meals. Promastigotes that reach the puncture wound are phagocytized by macrophages and other types of mononuclear phagocytic cells. Promastigotes transform in these cells into the tissue stage of the parasite (i.e., amastigotes), which multiply by simple division and proceed to infect other mononuclear phagocytic cells. Parasite, host, and other factors affect whether the infection becomes symptomatic and whether cutaneous or visceral leishmaniasis results. Sandflies become infected by ingesting infected cells during blood meals. In sandflies, amastigotes transform into promastigotes, develop in the gut and migrate to the proboscis.

LEISHMANIA

- Human infection is caused by about 21 of 30 species that infect mammals.
- ***L. donovani* complex** :
(*L. donovani*, *L. infantum* [*L. chagasi*]);
- ***L. mexicana* complex**
- (*L. mexicana*, *L. amazonensis*, and *L. venezuelensis*);
- morphologically indistinguishable
- 2 forms
- **cutaneous leishmaniasis**
- one or more sores on their skin (volcano)
- painless or painful
- **visceral leishmaniasis (kala-azar)**
- fever, weight loss, and an enlarged spleen and liver (spleen is bigger than the liver).
- anemia, low white blood cell count, and low platelet count
- important opportunistic infection - HIV.

<https://www.cdc.gov/parasites/leishmaniasis/biology.html>

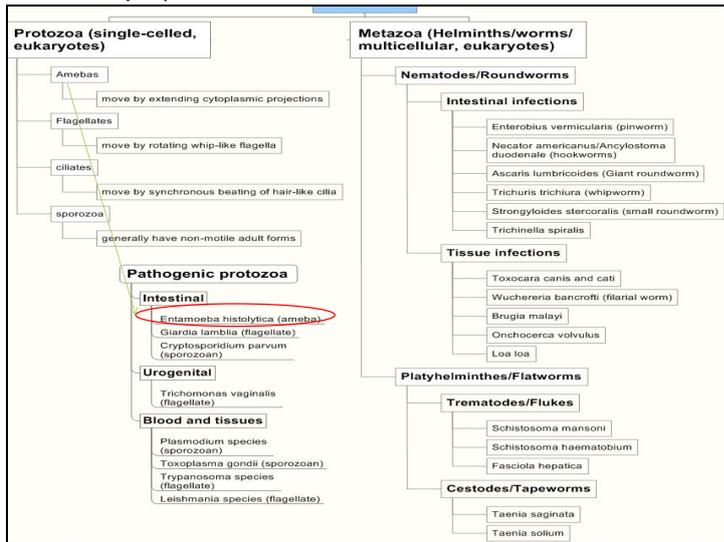
Leishmaniasis is a vectorborne disease that is transmitted by sand flies and caused by obligate intracellular protozoa of the genus *Leishmania*. Human infection is caused by more than 20 species. These include:

- *L. donovani* complex with 2 species (*L. donovani*, *L. infantum* [also known as *L. chagasi* in the New World]);
- *L. mexicana* complex with 3 main species (*L. mexicana*, *L. amazonensis*, and *L. venezuelensis*); *L. tropica*; *L. major*; *L. aethiopia*;

- subgenus *Viannia* with 4 main species (*L. [V.] braziliensis*, *L. [V.] guyanensis*, *L. [V.] panamensis*, and *L. [V.] peruviana*).

The most common form is **cutaneous leishmaniasis**, which causes skin sores. The sores typically develop within a few weeks or months of the sand fly bite.

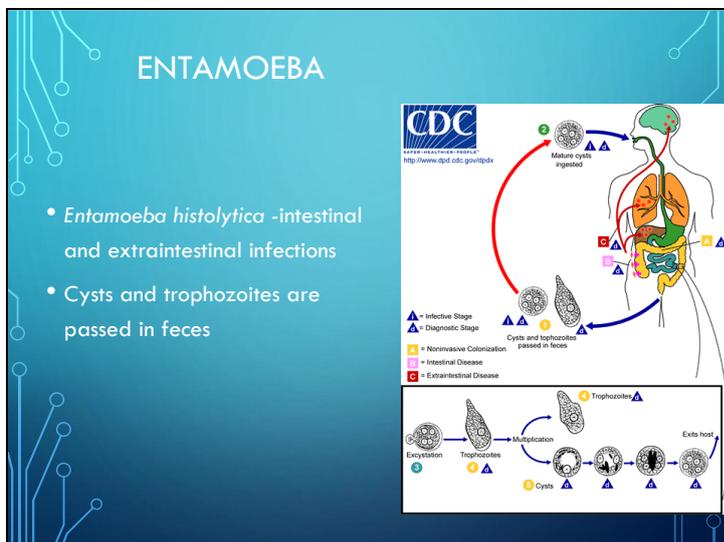
The other main form is **visceral leishmaniasis**, which affects several internal organs (usually spleen, liver, and bone marrow) and can be life threatening. The illness typically develops within months (sometimes as long as years) of the sand fly bite. Affected people usually have fever, weight loss, enlargement (swelling) of the spleen and liver, and low blood –anemia, leukopenia, and thrombocytopenia.



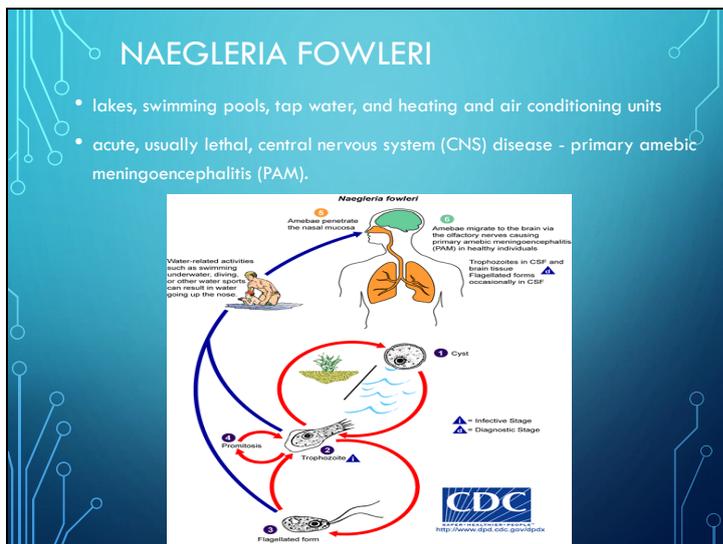
One intestinal ameba is a clear human pathogen, *E. histolytica*, and must be differentiated from 3 other nonpathogenic species:

E. dispar, *E. hartmanni*, and *E. coli*.

Amebas are shapeless mass of moving cytoplasm - divided in to granular endoplasm and clear ectoplasm. They move by pushing out the ectoplasm to form pseudopodia (false feet) into which the endoplasm then flow. Amoebae reproduce asexually by simply dividing into two (binary fission) Two stages exist for most amoebae: actively replicating trophozoite and dormant, stable cyst. The cyst stage is infectious. Detection and identification of most amoebae is by recognition of the cyst or trophozoite forms in stool specimens. The exception is *E. histolytica* where antigen detection tests have also been developed (most commonly enzyme immunoassays, EIAs).



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 in fecally contaminated food, water, or hands. Excystation occurs in the small intestine and trophozoites are released, which migrate to the large intestine. The trophozoites multiply by binary fission and produce cysts, and both stages are passed in the feces. 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 (: noninvasive infection) of individuals who are asymptomatic carriers, passing cysts in their stool. In some patients the trophozoites invade the intestinal mucosa (: intestinal disease), or, through the bloodstream, extraintestinal sites such as the liver, brain, and lungs (: 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).



Naegleria fowleri (commonly referred to as the “brain-eating amoeba” or “brain-eating ameba”), is a free-living microscopic amoeba, (single-celled living organism). It can cause a rare and devastating infection of the brain called primary amebic meningoencephalitis (PAM).

Naegleria fowleri has three stages in its life cycle: cysts, trophozoites, and **flagellated forms**.

