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Ferhat Abbas University, Setif 1
Faculty of Natural and Life Sciences
Department of Animal Biology and Physiology
Field: Natural and Life Sciences
Major: Biology
Specialty: Parasitology

Course Handout :

Protozoan and Metazoan Parasites

Content intended for 3rd-year students, Bachelor in Parasitology.

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Teaching Objectives

This course aims to develop knowledge of parasitic species that infest humans through the study of their morpho-anatomy, development, biological cycle, and classification within the animal kingdom.

It also aims to:

- ✓ Provide the student with an overview of the diversity of parasitic forms;
- ✓ Make the student aware of the importance of the host–parasite balance for the survival of species;

- ✓ Explain the role of environmental factors in the epidemiology of parasitic infections;
- ✓ Understand the interactions between vectors, hosts, and pathogenic agents that determine the spread and emergence of parasitic diseases.

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Practical Work:

➤ Presentation of representative Protozoa and Metazoa.

Examples: *Trypanosomes* / *Leishmania* / *Amoebae* / *Cestodes* / *Trematodes* / *Nematodes* / *Arthropods*.

➤ Observation of prepared slide specimens of Protozoa, Arthropods, and Mollusks.

Principle: Identification of parasitic species that affect humans through the study of their morphology, based on diagnostic criteria.

List of Abbreviations

DNA: deoxyribonucleic acid

DH: definitive host

IH: intermediate host

PH: paratenic host

CL: cutaneous leishmaniasis

DCL: diffuse cutaneous leishmaniasis

LCL: localized cutaneous leishmaniasis

MCL: mucocutaneous leishmaniasis

VL: visceral leishmaniasis, or Kala-azar

STD: sexually transmitted disease

PR: parasite reservoir

RES: reticuloendothelial system

I – GENERALITIES ON PARASITISM

1.1 – Definitions

Parasitology is the science that studies parasites.

A **parasite** (from Greek *para* = beside, *sitos* = food) is an animal, plant, or bacterium that, during part or all of its life cycle, lives at the expense of an individual of another species called the **host**, and may sometimes alter the host's health.

Characteristics of a Parasite

A parasite is always smaller than its host.

The parasite lives at the expense of another living organism, the host, which constitutes a source of nutrients and energy.

The parasite is carried by the host, a means of transport.

The parasite is protected by the host, a sheltering habitat.

The parasite causes harmful effects to its host but never kills it immediately.

Parasitism

Parasitism is a type of biological association in which one organism, the parasite, depends on another organism, the host, for nutrition, shelter, and/or reproduction, causing harm to the host without killing it right away.



Reservoir of Parasites

A sick human or a healthy carrier of parasites may play this role, the infected person therefore becoming a risk to the community. Sometimes the external environment, as well as many animals and plants, can act as a reservoir and ensure the survival and transformation of the parasite.

Different Hosts

The parasite transiently or permanently colonizes several types of hosts:

- **The definitive host**, which harbors the adult or sexual forms.
- **The intermediate host**, which is essential for the maturation and/or multiplication of a

parasite; there are two types of intermediate hosts:

➤ **Active intermediate host**, an arthropod vector in the strict sense, ensuring *active* transport between the reservoir and a susceptible subject (fly, mosquito...).

➤ **Passive intermediate host**: either a low-mobility host that ensures the dissemination of the infective form in its usually aquatic environment (mollusks for schistosomiasis), or a host that is ingested (cyclops, fish). Some plants can be considered “supports” of forms that have already undergone development inside a previous intermediate host (mollusk then wild watercress in the case of fascioliasis).

The role of humans in parasitic life cycles may be normal (*Taenia*) or accidental, replacing an animal: in this case, it is a **true parasitic dead-end**, with an incomplete life cycle in humans (toxocariasis), or a **cul-de-sac**, where the parasitic cycle continues only if the human host is itself eaten (trichinellosis). Parasitic cycles with a single host are called **monoxenous** (*Trichinella*), and **heteroxenous** when they involve several hosts (flukes).

Localization

Parasites may be **ectoparasites** or **endoparasites**, found **on** (lice, fleas) or **within** their hosts.

They may be intra- and/or extracellular: during their cycle, some parasitic forms must carry out part of their metabolism at the expense of a host cell — red or white blood cells, hepatic or intestinal cells.

Their localization and migration vary: parasites without their own means of movement may be transported by the airway, intestinal tract, or bloodstream. Others can crawl or move using pseudopods, suckers, cilia, flagella, or an undulating membrane, and can actively penetrate the skin or mucous membranes.

Modes of Infestation

Free infective forms in nature may be transmitted:

- **Orally** (flukes),
- **Transcutaneously** (schistosomes),
- **By airborne route** (pinworm eggs, fungal spores),
- **Sexually** (*Trichomonas vaginalis*).

Other infective forms may be transmitted:

- **By a passive intermediate host, orally** (cyclops and Guinea worm, fish and flukes, pork and

Taenia / Trichina),

- **By an active intermediate host, via biting** (filariasis, leishmaniasis, sleeping sickness — except malaria, in which the active vector, the mosquito, is the definitive host),
- **Via feces of the vector** (kissing bugs and Chagas disease).
- A mother may transmit parasites to her child **transplacentally** (toxoplasmosis).
- Transmission through **blood transfusion** is possible (malaria, trypanosomiasis, etc.).
- Transplantation of a parasitized organ is a rare but possible mode of contamination (toxoplasmosis, malaria...).

Fecal Hazard

The fecal hazard refers to a sanitary risk of transmission of an infectious agent (viral, bacterial, parasitic, or fungal) carried by the excreta of sick or asymptomatic humans or animals, following an **orofecal cycle**, through food, water, or an inert (object) or living (flies, hands, etc.) vehicle.

Transmission of infectious agents occurs via:

- **Indirect transmission**, through ingestion of water or food contaminated by infectious agents capable of surviving in the external environment;
- **Direct transmission**, from a carrier to a healthy individual through dirty hands or intimate contact; in this case, the infectious agent is not necessarily highly resistant in the environment.

1.2 – Host–Parasite Interactions

Different types of interspecific relationships:

- **Parasitism**: unilateral physiological dependence.
- **Mutualism**: reciprocal exchange of benefits.
- **Inquilinism**: sharing of habitat without feeding on the host.
- **Phoresy**: temporary transport relationship.
- **Parasitoidism**: long-term interaction ending in the death of the host.
- **Symbiosis**: reciprocal benefits for both organisms.

Lifestyle of Parasites

- **Facultative parasitism**: organisms that can live as parasites or lead a free life.
- **Obligate parasitism**: the parasite must spend part or all of its life inside a living organism.
- **Accidental parasitism**: parasites accidentally found in an unusual host, in which they survive

for some time.

- **Opportunistic parasitism:** non-pathogenic organisms that can become parasitic and pathogenic if the host's susceptibility increases.

Parasitic Specificity

This refers to the more or less strict fidelity linking a parasite to its host(s). Parasites may be:

- **Oioxenous:** affinity for a single host species.
- **Stenoxenous:** narrowly adapted to one or a few closely related host species and unable to live in others.
- **Euryxenous:** low specificity, readily switching among unrelated host species.

Parasitic Adaptation

- **Well-adapted parasite** → causes a chronic, mild disease.

Example: *Taenia saginata*

- **Poorly adapted parasite** → causes an acute, severe, sometimes fatal disease.

Example: *Plasmodium falciparum*

Limits of Parasitism

• **Parasite:** lives at the expense of the host (without intending to kill it, benefiting from it) — different from:

- **Predator:** feeds on prey.
- **Saprophyte:** feeds on decomposing matter without harming the host (the host's death is not caused by the saprophyte).
- **Commensal:** lives in the same place (ecological niche, biotope) as another organism with mutual indifference (no benefit or harm).
- **Symbiont:** lives in close collaboration with another organism, bringing reciprocal benefits.
- **Free-living organism:** able to meet its own needs independently.

1.3 – Evolutionary Cycles of Parasites

The parasitic cycle refers to the series of successive stages that allow the passage from one generation of a parasite to the next.

From simplest to most complex, we distinguish:

➤ **Direct or monoxenous cycle:** with a single host, where the parasite passes directly from an infested human to a healthy human. There is often a free-living phase during which the infective form develops (matures).

Examples: Amoebae, *Giardia*, *Trichomonas*, intestinal nematodes.

The cycle may be short and direct, without a mandatory passage through the external environment (the parasite becomes immediately infective - or auto-infective - as soon as it leaves the host).

The degree of infestation depends on the parasite's resistance in the external environment (e.g., *20 days for an amoebic cyst*).

Examples: *Enterobius vermicularis* (pinworms), *Entamoeba histolytica*.

The cycle may also be long and direct, requiring the maturation of a parasitic stage in the external environment under specific conditions of humidity, temperature, and soil composition. Maturation varies according to climatic conditions (*high humidity and temperatures between 20°C and 25°C*).

It is characterized by the formation of an embryo inside the egg, by the hatching of the egg producing a larva, and by the transformation of the larva into an infective larva.

Examples: *Ascaris lumbricoides* (Ascaris), *Ancylostoma duodenale* and *Necator americanus* (hookworms) (Figure 1).

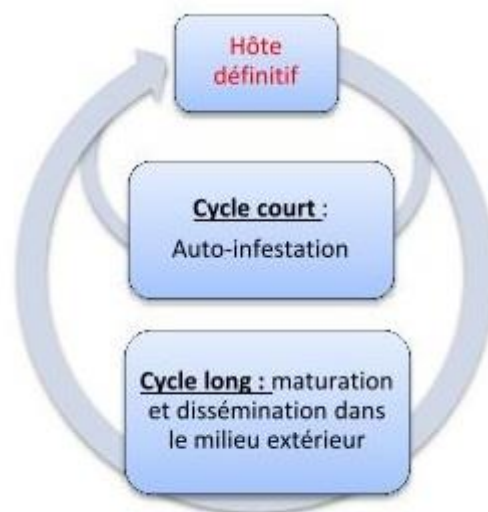


Figure 1. Direct, monoxenous cycle with a single host.

➤ **The indirect cycle with a definitive host and one or several intermediate hosts**

(heteroxenous), and sometimes a free-living phase between the different hosts.

The parasite passes through two or more hosts, and the cycle develops with one or several successive intermediate hosts or vectors (which are obligatory transformers of the pathogen into an infective form):

mosquitoes (malaria, lymphatic filariasis), fish (bothriocephalus), mollusks (flukes and schistosomes), cattle, pigs (*Taenia*), etc.

- **Dixenous cycle** = DH + IH₁
- **Three-host cycle** = DH + IH₁ + IH₂
- **Four-host cycle** = DH + IH₁ + IH₂ + PH

Example of a two-host cycle: *Taenia saginata* involving humans and cattle (Figure 2).

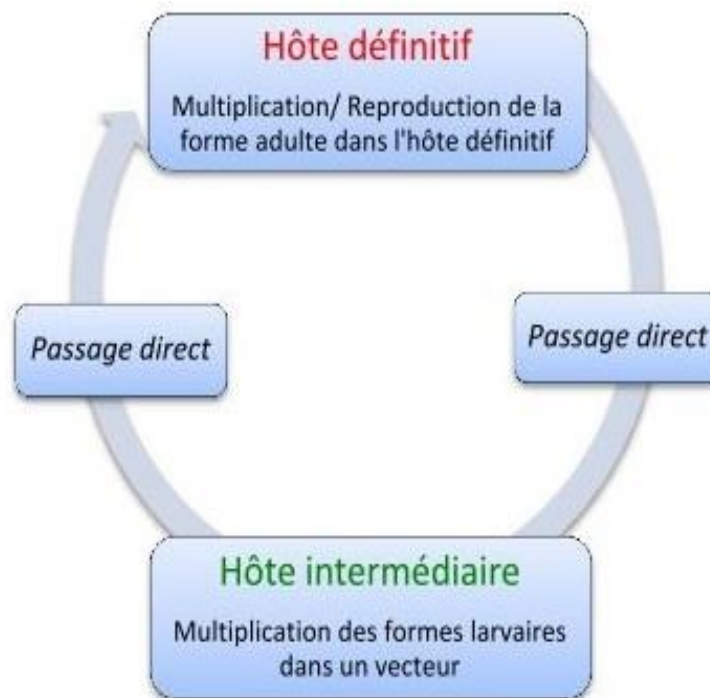


Figure 2. Heteroxenous cycle with a single intermediate host.

Example of a three-host cycle: *Diphyllobothrium latum* involving a crustacean, a fish, and humans or another mammal (piscivores) (Figure 3).

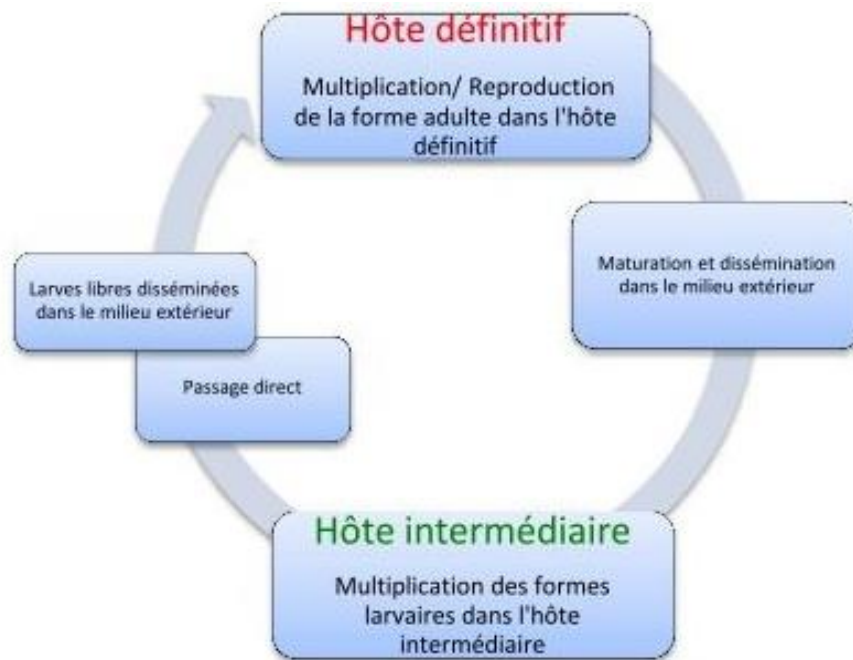


Figure 3. Heteroxenous cycle with multiple intermediate hosts.

Knowledge of parasite life cycles is essential because it allows us to deduce:

- the modes of human infestation,
- the clinical manifestations of parasitic disease,
- the diagnostic methods,
- the therapeutic indications,
- the possible prophylactic measures,
- the strategies of control.

Elements of the Parasitic Life Cycle

- **The parasite:** Protozoa or Metazoa.
- **The definitive host:** the host that harbors the adult form. Depending on the cycle, this may be humans or another mammal or animal.
- **The intermediate host(s):** The number of intermediate hosts depends on the type of parasitic cycle - none for monoxenous cycles, one or more for heteroxenous cycles. Generally, the intermediate host harbors the larval stages. The intermediate host is a living organism (human,

other mammals, crustacean, mollusk, arthropod...) in which the parasite must obligatorily remain in order to undergo the transformations that lead to its infective form.

According to the mode of transmission, we distinguish:

- **Passive intermediate hosts**, which harbor the infective form of the parasite or earlier stages and do not actively acquire or transmit the parasite to the reservoir or to a receptive host.

Example: *Lymnaea* (mollusk) for *Fasciola hepatica* (the large liver fluke), pig for *Taenia solium*.

- **Active intermediate hosts**, which seek out the parasite from the reservoir and, after transformation—particularly into infective forms—transmit it by inoculation.

- **The waiting host:** a host that harbors the larval stage of a parasite without transformation. The parasite waits until this host becomes prey for a predator in which it will continue its development.

Example: *Diphyllobothrium latum*.

- **The vector:** an animal responsible for the transmission of parasites.

- **Biological vector:** a blood-feeding and biting arthropod that acquires the parasite from the host, ensures its maturation and/or multiplication, preserves it, transports it, and finally inoculates it into a new host. It is therefore an active intermediate host.

- **Mechanical vector:** its role is limited to simple passive transport.

- **The reservoir of infection:** a living organism, or sometimes an inert substrate (soil, water), capable of maintaining a parasite for long periods and from which the parasite can be transmitted to a susceptible individual.

Three types of parasite reservoirs are recognized:

- **Human reservoir:**

- Mandatory in monoxenous cycles (example: *Enterobius vermicularis*),

- but also in some heteroxenous cycles - parasitic diseases in which humans are the reservoir are called *anthroponoses*.

- **Animal reservoir:** domestic or wild mammals, depending on the cycle, may serve as reservoirs:

- Domestic animals: e.g., cattle and sheep as reservoirs of *Fasciola hepatica*

– Wild animals: e.g., fox as reservoir of *Echinococcus multilocularis*

Parasitic diseases in which only animals (excluding humans) are reservoirs are called *zoonoses*. *Anthropozoonoses* are parasitic diseases affecting both humans and animals. In some anthropozoonoses where animals serve as reservoirs, humans may be only accidental hosts with no epidemiological role.

Example: fascioliasis due to *Fasciola hepatica*, unilocular hydatidosis due to *Echinococcus granulosus*.

➤ **Inert reservoir (soil, water):** the soil may serve as a parasite reservoir.

Example: mature oocysts of *Toxoplasma gondii*.

Position of Humans in Parasitic Life Cycles

- normal stage (e.g., *Taenia*)
- accessory stage: accidental host showing parasite development (e.g., *Dipylidium caninum*)
- **dead-end / bottleneck stage**, which may be:
 - ✓ **true dead-end:** development of the parasite stops (e.g., *Larva migrans*)
 - ✓ **conditional dead-end:** development continues only if the human host is eaten (e.g., cysticercosis).

Determinant Conditions of an Infesting Cycle

The maintenance of an epidemiological chain requires:

- the existence of a parasite reservoir (ill human, animal reservoir, or environmental reservoir),
- the possible presence of one or more essential intermediate hosts or vectors ensuring transformation and transmission of the parasite to humans,
- ecological conditions (climate, soil geophysics, fauna and flora),
- ethological conditions (behavior, sociocultural habits, professional exposure), economic and political context,
- host susceptibility (genetics, age, comorbidities, natural or acquired immune status).

General Classification of Parasites

Parasites are classified into four major groups (Figure 4):

- **Protozoa** (unicellular organisms capable of movement): depending on the case, they move by pseudopods (rhizopods), flagella, an undulating membrane, cilia, or cytoskeletal twisting

movements. They appear in an asexual or potentially sexual form, mobile and capable of division, or encysted, intra- or extracellular.

- **Helminths** (worms): metazoans (multicellular organisms with differentiated tissues); they exist in adult forms (male and female), larval, embryonic, or ovular stages.

- **Arthropods** (insects, arachnids and crustaceans), **mollusks**, **pararthropods** (porocephalans), or **annelids**: these are metazoans that may appear as adult forms (imago; male and female), eggs, larvae, or nymphs.

- **Micromycetes** (Kingdom Fungi): microscopic fungi identified as isolated or clustered spores, or as free or tissue filaments.

The different forms that parasites may adopt throughout their developmental cycles have highly diversified localizations such as body cavities, tissues, biological fluids, etc. (Table 1). These parasites may therefore induce parasitic diseases of varying severity in the hosts that harbor them (Table 2).

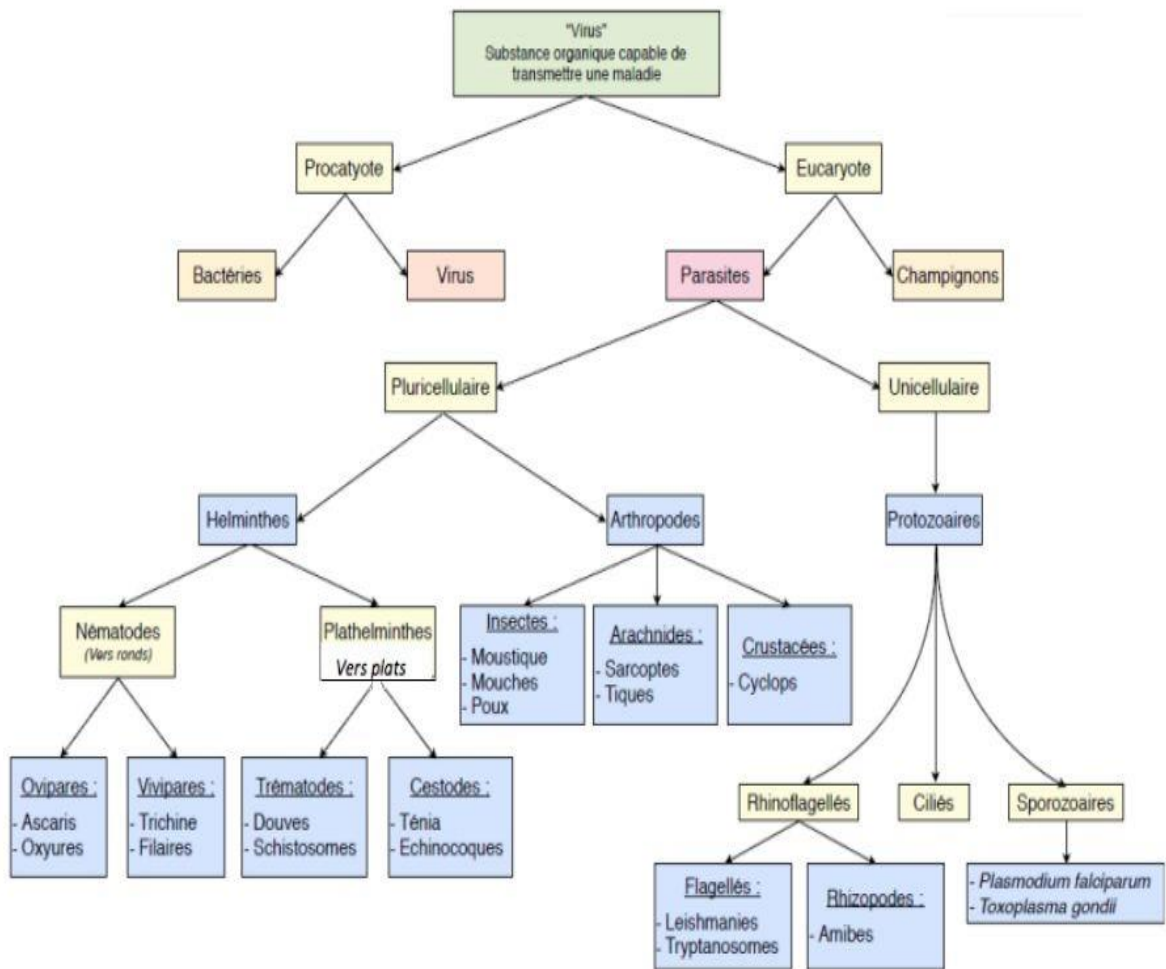


Figure 4. Simplified classification of parasites

Table 1. Parasitic forms and localization

Principaux parasites	Formes parasitaires	Localisation
<i>Plasmodium</i>	Formes intraglobulaires	Globules rouges
<i>Toxoplasma</i>	Trophozoite / Kyste	Globules blancs / cerveau
Amibes <i>Entamoeba</i>	Forme végétative /Kyste	Selles
<i>Naegleria/ Acanthamoeba</i>	Forme végétative /Kyste	Méninges / Cornée
<i>Trypanosoma</i>	Forme libre	Sang/Ganglion / LCR
<i>Leishmania</i>	Forme intracellulaire	Globules blancs / tissus
<i>Trichomonas</i>	Forme végétative	Urogénitale
<i>Balantidium</i>	Forme végétative /Kyste	Selles
<i>Giardia</i>	Forme végétative /Kyste	Selles
Microsporidies	Spore	Muqueuse intestinale
Cryptosporidies	Kyste	Muqueuse intestinale

