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
Trypanosoma
Disease: Sleeping sickness
Chagas disease

T. rhodesiense,
T. gambiense
and
T. cruzi

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 over 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 Lymphkonten), 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. 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 procyclic 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 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

- 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 (Appetitilosigkeit), 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.

If you believe you have been bitten by this bug, you should contact your personal physician.
An infected triatomine insect vector (or "kissing" bug) takes a blood meal and releases *trypomastigotes* in its feces near the site of the bite wound. Trypomastigotes enter the host through the wound or through intact mucosal membranes, such as the conjunctiva. Common triatomine vector species for trypanosomiasis belong to the genera *Triatoma*, *Rhodnius*, and *Panstrongylus*. Inside the host, the trypomastigotes invade cells, where they differentiate into intracellular amastigotes. The amastigotes multiply by binary fission and differentiate into trypomastigotes, and then are released into the circulation as bloodstream trypomastigotes. Trypomastigotes infect cells from a variety of tissues and transform into intracellular amastigotes in new infection sites. Clinical manifestations can result from this infective cycle. The bloodstream trypomastigotes do not replicate (different from the African trypanosomes). Replication resumes only when the parasites enter another cell or are ingested by another vector. The "kissing" bug becomes infected by feeding on human or animal blood that contains circulating parasites. The ingested trypomastigotes transform into epimastigotes in the vector's midgut. The parasites multiply and differentiate into infective metacyclic trypomastigotes in the hindgut. *Trypanosoma cruzi* can also be transmitted through blood transfusions, organ transplantation, transplacentally, and in laboratory accidents.

**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
Giardia
Disease: Giardiasis

200 million people infected each year

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). 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.

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. 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).
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
Disease: Leishmaniasis

Leishmania spp.
Sandfly
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 *spleen* (*Milz*) and liver
- Important opportunistic infection with HIV
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!
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. 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.
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). 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.

*Plasmodium falciparum: Ring Stage Parasites*
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.
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.
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.

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)