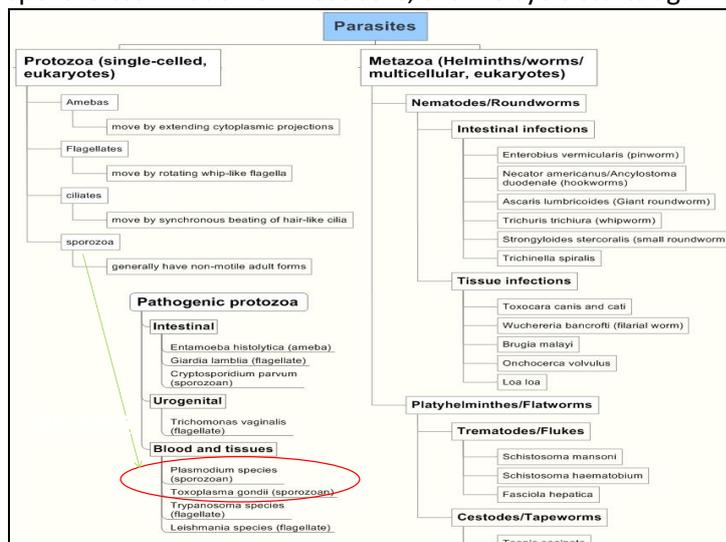
The trophozoites replicate by promitosis (nuclear membrane remains intact).

N. fowleri is found in fresh water, soil, thermal discharges of power plants, geothermal wells, and poorly-chlorinated swimming pools. Trophozoites can turn into temporary non-feeding **flagellated forms** which usually revert back to the trophozoite stage. Trophozoites infect humans or animals by penetrating the nasal mucosa and migrating to the brain via the olfactory nerves causing primary amebic meningoencephalitis (PAM). *N. fowleri* trophozoites are found in cerebrospinal fluid (CSF) and tissue, while flagellated forms are occasionally found in CSF. Cysts are not seen in brain tissue.

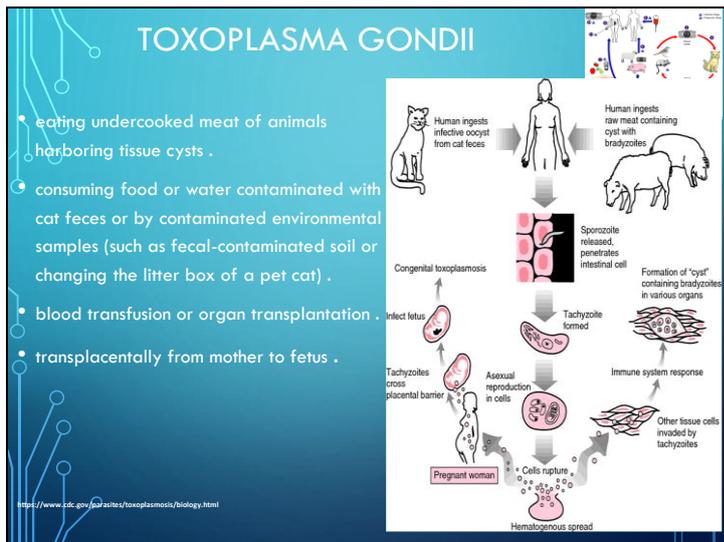
SPOROZOA

- super-class
- complex life cycle with alternating
- sexual and
- asexual reproductive phases (two different hosts)
- Coccidia -intracellular parasites,
 asexually = schizogony (merogony)
 sexually = sporogony.
- Class Coccidia
 - Isospora and Toxoplasma
- class Haematozoa
 - malarial parasites- Plasmodium species.

The Sporozoa reproduction cycle has both asexual and sexual phases. The asexual phase is termed schizogony (from the Greek, meaning generation through division), in which merozoites (daughter cells) are produced through multiple nuclear fissions. The sexual phase is known as sporogony (i.e., generation of spores) and is followed by gametogony or the production of sexually reproductive cells termed gamonts. Each pair of gamonts form a gamontocyst where the division of both gamonts, preceded by repeated nuclear divisions, originates numerous gametes. Gametes fuse in pairs, forming zygotes that undergo meiosis (cell division), thus forming new sporozoites. When sporozoites invade new host cells, the life cycle starts again.



Sporozoans are organisms that are characterized by being one-celled, non-motile, parasitic, and spore-forming. Sporozoans do not have flagella, cilia, or pseudopodia. They are capable of gliding movements. All sporozoa are obligate parasites, they form temporary non-motile spores which contain infective cells. Most of them have an alternation of sexual and asexual stages in their life cycle. An example of sporozoa is the *Plasmodium falciparum*, which is the causative agent of malaria.



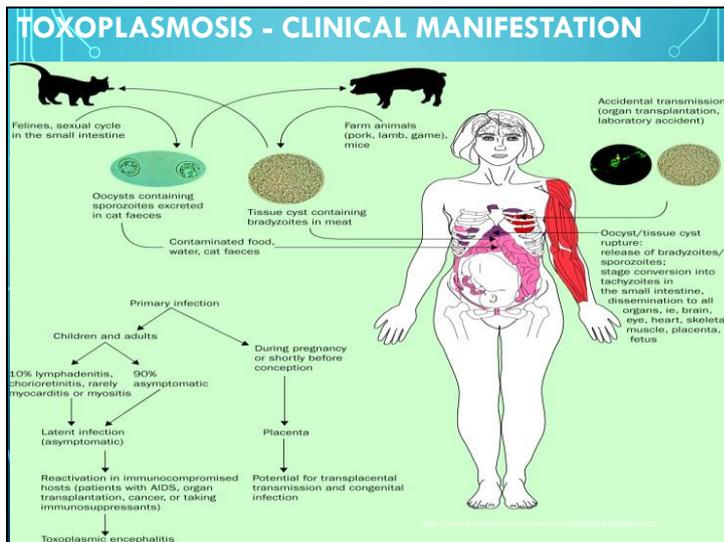
Toxoplasma gondii is a protozoan parasite that infects most species of warm-blooded animals, including humans, and causes the disease toxoplasmosis.

The only known definitive hosts for *Toxoplasma gondii* are members of family Felidae (domestic cats and their relatives). Cats become infected after consuming intermediate hosts harboring tissue cysts . Cats may also become infected directly by ingestion of sporulated oocysts. Animals bred for human consumption and wild game may also become infected with tissue cysts after ingestion of sporulated oocysts in the environment .Unsporulated oocysts are shed in the cat's feces . Although oocysts are usually only shed for 1-2 weeks, large numbers may be shed.

Oocysts take 1-5 days to sporulate in the environment and become infective. Intermediate hosts in nature (including birds and rodents) become infected after ingesting soil, water or plant material contaminated with oocysts . Oocysts transform into tachyzoites shortly after ingestion. These tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites. Humans can become infected by any of several routes:

- Eating undercooked meat of animals harboring tissue cysts .
- Consuming food or water contaminated with cat feces or by contaminated environmental samples (such as fecal-contaminated soil or changing the litter box of a pet cat) .
- Blood transfusion or organ transplantation .
- Transplacentally from mother to fetus .

In the human host, the parasites form tissue cysts, most commonly in skeletal muscle, myocardium, brain, and eyes; these cysts may remain throughout the life of the host. Diagnosis is usually achieved by serology, although tissue cysts may be observed in stained biopsy specimens . Diagnosis of congenital infections can be achieved by detecting *T. gondii* DNA in amniotic fluid using molecular methods such as PCR .



Infection with *Toxoplasma* in immuno-competent persons is generally an asymptomatic infection. However, 10% to 20% of patients with acute infection may develop: flu-like illness, cervical lymphadenopathy, atypical pneumonia, acute encephalitis, chorioretinitis. Symptoms usually resolve within a few months to a year.