Trichocéphales	Œuf	Selles
Oxyures	Adulte / œuf	Selles
<i>Ascaris</i>	Adulte / œuf	Selles
Ankylostomes	Œuf	Selles
Anguillules	Larve	Selles
Trichine	Pseudo-kyste	Muscles
<i>Wuchereria bancrofti</i>	Adulte ♀ / Microfilaire	Vaisseaux lymphatiques
<i>Loa loa</i>	Adulte ♀ / Microfilaire	Conjonctive
<i>Onchocerca volvulus</i>	Adulte ♀ / Microfilaire	Peau
<i>Mansonella sp.</i>	Microfilaire	Sang / Peau
Filaire de Médine	Adulte femelle	Peau
<i>Fasciola hepatica</i>	Adulte / œuf	Canaux biliaires / selles
Petite douve du foie	Œuf	Selles
<i>Paragonimus</i>	Œuf	Selles / crachats
<i>Schistosoma sp.</i>	Œuf	Selles / urines
<i>Taenia</i> du porc et du bœuf	Adulte / œuf	Selles
<i>Hymenolepis nana</i>	Adulte/œuf	Selles
Bothriocéphale	Adulte/œuf	Selles
<i>Candida sp</i>	Levures	Digestive / sang / peau
<i>Aspergillus</i>	Filaments, spores	Broncho-alvéolaire / sang
Cryptocoque	Levures	LCR, broncho-alvéolaire
Dermatophytes	Filaments	Peau, phanères
<i>Pneumocystis jirovecii</i>	Forme végétative /Kyste	Broncho-alvéolaire
Poux	Adulte / Lente	Poils
Punaises	Adulte	Literie
Puces	Adulte	Sols
Anophèles	Œuf / adulte	Eau/air
Phlébotome	Adulte	Canopée
Simulie	Adulte	Forêt / savane
<i>Chrysops</i> (Taon)	Adulte	Peau
Glossines	Adulte	Peau
Myiases	Larve	Ectoparasite
<i>Sarcoptes scabiei</i>	Œuf / adulte	Peau
Ixodidés / Argasidés	Adulte	Ectoparasite

Table 2. Classification of parasites and corresponding diseases

PROTOZOAIRES	
Embranchement des Apicomplexa (sporozoaires)	
<i>Plasmodium falciparum</i>	Paludisme
<i>Plasmodium vivax</i>	
<i>Plasmodium ovale</i>	
<i>Plasmodium malariae</i>	
<i>Plasmodium knowlesi</i>	
<i>Toxoplasma gondii</i>	Toxoplasmose
<i>Sarcocystis hominis</i>	Coccidioses intestinales
<i>Isospora belli</i>	
<i>Cryptosporidium sp.</i>	
<i>Cyclospora cayetanensis</i>	
Embranchement des Rhizoflagellés	
Classe des Rhizopodes	
<i>Entamoeba histolytica (amibe dysentérique)</i>	Amoebose intestinale et tissulaire
<i>Entamoeba dispar</i>	Amibes non ou peu pathogènes
<i>Entamoeba hartmanni</i>	
<i>Entamoeba coli</i>	
<i>Endolimax nanus</i>	
<i>Iodamaeba butschlii</i>	
<i>Naegleria fowleri</i>	Méningoencéphalites et kératites amibiennes
<i>Acanthamoeba spp.</i>	
Classe des Flagellés	
<i>Trypanosoma brucei gambiense</i>	Trypanosomoses africaines (maladie du sommeil)
<i>Trypanosoma brucei rhodesiense</i>	
<i>Trypanosoma cruzi</i>	Trypanosomose américaine (maladie de Chagas)
<i>Leishmania donovani</i>	Leishmaniose viscérale de l’Ancien Monde (kala-azar)
<i>Leishmania infantum</i>	
<i>Leishmania tropica</i>	Leishmaniose cutanée de l’Ancien Monde
<i>Leishmania major</i>	
<i>Leishmania brasiliensis</i>	Leishmaniose cutanée ou cutanéomuqueuse

<i>Leishmania mexicana</i>	américaine
<i>Giardia intestinalis</i> ou <i>Giardia duodenalis</i>	Giardiose intestinale (autrefois « lambliaose »)
<i>Trichomonas hominis</i>	Flagelloses intestinales non pathogènes
<i>Chilomastix mesnili</i>	
<i>Trichomonas vaginalis</i>	Trichomonose urogénitale
Embranchement des Ciliés	
<i>Balantidium coli</i>	Balantidiose
Position incertaine	
<i>Encephalitozoon intestinalis</i>	Microsporidioses
<i>Enterocytozoon bienewisi</i>	
<i>Blastocystis hominis</i>	Blastocystose, rarement pathogène
HELMINTHES	
Embranchement des Némathelminthes (vers ronds)	
Classe des Nématodes, ovipares	
<i>Trichuris trichiura</i> (trichocéphale)	Trichocéphalose
<i>Enterobius vermicularis</i> (oxyure)	Oxyurose
<i>Ascaris lumbricoides</i> (ascaris)	Ascariidiose
<i>Ancylostoma duodenale</i> (ankylostome)	Ankylostomoses
<i>Necator americanus</i> (ankylostome)	
<i>Strongyloides stercoralis</i> (anguillule)	Anguillulose
<i>Toxocara canis</i>	<i>Larva migrans</i> viscérale (toxocarose)
<i>Ancylostoma brasiliensis</i>	<i>Larva migrans</i> cutanée (larbish)
<i>Anisakis</i> spp.	Anisakiose
Classe des Nématodes, vivipares	
<i>Trichinella spiralis</i> (trichine)	Trichinellose
<i>Wuchereria bancrofti</i>	Filariose lymphatique de Bancroft
<i>Brugia malayi</i>	Filariose lymphatique de Malaisie
<i>Loa loa</i>	Loaose
<i>Onchocerca volvulus</i> (onchocerque)	Onchocercose
<i>Dracunculus medinensis</i> (ver de Guinée)	Dracunculose
Embranchement des Plathelminthes (vers plats)	
Classe des Trématodes	

Douves	
<i>Fasciola hepatica</i> (grande douve du foie)	Distomatoses hépatobiliaires
<i>Dicrocoelium dentriticum</i> (petite douve du foie)	
<i>Opisthorchis felineus</i>	Distomatoses intestinales
<i>Heterophyes heterophyes</i>	
<i>Paragonimus westermani</i>	Distomatoses pulmonaires
Schistosomes	
<i>Schistosoma haematobium</i>	Schistosomose (bilharziose) urogénitale
<i>Schistosoma mansoni</i>	Schistosomoses intestinales
<i>Schistosoma intercalatum</i>	
<i>Schistosoma guineensis</i>	
<i>Schistosoma japonicum</i>	Schistosomoses artérioveineuses extrême-orientales
<i>Schistosoma mekongi</i>	
Classe des Cestodes	
<i>Taenia saginata</i> (ténia du bœuf)	Tœniasis intestinal
<i>Taenia solium</i> (ténia du porc)	Tœniasis intestinal et cysticercose
<i>Diphyllobothrium latum</i>	Bothriocéphalose
<i>Hymenolepis nana</i>	Hyménolépiose
<i>Echinococcus granulosus</i>	Échinococcose hydatique
<i>Echinococcus multilocularis</i>	Échinococcose alvéolaire

II- PARASITIC PROTOZOA

The simplest organisms are unicellular animals (protozoa), which include many parasitic forms. Among the flagellates, only the Polymastigines and the Diplomonadines include free-living forms; Sporozoa are all parasitic.

2.1- Classification

The primary purpose of classifications was to assist in the identification of living organisms. As plants were more difficult to identify than animals, they were the first to be classified.

Species were classified either by grouping those that shared common characteristics or by dividing all living organisms roughly from the outset.

The classification of plants proposed by Carl Von Linné in the 18th century used both methods. He divided organisms into kingdom, class, order, but grouped species into genera and thereby invented binomial nomenclature—designating a species not by a single name but by two.

Why can living organisms be classified?

Because, according to Darwin, they are the result of evolution. Classification must therefore reflect the evolutionary history of living organisms. Until the mid-20th century, phylogeny (who is more closely related to whom) and genealogy (who descends from whom) were often mixed. In 1950, Willi Hennig introduced a rigorous and quantifiable method: **phylogenetic** or **cladistic classification**.

His method consists of creating groups of species that share a common characteristic not found in species outside the group. This shared characteristic is created by mutation; it is an innovation typical of the group, called an apomorphy, or more simply, a derived character. This innovation appeared for the first time in the common ancestor of the entire group.

The derived state of a character contrasts with its ancestral state, that is, its state outside the group.

For example, the Clade Aves (Birds) is defined by the derived character “feathers.”

Possessing four limbs is the derived character of the clade Tetrapoda. Tetrapods include the clade Mammalia, but also organisms that have four limbs but no mammary glands, such as the lizard or the frog.

The cladogram below (Figure 5) highlights the three domains of life: **Eubacteria**, **Archaeobacteria** and **Eukaryotes**.

Eukaryotes represent a particular case because their organelles (chloroplasts and mitochondria)

contain ribosomes that are more closely related to those of Archaeobacteria and Eubacteria than to those of the hyaloplasm of Eukaryotic cells.

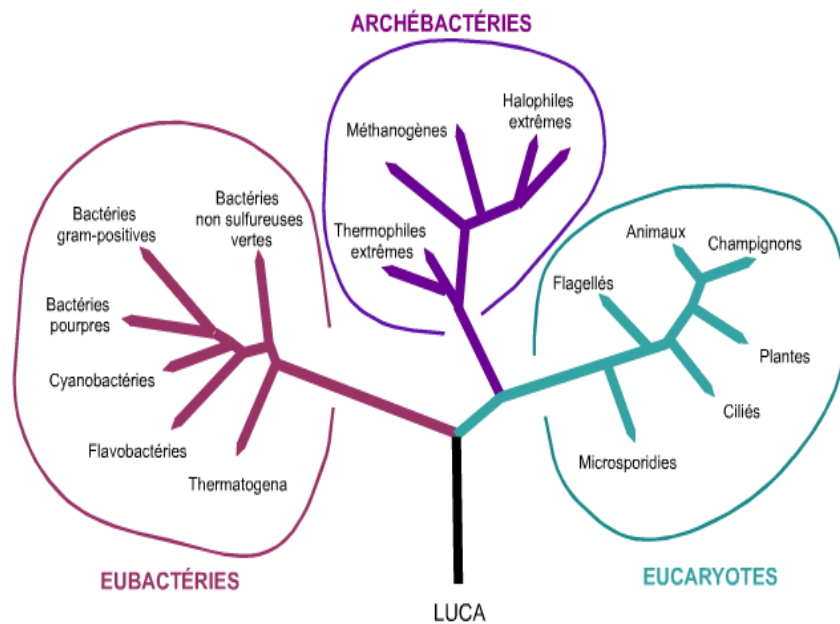


Figure 5. Phylogenetic tree of living organisms established from ribosomal RNA comparison. LUCA is the Last Universal Common Ancestor of all living organisms.

2.2– Study of the different Phyla

2.2.1 – Phylum Sarcomastigophora

Subphylum Mastigophora

Class Zoomastigophora (or Zooflagellates)

Flagellates are protozoa (unicellular eukaryotic microorganisms) equipped with flagella that serve as locomotor organelles. Several genera can be distinguished.

The classification of these groups can be represented as follows:

- **Kingdom:** Protista
- **Phylum:** Sarcomastigophora
- **Subphylum:** Mastigophora
- **Class:** Zoomastigophora (one or several flagella)

These organisms are subdivided into two distinct groups:

- **Polyflagellates**, which are found in cavities (intestinal, urogenital)
- **Monoflagellates**, found in the blood and tissues

2.2.1.1 – Order Kinetoplastida

Organisms belonging to this group are characterized by the presence of a **kinetoplast** (a differentiated part of the mitochondrion containing one-quarter of the cellular DNA; the giant mitochondrion of trypanosomatids).

The kinetoplast is always connected to the **blepharoplast** (or **kinetosome**) of the flagellum.

Suborder Trypanosomatina

Family Trypanosomatidae

Genus *Trypanosoma*

Examples of species: *T. brucei*, *T. gambiense*, *T. rhodesiense*, *T. cruzi*.

Species of the genus *Trypanosoma* are heterotrophic parasites whose developmental cycle alternates between two hosts (dixenous cycle):

- ✓ A vertebrate host (human or mammals), where they are found in the bloodstream
- ✓ An invertebrate host (insect), where they are located in the digestive tract or salivary glands

They possess a single flagellum that runs along the cell surface. This flagellum, described as recurrent, appears as an “undulating membrane” when attached to the body of the parasite.

a) Morphology of *T. gambiense*

The parasite may appear under different morphological forms depending on the host and its localization. It can present as **amastigote**, **promastigote**, **epimastigote**, or **trypomastigote** (Figure 6).

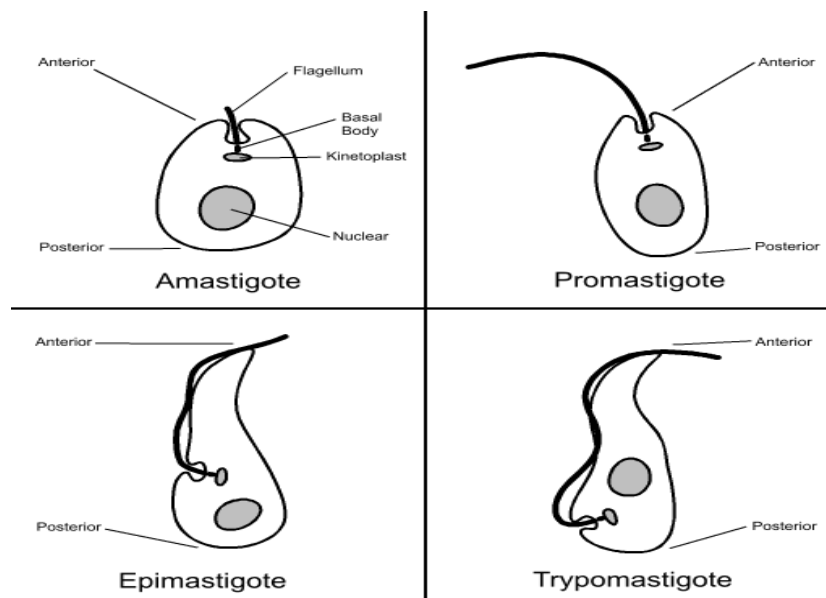


Figure 6. Different forms of the trypanosome.

b) Developmental cycle

This parasite, transmitted to mammals by the tsetse fly (*Glossina*), causes African trypanosomiasis, also known as sleeping sickness (as opposed to American trypanosomiasis, or Chagas disease, caused by *Trypanosoma cruzi*). Some parasitologists classify it as a subspecies of *Trypanosoma brucei brucei* (which is non-pathogenic).

In this case, we are dealing with *T. brucei gambiense* or *T. brucei rhodesiense*, which are pathogenic species.

• In humans and other mammals

The slender trypomastigote form is found in the blood, cerebrospinal fluid, and lymph nodes after the bite of the tsetse fly.

Amastigote (or micromastigote) forms may be detected in the choroid 48 hours after infection. They reportedly transform rapidly into spheromastigotes, which then give rise to bloodstream trypomastigotes. These forms multiply actively through binary fission and produce stumpy trypomastigotes.

• In the tsetse fly (which is both vector and intermediate host)

The *Glossina* fly, feeding on an infected host, ingests stumpy trypomastigotes, which develop in the midgut of the vector and produce free trypomastigotes.

These then transform into epimastigotes, with the kinetoplast positioned anterior to the nucleus. After multiple binary divisions, the epimastigotes give rise to metacyclic trypomastigotes, which migrate from the gut to the salivary glands of the insect.

If a metacyclic trypomastigote is injected into a human during the next blood meal of the vector, the cycle starts again (Figure 7).

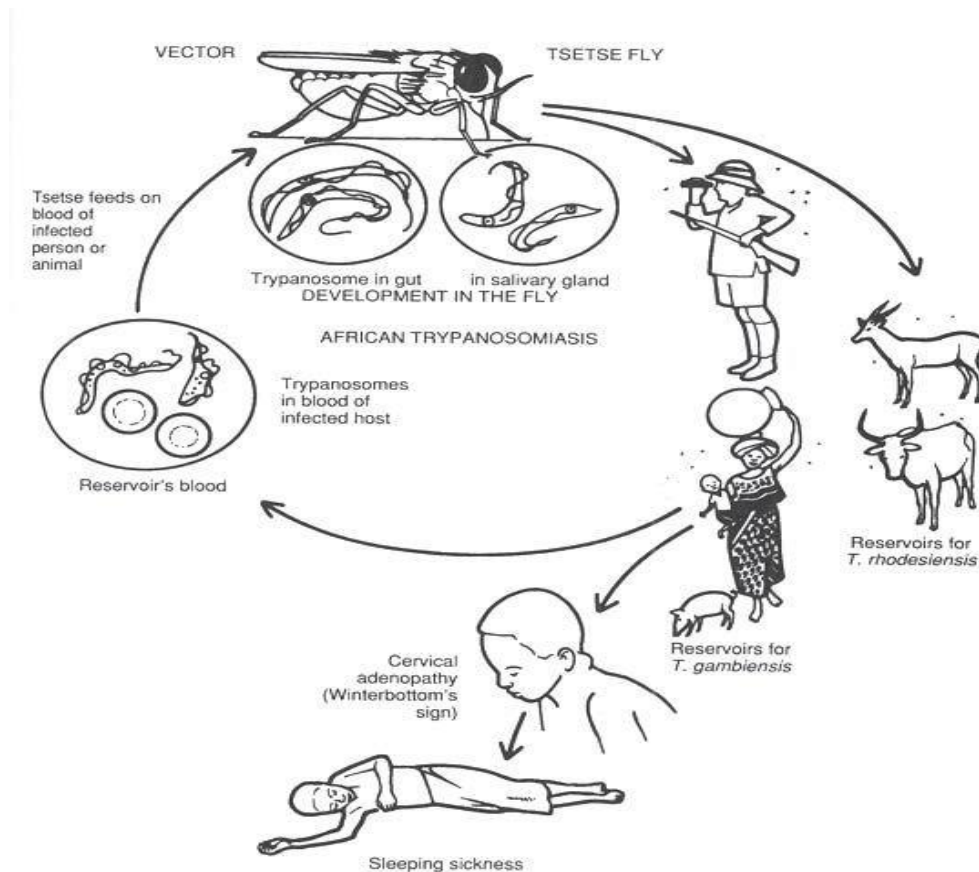


Figure 7. Life cycle of *T. brucei gambiense*

c) Sexual multiplication

Syngamy (the sexual process involving the fusion of two gametes, one male and one female, to form a zygote) occurs shortly after the ingestion of the stumpy forms, and meiosis takes place in the salivary glands.

Suborder Trypanosomatina

Family Trypanosomatidae

Genus *Leishmania*

Examples of species: *L. major*, *L. infantum*, *L. braziliensis*, *L. donovani*, *L. chagasi*.

The amastigote form is found in the definitive (vertebrate) host,

The promastigote form is present in the insect intermediate host (*Phlebotomus* in the Old World and *Lutzomyia* in the New World).

a) Morphology

Two distinct forms exist:

- **Amastigote forms** (= without flagellum), which are ovoid, 2–3 μm in diameter, with a nucleus and a kinetoplast, with or without the short root of a flagellum (rhizoplast).

Reminder: the kinetoplast is a giant mitochondrion containing DNA, and the cytoskeleton is composed of peripheral microtubules (Figure 8).

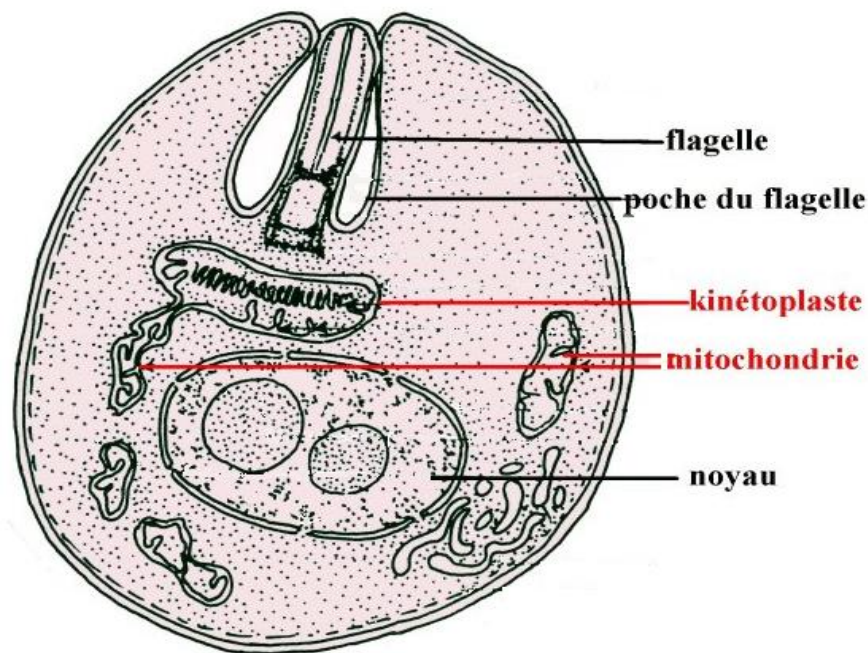


Figure 8. Amastigote form of *Leishmania* sp.

Multiplication occurs by binary fission. These forms are non-motile, obligatorily intracellular. They are present in the vertebrate definitive host.

- **Promastigote forms** are elongated, flagellated, measuring 8–24 \times 4–5 μm and highly motile, moving with the flagellum forward;
The kinetoplast is located at the base of the flagellum;
The flagellum measures 10 -15 μm (Figure 9). Multiplication occurs by binary fission. These forms are found in the digestive tract of intermediate hosts and in *in vitro* culture.

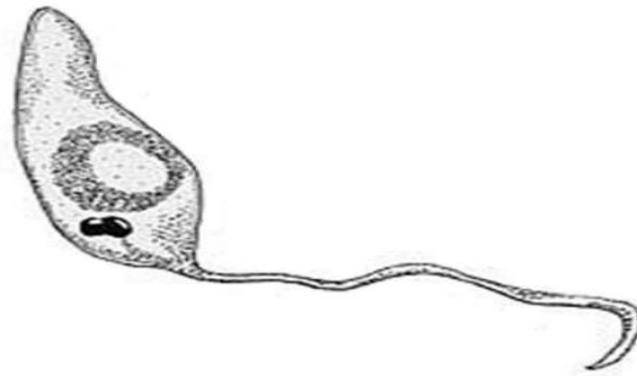


Figure 9. Promastigote form of *Leishmania* sp.

b) Evolutionary cycle:

The cycle is indirect.