In immunodeficient patients or infants (congenital) infection lead to:

- 1- Toxoplasmic encephalitis (hydrocephalus)
- 2- Myocarditis.
- 3- Retinochoroiditis (Ocular Toxoplasma infection)

(congenital) infection - in acute primoinfection of mother during pregnancy. Symptoms depends on the length of gravidity during primoinfection. Therapy can decrease the symptomatology

Acute diagnosis is important. New borne can have:

- subclinical symptoms (without therapy usually getting worse),
- syndrome of i.u. toxoplasmosis – hydrocephalus, calcifications in brain and liver, cataracta, microcephalus.

TOXOPLASMA – LABORATORY DIAGNOSIS

- **Observation of parasites - bronchoalveolar lavage** material from immunocompromised patients, **lymph node biopsy**.
- **Isolation** - from **blood** or other body fluids, by intraperitoneal inoculation into mice or tissue culture. The mice should be tested for the presence of *Toxoplasma* organisms in the peritoneal fluid 6 to 10 days post inoculation; if no organisms are found, serology can be performed on the animals 4 to 6 weeks post inoculation.
- **Detection - PCR**, - detecting congenital infections in utero.
- **Serologic testing is the routine method of diagnosis.**

The diagnosis of toxoplasmosis is typically made by serologic testing. A test that measures immunoglobulin G (IgG) is used to determine if a person has been infected. If it is necessary to try to estimate the time of infection, which is of particular importance for pregnant women, a test which measures immunoglobulin M (IgM) is also used along with other tests such as an avidity test.

Diagnosis can also be made by direct observation of the parasite in stained tissue sections, cerebrospinal fluid (CSF), or other biopsy material. These techniques are used less frequently because of the difficulty of obtaining these specimens. Parasites can also be isolated from blood or other body fluids (for example, CSF) but this process can be difficult and requires considerable time. Molecular techniques that can detect the parasite's DNA in the amniotic fluid can be useful in cases of possible mother-to-child (congenital) transmission. Ocular disease is diagnosed based on the appearance of the lesions in the eye, symptoms, course of disease, and often serologic testing. *Piccture: Toxoplasma gondii* tachyzoites, stained with Giemsa, from a smear of peritoneal fluid obtained from a laboratory-inoculated mouse.

TOXOPLASMA - SEROLOGY		
IgG result	IgM result	Report/interpretation for humans*
Negative	Negative	No serological evidence of infection with <i>Toxoplasma</i> .
Negative	Equivocal	Possible early acute infection or false-positive IgM reaction. Obtain a new specimen for IgG and IgM testing. If results for the second specimen remain the same, the patient is probably not infected with <i>Toxoplasma</i> .
Negative	Positive	Possible acute infection or false-positive IgM result. Obtain a new specimen for IgG and IgM testing. If results for the second specimen remain the same, the IgM reaction is probably a false-positive.
Equivocal	Negative	Indeterminate: obtain a new specimen for testing or retest this specimen for IgG in a different assay.
Equivocal	Equivocal	Indeterminate: obtain a new specimen for both IgG and IgM testing.
Equivocal	Positive	Possible acute infection with <i>Toxoplasma</i> . Obtain a new specimen for IgG and IgM testing. If results for the second specimen remain the same or if the IgG becomes positive, both specimens should be sent to a reference laboratory with experience in diagnosis of toxoplasmosis for further testing.
Positive	Negative	Infected with <i>Toxoplasma</i> for more than 1 year.
Positive	Equivocal	Infected with <i>Toxoplasma</i> for probably more than 1 year or false-positive IgM reaction. Obtain a new specimen for IgM testing. If results with the second specimen remain the same, both specimens should be sent to a reference laboratory with experience in the diagnosis of toxoplasmosis for further testing.
Positive	Positive	Possible recent infection within the last 12 months, or false-positive IgM reaction. Send the specimen to a reference laboratory with experience in the diagnosis of toxoplasmosis for further testing.

*except infants

Antibody Detection

The detection of *Toxoplasma*-specific antibodies is the primary diagnostic method to determine infection with *Toxoplasma*. *Toxoplasma* antibody detection tests are performed by a large number of laboratories with commercially available kits. An algorithm for the immunodiagnosis of toxoplasmosis for individuals greater than one year of age is shown table below. The IFA and EIA tests for IgG and IgM antibodies are the tests most commonly used today. Persons should be initially tested for the presence of *Toxoplasma*-specific IgG antibodies to determine their immune status. A positive IgG titer indicates infection with the organism at some time. If more precise knowledge of the time of infection is necessary, then an IgG positive person should have an IgM test performed by a procedure with minimal nonspecific reactions, such as IgM-capture EIA. A negative IgM test essentially excludes recent infection, but a positive IgM test is difficult to interpret because *Toxoplasma*-specific IgM antibodies may be detected by EIA for as long as 18 months after acute acquired infection.

A major problem with *Toxoplasma*-specific IgM testing is lack of specificity. Two situations occur frequently:

- persons with a positive IgM but negative IgG
- individuals with positive IgG and IgM results.

In the first situation, a positive IgM result with a negative IgG result in the same specimen should be viewed with great suspicion; the patient's blood should be redrawn two weeks after the first and tested together with the first specimen. If the first specimen was drawn very early after infection, the patient should have highly positive IgG and IgM antibodies in the second sample. If the IgG is negative and the IgM is positive in both specimens, the IgM result should be considered to be a false positive and the patient should be considered to be not infected.

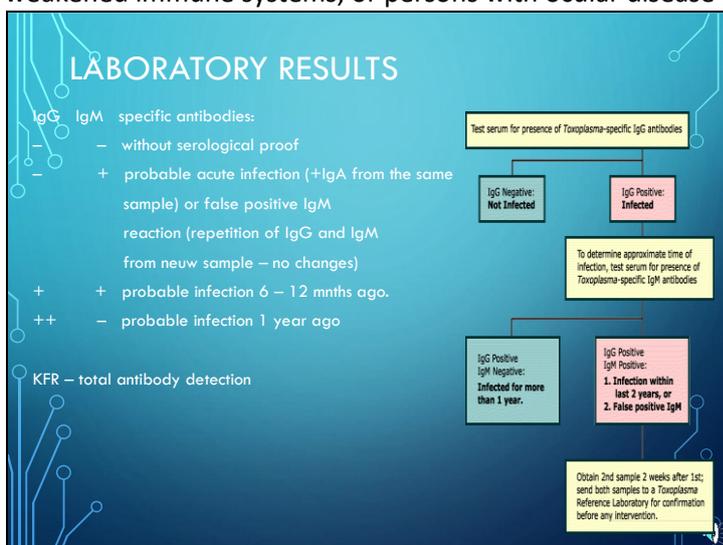
In the second situation, a second specimen should be drawn and both specimens submitted together to a reference lab which employs a different IgM testing system for confirmation. Prior to initiation

of patient management for acute toxoplasmosis, all IgG/IgM positives should be submitted to a reference lab for IgG avidity testing.

If the patient is pregnant, and IgG/IgM positive, an IgG avidity test should be performed. A high avidity result in the first 12 to 16 weeks of pregnancy (time dependent upon the commercial test kit) essentially rules out an infection acquired during gestation. A low IgG avidity result should not be interpreted as indicating recent infection, because some individuals have persistent low IgG avidity for many months after infection. Suspected recent infection in a pregnant woman should be confirmed prior to intervention by having samples tested at a toxoplasmosis reference laboratory. If the patient has clinical illness compatible with toxoplasmosis but the IgG titer is low, a follow-up titer two to three weeks later should show an increase in antibody titer if the illness is due to acute toxoplasmosis, assuming the host is not severely immunocompromised.

Newborn infants suspected of congenital toxoplasmosis should be tested by both an IgM- and an IgA-capture EIA. Detection of *Toxoplasma*-specific IgA antibodies is more sensitive than IgM detection in congenitally infected babies. *Toxoplasma*-specific IgG antibody levels in AIDS patients often are low to moderate, but occasionally no specific IgG antibodies can be detected. Tests for IgM antibodies are generally negative.