DH (Definitive host): humans and animals (dogs, wild rodents)

Amastigote forms are located in the cells of the Reticuloendothelial System (RES), multiplication occurring by binary fission and spreading progressively from cell to cell.

IH (Intermediate host): blood-sucking insects of the sand fly group (genus *Phlebotomus* in the Old World and *Lutzomyia* in the New World). Only the female is hematophagous.

The parasite is ingested during a bite (during a blood meal) and reaches the digestive tract of the insect. The amastigote forms then transform into promastigotes.

The parasites multiply and migrate to the anterior part of the digestive tract (proventriculus or pharynx, depending on the sub-genus), becoming infectious promastigote forms (Figure 10).

During a subsequent bite, the insect regurgitates and injects the promastigote forms into the wound; transformation into intracellular amastigote forms occurs within a few minutes.

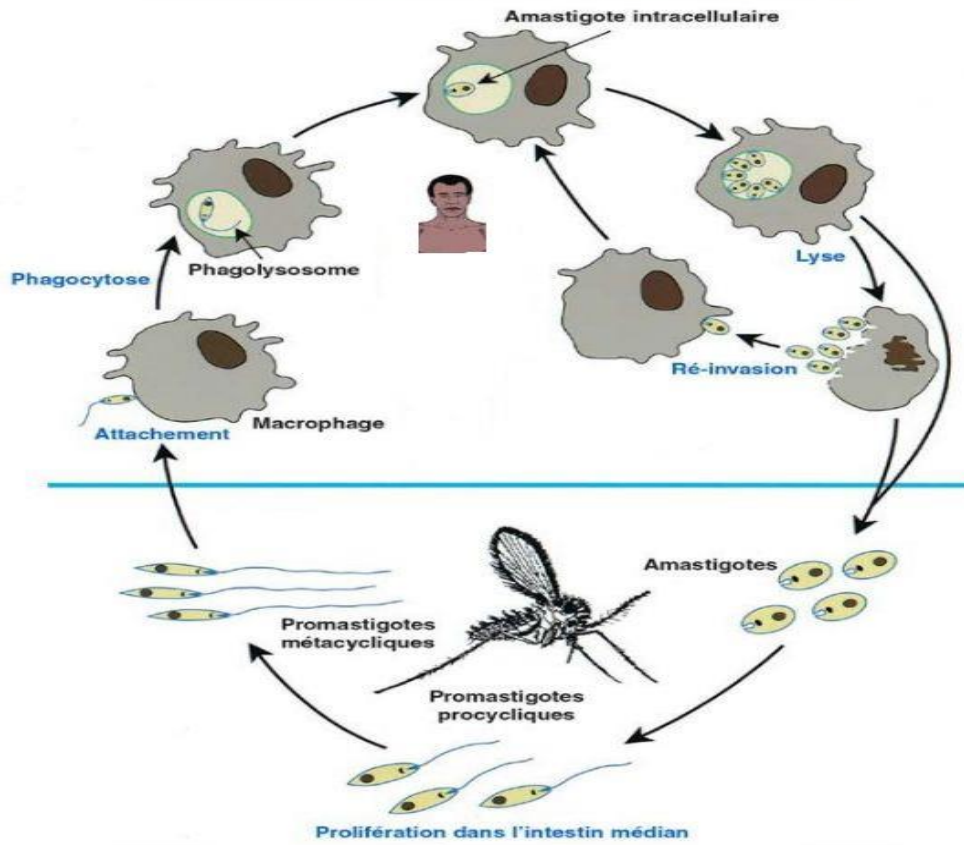


Figure 10. Evolutionary cycle of leishmaniasis

c) Epidemiological aspects:

– **IH “Sand fly” (Phlebotomine):** Diptera, Nematocera, Psychodidae. Only the female is hematophagous (Figure 11). The biological cycle is terrestrial (larvae develop in moist soil). Sand flies are nocturnal and crepuscular. Their flight, clumsy, is silent. They are mainly found in holes, cracks, burrows, and woodland edges.

They are very small, smaller than mosquitoes, about 2 mm in length.

They are present during summer in temperate zones (from May to October) and throughout the year in tropical regions.

Multiplication of the parasite in the lumen of the digestive tract lasts 8 to 20 days.

Infection occurs by regurgitation of the infectious promastigote forms from the pharynx during intense suction efforts.



Figure 11. Female sand fly (phlebotomine) taking a blood meal.

– Anthrozooses in general, and anthroponoses in certain foci. A disease of wild rodents or dogs, depending on the case, transmitted accidentally to humans.

Endemo-epidemic or sporadic. Rural or urban.

This parasitic infection is quite frequent in certain countries (India, Nepal, Bangladesh, Sudan, Ethiopia, Brazil, Afghanistan and Iran).

It is also widely distributed in North Africa (Figure 12).

Two clinical entities exist:

- Visceral leishmaniasis (VL), or Kala-azar, which is fatal;
- Tegumentary leishmaniases, including:
 - Localized cutaneous leishmaniasis (LCL), spontaneously curable;
 - Diffuse cutaneous leishmaniasis (DCL), resistant to all treatments;
 - Mucocutaneous leishmaniasis (MCL), severely mutilating.

Cutaneous leishmaniasis has various local names depending on the region: “Oriental sore” or “Biskra boil” on the Mediterranean coasts, and “Aleppo boil” in the Middle East.

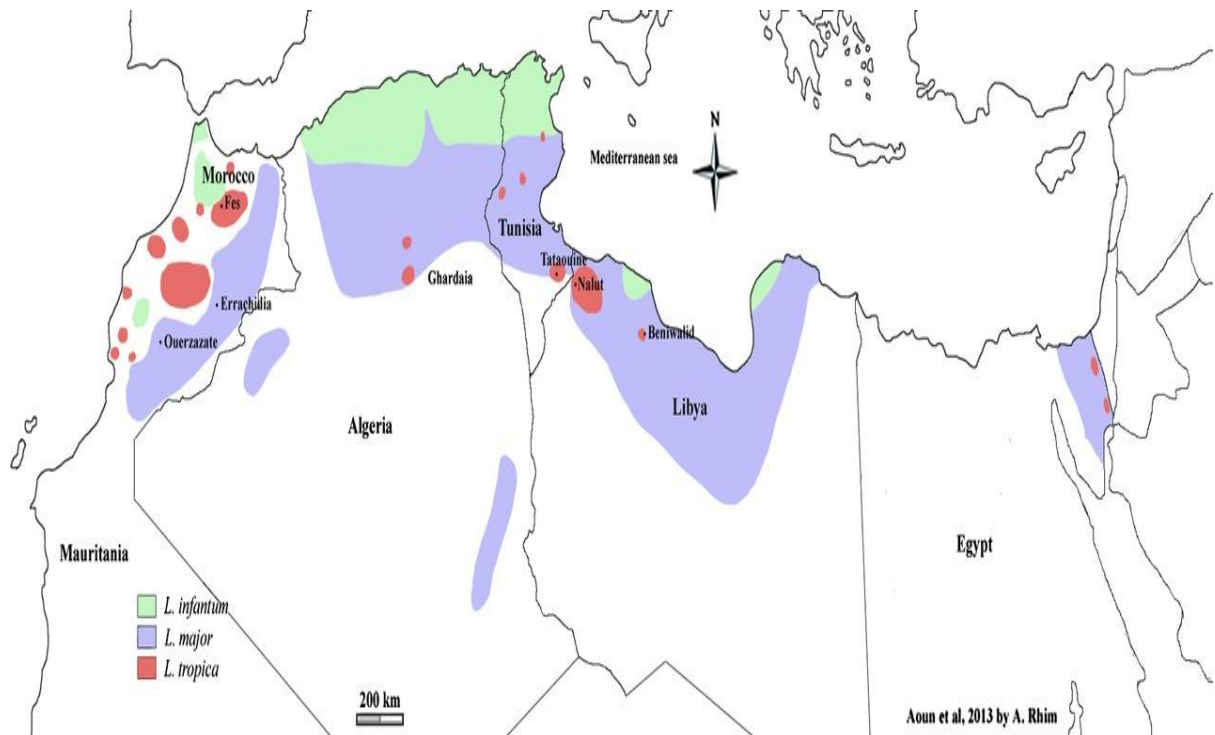


Figure 12. Geographical distribution of cutaneous leishmaniasis cases due to *Leishmania infantum*, *Leishmania major*, and *Leishmania tropica* in North African countries.

2.2.1.2 – Order Retortamonadida

Genus *Chilomastix*

Chilomastix mesnili is a flagellated protozoan parasite of pigs and primates in the broad sense (including humans). Synonyms include *Chilomastix suis* and *Chilomastix hominis*.

It is a common intestinal parasite causing chilomastosis, although its pathogenic role remains debated.

Living on the mucosa of the large intestine (colon), the vegetative form multiplies actively there and proliferates during episodes of diarrhea. When intestinal function returns to normal, it encysts and disappears until the next episode.

The trophozoite can be detected by stool parasitological examination (soft stools or diarrhea).

a) Morphology

Chilomastix mesnili, in its vegetative form, is a very mobile, pear-shaped parasite.

The trophozoite measures 12–18 μm in length and 5–6 μm in width.

It has no axostyle, no undulating membrane, but possesses an external spiral groove.

The posterior end is tapered and flexible.

The trophozoite bears three unequal anterior flagella and a small recurrent flagellum located free within the anterior cystosomal pouch (*Figure 13*).

The cyst, smaller in size (6–8 μm \times 4–6 μm), is rounded (lemon-shaped) with a small anterior knob.

Its double wall contains a large nucleus on one side and flagellar remnants on the other.

The cysts of *Chilomastix mesnili* are pear-shaped and small (6–10 μm), with a thin and refringent wall.

They show a nucleus and a juxtannuclear organelle (cytostome) where the flagellum is inserted; however, this structure is difficult to visualize (*Figure 13*).

The internal contents of the cyst are more easily observed when using iron-hematoxylin staining.

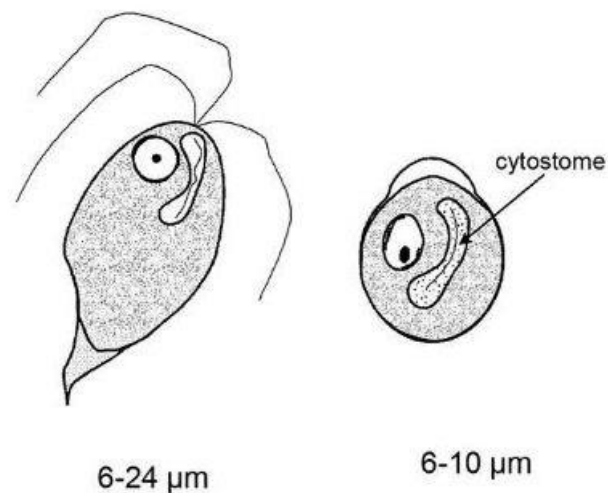


Figure 13. Forms of *Chilomastix mesnili* (trophozoite and cyst)

b) Life cycle

The cyst is resistant to environmental pressures and is responsible for the dissemination of *Chilomastix*.

Both cysts and trophozoites can be found in fecal samples (diagnostic stages).

Infection occurs through the ingestion of cysts contained in contaminated water or food, or via the fecal - oral route (contaminated hands or objects).

In the large intestine (and possibly the small intestine), excystation releases trophozoites.

Chilomastix resides in the cecum and/or colon.

It is generally considered a commensal organism, and its pathogenic role remains uncertain (*Figure 14*).

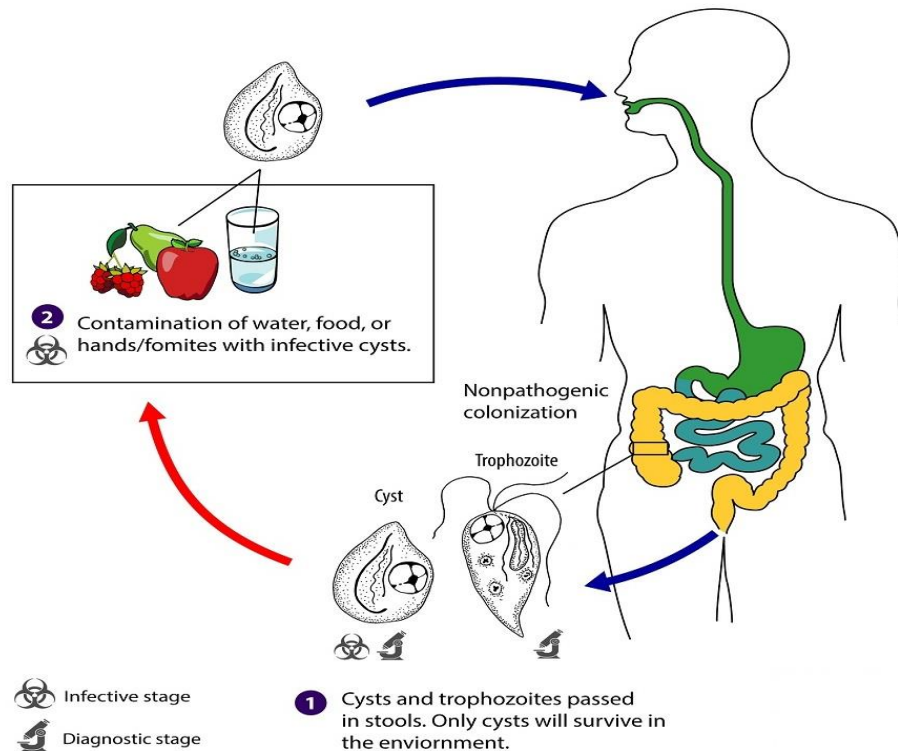


Figure 14. Life cycle of *Chilomastix mesnili*.

c) Epidemiology

Chilomastix mesnili is a cosmopolitan parasite that is more frequently encountered in temperate climates.

Chilomastosis is more prevalent in regions with inadequate sanitation.

Clinically, *Chilomastix mesnili* is considered non-pathogenic. However, the presence of cysts and/or trophozoites in stool samples may indicate fecal contamination of a water or food source and therefore does not exclude the possibility of other parasitic infections.

2.2.1.3 – Order Diplomonadida

Suborder: *Diplomonadina*

Genus: *Giardia*

a) Morphology

Giardia intestinalis exists in two known forms:

- **Trophozoite:** symmetrical “kite-shaped” body tapered posteriorly, measuring 10 to 20 $\mu\text{m} \times 6\text{--}10 \mu\text{m}$ and dorsoventrally flattened.

It possesses eight flagella (six anterior + two posterior), two nuclei, two parabasal bodies (Golgi

apparatus), and an anterior ventral adhesive disc.

The vegetative form is highly motile (*Figure 15*).

- **Cyst:** this form measures $12 \times 8 \mu\text{m}$, is ovoid, and has a thin, clear, smooth, refringent wall.

The cyst contains two nuclei at shedding, a flagellar bundle aligned along the axis, and two comma-shaped parabasal bodies.

After 24–48 hours in the environment, it contains four nuclei (*Figure 15*).

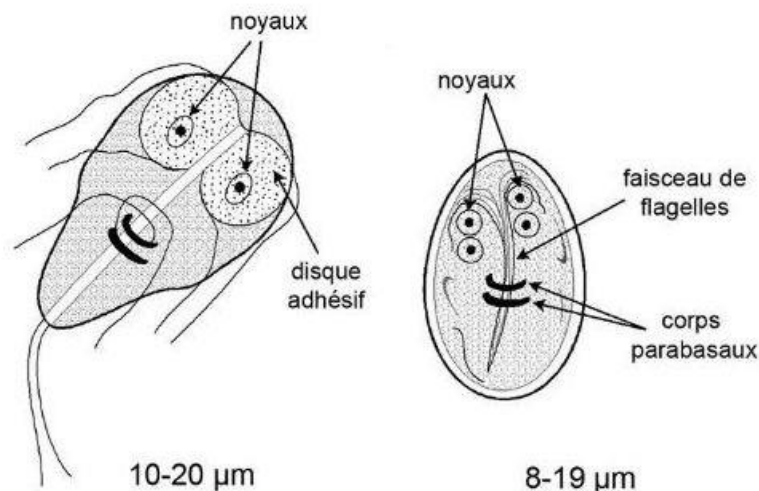


Figure 15. Forms of *Giardia intestinalis* (trophozoite and cyst)

b) Life cycle

The life cycle is direct. The definitive host (DH) is humans (and other animals).

Multiplication occurs by binary fission in the lumen of the duodenal region, in the flagellated form.

Cysts, formed irregularly, are passively eliminated with the feces. Maturation takes place in the external environment.

Infection occurs through ingestion of four-nuclei cysts via contaminated water or food (fecal - oral transmission).

Excystation takes place in the duodenum, where trophozoites attach to the intestinal epithelium (*Figure 16*).

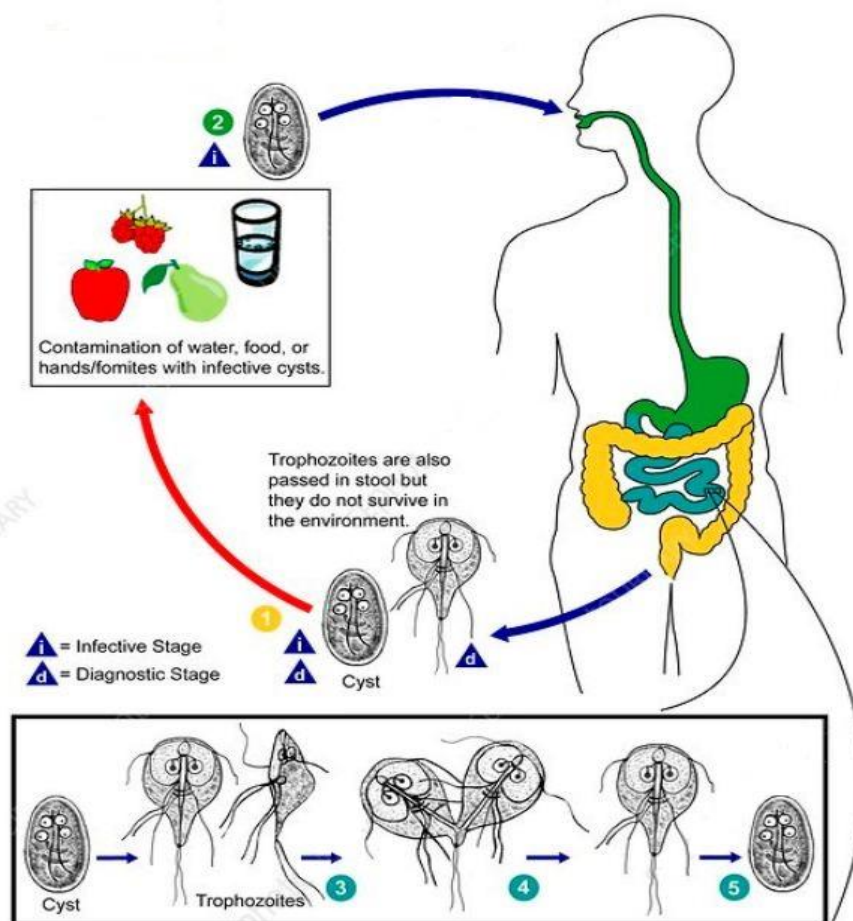


Figure 16. Forms of *Giardia intestinalis*

c) Epidemiology

Giardiasis is a cosmopolitan infection, associated with fecal contamination, in which transmission of cysts occurs through contaminated food, polluted water, and dirty hands. The reservoir of the parasite is humans (and sometimes animals).

Predisposing factors include hypochlorhydria, immune deficiency, and young age.

The prevalence ranges from 1.5% to 20% in humans, depending mainly on hygiene levels; prevalence decreases in breast-fed young children (reported in studies from Mexico).

The cyst is highly resistant to cold (2 months at +8°C) and to chlorine.

2.2.1.4 – Order Trichomonadida

Genus *Trichomonas* (*T. vaginalis*)

Urogenital trichomoniasis is a sexually transmitted infection (STI), benign, cosmopolitan and common, caused by *Trichomonas vaginalis*, a flagellated protozoan parasite of the urogenital tract.

a) Morphology

The trophozoite is oval to spherical, measuring 7–30 μm long \times 5–12 μm wide. It presents an axostyle, an oval nucleus located at the anterior part of the body, and a blepharoplast from which emerge four free anterior flagella and one short posterior flagellum forming a short undulating membrane, extending up to two-thirds of the body length (*Figure 17*).

The flagella ensure motility; the trophozoite rotates on itself. When temperature decreases, the vegetative form rounds up and mobility diminishes.

Reproduction occurs by binary fission, and no sexual reproduction takes place.

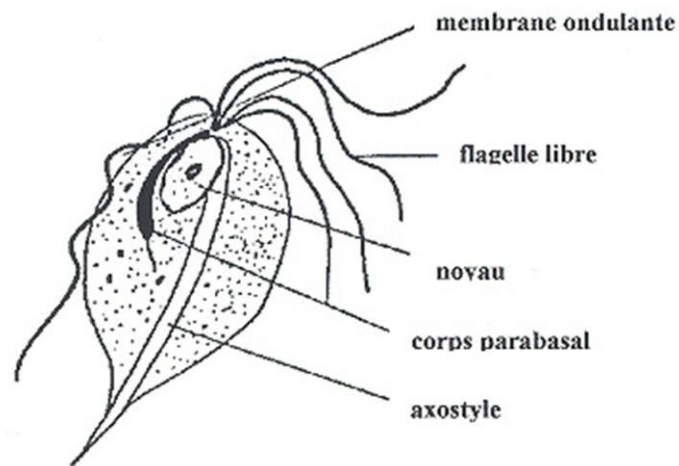


Figure 17. Trophozoite of *Trichomonas vaginalis*

b) Life cycle

The parasitic cycle is direct (*Figure 18*).

The parasite, strictly human, is located in the male and female genital tract.

Trichomonas vaginalis exists only in the trophozoite (vegetative) form, and dies rapidly in the external environment.

There is no cystic form responsible for dissemination in the external environment in *Trichomonas* spp.