Treatment is not needed for a healthy person who is not pregnant. Symptoms will usually go away within a few weeks. Treatment may be recommended for pregnant women, persons who have weakened immune systems, or persons with ocular disease or severe illness.



TOXOPLASMOSIS - TREATMENT

- **Healthy people (nonpregnant)** - rarely indicated
- **Pregnant women, newborns, and infants** – spiramycin, pyrimethamine, leucovorin
- **Persons with ocular disease**
- **Persons with compromised immune systems**

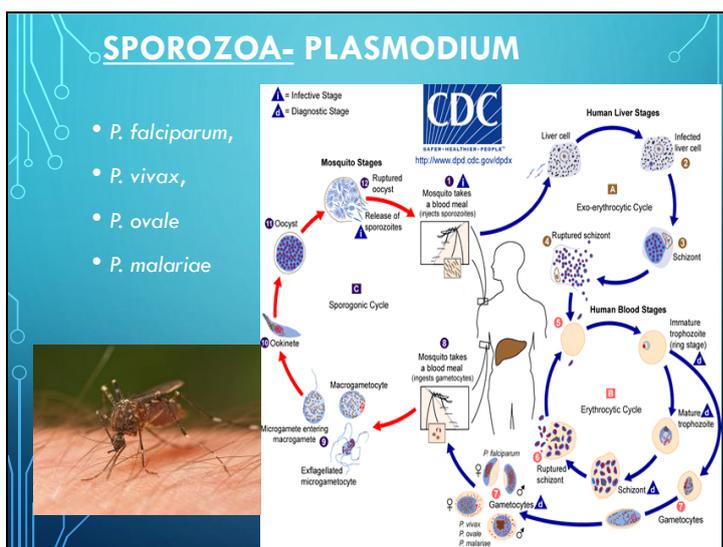
Treatment of immunocompetent adults with lymphadenopathic toxoplasmosis is rarely indicated; this form of the disease is usually self-limited. If visceral disease is clinically evident or symptoms are severe or persistent, treatment may be indicated for 2 to 4 weeks.

Management of maternal and fetal infection varies depending on the treatment center. In general, spiramycin is recommended for women whose infections were acquired and diagnosed before 18 weeks gestation and infection of the fetus is not documented or suspected. Spiramycin acts to reduce transmission to the fetus and is most effective if initiated within 8 weeks of seroconversion. Pyrimethamine, sulfadiazine and leucovorin are recommended for infections acquired at or after 18 weeks gestation or infection in the fetus is documented or suspected. PCR is often performed on the amniotic fluid at 18 gestation weeks to determine if the infant is infected. Congenitally infected newborns are generally treated with pyrimethamine, sulfonamide, and leucovorin for 12 months.

Treatment for ocular diseases should be based on a complete ophthalmologic evaluation. The decision to treat ocular disease is dependent on numerous parameters including acuteness of the lesion, degree of inflammation, visual acuity, and lesion size, location, and persistence. Healed lesions should not be treated.

Toxoplasmosis in immunodeficient patients is often fatal if not treated. Treatment is recommended for at least 4 to 6 weeks beyond resolution of all clinical signs and symptoms, but may be required for 6 months or longer. Relapses are known to occur in AIDS patients and maintenance therapy is recommended until a significant immunologic improvement is achieved in response to antiretroviral therapy. Pyrimethamine, folinic acid (leucovorin), and sulfadiazine are standards of therapy for immunodeficient patients.

Currently recommended treatment drugs for toxoplasmosis target the tachyzoite stage of the parasite and do not eradicate encysted parasites in the tissues. Pyrimethamine, considered the most effective drug against toxoplasmosis, is a standard component of therapy. Pyrimethamine is a folic acid antagonist and can cause dose-related suppression of the bone marrow, which is mitigated by concurrent administration of folinic acid (leucovorin). Leucovorin protects the bone marrow from the toxic effects of pyrimethamine. A second drug, such as sulfadiazine or clindamycin (if the patient has a hypersensitivity reaction to sulfa drugs), should also be included. The fixed combination of trimethoprim with sulfamethoxazole has been used as an alternative, as well as other drugs such as atovaquone and pyrimethamine plus azithromycin, which have not been extensively studied.



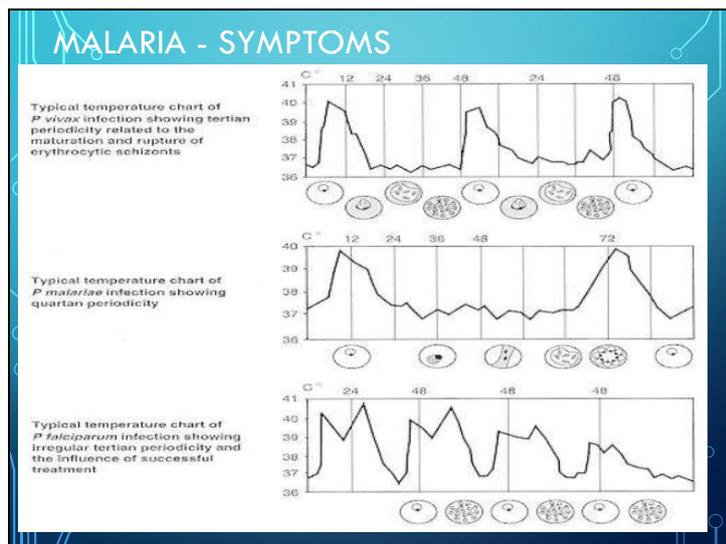
Malaria is a serious and sometimes fatal disease caused by a parasite that commonly infects a certain type of mosquito which feeds on humans. The natural history of malaria involves cyclical infection of humans and female *Anopheles* mosquitoes.

The malaria parasite life cycle involves two hosts. During a blood meal, a malaria-infected female *Anopheles* mosquito inoculates sporozoites into the human host. Sporozoites infect liver cells and mature into schizonts, which rupture and release merozoites. (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), the parasites undergo asexual multiplication in the erythrocytes (erythrocytic schizogony). Merozoites infect red blood cells. The ring stage trophozoites mature into schizonts, which rupture releasing merozoites. Some parasites differentiate into sexual erythrocytic stages (gametocytes). 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. The parasites' multiplication in the mosquito is known as the sporogonic cycle. While in the mosquito's stomach, the microgametes penetrate the macrogametes generating zygotes. The zygotes in turn become motile and elongated (ookinetes) which invade the midgut wall of the mosquito where they develop into oocysts. The oocysts grow, rupture, and release sporozoites, which make their way to the mosquito's salivary glands. Inoculation of the sporozoites into a new human host perpetuates the malaria life cycle.

Human Malaria					
Stages Species	Ring	Trophozoite	Schizont	Gametocyte	
<i>P. falciparum</i>					<ul style="list-style-type: none"> Parasitised red cells (pRBCs) not enlarged. RBCs containing mature trophozoites sequestered in deep vessels. Total parasite biomass = circulating parasites + sequestered parasites.
<i>P. vivax</i>					<ul style="list-style-type: none"> Parasites prefer young red cells pRBCs enlarged Trophozoites are amoeboid in shape. All stages present in peripheral blood.
<i>P. malariae</i>					<ul style="list-style-type: none"> Parasites prefer old red cells. pRBCs not enlarged Trophozoites tend to have a band shape. All stages present in peripheral blood.
<i>P. ovale</i>					<ul style="list-style-type: none"> pRBCs slightly enlarged and have an oval shape, with tufted ends. All stages present in peripheral blood.
<i>P. knowlesi</i>					<ul style="list-style-type: none"> pRBCs not enlarged. Trophozoites, pigment spreads inside cytoplasm, like <i>P. malariae</i>, band form may be seen Multiple invasion & high parasitaemia can be seen like <i>P. falciparum</i> All stages present in peripheral blood.

There are 5 *Plasmodium* species that cause malaria in human: *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium malariae*, *Plasmodium ovale*, and *Plasmodium knowlesi*. The 2 most common species are *P. falciparum* and *P. vivax*. *P. falciparum* is the most severe form and is responsible for most malaria-related deaths globally.