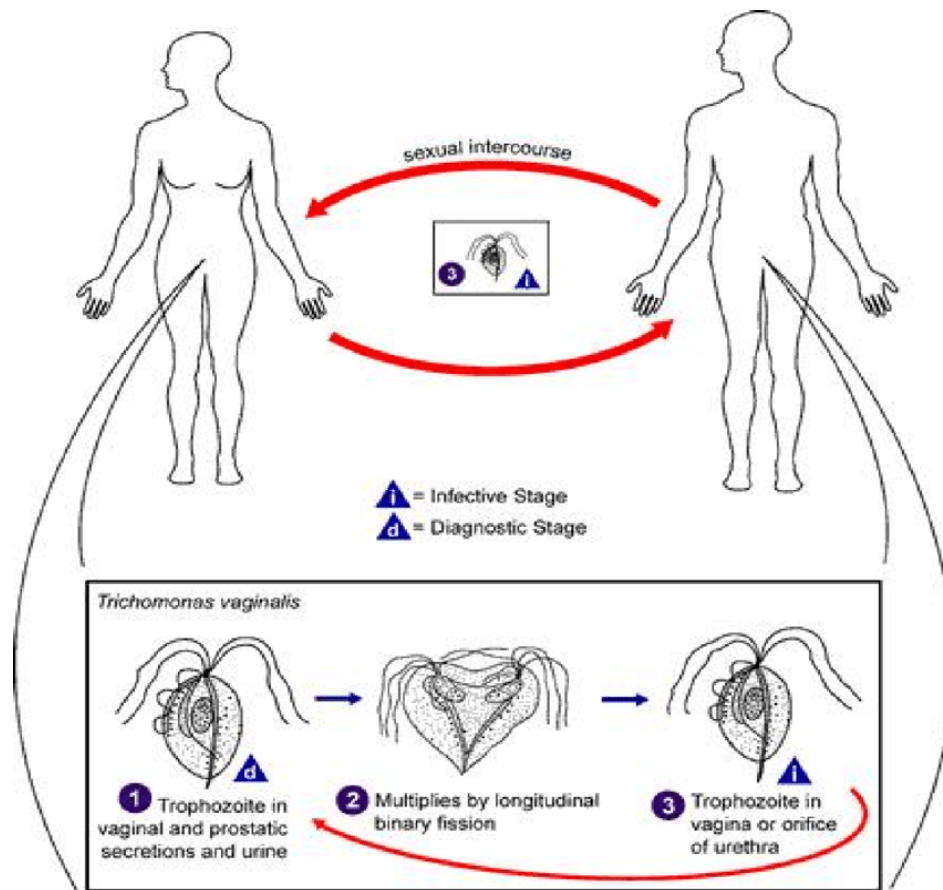


Figure 18. Life cycle of *Trichomonas vaginalis*

Highly sensitive to desiccation, transmission from one individual to another can only occur in a moist environment. The parasite can survive 1-2 hours on a moist surface, and up to 24 hours in urine or semen.

Transmission is direct, most often through sexual contact.

Optimal growth conditions for the parasite are a temperature of 35–37°C, pH between 5.5 and 6, and anaerobic conditions.

c) Epidemiology

- Parasite reservoir: male individuals of the human species
- Low resistance of trophozoites in the external environment
- Prevalence: 15–25% among adult women (up to 50% in certain African countries and Haiti)
- Urogenital trichomoniasis is a sexually transmitted infection (STI), cosmopolitan and very common

Subphylum Sarcodina

Superclass Rhizopoda

2.2.1.5 – Order Amoebida

Family Entamoebidae

Genus *Entamoeba* (*E. histolytica*)

Amoeba (from Greek ameibein, meaning *to change*, referring to protozoa that change shape)

a) Morphology

The **trophozoite** is the vegetative and multiplying form. In *Entamoeba histolytica*, the trophozoite displays dimorphism (*Figure 19*):

- **Minuta form:** 10–15 μm , finely granular endoplasm, refringent ectoplasm, peripheral nucleus (3–4 μm) with a small central karyosome and peripheral chromatin in a thin rim or dot-like pattern.

In a fresh mount, movement is lively and pseudopods are long.

Optimal temperature for this form is below 37°C.

- **Histolytica form:** 20–30 μm (up to 40 μm), same general appearance as the minuta form, but with additional features: presence of erythrocytes in digestion inside cytoplasmic vacuoles.

This form withstands 37°C well.

The cyst represents the passive dissemination and resistance form in the external environment. It is spherical, refringent, colorless, with thin walls, measuring 10–14 μm in diameter, containing four nuclei at maturity, and a thick chromatoid body with rounded ends.

Immature cysts contain one or two nuclei and a vacuole, and are larger (*Figure 19*).

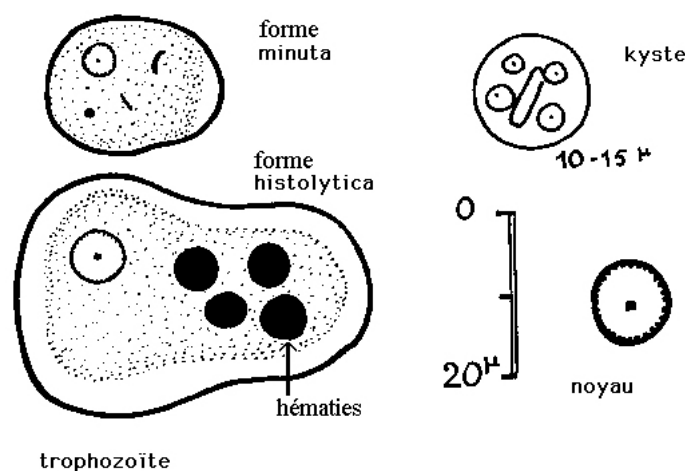


Figure 19. Forms of *Entamoeba histolytica*.

b) Developmental cycle:

The cycle is direct.

These are obligatory monoxenous parasites of humans. Their passive transmission occurs through ingestion of mature cysts.

⇨ **Non-pathogenic cycle.** After ingestion of a mature 4-nuclei cyst, excystation occurs with the release of 8 *minuta*-type amoebulae, which multiply by binary fission in contact with the colonic mucosa.

Intermittent and irregular elimination into the external environment occurs in the form of fecal cysts.

⇨ **Accidental pathogenic cycle.** *Minuta* forms in the colon transform into *histolytica* forms, which cause mucosal abscesses; they multiply by scissiparity within the abscesses.

No cyst formation occurs during this stage, and therefore there is no direct epidemiological role. The return of the parasite to the *minuta* form after approximately 3 weeks signals the end of the amoebic crisis.

Hematogenous and/or lymphatic metastasis from a colonic abscess may lead to extra-colonic localizations (liver, lung, brain, etc.).

In extra-colonic localizations, there is no return to the *minuta* form (Figure 20).

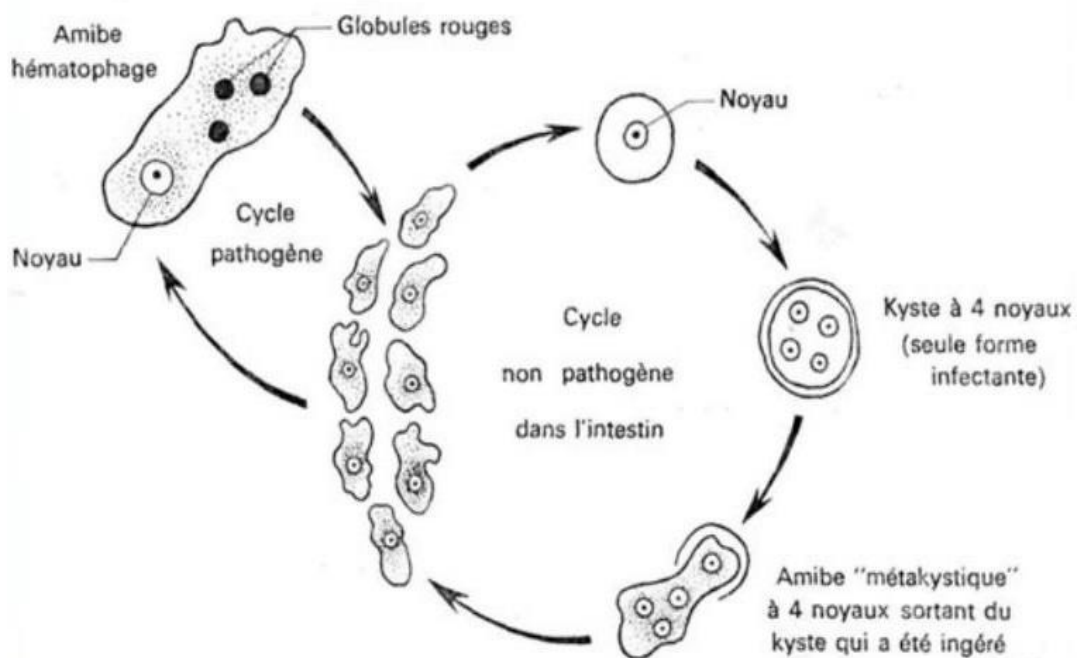


Figure 20. *Entamoeba histolytica* life cycle in the human host.

c) Epidemiology:

Amoebiasis is a disease linked to human fecal contamination — meaning fecal pollution of the environment caused by human excreta that contaminate water, raw vegetables, hands, soil, and are transported by flies (Figure 21).

The reservoir of the parasite is humans.

The cyst survives for at least 15 days in water at 18°C, 10 days in stools, and 24 hours in dry conditions. It is highly resistant to chemical agents.

The vegetative forms are too fragile to survive in nature and therefore play no epidemiological role.

Amoebae are cosmopolitan, but the parasitosis is endemic in hot and humid countries, especially in developing countries where fecal hygiene is poorly practiced and where human excreta are used as fertilizer.

The prevalence of this parasitosis is worldwide: about 10% of the world population, nearly 600 million carriers, with 90% being asymptomatic carriers, who represent the main source of transmission.

Experimental transmission to animals is possible in young cats through rectal inoculation and anal collodion application. In rats and hamsters, intra-caecal or intra-hepatic inoculation is possible. Amoebae develop abscesses but never produce minuta forms nor cysts in these animals.

Clinically, two species of amoebae are distinguished:

- *Entamoeba histolytica*, the virulent form, responsible for clinical disease;
- *Entamoeba dispar*, avirulent and non-pathogenic, likely more common in temperate regions.

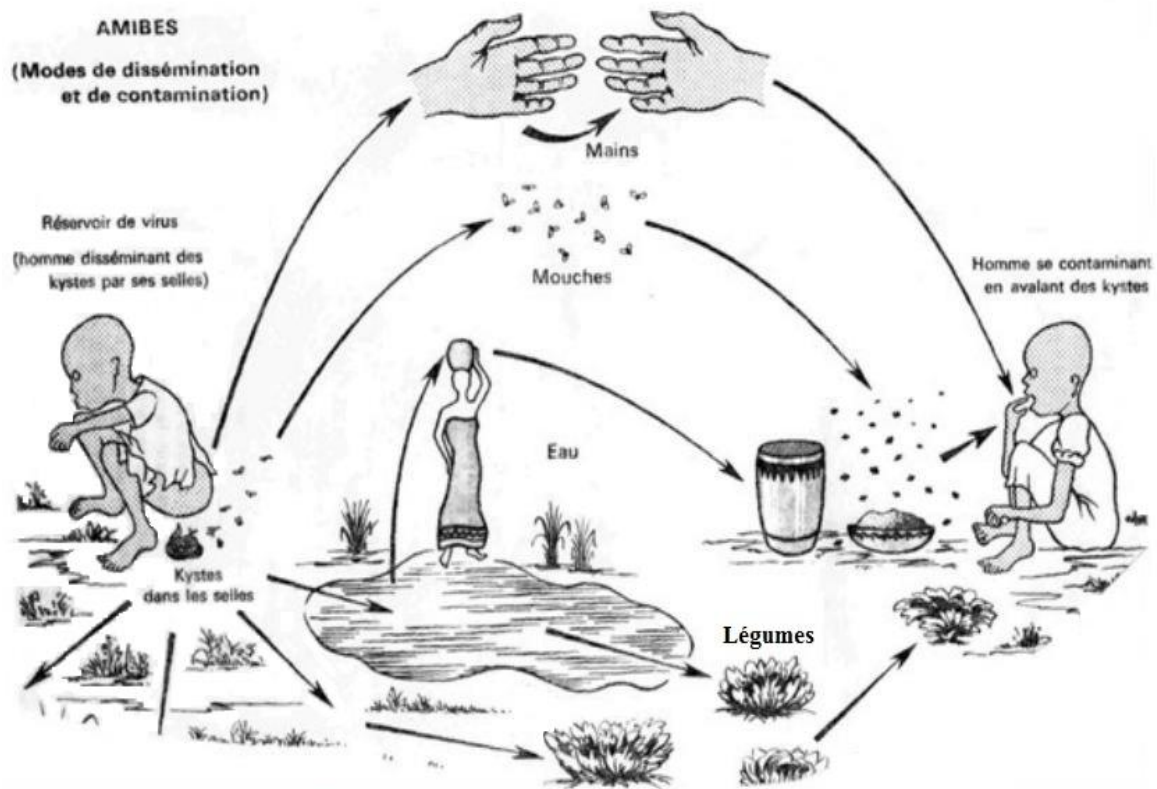


Figure 21. Dissemination of *Entamoeba histolytica*

2.2.2 – Phylum Apicomplexa

Class *Sporozoea*

Subclass *Coccidia*

Order *Eimeriida*

2.2.2.1 – Suborder Eimeriina

Family *Sarcocystidae*

Genus *Toxoplasma* (*T. gondii*)

Toxoplasmosis is a cosmopolitan protozoan infection caused by *Toxoplasma gondii*, an intracellular parasite. The congenital form is responsible for very severe malformations, whereas the acquired form is benign in 95% of cases.

a) Morphology:

Vegetative form:

Under light microscopy, the tachyzoite appears crescent-shaped (arc-shaped — *toxon* in Greek), measuring 5-8 μm long and 2-3 μm wide. The anterior end is pointed compared to the posterior end, which is broader and rounded.

The parasite shows a clear cytoplasm surrounded by a thin membrane and a single large nucleus. A large karyosome occupies the entire nuclear surface.

The tachyzoite exhibits lateral movements of the apical region that allow it to penetrate host cells of the reticulo-histiocytic system (Figure 22). This form is responsible for the acute septicemic phase and for fetal contamination.

Tissue cyst:

This is the latent and resistant form of the parasite, located in nervous and muscular tissue throughout the lifetime of the intermediate host. It appears as spherical formations ranging from 10 μm to several tens of microns in diameter. Its thick wall surrounds a large number of bradyzoites with slow metabolic activity and reduced division.

Oocyst:

This is an ovoid resistant form in the external environment (maturation in soil), measuring 10–12 μm .

- In the non-sporulated stage, it contains a granular mass of cells.
- When mature, the oocyst has a two-layered wall enclosing two sporocysts, each containing four sporozoites (Figure 22).

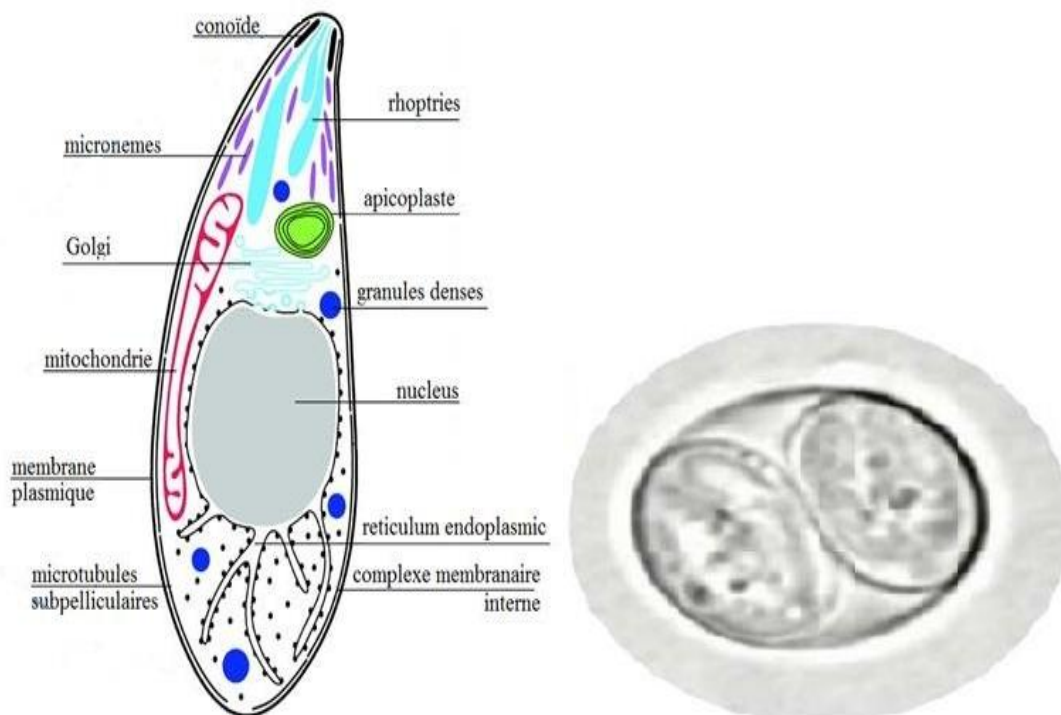


Figure 22. Tachyzoite and oocyst forms of *Toxoplasma gondii*.

b) Life cycle of toxoplasmosis:

As with all sporozoans, the life cycle is divided into two major phases: gametogony and schizogony.

The first phase is sexual and takes place entirely in the definitive host (felids), while the second is asexual and begins in the cat but is completed in the intermediate host, which is a warm-blooded animal (mammal or bird) (Figure 23).

• Schizogonic phase:

This is the phase during which the production of toxoplasms is the highest. It takes place almost entirely in warm-blooded intermediate hosts. This step is responsible for the acquired and congenital complications of the disease.

In the intermediate host:

After ingestion of the infective form, the sporozoites or bradyzoites transform into tachyzoites, cross the intestinal lamina propria and disseminate via the bloodstream or lymphatic system throughout the reticulo-histiocytic system.

Inside the host cells, they multiply actively and cause septicemia.

Entry into host cells occurs actively through parasite motility, which requires energy. Once inside, the toxoplasma multiplies by endodyogeny (two daughter cells form inside each parasite). It is during this stage that fetal contamination may occur.

The immune system responds by producing specific antibodies. The parasite then becomes encysted in tissues with low immunoglobulin concentrations, mainly the nervous system and muscle.

The cysts remain quiescent in the organism of the intermediate host for its entire lifetime. This quasi-constant immunological stimulation in the tissues is responsible for a residual and protective antibody level in the bloodstream.

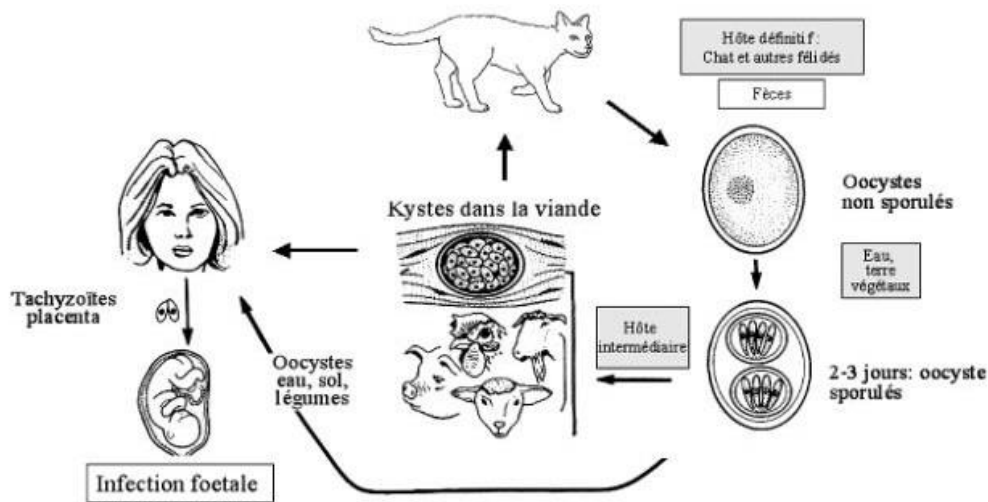


Figure 23. Diagram of the life cycle of *Toxoplasma gondii*

In the definitive host:

The asexual schizogonic phase continues in the cat after ingestion of cysts contained in tissues. Digestion releases the bradyzoites, which transform into tachyzoites in the cat's digestive tract. They then penetrate intestinal cells where they multiply.

These cells eventually burst, releasing dozens of toxoplasms that invade other enterocytes. After several intestinal schizogonies, sexual parasitic forms appear: gametocytes.

• Gametogonic phase:

Female macrogametes (5-7 μm) and male microgametes (3 μm), which are falciform and possess three flagella, are formed. The production of the unsporulated oocyst occurs in the external environment within the feces of the feline.

Oocyst maturation requires oxygen and humidity.

Oocysts are dissemination forms found in the environment and represent a very important soil reservoir, responsible for the contamination of herbivores.

c) Epidemiology:

Toxoplasmosis is a cosmopolitan zoonosis. Humans can be infected by all parasitic forms. Contamination of humans and herbivores may occur through raw vegetables, fruits or water contaminated with oocysts, or through contact with cats or their litter.

Congenital infection occurs via tachyzoites, which cause placental lesions. Fetal malformations are more severe when infection occurs early in pregnancy.

Congenital transmission, the most serious form, depends largely on dietary and culinary habits of populations.

The main sources of human infection are:

- **Ingestion of mature oocysts from soil** (contaminated water, soiled food, dirty hands).

Oocysts can remain infective in the environment for several months.

They are sensitive to desiccation and to heat (50°C for half an hour) and are also killed by freezing.

They survive one hour in absolute alcohol, 0.5N sulfuric acid, 6% sodium hydroxide, and one day in 10% formalin, demonstrating their strong resistance to harsh environmental conditions.

About 2% of cats shed oocysts for 1 to 3 weeks (mostly young cats). Sporulation conditions: 1 to 5 days at 20°C in the presence of oxygen. Oocysts remain viable for months in soil and resist HCl.

- **Ingestion of viable cysts in raw or undercooked meat** is also a major oral route of infection. Prevalence in butchered meat: 80% of adult sheep and goats infected; <40% in pigs. Cysts can survive several days at room temperature. They remain viable for several months at +4°C. Freezing at -20°C kills bradyzoites within thirty minutes. Cooking to +70°C or microwave heating destroys infective forms.

2.2.2.2 – Suborder Haemosporina

Phylum: APICOMPLEXA ; Class: SPOROZOEIA ; Subclass: COCCIDIA ; Order:

EUCOCCIDA ; Suborder: HAEMOSPORINA ; Family: PLASMODIIDAE

Genus *Plasmodium* (*P. falciparum*, *P. vivax*, *P. malariae*, *P. ovale*, and *P. knowlesi*)

Malaria is a parasitic disease caused by hematozoa of the genus *Plasmodium*, transmitted by mosquitoes of the genus **Anopheles**. This disease is of major importance not only to populations living in endemic areas (intertropical zone) but also to travellers.

Global significance

- In 2009, malaria remained the world's leading parasitic endemic disease. Nearly half of the world's population lives in endemic areas.
- The annual number of malarial episodes, although decreasing, is estimated to range from 250 to 500 million, resulting in the death of about 750,000 to 1 million people, mostly young children living in sub-Saharan Africa.

- Malaria represents a heavy financial burden for affected populations and is therefore a major obstacle to the development of concerned countries, particularly in Africa.