The most characteristic symptom of malaria is fever. Other common symptoms include chills, headache, myalgias, nausea, and vomiting. Diarrhea, abdominal pain, and cough are occasionally seen. As the disease progresses, some patients may develop the classic malaria paroxysm with bouts of illness alternating with symptom-free periods.

The malaria paroxysm comprises three successive stages.

The first is a 15-to-60-minute cold stage characterized by shivering and a feeling of cold. Next comes the 2-to-6 hour hot stage, in which there is fever, sometimes reaching 41°C, flushed, dry skin, and often headache, nausea, and vomiting. Finally, there is the 2-to-4 hour sweating stage during which the fever drops rapidly and the patient sweats.

In all types of malaria the periodic febrile response is caused by rupture of mature schizonts.

In *P. vivax* and *P. ovale* malaria, a brood of schizonts matures every 48 hr, so the periodicity of fever is tertian - "tertian malaria".

In *P. malariae* disease, fever occurs every 72 hours - "quartan malaria".

The fever in *P. falciparum* malaria may occur every 48 hours, but is usually irregular, showing no distinct periodicity.

MALARIA

- **ovale malaria** a mild form due to *Plasmodium ovale*, with recurring tertian febrile paroxysms and a tendency to end in spontaneous recovery.
- **quartan malaria** that in which the febrile paroxysms occur every 72 hours, or every fourth day counting the day of occurrence as the first day of each cycle; due to *Plasmodium malariae*.
- **quotidian malaria** vivax malaria in which the febrile paroxysms occur daily.
- **tertian malaria** vivax malaria in which the febrile paroxysms occur every 42 to 47 hours, or every third day counting the day of occurrence as the first day of the cycle.
- **vivax malaria** that due to *Plasmodium vivax*, in which the febrile paroxysms commonly occur every other day (*tertian m.*), but may occur daily (*quotidian m.*), if there are two broods of parasites segmenting on alternate days.

In *P. vivax* and *P. ovale* malaria, a brood of schizonts matures every 48 hr, so the periodicity of fever is tertian - "tertian malaria".

In *P. malariae* disease, fever occurs every 72 hours - "quartan malaria".

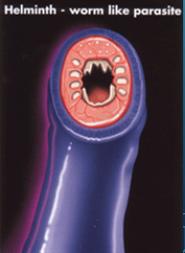
The fever in *P. falciparum* malaria may occur every 48 hours, but is usually irregular, showing no distinct periodicity.

MALARIA TREATMENT		
VIVAX	Chloroquine for 3days + Primaquine for 14days	
FALCIPARUM	Artemether for 3days + Lumefantrine for 3days + Primaquine single dose on Day2	Artemether for 3days + Sulfadoxine 3d +Pyridoxine 3d + Primaquine single dose on Day2
MIXED INFECTION	Same as falciparum + Add primaquine for 14days	
SEVERE FALCIPARUM MALARIA	DOC: Artesunate i.v for 48hrs followed by oral ACT	In pregnancy : Quinine i.v for 48hrs followed by Quinine + Doxycycline/clindamycin

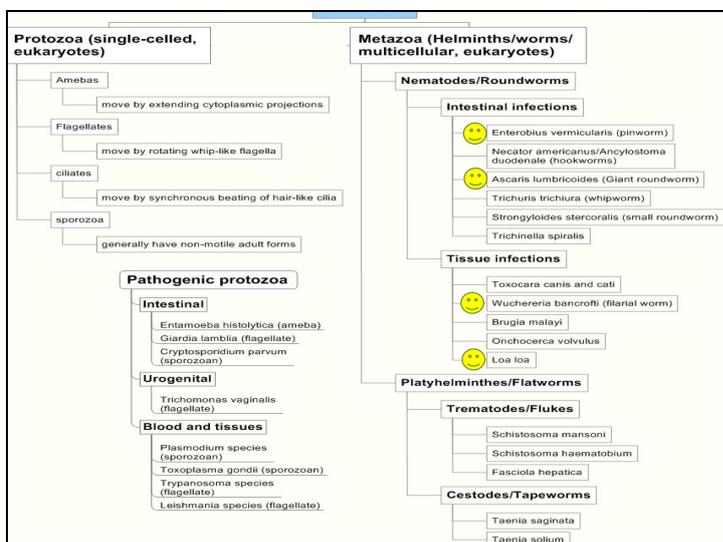
Chloroquine was, until recently, the most widely used anti-malarial. Chloroquine enters the red blood cell by simple diffusion, inhibiting the parasite cell and digestive vacuole. Primaquine is lethal to *P. vivax* and *P. ovale* in the liver stage, and also to *P. vivax* in the blood stage through its ability to do oxidative damage to the cell. However, the exact mechanism of action is not fully understood. Artemether is an artemisinin derivative that interact with ferriprotoporphyrin IX (heme) or ferrous ions in the acidic parasite food vacuole, and generates cytotoxic radical species. **Lumefantrine** (or **benflumetol**) is an antimalarial drug. It is only used in combination with artemether.

CLASSIFICATION OF PARASITIC HELMINTHS

- **Helminths**
 - A) *Nemathelminthes*
 - Nematoda (Round Worms)**
 - B) *Platyhelminthes*
 - Cestoda (Tapeworms)**
 - Trematoda (Flukes)**



Helminth - worm like parasite



NEMATODES

1. Intestinal

- i. **Small intestine only:** *Ascaris lumbricoides* (Common round worm), *Ancylostoma duodenale* (The old world hook worm), *Necator americanus* (American hookworm)
- ii. **Caecum and vermiform appendix:** *Enterobius vermicularis* (Threadworm or pin worm), *Trichuris trichuria* (Whipworm).

2. Somatic (inside the tissues and organs)

- i. **Lymphatic system:** *Wuchereria bancrofti*, *Brugia malayi*
- ii. **Subcutaneous tissue:** *Onchocerca volvulus*, *Dracunculus medinensis*
- iii. **Lungs:** *Strongyloides stercoralis*
- iv. **Conjunctiva:** *Loa loa*



Their body is elongated, cylindrical and unsegmented. Sexes are separate (dieocious). They also lack hooks and suckers. They possess the complete alimentary canal and body cavity.

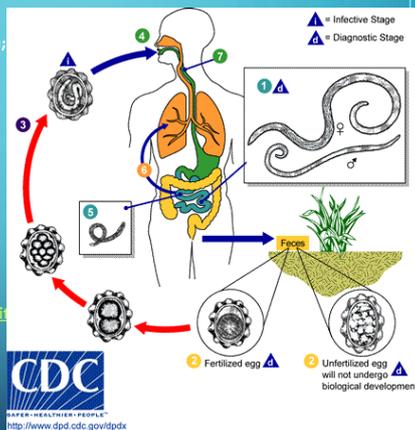
NEMATODES - ASCARIS LUMBRICOIDES

- Adult females: 20 to 35 cm;
- adult male: 15 to 30 cm

Diagnostic findings

- [Microscopy](#)
- [Macroscopy](#)
- [Morphologic comparison with other intestinal parasites](#)

Treatment: albendazole
mebendazole



Ascaris species are very large (adult females: 20 to 35 cm; adult males: 15 to 30 cm) nematodes (roundworms) that parasitize the human intestine. Adult worms live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the feces. Unfertilized eggs may be ingested but are not infective. Fertile eggs embryonate and become infective after 18 days to several weeks, depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed, the larvae hatch, invade the intestinal mucosa, and are carried via the portal, then systemic circulation to the lungs. The larvae mature further in the lungs (10 to 14 days), penetrate the alveolar walls, ascend the bronchial tree to the throat, and are swallowed. Upon reaching the small intestine, they develop into adult worms. Between 2 and 3 months are required from ingestion of the infective eggs to oviposition by the adult female. Adult worms can live 1 to 2 years.

People infected with *Ascaris* often show no symptoms, regardless of the species of worm. If symptoms do occur they can be light and include abdominal discomfort. Heavy infections can cause intestinal blockage and impair growth in children. Other symptoms such as cough are due to migration of the worms through the body.

Anthelmintic medications (drugs that rid the body of parasitic worms), such as albendazole and mebendazole, are the drugs of choice for treatment. Infections are generally treated for 1-3 days.

NEMATODES- ENTEROBIUS VERMICULARIS

- Enterobiasis
- asymptomatic.
- perianal pruritus, especially at night, which may lead to excoriations and bacterial superinfection.
- Occasionally, invasion of the female genital tract with vulvovaginitis and pelvic or peritoneal granulomas can occur.
- Other symptoms include anorexia, irritability, and abdominal pain.
- ("Scotch test", cellulose-tape slide test)

Pinworm infection is caused by a small, thin, white roundworm called *Enterobius vermicularis*. Although pinworm infection can affect all people, it most commonly occurs among children, institutionalized persons, and household members of persons with pinworm infection. Gravid adult female *Enterobius vermicularis* deposit eggs on perianal folds. Self-infection occurs by transferring infective eggs to the mouth with hands that have scratched the perianal area. Person-to-person transmission can also occur through handling of contaminated clothes or bed linens. Enterobiasis may also be acquired through surfaces in the environment that are contaminated with pinworm eggs (e.g., curtains, carpeting). Some small number of eggs may become airborne and inhaled. These would be swallowed and follow the same development as ingested eggs. Following ingestion of infective eggs, the larvae hatch in the small intestine and the adults establish themselves in the colon. The time interval from ingestion of infective eggs to oviposition by the adult females is about one month. The life span of the adults is about two months. Gravid females migrate nocturnally outside the anus and oviposit while crawling on the skin of the perianal area. The larvae contained inside the eggs develop (the eggs become infective) in 4 to 6 hours under optimal conditions. Retroinfection, or the migration of newly hatched larvae from the anal skin back into the rectum, may occur but the frequency with which this happens is unknown.

ENTEROBIUS VERMICULARIS

1-Scotch tape technique

Principle:
the eggs of *E. vermicularis* can be collected from the perianal skin using a piece of transparent adhesive tape.

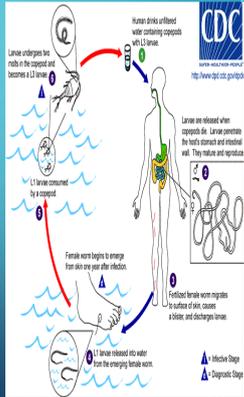
Many affected individuals do not observe visible pinworms (low parasite burden and no symptoms). Since oviposition occurs on the anal folds, the worm eggs that are invisible to the naked eye can be swabbed using commercially available adhesive cellulose tape (“Scotch tape”) in the morning prior to defecation and before washing the genital area (Scotch tape test). To this end, one presses the adhesive side of the tape (previously cut to size, e.g., 10 × 2 cm) against the anal and perianal region several times. The tape is then affixed to a suitable slide, adhesive side down. Microscopic detection of the characteristic worm eggs confirms infection.

NEMATODES - DRACUNCULUS MEDINENSIS

guinea worm disease
<https://www.youtube.com/watch?v=cd9rmy7cB6A>
 B6A

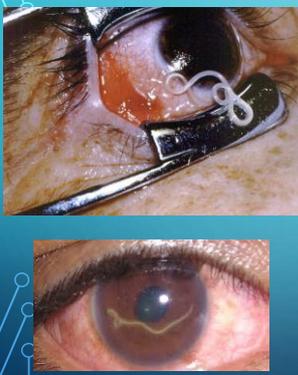
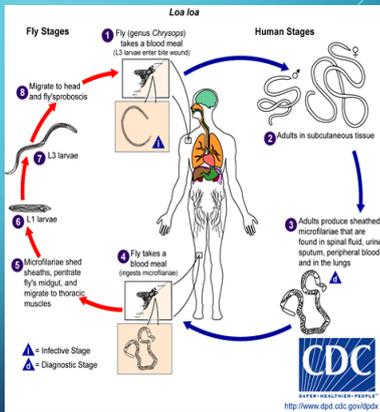


- The female guinea worm induces a painful blister
- (A) after rupture of the blister, the worm emerges as a whitish filament
- (B) in the center of a painful ulcer which is often secondarily infected.

Humans become infected by drinking unfiltered water containing copepods (small crustaceans) which are infected with larvae of *D. medinensis*. Following ingestion, the copepods die and release the larvae, which penetrate the host stomach and intestinal wall and enter the abdominal cavity and retroperitoneal space. After maturation into adults and copulation, the male worms die and the females (length: 70 to 120 cm) migrate in the subcutaneous tissues towards the skin surface. Approximately one year after infection, the female worm induces a blister on the skin, generally on the distal lower extremity, which ruptures. When this lesion comes into contact with water, a contact that the patient seeks to relieve the local discomfort, the female worm emerges and releases larvae. The larvae are ingested by a copepod and after two weeks (and two molts) have developed into infective larvae. Ingestion of the copepods closes the cycle.

NEMATODES- LOA LOA

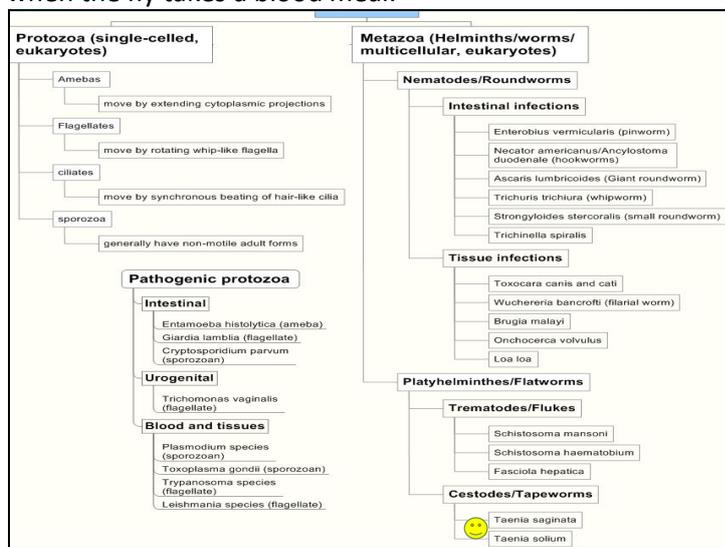



The vector for *Loa loa* filariasis are flies from two species of the genus *Chrysops*, *C. silacea* and *C. dimidiata*. During a blood meal, an infected fly (genus *Chrysops*, day-biting flies) introduces third-

stage filarial larvae onto the skin of the human host, where they penetrate into the bite wound. The larvae develop into adults that commonly reside in subcutaneous tissue.

The female worms measure 40 to 70 mm in length and 0.5 mm in diameter, while the males measure 30 to 34 mm in length and 0.35 to 0.43 mm in diameter. Adults produce microfilariae measuring 250 to 300 µm by 6 to 8 µm, which are sheathed and have diurnal periodicity.

Microfilariae have been recovered from spinal fluids, urine, and sputum. During the day they are found in peripheral blood, but during the noncirculation phase, they are found in the lungs. The fly ingests microfilariae during a blood meal. After ingestion, the microfilariae lose their sheaths and migrate from the fly's midgut through the hemocoel to the thoracic muscles of the arthropod. There the microfilariae develop into first-stage larvae and subsequently into third-stage infective larvae. The third-stage infective larvae migrate to the fly's proboscis and can infect another human when the fly takes a blood meal.