For these reasons, the fight against malaria constitutes - along with the fight against HIV/AIDS and tuberculosis - one of the “Millennium Development Goals” defined by the United Nations. The Global Fund aims to provide requesting countries with antimalarial drugs.

a) Pathogen:

Malaria is transmitted by a protozoan of the genus *Plasmodium*. More than 140 *Plasmodium* species exist and infect various animal species, but only five infect humans:

Plasmodium falciparum, *P. vivax*, *P. ovale*, *P. malariae* and *P. knowlesi*, the latter being a parasite normally infecting Asian macaques and recently crossing over to humans.

These five species differ in biological and clinical characteristics, geographic distribution and their ability to develop resistance to antimalarial drugs.

A distinction must immediately be made between *P. falciparum* and the other species.

P. falciparum is the most widespread species worldwide, the most prone to drug resistance, and the one responsible for potentially fatal clinical forms.

Plasmodium falciparum

In equatorial regions, transmission occurs throughout the year with seasonal peaks. In subtropical regions, it occurs only during hot and humid periods. Transmission stops when the temperature drops below 18°C.

This also explains why, regardless of latitude, malaria is not transmitted at high altitude (above 1500 m in Africa and 2500 m in America and Asia).

The evolution of the infection is continuous after an incubation period of 7 to 12 days. Late relapses - seen with other species - do not occur.

More than 90% of *P. falciparum* malaria cases occur within two months of return from an endemic area.

P. falciparum (Figure 24) is responsible for severe clinical forms, particularly cerebral malaria. It is the species most frequently observed in France, responsible for more than 80% of so-called imported malaria, i.e., contracted in endemic areas but appearing later in metropolitan France after return.

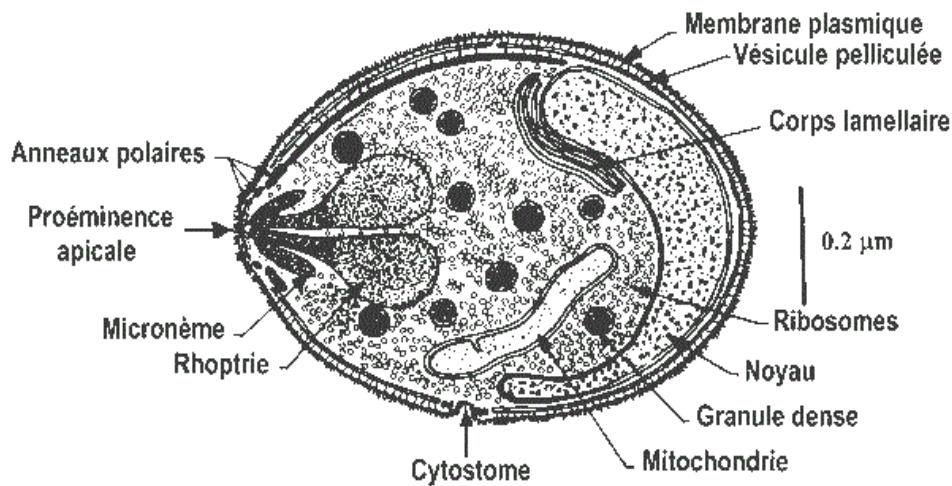


Figure 24. Ultrastructure of the merozoite of *P. falciparum*

Plasmodium vivax

Very widely distributed in South America and Asia, it is much more rarely observed in Africa. Erythrocytes of the Duffy-negative blood group (found in the majority of individuals originating from West Africa) lack the membrane receptor necessary for infection by *P. vivax*. Its transmission stops below 15°C.

Its incubation period is 11 to 13 days, but relapses (recrudescence attacks) can occur for 3 to 4 years. Infection with *P. vivax* is classically considered benign (benign tertian fever, meaning due to an erythrocytic cycle of 48 hours), but in endemic regions it can have serious repercussions on the health of populations, particularly through anemia in children. In addition, some drug resistance to *P. vivax* to chloroquine is beginning to emerge.

Plasmodium ovale

It occurs in Central and West equatorial Africa (and in certain Pacific regions) and causes benign tertian fever, like *P. vivax*, to which it is very similar. Its incubation period is at least 15 days but may be much longer, up to 4 years. Its progression is benign, but late relapses (5 years) may occur, as with *P. vivax*. In summary, *P. ovale* replaces *P. vivax* in areas where the latter species does not exist.

Plasmodium malariae

It occurs across the three continents, in a much more sporadic pattern. It differs from the other species by a longer incubation period (15 to 21 days), by a different periodicity of fever (erythrocytic cycle of 72 hours causing quartan fever) and especially by its capacity to induce very late recrudescences (up to 20 years after return from the endemic area).

The pathophysiological mechanisms responsible for these late recrudescences are not fully elucidated; some authors suggest the presence of latent merozoites in the lymphatic vessels. The infection is benign, but *P. malariae* may sometimes lead to renal complications.

Plasmodium knowlesi

It is found in Southeast Asia (particularly in Malaysia and Borneo), in forest environments because it is closely linked to the distribution of macaque monkeys, its usual host, and to the vector that bites both humans and monkeys. Morphologically, it is similar to *P. malariae*.

It differs from the other species by an erythrocytic cycle of 24 hours causing daily fever. Rare severe or even fatal forms have been reported, with high parasitemia. To date, no chemoresistance has been observed in this species.

b) Vector

Malaria is transmitted to humans through the bite of a culicid mosquito of the genus *Anopheles* (Figure 25) during its blood meal. Only the hematophagous female transmits the disease. It bites only after sunset, with maximum activity between 11 p.m. and 6 a.m. This explains why the use of mosquito nets is the most effective means of individual prevention.



Figure 25. *Anopheles gambiae* during a blood meal.

The larvae of *Anopheles* develop in water collections. The nature of the soil, the rainfall pattern, temperature and therefore altitude, natural vegetation or agriculture make water collections more or less favorable to the development of vector species. Some species have managed to adapt to particular environments such as urban areas. The development and longevity of *Anopheles* depend on temperature, with an optimum ranging between 20 and 30°C, for a lifespan of approximately 30 days.

There are numerous species of *Anopheles*, but not all are capable of transmitting malaria. The most dangerous ones are anthropophilic species, which prefer to take their blood meals on humans rather than animals, and endophilic species, which rest inside houses. Numerous climatic and/or environmental factors - natural (droughts, temperature variations, etc.) or due to human activity (draining marshes, building dams, irrigation, road construction, etc.) - can modify the distribution of *Anopheles* in a given region and therefore influence malaria transmission.

The distribution of *Anopheles* throughout the world largely exceeds that of malaria. If conditions favorable to their reimplantation occurred in currently eradicated areas, transmission could be re-established. In addition, insecticide resistance has emerged, limiting control measures.

c) Cycle

The cycle occurs successively in humans (asexual phase in the intermediate host) and in the mosquito *Anopheles* (sexual phase in the definitive host). In humans, the cycle is further divided into two phases: the hepatic or pre-erythrocytic phase (= exo-erythrocytic), corresponding to the incubation period and clinically asymptomatic, and the blood or erythrocytic phase, which corresponds to the clinical phase of the disease (Figure 26).

➤ In humans

● Pre-erythrocytic schizogony

The sporozoites inoculated by the female *Anopheles* during its blood meal remain for a maximum of about thirty minutes in the skin, lymph and bloodstream. Many are destroyed by macrophages, but some reach the hepatocytes. They transform into pre-erythrocytic schizonts or “blue bodies” (multinucleated forms), which after 7 to 15 days of maturation rupture and release thousands of merozoites into the blood (10,000 to 30,000 depending on the species). Hepatic

schizogony occurs only once in the cycle, since the hepatocyte can be infected only by sporozoites.

In infections caused by *P. vivax* and *P. ovale*, some intrahepatic sporozoites remain dormant (hypnozoites) and are responsible for delayed hepatic schizogony, releasing merozoites into the bloodstream several months after the mosquito bite, thus explaining late relapses observed with these two species. Hypnozoites do not exist in *P. falciparum* infection (no relapse), nor have they been demonstrated in *P. malariae* infection despite the occurrence of late relapses, nor apparently in *P. knowlesi*.

● Erythrocytic schizogony

Merozoites rapidly penetrate red blood cells. Penetration of the merozoite into the erythrocyte and its maturation into a trophozoite and then into a schizont takes 24, 48 or 72 hours (depending on the species) and leads to the destruction of the host red blood cell and the release of 8 to 32 new merozoites.

These merozoites invade new erythrocytes and initiate another replication cycle.

This part of the cycle corresponds to the clinical phase of the disease: parasitemia rises, the individual becomes febrile, producing a malaria attack. Without treatment, the parasites gradually evolve in synchrony (they become synchronous), and all erythrocytic schizonts reach maturation at the same time, causing the destruction of a large number of red blood cells periodically - every 24 hours (for *P. knowlesi*), 48 hours (tertian fever of *P. falciparum*, *P. vivax* or *P. ovale*), or 72 hours (quartan fever of *P. malariae*). In practice, the tertian fever due to *P. falciparum* is rarely synchronous.

After several erythrocytic cycles, some merozoites undergo about ten days of maturation accompanied by sexual differentiation: they transform into gametocytes (male or female), which will remain in circulation in the blood for 10 to 15 days.

➤ In the female *Anopheles*

The gametocytes, ingested by the mosquito during a blood meal on an infected person, transform into male and female gametes that fuse to form a free and mobile egg called an ookinete. This ookinete leaves the lumen of the digestive tract, then attaches to the outer wall of the stomach

and transforms into an oocyst. Parasitic cells multiply inside this oocyst, producing hundreds of sporozoites that then migrate to the mosquito's salivary glands. These sporozoites are the infective forms ready to be inoculated with the mosquito's saliva during a blood meal on a vertebrate host.

The duration of sporogonic development of *Plasmodium* varies depending on climatic conditions: between 9 and 20 days for *P. falciparum* (respectively at 30°C and 20°C), slightly faster for *P. vivax* under comparable temperatures, and longer for *P. malariae*.

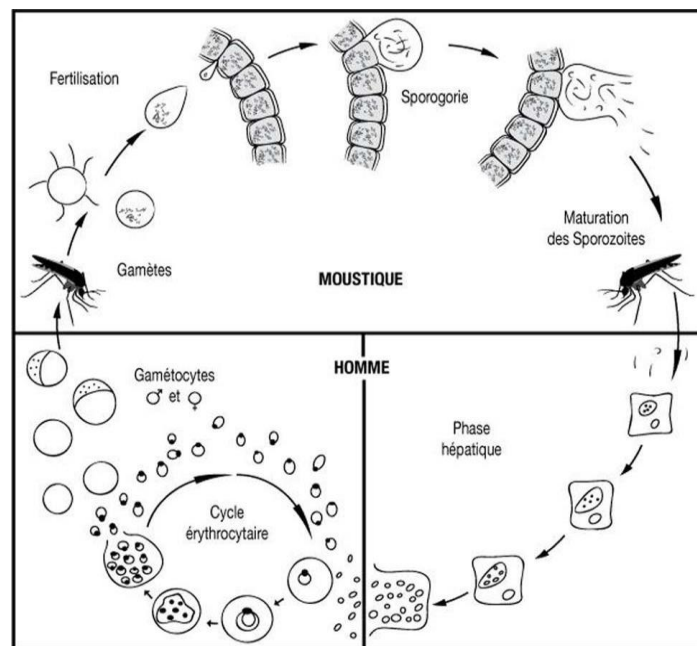


Figure 26. Life cycle of *Plasmodium* sp.

Regarding transmission modalities, malaria is transmitted at night through the bite of a mosquito - the female *Anopheles*.

The blood phase of the cycle allows for other possible modes of contamination: congenital transmission, transmission through blood transfusion, organ transplantation, or accidental transmission among healthcare workers handling contaminated blood. In practice, these transmission routes are extremely rare.

2.2.3 – Phylum Ciliophora

Order: Vestibuliferida

Genus: *Balantidium*

Balantidium coli is the largest and the only ciliated protozoan parasite of humans. *B. coli* belongs to the class *Ciliata*, the order *Vestibuliferida* and the family *Balantidiidae*. Balantidiasis (or balantidiosis) is a parasitic disease very common in pigs. In addition to pigs, which are the main reservoir, *B. coli* colonizes the large intestine of many mammals.

a) Morphology

Vegetative form:

Oblong, spheroid or tapered in shape, trophozoites measure 30–150 μm in length and 25–120 μm in width. They contain a peristome, two nuclei (a large kidney-shaped macronucleus and a spherical micronucleus), and two contractile vacuoles in the cytoplasm. The surface of the *B. coli* body is covered with longitudinal rows of cilia (Figure 27).

Reproduction occurs by asexual division. Trophozoites may be present in the anaerobic environment of the intestine and grow at temperatures between 20 and 40 $^{\circ}\text{C}$. The organism moves by rotation in the colon.

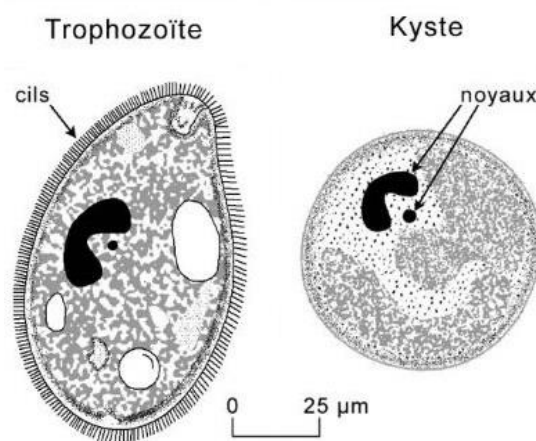


Figure 27. Trophozoite and cyst of *Balantidium coli*

The cyst:

The cysts, found most frequently in the feces, are spheroidal or ovoid and measure 40–60 μm in diameter. They also possess two nuclei: a macronucleus and a micronucleus (Figure 27).

b) Life cycle

Balantidiasis has a short, direct life cycle.

DH (definitive hosts) = pig, dog, human.

Its developmental cycle includes two stages: a ciliated trophozoite stage and an environmentally resistant cyst stage (Figure 28).

The cysts are infectious. After ingestion, the cysts lose their protective wall in the small intestine, and the trophozoites, located in the intestinal lumen, attach to the mucosa of the terminal ileum and colon.

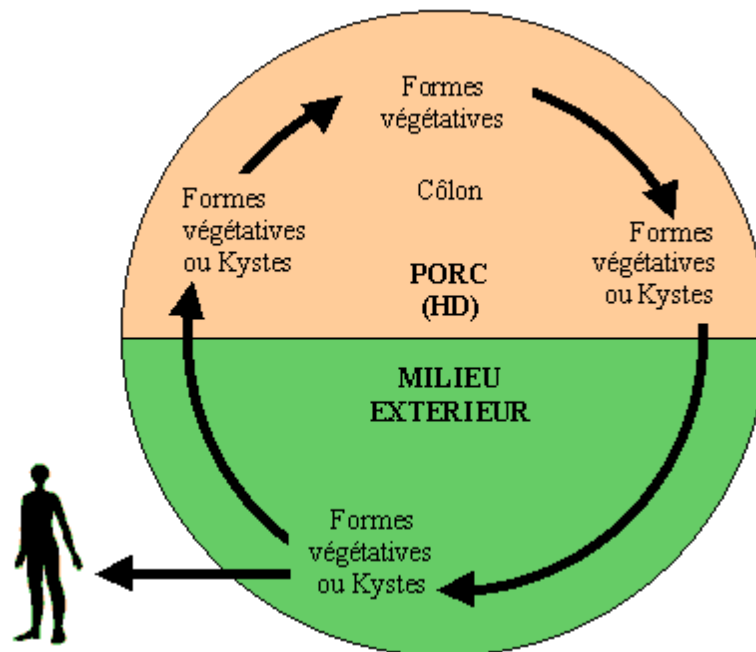


Figure 28. *Balantidium coli* life cycle

c) Epidemiology:

This disease is cosmopolitan, but with a higher prevalence in warm and humid regions of Africa, South America, Asia and the Pacific. Balantidiasis is a digestive zoonosis transmitted via the fecal–oral route.

Reservoir host (RH): The pig is the main reservoir host. However, *B. coli* is also found in humans, rats, monkeys, mice, insects, fish, and amphibians.

This parasitosis is endemic in tropical countries and mainly affects occupational groups in close contact with pigs.

Close contact with pigs or pig feces is the primary risk factor for humans. Transmission most often occurs through the fecal–oral route after ingestion of protozoan cysts from contaminated food or by direct contact with animals. Water is the main factor in the dissemination of *B. coli* infections. Rats may carry the pathogen, but it remains unknown whether transmission to humans is possible. Cockroaches may act as mechanical vectors, transferring fecal material to food.

Nevertheless, transmission to humans is probably underestimated in countries with high endemicity. Most cases are asymptomatic, but balantidiasis may cause a dysenteric syndrome that can be severe.

III. PARASITIC METAZOA

3.1 – Phylum Platyhelminthes

3.1.1 – Class Trematoda

Subclass Digenea

- **Genus *Fasciola* (*Fasciola hepatica*)**

Fascioliasis is a zoonosis caused by a hermaphroditic trematode, a flat non-segmented worm. This distomatosis, rare in humans, is transmitted through the consumption of semi-aquatic plants, particularly contaminated watercress. *Fasciola hepatica* is located in its adult form within the biliary ducts of numerous herbivorous animals and, occasionally, in humans.

Hepatobiliary distomatosis, or fascioliasis due to *Fasciola hepatica*, commonly known as the “large liver fluke,” is pathogenic in humans.

Other species of flukes are also capable of parasitizing humans, causing hepatic, pulmonary, or intestinal distomatoses depending on the localization of the adult worms. Each of these species has a specific contaminating food source; therefore, the route of infection is always oral.

a) Pathogen:

Fasciola hepatica, commonly known as the large liver fluke, is a hermaphroditic flatworm shaped like a small leaf, measuring 2–4 cm in length and approximately 1 cm at its greatest width in its adult stage (Figure 29). At its anterior extremity, it possesses two suckers that allow it to adhere to the epithelium of the biliary ducts.

The eggs, measuring 120 × 70 μm, are ovoid and operculated.

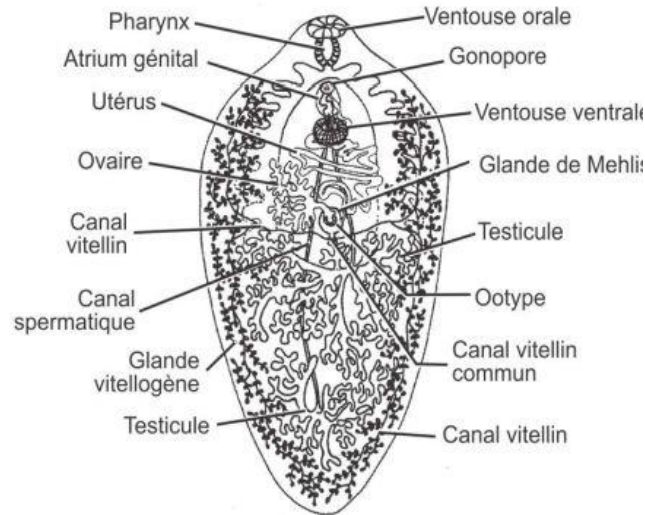


Figure 29. Anatomy of the liver fluke, *Fasciola hepatica*

b) Life cycle:

The adult worm parasitizes the intra- and extra-hepatic bile ducts of the definitive host: numerous mammals (particularly sheep and cattle), and humans occasionally.

The life cycle is summarized in Figure 30.

The adult worm lays operculated eggs that are excreted in the feces. In fresh water, these eggs become embryonated within approximately three weeks and release a ciliated embryo known as the miracidium.

The miracidium swims to locate the intermediate host, a freshwater snail of the genus *Galba* (*Galba truncatula*).

This snail lives along streams and drainage ditches in pastures. Infection of the snail occurs mainly at the beginning of summer.

Inside the snail, the miracidium transforms and multiplies into hundreds of larvae (polyembryony phenomenon), first producing rediae, which subsequently develop into cercariae. The cercariae are released into the external environment after a few weeks.

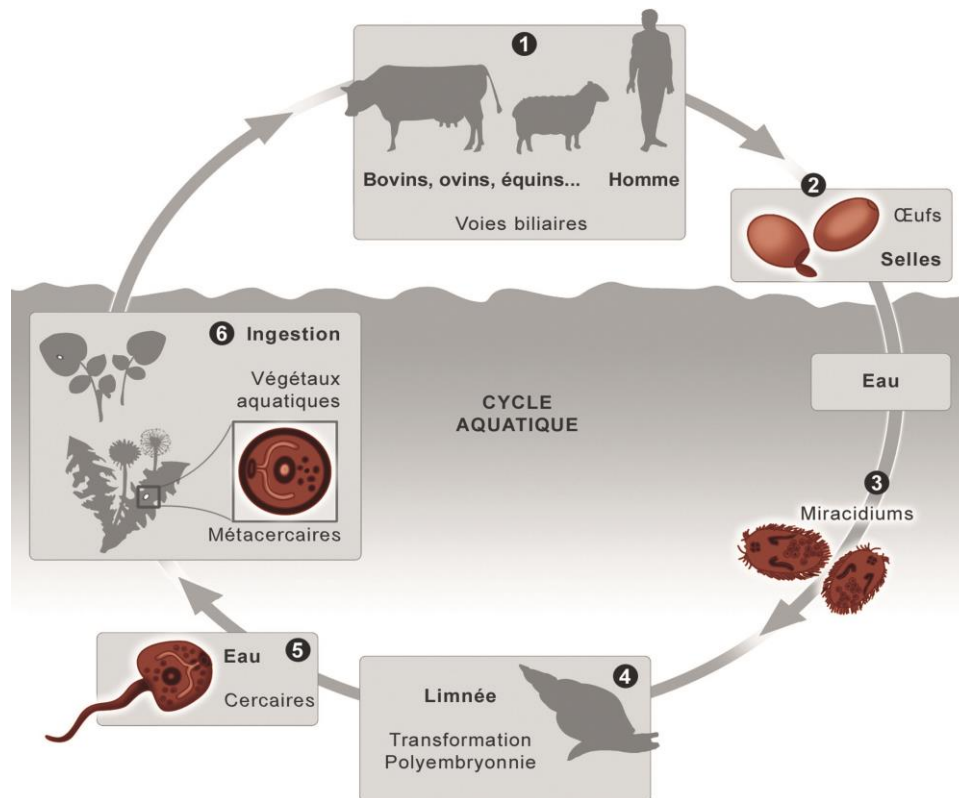


Figure 30. Life cycle of the liver fluke

The cercaria, equipped with a tail, swims in water, attaches itself to a semi-aquatic plant (watercress, dandelion, etc.) along streams, and develops into an encysted metacercaria. This larval form, surrounded by a thick protective capsule, is the resistant and infective stage of the parasite.

The definitive host (sheep, cattle, humans) becomes infected by ingesting vegetation on which metacercariae are attached. Once released from their cyst wall by digestive enzymes, the larvae transform into immature juvenile flukes that migrate toward the liver. They penetrate the intestinal wall, then move across the peritoneal cavity toward Glisson's capsule, which they perforate.

They subsequently migrate through the hepatic parenchyma, causing lesions, before settling in the bile ducts, where they reach maturity approximately three months after infection.

Adult flukes have a lifespan of about 3 to 5 years.

c) Epidemiology:

Fascioliasis is a cosmopolitan zoonosis found in all livestock-raising regions. Countries with high prevalence include Egypt, Iran, Argentina, and Andean countries.

It is also present in some regions of Algeria (e.g., Jijel, El Kala).

Humans acquire the disease by consuming raw wild watercress harvested from meadows or downstream from moist pastures where sheep, cattle, and other herbivores graze. Other plants have been implicated (lamb's lettuce, dandelions). Infection is often seasonal, beginning at the end of summer or in autumn. Rainy summers, which favor the proliferation of the snail intermediate host, increase the risk of infection.

▪ Genus *Schistosoma* (*Schistosoma spp.*)

Members of this genus belong to the phylum Platyhelminthes (flatworms), class Trematoda (non-segmented worms), and subclass Digenea.

Bilharziasis or schistosomiasis refers to parasitic diseases caused by gonochoric trematodes (separate sexes), blood-feeding flatworms that live in the mesenteric or pelvic venous system of mammals as adults, and develop in freshwater snails during their larval stages.

a) Pathogenic agents:

A marked sexual dimorphism exists within this group of parasitic worms.

The male is white and measures 10–15 mm long and approximately 1 mm wide. Although anatomically flat, the worm appears cylindrical (robust aspect) because the lateral edges are folded inward, forming a gynecophoric canal in which the female resides.

The female, cylindrical and filiform, is longer and more slender than the male, measuring 15-30 mm in length.

During copulation, the female remains lodged inside the male's gynecophoric canal (Figure 31).

Both sexes possess two suckers: an oral sucker, which opens into the esophagus and functions in feeding, and a ventral sucker, which serves as an attachment organ.

The furcocercariae, measuring 400 - 600 μm , have a pyriform (pear-shaped) head and a bifurcated tail (Figure 30).

The eggs vary in shape and size depending on the species:

- *S. haematobium* (120–160 × 40–60 μm): oval, with a terminal spine, and a transparent shell;
- *S. intercalatum* (150–250 × 60 μm): diamond-shaped, with a terminal spine, and a light yellow shell;
- *S. mansoni* (130–160 × 60–70 μm): oval, with a lateral spine, and a colorless or yellowish shell;
- *S. japonicum* (70 × 40 μm) and *S. mekongi* (60 × 50 μm): spherical, with a small lateral spine, and a colorless shell.

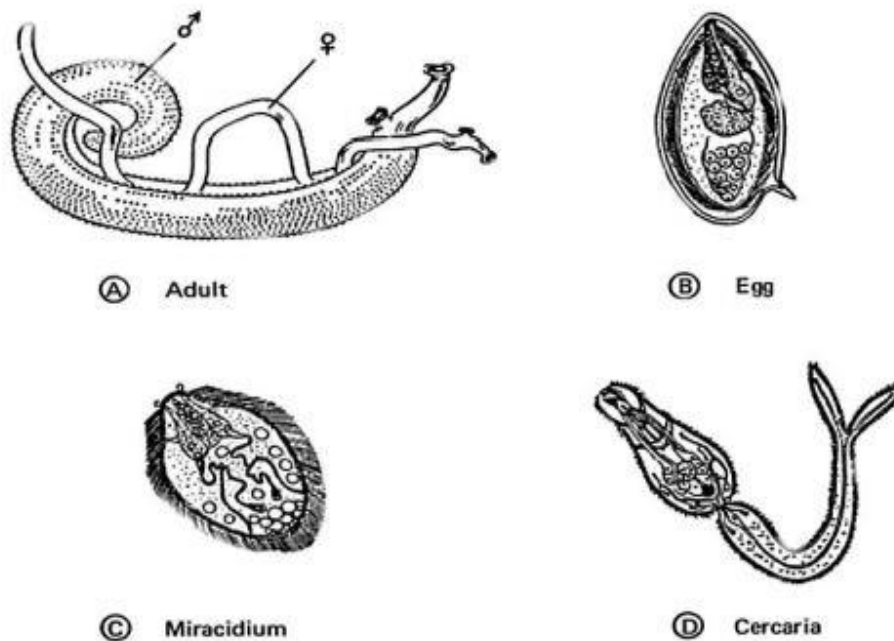


Figure 31. Different developmental stages of *Schistosoma mansoni*.

- *Schistosoma haematobium* is the causative agent of urogenital bilharziasis (tropism for the perivesical and peri-rectal venous plexuses). The female lays its terminal-spined eggs, in clusters, within the bladder and rectal walls. The eggs are eliminated mainly in the urine. The lifespan of *S. haematobium* exceeds 10 years. Humans are the only reservoir of the parasite. Intermediate hosts are freshwater mollusks, most often of the genera *Bulinus* and *Physopsis*.
- *Schistosoma mansoni* causes intestinal schistosomiasis and, sometimes, hepatosplenic schistosomiasis.

Adult schistosomes migrate to the inferior mesenteric venous plexuses. Egg-laying therefore occurs mainly in the intestinal wall, although the lateral-spined eggs frequently embolize to the liver or spleen.

The lifespan of adult worms is more than 10 years. Humans are the main reservoir, but about thirty animal species (mainly rodents) have been found naturally infected. Intermediate hosts are planorbids (freshwater mollusks).

- *Schistosoma japonicum*, the most pathogenic species, causes the severe arterio-venous schistosomiasis. In humans, adult worms live mainly in the superior mesenteric venous plexuses, although erratic couples may reach the pulmonary arteries. Egg-laying is particularly abundant. The lifespan of adults barely exceeds 5 years. This worm causes a zoonosis affecting humans and many wild and domestic animals. Intermediate hosts are amphibious mollusks of the genus *Oncomelania*.

- *Schistosoma intercalatum* and *Schistosoma guineensis* are responsible for rectal schistosomiasis. These two parasites, very close morphologically but with different geographic distributions, are rather poorly adapted to humans. Adult worms live mainly in the peri-rectal venous plexuses.

Their lifespan is poorly known. The intermediate host is a *Bulinus* snail.

b) Life cycle

The life cycle is essentially identical for the six species and requires the obligatory involvement of an intermediate host, which is a freshwater mollusk.

Females, located depending on the species in the fine venous branches of the intestine or the bladder, lay eggs that reach the intestine or the bladder via trans-tissue migration (spined eggs) and are excreted into the external environment in embryonated form, via the stool (*S. mansoni*, *S. japonicum*, *S. mekongi*, *S. intercalatum*, *S. guineensis*) or urine (*S. haematobium*).

If eggs become embolized in tissues, they become trapped and calcified.

If, on the contrary, eggs are excreted into the external environment and if conditions are favorable (fresh water, nearly neutral pH and temperature between 18 °C and 33 °C), they

release a ciliated larva: the *miracidium* (short-lived: a few hours), which must swim in search of the specific intermediate mollusk host of the schistosome species.

The miracidium penetrates the intermediate host (mollusk), and when the temperature is adequate (around 30 °C), larval development within the mollusk lasts one month. The parasite multiplies actively and produces (by polyembryony) sporocysts and then thousands of cercariae or fork-tailed cercariae (the infective larval stage), which are released into the external environment. These are highly mobile in fresh water.

Contamination of the definitive host (human) by cercariae occurs transcutaneously during contact with freshwater, even briefly (less than 10 minutes). The survival of cercariae is short (a few hours); they are attracted by chemotaxis and penetrate through the skin of any immersed body surface.

As the cercariae penetrate, they shed their tails and the anterior part, the schistosomulum, migrates through the organism via blood and lymphatic routes. After a pulmonary, cardiac and hepatic passage, the worms mature into adults in the portal system within 2–3 months. After mating, the worms ascend the portal circulation against the current. The fertilized females then separate from the males and migrate, according to species-specific tropism, into the fine visceral ramifications of a given venous plexus, where they lay their eggs (Figure 32).

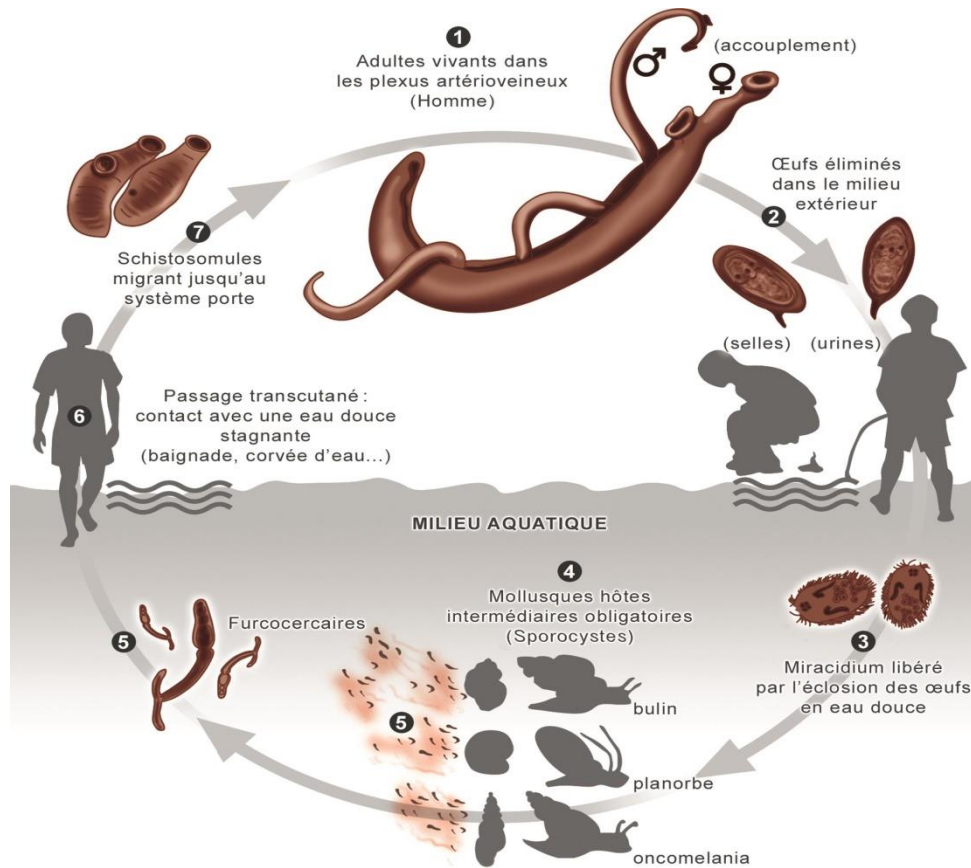


Figure 32. Evolutionary cycle of schistosomes.

c) Epidemiology

There are approximately 200 million cases of schistosomiasis worldwide. Six species are pathogenic to humans and occur endemically on three continents. Infection occurs through transcutaneous penetration of the parasite during bathing in stagnant freshwater, particularly during the warm hours of the day.

Schistosomiasis due to *S. haematobium* occurs throughout Africa, in Madagascar (western coast) and Mauritius. A few foci exist around the Mediterranean basin (Maghreb) and in the Middle East.

Schistosomiasis due to *S. mansoni* is the most widespread worldwide (tropical Africa, eastern coast of Madagascar, the Caribbean and South America, Arabian Peninsula).

Schistosomiasis due to *S. japonicum* is strictly Asian (China, Taiwan, Philippines, Sulawesi). Eradicated from Japan, the disease retains only the name of that country.

The distribution of *Schistosoma mekongi* is restricted to a few endemic foci (Thailand, Laos and Cambodia).

Strictly Asian, *S. mekongi* is also highly pathogenic. Its intermediate host is a mollusk (*Tricula aperta*), which does not survive drought.

Schistosomiasis due to *Schistosoma intercalatum* and *Schistosoma guineensis* is exclusively African (Equatorial West Africa: Congo for *S. intercalatum*; Central African Republic, Equatorial Guinea, Cameroon, Gabon, Nigeria, Angola, Congo, Chad and São Tomé for *S. guineensis*).

Class Monogenea

Only distinctive characteristics are mentioned, since this group does not contain species parasitic to humans.

3.1.2 – Class Cestodes

General morphology of adults

Cestodes, commonly known as tapeworms, are multicellular, flat helminths that are endoparasites of the digestive tract of vertebrates. Their morphology is highly specialized for parasitic life and for the direct absorption of nutrients from the host.

Adult cestodes have a body that is dorsoventrally flattened, elongated and ribbon-like, whitish or translucent, highly variable in length and ranging from a few millimeters to several meters depending on the species).

The adult body is generally divided into three main regions:

- **The scolex** (head with fixation organs) is the anterior region, responsible for attachment to the host's intestinal wall.

It may bear:

- Suckers
- A rostellum, which may be armed or unarmed with hooks
- Bothridia (slit-like attachment organs) in some groups

- **The neck** (proliferation or germinal zone)

- Narrow region directly behind the scolex
- Growth zone
- Continuously produces new segments (proglottids) by posterior budding

- The strobila

- Long chain of proglottids forming the majority of the body
- Each proglottid constitutes a reproductive unit
- Classified according to their maturity:
 - **Immature:** close to the neck, sexual organs not developed
 - **Mature:** complete reproductive system, usually hermaphroditic
 - **Gravid:** enlarged uterus filled with eggs for environmental dissemination

Tegument and nutrition

Cestodes possess no mouth and no digestive tract.

Their nutrition occurs through direct absorption of nutrients across the tegument.

The tegument:

- Is covered with microtriches (microscopic projections)
- Greatly increases the absorptive surface
- Protects against host digestive enzymes
- Actively transports carbohydrates and other nutrients

Reproductive system

Cestodes are typically protandrous hermaphrodites:

- Male organs develop before female organs
- Each mature proglottid contains:
 - Numerous testes
 - A bilobed ovary
 - A vitelline gland (yolk production)
 - A genital pore (lateral or median depending on the species)

A single individual can reproduce through self-fertilization, though cross-fertilization between proglottids may also occur.

Other organ systems

Cestodes exhibit a highly specialized internal organization adapted to endoparasitism. Although they lack a digestive system, they possess efficient reproductive, nervous, and excretory systems that ensure their survival, development, and propagation within the host.

The next table presents these organ systems and explains their functions.

System	Status
Digestive	Absent
Circulatory	Absent
Respiratory	Absent
Excretory	Present (protonephridia with flame cells)
Nervous	Present (scolex + longitudinal nerve cords)
Reproductive	Highly developed

The absence of digestive and circulatory systems represents an adaptation to intestinal parasitism, whereas the reproductive system is highly developed to ensure massive egg production.

Morphological adaptations to parasitism

Adult cestodes exhibit several adaptations facilitating parasitic life:

- Strong fixation to the intestinal mucosa
- Direct absorption of nutrients
- Massive reproductive capacity
- Complex life cycles ensuring persistence in the environment and among successive hosts

Schematic summary

The diagram below illustrates the major characteristics and organization of cestodes.

Region	Main function
Scolex	Attachment and host adaptation
Neck	Production of new proglottids
Strobila	Reproduction and egg dissemination
System	Status
Digestive	Absent
Circulatory	Absent
Respiratory	Absent
Excretory	Present (protonephridia with flame cells)
Nervous	Present (scolex + longitudinal nerve cords)
Reproductive	Highly developed

1 – Genus *Taenia*

Subclass **Eucestoda**, Order **Cyclophyllidea**, Family **Taeniidae**

Tapeworms, *Taenia saginata* and *Taenia solium*, are flatworms (platyhelminths) or cestodes, cosmopolitan, parasites of the human small intestine.

They are ribbon-shaped, segmented, hermaphroditic, and their development includes an adult stage and a larval stage.

The larval stages, or cysticerci, are harbored by the intermediate hosts (cattle, pigs). These larval forms are orally infective for humans (definitive host), in whom they produce taeniasis.

Selected model: *Taenia saginata*

a) Morphology

Adult worm: A strictly human parasite of the small intestine, *Taenia saginata* is a large, shiny white flatworm (4 to 10 m in length), ribbon-like and segmented into 1,000 to 2,000 rings or proglottids. It is generally isolated (solitary worm).

The head or scolex is pear-shaped, depressed at the apex, with a diameter of 1.5 to 2 mm. The scolex has four suckers but no rostellum nor hooks (unarmed tapeworm). Following the scolex,

the neck measures a few millimeters in length and produces the proglottids. The whole set of proglottids forms the body of the tapeworm or strobila (Figure 33).

The reproductive system consists of two ovarian lobes, a cluster of 300 to 1,200 testicular glands, and a highly developed ramified uterus filled with eggs.

The mature gravid proglottids, or cucurbitans, are actively expelled into the external environment, outside of the stool, either individually or in chains of varying length.

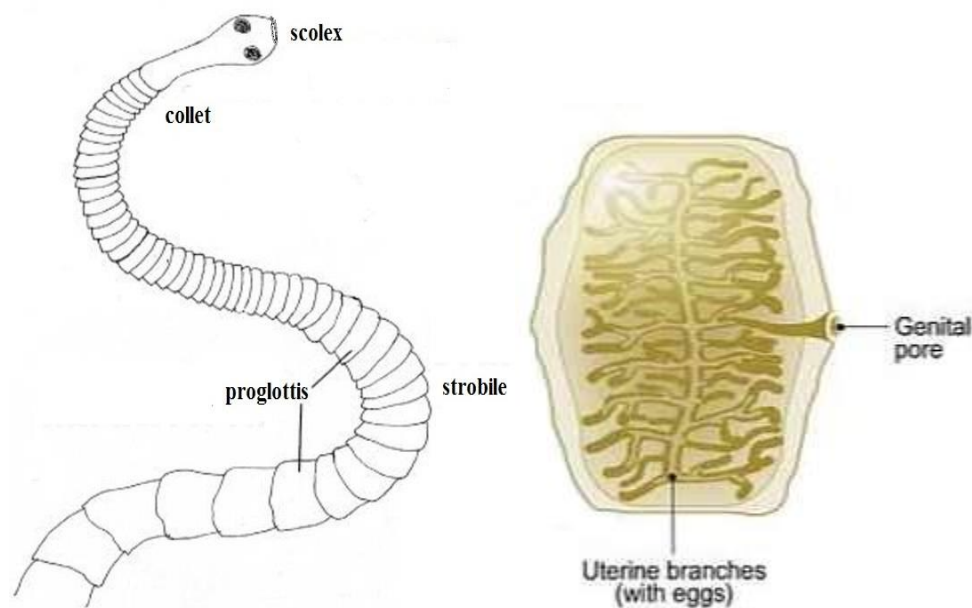


Figure 33. Morphology of an adult tapeworm.

Eggs: In the external environment, the eggs are released after the disintegration of the proglottid.

They possess two shells:

- an outer shell or vitelline membrane, fragile, thick, translucent, containing refringent granules, delimiting the actual egg, and often destroyed;
- an inner dark-brown resistant shell, delimiting an embryophore that contains an embryo provided with three pairs of hooks (hexacanth embryo or oncosphere) (Figure 34).

The lifespan of the adult tapeworm is very long, around several years (25 years or even 30 years) in the absence of treatment.

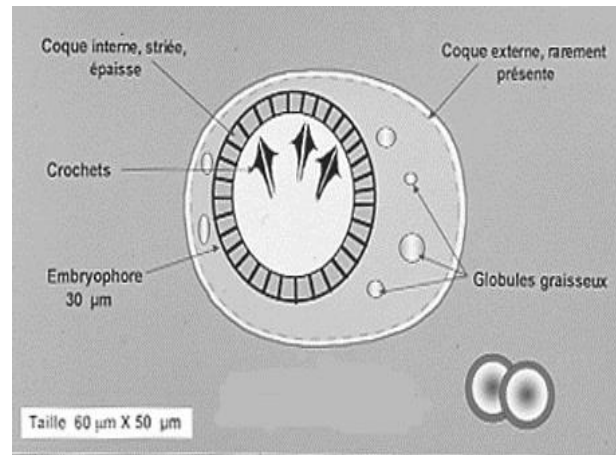


Figure 34. Egg of *Taenia saginata*.

Larval form:

The cysticercus larva is composed of an external membrane and an internal fibromuscular tissue containing calcareous corpuscles. The cysticercus has an invaginated scolex with four suckers. Its lifespan is on average 20 to 30 months, after which it becomes calcified.

Cysticerci can survive 40 days in carcasses at 4°C and 4 hours at -10°C. They are destroyed at 45°C.

b) Life cycle:

At maturity, the proglottids detach one by one from the strobila. Five to ten mobile segments are expelled spontaneously each day (rarely with the feces). By forcing the anal sphincter, they may release eggs or embryophores into the folds of the anal margin.

In the external environment, the segments undergo lysis and release the eggs (about 80,000 eggs per proglottid, more than 150 million per year) which are highly resistant in the environment and to physical and chemical agents. They are spread across soil, sewage systems, and scattered in pastures. They are also found in residual sludge used in agriculture.

After ingestion by cattle, the embryophores are digested by gastric juices, intestinal secretions, and bile. The released embryos cross the intestinal mucosa using their hooks and lytic secretions.

They are disseminated throughout the organism via blood and lymphatic circulation. They lodge in the interfascicular adipose tissue of muscles, where within 3 to 4 months, they form vesicles measuring 5-10 mm: the cysticercus larvae (Figure 35).

The same host may harbor numerous cysticerci at different developmental stages.

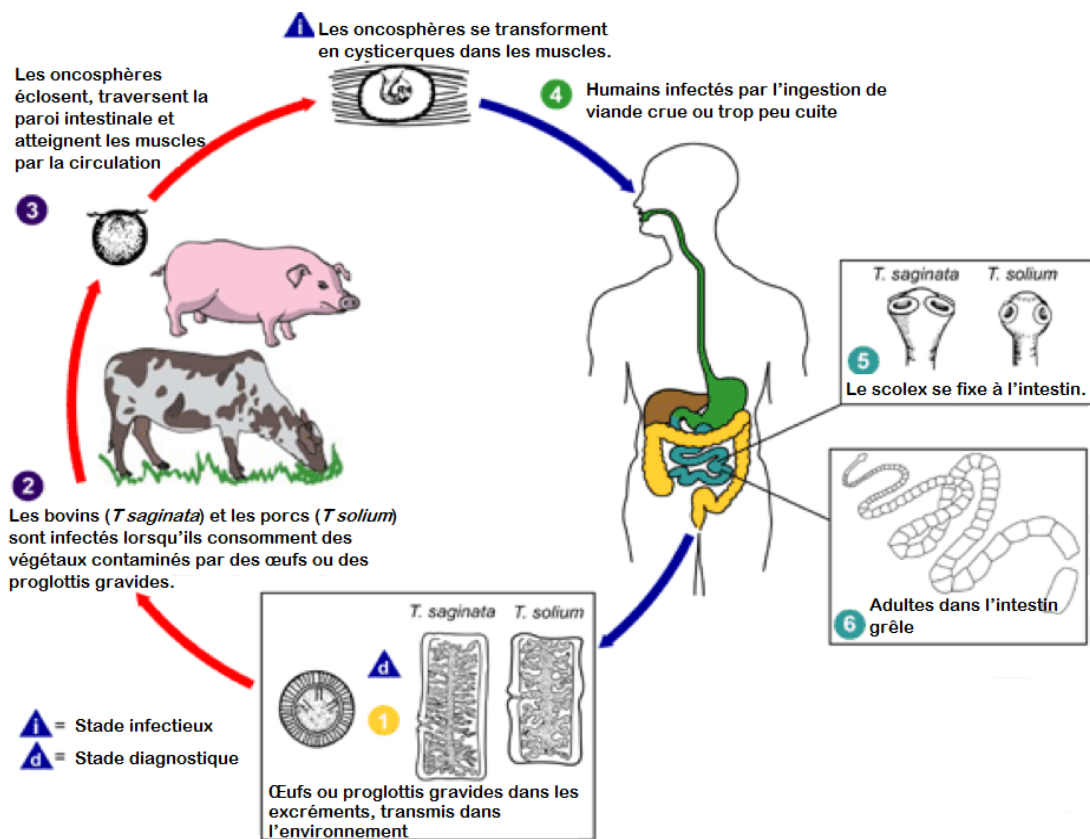


Figure 35. Life cycle of taeniasis.