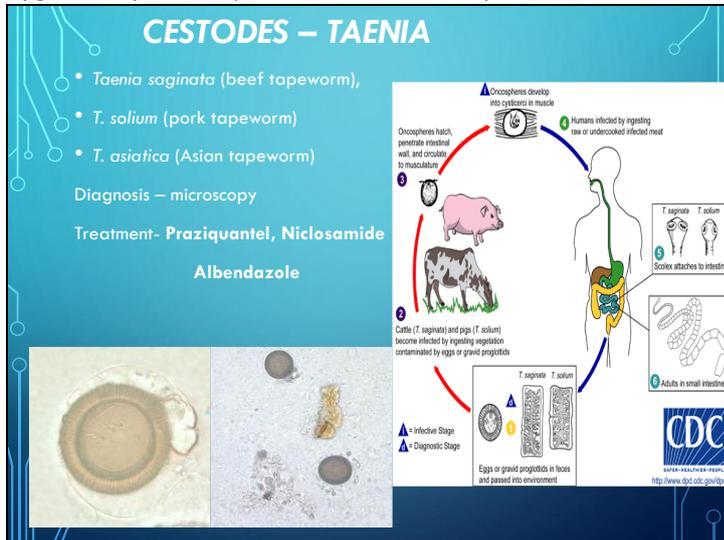
CESTODES



- tape-like,
- segmented
- hermaphrodite organism
- suckers in their head and in some species also hooks that attach the tapeworm to its host
- consists of a head (scolex) and many proglottids.
- Alimentary canal and body cavity are absent.
- *Diphyllobothrium*, *Taenia*, *Echinococcus*, *Hymenolepis*, etc

Cestoda is a class of parasitic worms in the flatworm phylum (Platyhelminthes). Most of the species—and the best-known—are those in the subclass Eucestoda; they are ribbon-like worms as adults, known as **tapeworms**. The adult tapeworm has a scolex (head), a short neck, and a strobila (segmented body) formed of proglottids. Tapeworms anchor themselves to the inside of the intestine of their host using their scolex, which typically has hooks, suckers, or both. They have no mouth, but absorb nutrients directly from the host's gut. The neck continually produces proglottids,

each one containing a reproductive tract; mature proglottids are full of eggs, and fall off to leave the host, either passively in the feces or actively moving. All tapeworms are hermaphrodites, with each individual having both male and female reproductive organs. Humans are subject to infection by several species of tapeworms if they eat undercooked meat such as pork (*Taenia solium*), beef (*T. saginata*), and fish (*Diphyllobothrium*), or if they live in, or eat food prepared in, conditions of poor hygiene (*Hymenolepis* or *Echinococcus* species).



Taeniasis is the infection of humans with the adult tapeworm of *Taenia saginata*, *T. solium* or *T. asiatica*. Humans are the only definitive hosts for these three species. Eggs or gravid proglottids are passed with feces ; the eggs can survive for days to months in the environment. Cattle (*T. saginata*) and pigs (*T. solium* and *T. asiatica*) become infected by ingesting vegetation contaminated with eggs or gravid proglottids . In the animal's intestine, the oncospheres hatch , invade the intestinal wall, and migrate to the striated muscles, where they develop into cysticerci. A cysticercus can survive for several years in the animal.

Humans become infected by ingesting raw or undercooked infected meat . In the human intestine, the cysticercus develops over 2 months into an adult tapeworm, which can survive for years. The adult tapeworms attach to the small intestine by their scolex and reside in the small intestine . The adults produce proglottids which mature, become gravid, detach from the tapeworm, and migrate to the anus or are passed in the stool (approximately 6 per day). The eggs contained in the gravid proglottids are released after the proglottids are passed with the feces.

Most people with tapeworm infections have no symptoms or mild symptoms. Patients with *T. saginata* taeniasis often experience more symptoms than those with *T. solium* because the *T. saginata* tapeworm is larger in size (up to 10 meters (m)) than *T. solium* (usually 3 m). Tapeworms can cause digestive problems including abdominal pain, loss of appetite, weight loss, and upset stomach. The most visible symptom of taeniasis is the active passing of proglottids (tapeworm segments) through the anus and in the feces. In rare cases, tapeworm segments become lodged in the appendix, or the bile and pancreatic ducts.

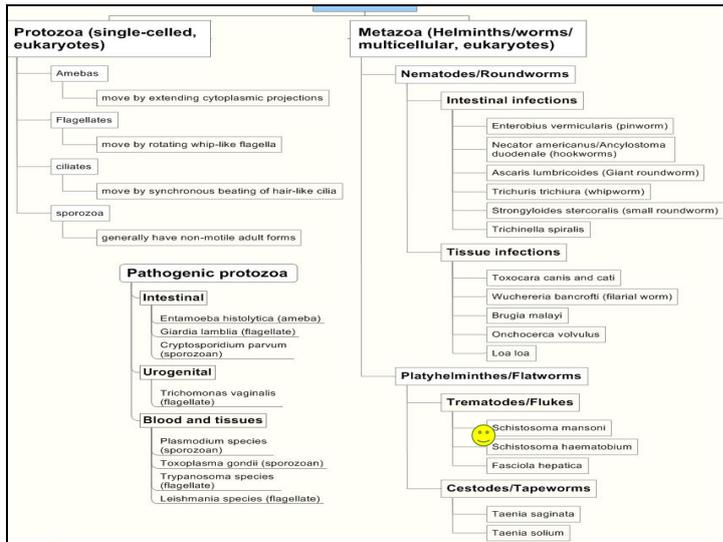
Infection with *T. solium* tapeworms can result in human cysticercosis, which can be a very serious disease that can cause seizures and muscle or eye damage.

Diagnosis of *Taenia* tapeworm infections is made by examination of stool samples; individuals should also be asked if they have passed tapeworm segments. Stool specimens should be collected on three different days and examined in the lab for *Taenia* eggs using a microscope. Tapeworm eggs can be detected in the stool 2 to 3 months after the tapeworm infection is established.

Praziquantel is the medication most often used to treat active taeniasis, given orally once.

Niclosamide is an alternative, given at 2 g orally once for adults and 50 mg/kg orally once for

children. **Albendazole**, given for three days, may be used as another option for the treatment of taeniasis.



TREMATODES

- leaf-like
- unsegmented
- Sexes are not separate (except Schistosomes- diecious).
- Alimentary canal -present but is not complete (anus absent).
- The body cavity is absent.
- *Schistosoma*, *Gastrodiscoides*, *Fasciolopsis*, *Fasciola*, *Clonorchis*, *Heterophyes*, etc.

Trematoda is a class within the phylum Platyhelminthes. It includes two groups of parasitic flatworms, known as **flukes**. Trematodes are flattened oval or worm-like animals, usually no more than a few centimetres in length, although species as small as 1 millimetre) are known. Their most distinctive external feature is the presence of two suckers, one close to the mouth, and the other on the underside of the animal. Almost all trematodes infect molluscs as the first host in the life cycle, and most have a complex life cycle involving other hosts.

TREMATODES- SCHISTOSOMA

- *Schistosoma haematobium*,
- *S. japonicum*,
- *S. mansoni*.

Diagnosis – microscopy

Treatment - praziquantel

Life Cycle Diagram:

- Eggs hatch releasing miracidia
- Miracidia penetrate snail tissue
- Sporocysts in snail (successive generations)
- Cercariae released by snail into water and free-swimming
- Penetrate skin
- Cercariae lose tails during penetration and become schistosomulae
- Circulation
- Migrate to portal blood in liver and mature into adults
- Paired adult worms migrate to:
 - A mesenteric venules of bowel/rectum (laying eggs that circulate to the liver and shed in stools)
 - B mesenteric plexus of bladder
- in feces (A) or in urine (B)
- Eggs hatch releasing miracidia

Legend:

- ▲ = Infective Stage
- △ = Diagnostic Stage

Species:

- A S. japonicum
- B S. haematobium
- C S. mansoni

Source: <http://www.dpd.cdc.gov/dpd>

Schistosomiasis, also known as **snail fever** and **bilharzia**,¹ is a disease caused by parasitic flatworms called schistosomes.¹ The urinary tract or the intestines may be infected.