2 – Genus *Echinococcus*

Subclass **Eucestoda**, Order **Cyclophyllidea**, Family **Taeniidae**

Name of the parasitic disease: **echinococcosis, cystic echinococcosis, hydatidosis, cystic hydatidosis.**

a) Morphology:

Adult worm: Adult forms of *Echinococcus granulosus* are found only in the small intestine of definitive hosts, which are carnivores (dogs and other canids).

Adult worms measure 3 to 7 mm in length and contain only three proglottids. The body consists of an armed scolex, a short neck, and proglottids; the mature segment is the middle one, and the gravid segment is the terminal one (Figure 36).

Several hundred adults may parasitize the same animal.

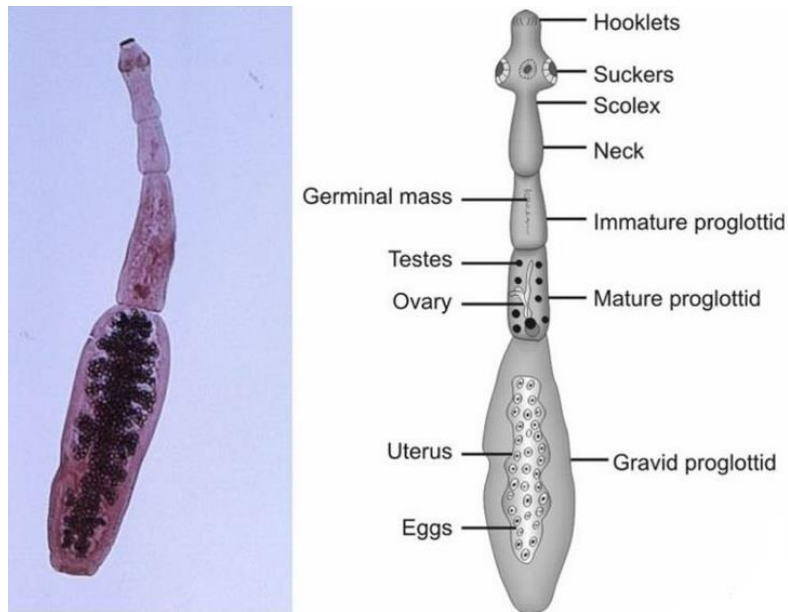


Figure 36. Adult *Echinococcus granulosus*.

Eggs:

The eggs are found in the feces of the definitive host. They are spherical and measure 30 to 50 μm in diameter. The embryo (oncosphere) is protected by a thick and impermeable embryophore, which makes the eggs extremely resistant (Figure 37).

The gravid segments (and sometimes the whole worm) are expelled in the feces. The eggs are immediately infective for the intermediate host.

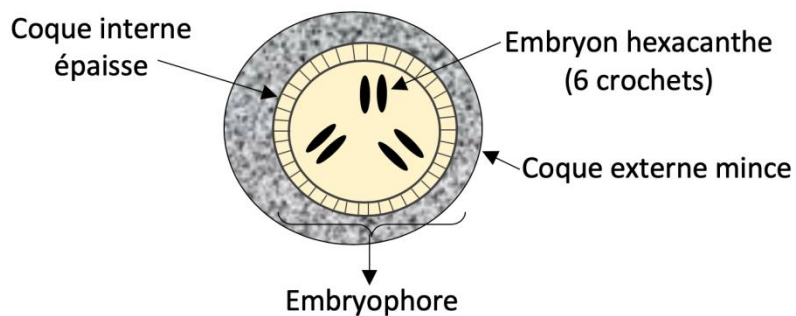


Figure 37. Egg of *Echinococcus granulosus*.

Larval form:

When the intermediate host (livestock) or humans (accidental host) ingest eggs, the oncospheres hatch and become activated. They are then transported through the bloodstream to the liver and other target organs. Once the oncospheres reach their final destination, they transform into

hydatid cysts, which enlarge and produce protoscoleces or daughter hydatids on the inner wall of the cysts (Figure 38).

The dog becomes infected by ingesting the liver of a sheep (or another herbivore) containing a hydatid cyst filled with numerous infective protoscoleces.

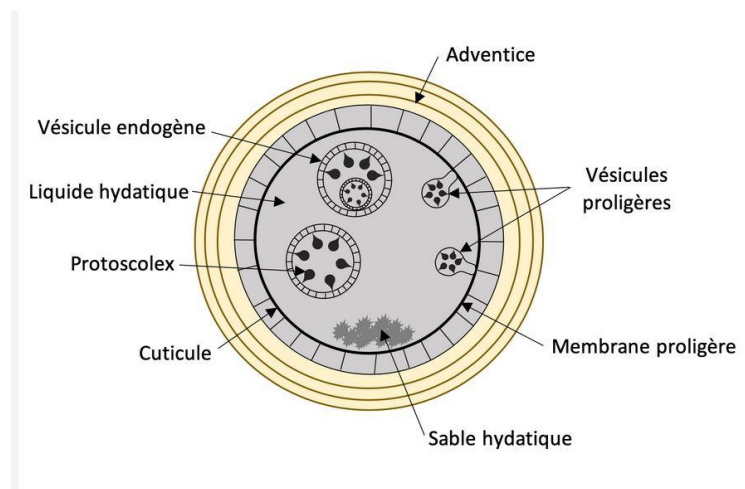


Figure 38. Hydatid larva of *Echinococcus granulosus*.

b) Biological cycle:

The cycle is dioxenic. The preferred intermediate host is the sheep (as well as many other herbivores) (Figures 39 and 40).

Humans take part in the cycle as intermediate hosts. Therefore, they harbor the *echinococcus* larva, which develops at the expense of hepatic tissue, and sometimes in other organs (lungs, bones, etc.).

Humans may acquire the infection if they are exposed to dogs infected after eating the flesh of infected sheep. Transmission to humans occurs through the ingestion of eggs shed in the dog's feces. These eggs may be present on the dog's fur, tongue, and in the surrounding environment.

In general, the cycle includes a stage within the definitive host (scolex evagination → adults), followed by a stage in the environment (eggs), and finally a stage within the intermediate host (cysticercus / hydatid larva) (Figure 41).

As a result, the only protection against this zoonosis is strict hand hygiene after any contact with dogs. Because the disease is a completely asymptomatic taeniasis in dogs, every dog must be considered as a potential carrier of the parasite.

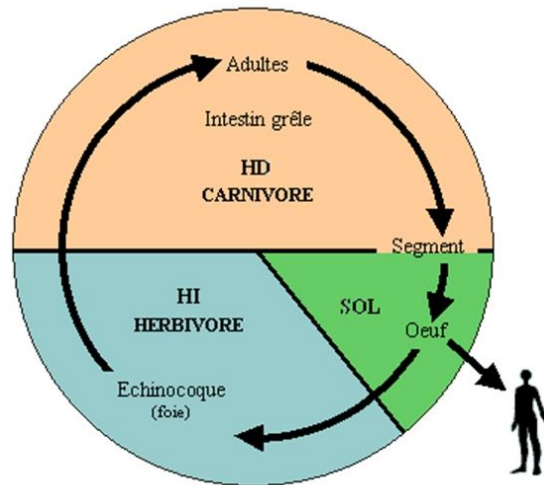


Figure 39. Evolutionary diagram of *Echinococcus granulosus*.

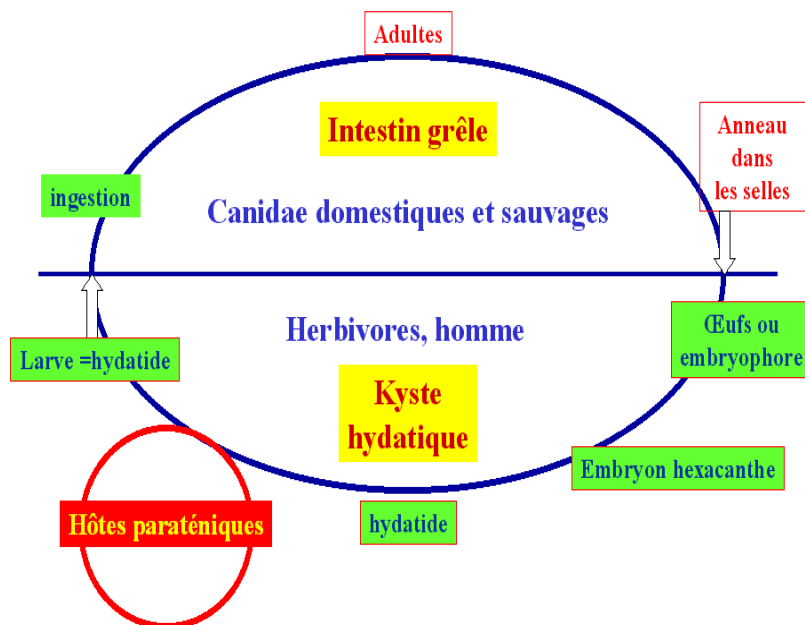


Figure 40. Life cycle of *Echinococcus granulosus*

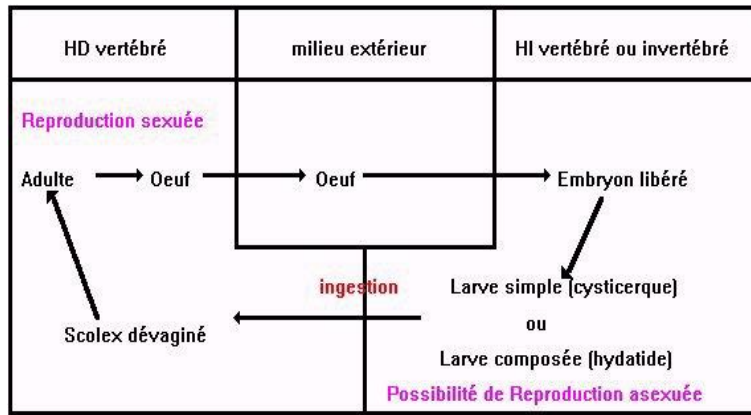


Figure 41. Life cycle of Cyclophyllidean Cestodes.

N.B.: An organ evaginates when it turns inside out like the finger of a glove.

3 – Genus *Diphyllobothrium*

Subclass: **Eucestoda** ; Order: **Pseudophyllidea** ; Family: **Diphyllobothriidae**.

Also called “**bothriocephaliosis**”, **diphyllobothriosis** is an intestinal parasitic disease caused by the cestode *Diphyllobothrium latum*.

a) Morphology:

In its adult form, this tapeworm stands out from other cestodes due to its very large size (on average 2 to 8 meters, and in some cases up to 20 meters), and its 3,000 to 4,000 trapezoidal segments, where the central rosette in each segment corresponds to the uterus (Figure 42).

The scolex bears two types of suckers, the ventral and dorsal bothridia (Figure 43).



Figure 42. Trapezoidal segment.

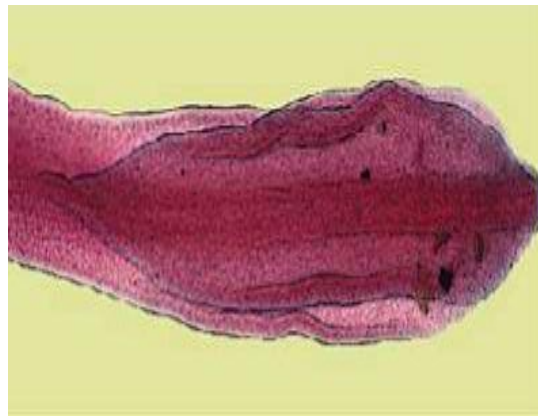


Figure 43. Scolex of *Diphyllobothrium latum*.

Eggs:

The egg ($70 \times 40 \mu\text{m}$), short and ovoid in shape (resembling a chicken egg), is equipped with an operculum and covered with a light yellow shell. It contains a single cell (the egg is non-embryonated when laid) (Figure 44).

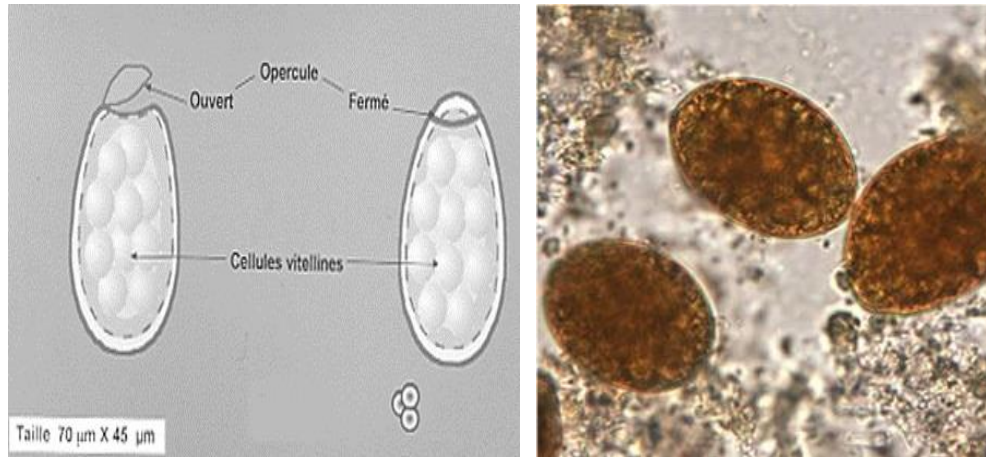


Figure 44. Egg of a broad tapeworm.

b) Life cycle of the parasite

The definitive host (ichthyophagous or piscivorous mammal: humans, dogs, cats, pigs, foxes, bears) becomes infected by ingesting raw or undercooked flesh of a predatory fish. The parasite attaches to the intestinal mucosa by means of its bothridia, grows, and can produce eggs by the fifth week. It releases eggs in the feces of the definitive host. When these eggs come into contact with water under favorable conditions (oxygen, light, pH 4–9, temperature 8–20 °C), they mature and eventually hatch after seven to twenty days, releasing a larva called a *coracidium* (a ciliated free-swimming hexacanth embryo).

The *coracidium* is ingested by a small planktonic crustacean - a copepod (*Cyclops* or *Eudiaptomus*) - considered as the first intermediate host (IH1). There it develops and, after 16 to 18 days, becomes a *proceroid* larva (worm-shaped larva with six hooks) in the general cavity.

The copepod is then ingested by a small fish (roach: IH2), which is itself eaten by a predatory fish (trout, perch, burbot, pike... : IH3). In this predatory fish, the proceroid larva migrates to variable locations depending on the intermediate host (muscles in perch and burbot, abdominal cavity in pike...) and, after two to three months, develops into a *plerocercoid* larva in the viscera and muscles of the fish. It is at this larval stage that the two bothridia appear.

Humans seem to be the natural definitive host of this parasite, since its development is completed only in humans (Figures 45 and 46).

D. latum feeds passively (absorbing nutrients from the host's intestinal chyme) and consumes a large amount of vitamin B₁₂. Vitamin B₁₂ injections may be necessary in cases of severe anemia.

Three-host parasite (trixenic parasite):

- Intermediate host 1: lake copepod
- Intermediate host 2: salmonid or percid fish
- Paratenic (waiting) host, non-obligatory: pike

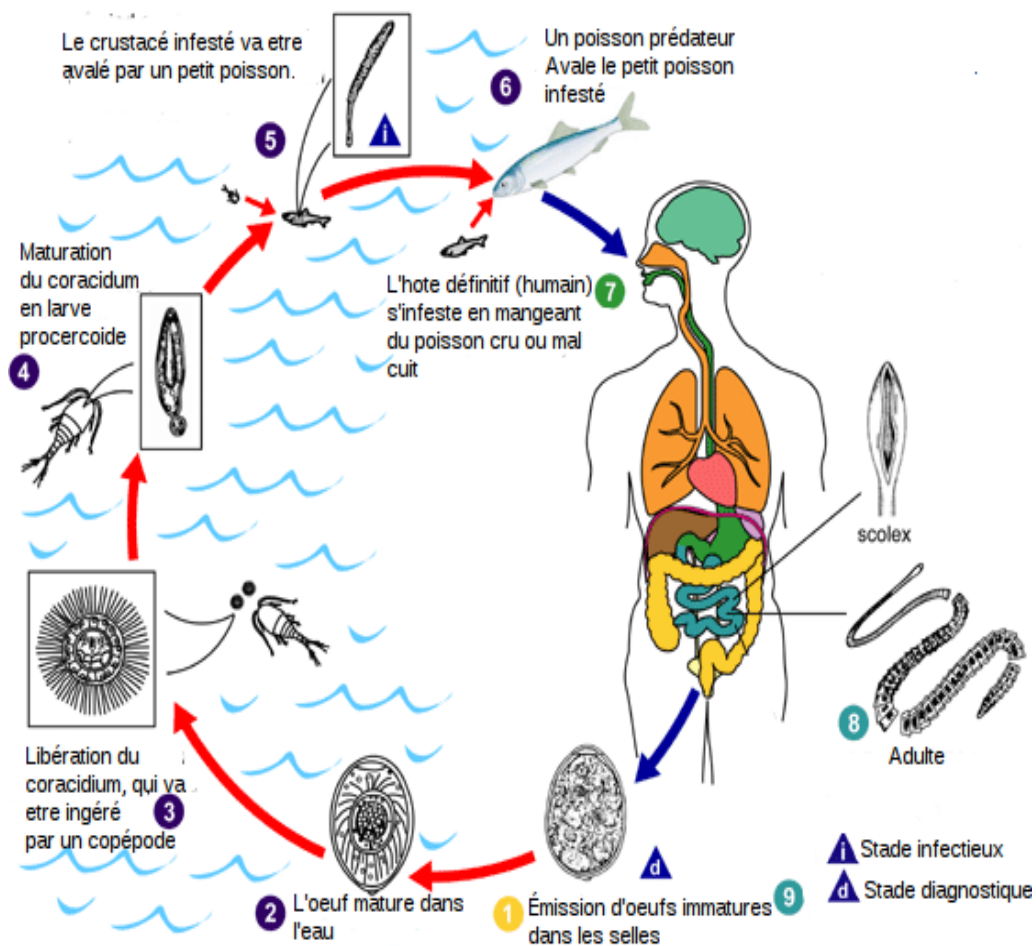


Figure 45. Evolutionary life cycle of *Diphyllobothrium latum*.

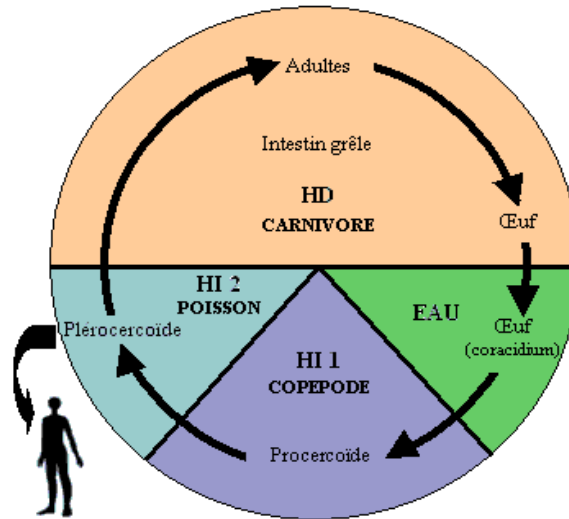


Figure 46. Evolutionary diagram of *Diphyllbothrium latum*.

c) Epidemiology

This is a rare cestodosis. Contamination occurs through the ingestion of raw or undercooked fish.

Diphyllbothrium latum is found only under specific environmental conditions: freshwater (pH between 4 and 9) and cold climates (8 to 20 °C).

The infective larva contained in fish does not survive cooking above 56 °C.

Clinical signs

Diphyllbothriosis is often asymptomatic. Symptoms are mainly digestive (constipation, hunger sensation, bloating, and abdominal pain) and appear three to six weeks after ingestion of infected fish. They are due to the attachment of the parasite by its bothridia, followed by its displacement, which destroys intestinal villi and creates an inflammatory lesion.

After several years, bothriocephalic anemia (Biermer-type) may occur, caused by vitamin B12 deficiency, as the parasite absorbs a large quantity of this vitamin.

Diagnosis is direct (eggs in feces).

The plerocercoid larva (infective stage) is destroyed by heat (2 minutes at 60 °C), freezing (20 days at 0 °C, 3 days at -5 °C), and dry salting within a few days.

3.1.3 – Class Monogenea

Monogeneans are Platyhelminths that are essentially ectoparasites of fish. This group does not include any species parasitic to humans.

a) Morphology

They are characterized by a particular attachment organ located in the posterior region of the body: the haptor. This organ is the key structure on which the very existence of the parasite depends, as it ensures the host–parasite relationship.

Throughout evolution, this attachment organ has adapted to various microhabitats: the skin, the gills, and sometimes even the coelomic cavity and the stomach.

These are ectoparasites of aquatic organisms (mainly fish, on which they live on the gills and fins, and sometimes in the buccal cavity), with some notable exceptions: some species are found in the urinary bladder of certain amphibians (*Polystomatidae*), and one species (*Oculotrema hippopotami*) lives on the eye of hippopotamuses.

Their size ranges from 0.5 to 6 mm, although some species can reach 30 mm. The estimated number of monogenean species is 25,000—approximately as many as the number of teleost fishes they parasitize.

Eggs are produced individually. They generally have polar filaments of variable length. A few rare species of monogeneans attach their eggs to the gills of the host.

Monogeneans are mostly hermaphroditic (Figure 47).

The male apparatus includes a testis, although some species possess several. The vas deferens opens into the copulatory organ. With rare exceptions (e.g., *Diplozoidae*), the spermatozoa are not flagellated.

The female apparatus consists of a single ovary. The vitelline glands are generally highly developed. The vagina, when present, opens on either the dorsal or ventral surface of the body, or even laterally. Some species have a double vagina.