Eggs are eliminated with feces or urine. Under optimal conditions the eggs hatch and release miracidia, which swim and penetrate specific snail intermediate hosts. The stages in the snail include 2 generations of sporocysts and the production of cercariae. Adult worms in humans reside in the mesenteric venules in various locations. *S. haematobium* most often occurs in the venous plexus of bladder, but it can also be found in the rectal venules.

The females (size 7 to 20 mm; males slightly smaller) deposit eggs in the small venules of the portal and perivesical systems. The eggs are moved progressively toward the lumen of the intestine (*S. mansoni* and *S. japonicum*) and of the bladder and ureters (*S. haematobium*), and are eliminated with feces or urine, respectively.

Pathology of *S. mansoni* and *S. japonicum* schistosomiasis includes: Katayama fever, hepatic perisinusoidal egg granulomas, Symmers' pipe stem periportal fibrosis, portal hypertension, and occasional embolic egg granulomas in brain or spinal cord.

Pathology of *S. haematobium* schistosomiasis includes: hematuria, scarring, calcification, squamous cell carcinoma, and occasional embolic egg granulomas in brain or spinal cord.

Human contact with water is thus necessary for infection by schistosomes. Various animals, such as dogs, cats, rodents, pigs, horses and goats, serve as reservoirs for *S. japonicum*, and dogs for *S. mekongi*.

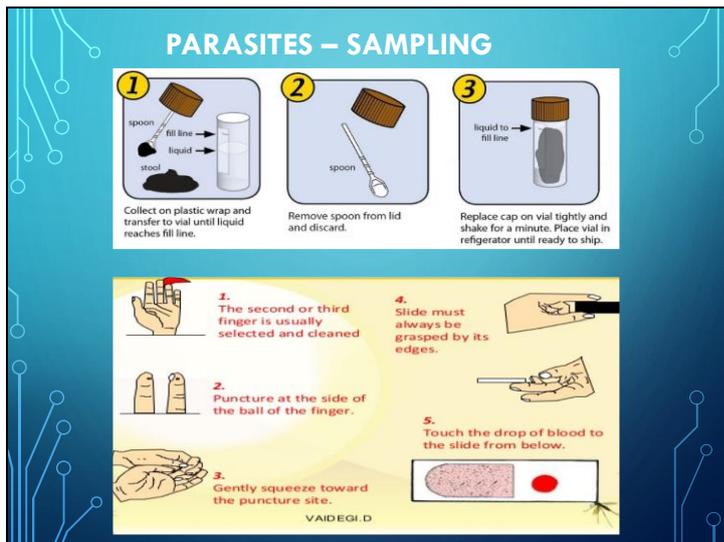
Diagnosis - Stool or urine samples can be examined microscopically for parasite eggs (stool for *S. mansoni* or *S. japonicum* eggs and urine for *S. haematobium* eggs). The eggs tend to be passed intermittently and in small amounts and may not be detected, so it may be necessary to perform a blood (serologic) test.

Treatment - Infections with all major *Schistosoma* species can be treated with praziquantel. The timing of treatment is important since praziquantel is most effective against the adult worm and requires the presence of a mature antibody response to the parasite. For travelers, treatment should be at least 6-8 weeks after last exposure to potentially contaminated freshwater.

PARASITES – LABORATORY DIAGNOSIS

Currently diagnosis is mostly based on microscopic exam	Alternative testing methods:
Specimen types:	◦ Serology for select pathogens
◦ Stool	◦ Fluorescent stains for select pathogens
◦ Non-stool	◦ Molecular assays for select pathogens
◦ Perianal specimen	
◦ Sigmoidoscopic specimen	
◦ Duodenal aspirates	
◦ Liver abscess	
◦ Sputum	
◦ Urine	
◦ Urogenital	
◦ Blood	
◦ Tissue	

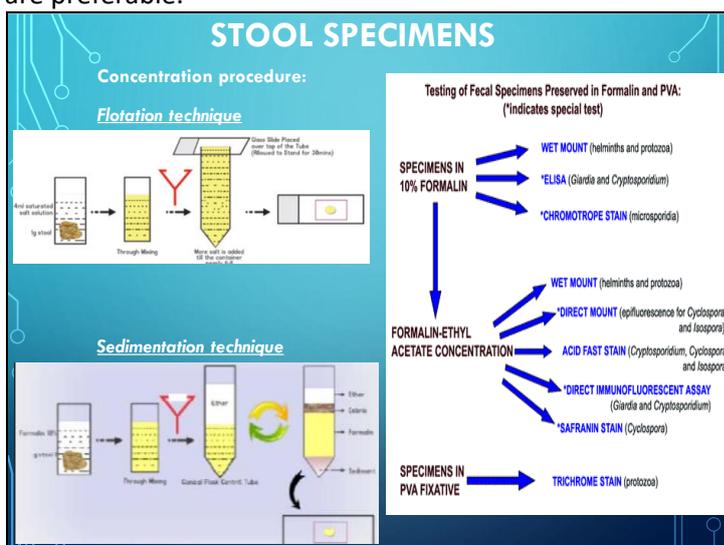
Diagnostic parasitology includes laboratory procedures that are designed to detect organisms within clinical specimens using morphologic criteria and visual identification. Some clinical specimens, such as those from the intestinal tract, contain numerous artifacts that complicate the differentiation of parasites from surrounding debris. Final identification is usually based on light microscopic examination of stained preparations, often using high magnification techniques such as oil immersion (1000×)



Stool - Collect the stool in a dry, clean, leakproof container. Make sure no urine, water, soil or other material gets in the container. **Faecal specimens** should be collected in the early stages of the disease, when pathogens are present in the highest number, and preferably before antimicrobial treatment is started, if appropriate. The specimen should be collected in the morning to reach the laboratory before noon, so that it can be processed the same day. In parasitology, a fresh stool specimen is preferred to a rectal swab.

Blood samples - Whenever possible, specimens should be collected before treatment is initiated. When malaria and babesiosis are suspected, blood smears should be obtained and examined without delay. Since the parasitemia may fluctuate, multiple smears might be needed. These can be taken at 8 to 12 hour intervals for 2 to 3 days.

Venous blood samples provide sufficient material for performing a variety of diagnostic tests, including concentration procedures (filariasis, trypanosomiasis). However, in some parasitic diseases (e.g., for diagnosis of malaria in particular), anticoagulants in the venous blood specimen can interfere with parasite morphology and staining characteristics; this problem can be further compounded by excessive delays prior to making the smears. In such cases, capillary blood samples are preferable.



Examination of fresh specimens permits the observation of motile trophozoites, but this must be carried out without delay. Liquid (diarrheic) specimens (which are more likely to contain trophozoites) should be examined within 30 minutes of passage (not within 30 minutes of arrival in

the laboratory!), and soft specimens (which may contain both trophozoites and cysts) should be examined within one hour of passage. If delays cannot be avoided, the specimen should be preserved to avoid disintegration of the trophozoites.

Specimens preserved in formalin can be tested directly (wet mount, immunoassay, chromotrope stain, UV fluorescence) or can be concentrated prior to further testing.

Concentration procedure separate parasites from fecal debris and increase the chances of detecting parasitic organisms when these are in small numbers. They are divided into flotation techniques and sedimentation techniques.

Flotation techniques (most frequently used: zinc sulfate or Sheather's sugar) use solutions which have higher specific gravity than the organisms to be floated so that the organisms rise to the top and the debris sinks to the bottom. The main advantage of this technique is to produce a cleaner material than the sedimentation technique. The disadvantages of most flotation techniques are that the walls of eggs and cysts will often collapse, thus hindering identification. Also, some parasite eggs do not float.

Sedimentation techniques use solutions of lower specific gravity than the parasitic organisms, thus concentrating the latter in the sediment. Sedimentation techniques are recommended for general diagnostic laboratories because they are easier to perform and less prone to technical errors. The sedimentation technique used at CDC is the formalin-ethyl acetate technique, a diphasic sedimentation technique that avoids the problems of flammability of ether, and which can be used with specimens preserved in formalin, MIF or SAF.



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