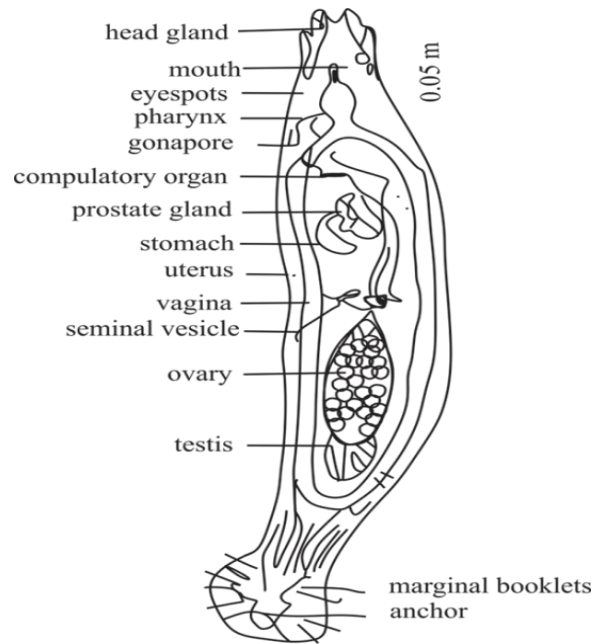


Figure 47. Anatomy of *Gyrodactylus* sp.

b) Life cycle of monogeneans

Monogeneans are generally characterized by their direct life cycle (egg, larva, adult) with a single host (Figure 48).

The life cycle includes a ciliated larva emerging from the egg, the oncomiracidium, which actively searches for a suitable host on which it attaches and develops into an adult.

In the case of the gill worm causing Dactylogyrosis, the adult releases eggs that either attach to the gills of the host or disperse into the environment. These eggs hatch rapidly (within a few days) to produce ciliated larvae capable of actively searching for their hosts. Once attached - often at the base of the gills - the larva metamorphoses into an adult.

Adults tend to settle at the extremity of the gills, which facilitates the expulsion of eggs.

However, certain genera such as *Gyrodactylus* (Figure 47) deviate from this pattern. Also known as “killer Russian dolls”, *Gyrodactylus* are viviparous and exhibit polyembryony, in which a single individual can carry several developing generations ready to be released into the environment.

Gyrodactylus, or skin worms, are metazoan parasites that attach to the skin of fish using their hooks, causing Gyrodactylosis. These flatworms measure 0.5 mm to 1 mm.

As monogenean trematodes, they attach to a single host and cause small lesions by grazing on the epithelial tissue.

Externally, the skin worm resembles the gill worm, although the latter is smaller (0.1 to 0.3 mm) and lays eggs.

In small numbers, they cause little damage. However, because these worms multiply very rapidly, they become a serious threat, causing severe damage to skin tissues and creating entry points for secondary pathogens.

Gyrodactylus is viviparous: it gives birth to a new individual without laying eggs.

Under favorable conditions, these parasites multiply very quickly on the surface of the infected fish without ever needing to leave the host.

A *Gyrodactylus* that leaves its host survives only around twelve hours in open water.

The rate of reproduction increases with temperature.

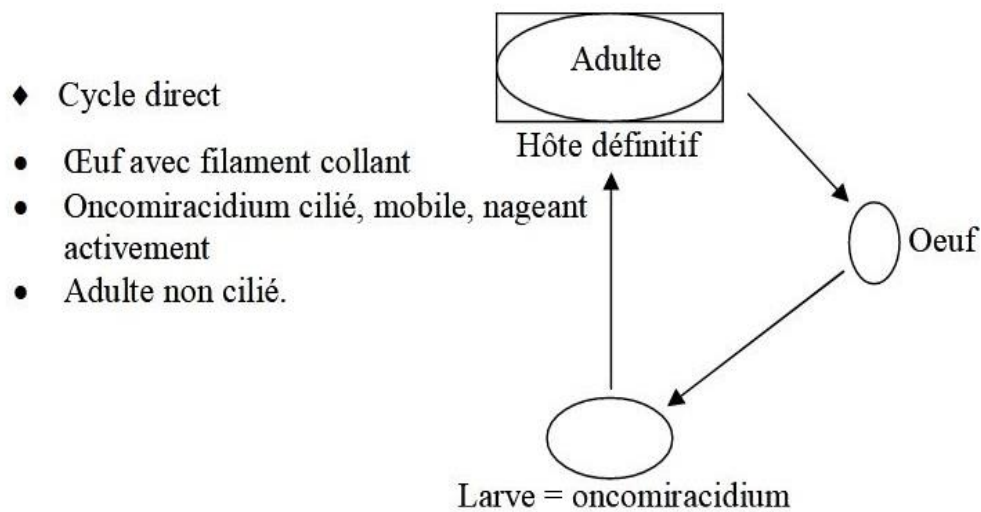


Figure 48. Typical developmental cycle of Monogeneans.

The presence of these parasites is common in ponds. Poor water quality, poor sanitary conditions, inadequate filtration, insufficient water flow, excessive organic matter, overcrowding of fish, and stress are all factors that promote these infestations.

3.2 – Phylum Nematoda

Nemathelminths are cylindrical, unsegmented worms with tapered extremities, covered with a strong and flexible cuticle. Their growth occurs through molting.

There is no circulatory or respiratory system. The digestive tract is complete.

Nematodes possess phasmids, which are sensory organs located in the caudal region. Their presence is used in classification.

The reproductive system is tubular and the sexes are separate (gonochoric worms); the female is usually larger than the male.

In the male, the testis is coiled, and the copulatory apparatus includes rigid chitinous spicules and sometimes a caudal bursa. The posterior extremity is typically curved ventrally.

In the female, there are two ovaries and fertilization is internal, with oviparity or ovoviviparity. The posterior extremity of the body is straight and blunt.

Genus *Enterobius*

Enterobius vermicularis is responsible for oxyuriasis, a cosmopolitan parasitosis transmitted via the fecal–oral route. It is a strictly human parasite, and children are most frequently affected. Transmission occurs by ingestion of embryonated eggs (digestive route) with frequent self-infection, or by inhalation of eggs (respiratory route).

Contamination may be intra-individual (self-infection cycle) or inter-individual, the latter being the most significant.

Group living, thumb-sucking, and nail-biting are major risk factors.

a) Morphology

Adult pinworms are oviparous, white, roundworms, highly mobile, with a cephalic swelling. They possess a mouth surrounded by three retractile cutting lips (allowing attachment to the intestinal mucosa) and two lateral longitudinal ridges.

The male (5 mm long × 0.2 mm) has a posterior extremity ventrally curved and bears a spicule.

The female (1 cm long × 0.5 mm) has a posterior extremity tapered and translucent, with a ventral vulva located in the anterior third of the body. Only the female is visible.

The eggs, colorless and oval-shaped, measure $55 \times 30 \mu\text{m}$. The asymmetric shell (with one flattened side), thick and smooth, contains a mobile embryo (Figure 49).

They can persist in the environment for 3 to 4 days.

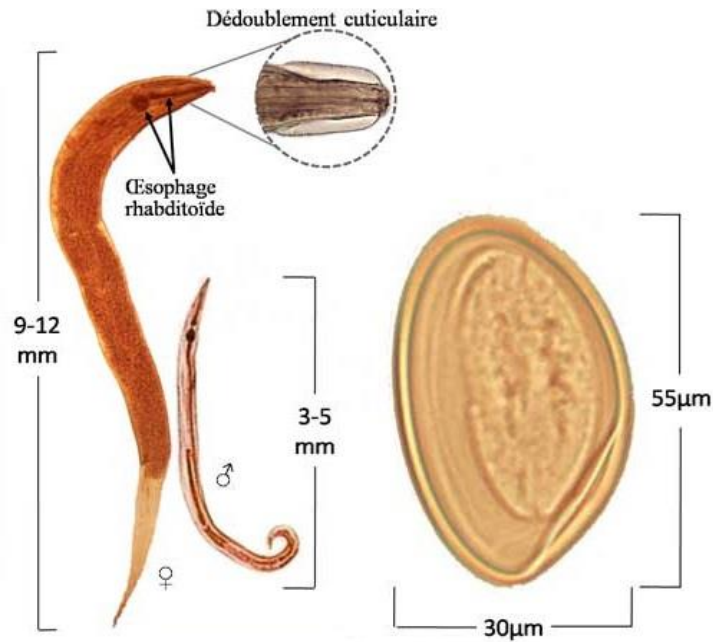


Figure 49. Morphology of *Enterobius vermicularis*.

b) Life cycle

The life cycle is short and monoxenic (duration: 28 days) (Figure 50).

The adults live and mate in the cecum of humans, and may sometimes cause appendicitis.

After mating, the males die and are eliminated in the feces, whereas the females migrate to the rectum, attach themselves, and lay eggs. This leads to itching or anal pruritus.

The eggs remain glued around the anus and develop into embryos that are immediately infective.

The child becomes infected by putting contaminated fingers into the mouth (self-infection, “dirty-hands disease”), or by inhaling dust containing eggs.

Once the eggs reach the digestive tract, digestive secretions break the shell, releasing the larvae, which reach the adult stage in about thirty days.

Adults feed on bacteria in the intestinal tract and are not pathogenic unless their population becomes excessively high, in which case they may cause intestinal obstruction.

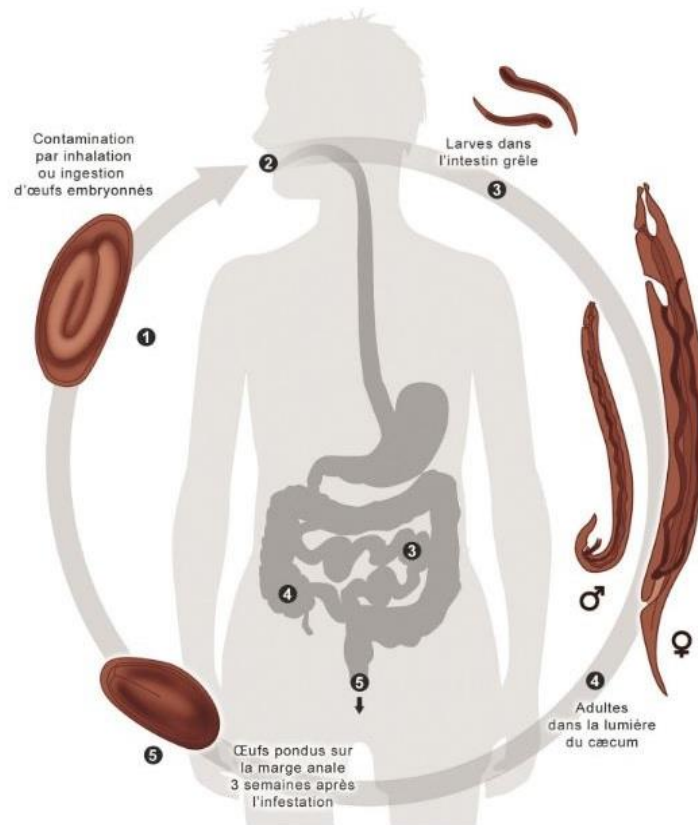


Figure 50. Life cycle of *Enterobius vermicularis*.

Contamination is direct or indirect fecal-oral (contaminated food) through the ingestion of embryonated eggs.

The eggs are digested in the small intestine, which leads to the release of a larva. Within 2-4 weeks, and after several molts, the eggs develop into adult forms.

Adult pinworms mate in the intestine, and the gravid females migrate to the anal margin (actively crossing the anal sphincter) to lay eggs (preferably at night), producing up to 10,000 eggs in 20 minutes.

The eggs do not require maturation in the external environment; they are immediately embryonated, which allows self-infestation.

Genus *Trichuris*

Phylum Nematoda, Class Enoplea, Order Trichocephalida, Family Trichuridae.

Trichocephalosis is a cosmopolitan cecal nematodosis caused by *Trichuris trichiura* (or *Trichocephalus trichiurus*).

Whipworms (from *thrix* = hair and *cephale* = head) are hematophagous worms (5 µL of blood per worm per day).

a) Morphology

Adult *T. trichiura* parasites are characterized by a very thin anterior (cephalic) end, which burrows into the host's mucosa, and a thicker posterior end, which remains in the intestinal lumen.

The worms are white and measure approximately 30 to 50 mm long (Figure 51).

The male worm has a posterior extremity rolled into a hook and is smaller than the female.

The **female worm** has a broader and straight posterior extremity and lays 3,000 to 20,000 unembryonated eggs per day. These eggs embryonate in nature within 3 weeks.

Adult worms live for a long time in their host, typically up to 5 years. The most severe infections occur in children aged 5 to 15 years, with both frequency and intensity decreasing in adulthood.

The **egg**, lemon-shaped, has a prominent mucous plug at each pole and a double shell: the outer shell is smooth and brown, and the inner shell is yellow.

Its dimensions are 50–60 μm \times 25–30 μm .

The egg is unembryonated when laid.

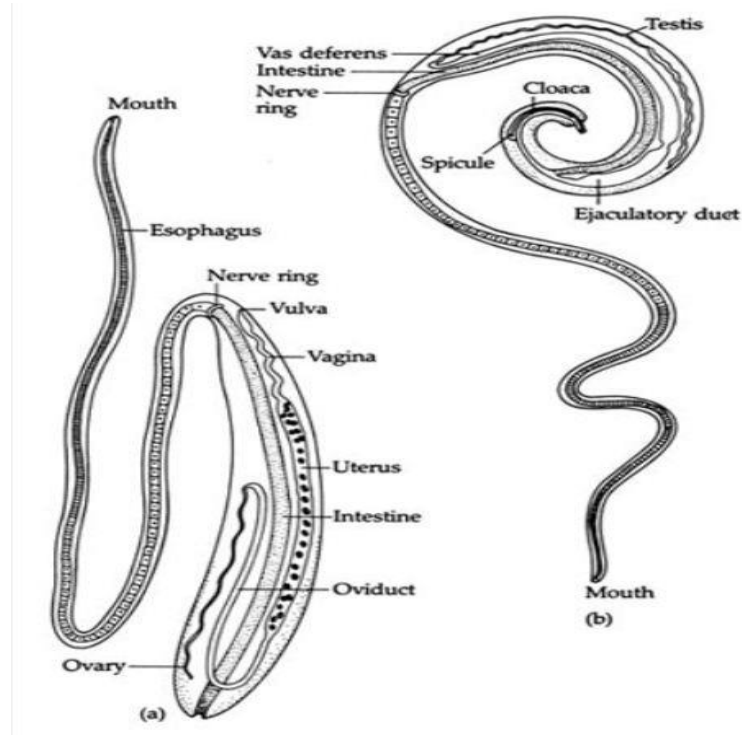


Figure 51. Morphology of *Trichuris trichiura*.

b) Life cycle:

The life cycle of the whipworm (Figure 52) is direct (monoxenous) and humans are the main host, although cases of infection have been reported in chimpanzees, pigs, lemurs, and other

monkeys. It is a parasitic disease linked to fecal contamination (contaminated soil, fruits, and vegetables).

Contamination occurs through ingestion of embryonated eggs (L1 larva) present on hands or in soil (eggs must remain for at least 15 to 30 days in warm and humid soil before becoming infectious), or on contaminated food or water.

After ingestion of *Trichuris* eggs, the hatched larvae molt in the mucosa of the small intestine within 2 to 3 weeks (L2, L3 and L4 stages), then migrate to the cecum and the colon where they burrow into the epithelium and become adult whipworms within approximately 12 weeks. The adult worms are located in the colon and cecum; their anterior end is embedded in the glands, while the posterior end floats freely in the lumen.

Eggs become detectable in stool 70 to 90 days after ingestion, and carriers may excrete eggs for years if not treated.

After being passed in feces, maturation and embryonation take place in soil (3 weeks or more depending on conditions), and the L1 larva remains inside the egg.

The lifespan of the parasite is 5 to 10 years, and a female lays approximately 30,000 eggs per day.

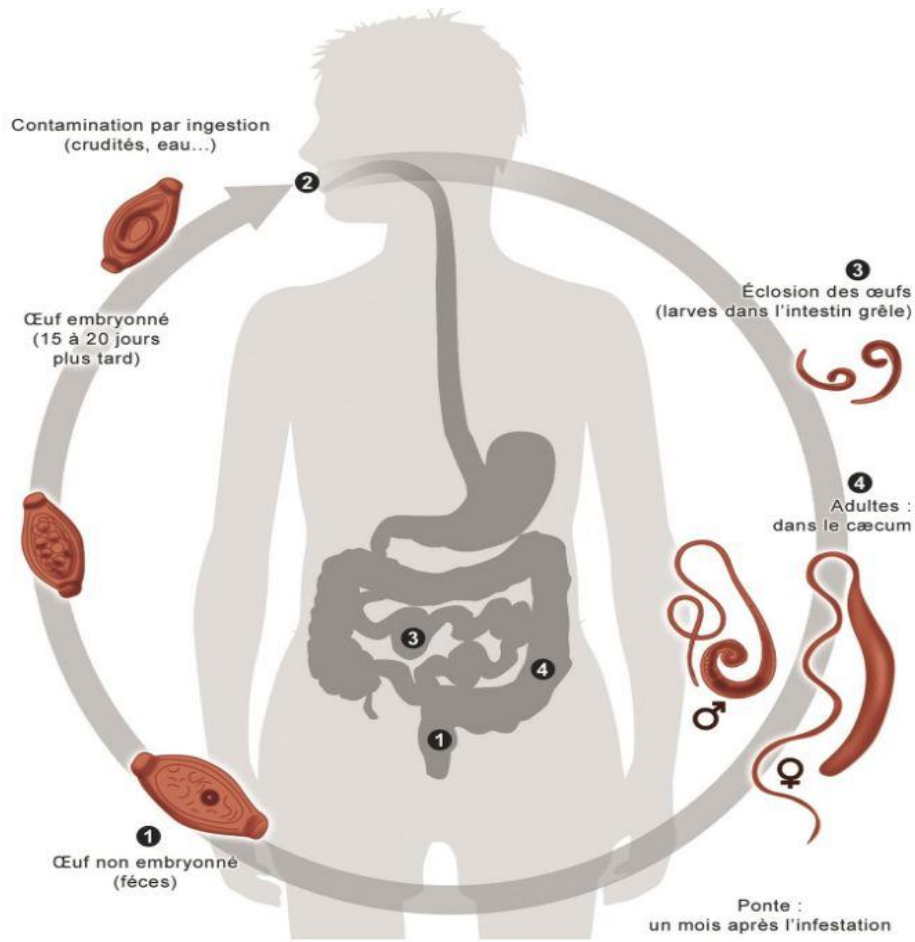


Figure 52. Life cycle of *Trichuris trichiura*

Genus *Trichinella*

The genus *Trichinella* belongs to the phylum Nematoda, class Enoplea, order Trichocephalida and family Trichinellidae.

Trichinellosis (or trichinosis) is a cosmopolitan zoonosis mainly distributed in temperate regions and caused primarily by *Trichinella spiralis*, a viviparous nematode of the small intestine, contracted through the ingestion of raw or undercooked meat contaminated with infective larvae.

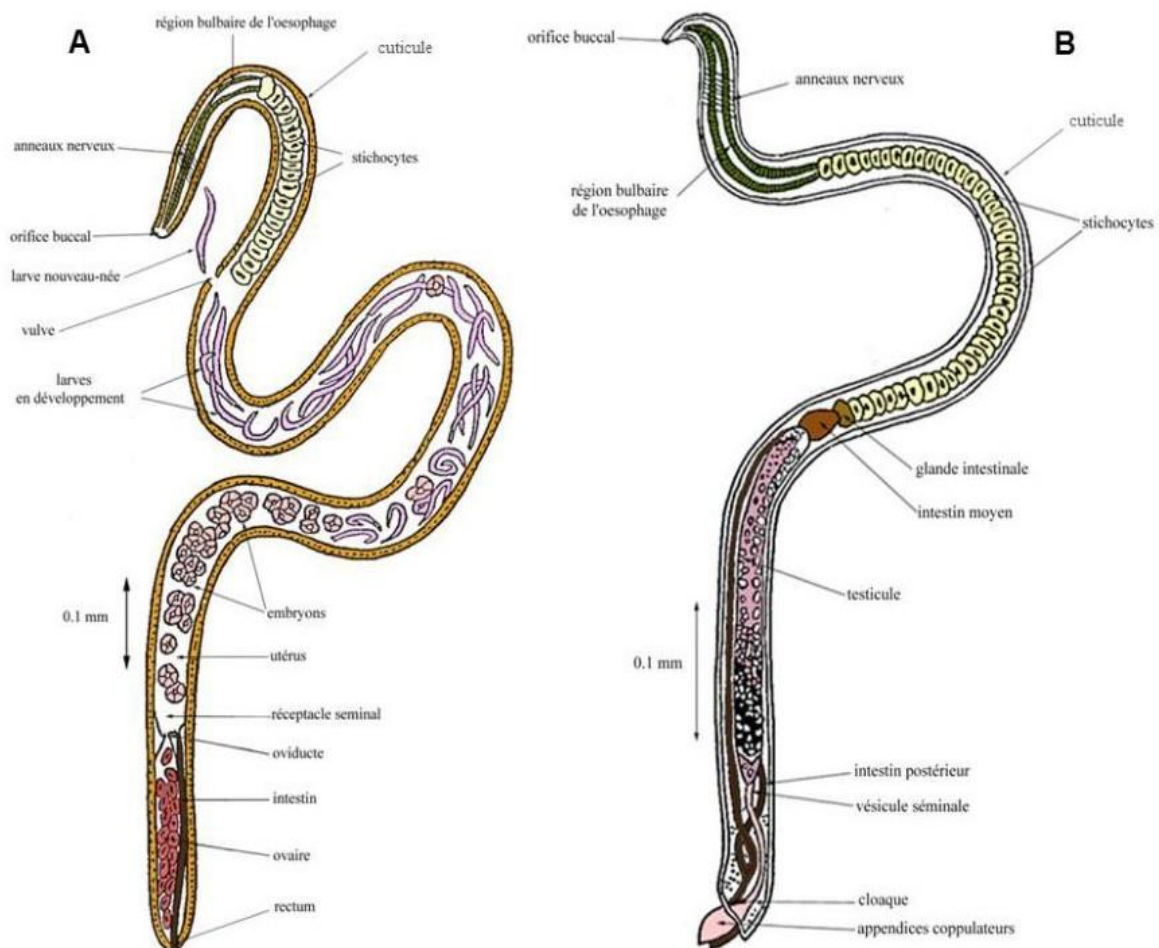
This species is most closely associated with domestic pigs but also infects many wild mammals (e.g., wild boars, rodents) and domestic mammals (e.g., horses, dogs). It has a worldwide distribution and is the main agent responsible for human trichinellosis. This parasitosis can be fatal.

In rats, infestation is propagated through the cannibalistic behavior characteristic of this species: the intermediate host and the definitive host may be the same species.

a) Morphology:

The male *Trichinella spiralis* measures 1.5 mm × 36 µm and presents characteristic copulatory appendages.

The whitish female is twice as long as the male (3 mm × 36 µm) and, with its tapered anterior extremity, resembles that of the whipworm. The adult female contains numerous newborn larvae visible inside its uterus (Figure 53).



**Figure 53. Representation of the Adult Stage at Day 5 of *Trichinella spiralis*.
(A: Female; B: Male)**

b) Life cycle:

Trichinella spiralis is a nematode with an auto-heteroxenous cycle, meaning that it acts as both intermediate host and definitive host. The entire parasitic cycle occurs through an intestinal phase followed by a muscular phase, with an intermediate lymphatic-blood migration phase (Figure 54).

This parasite is found in the small intestine of numerous animals (dog, cat, rat, pig, wild boar, warthog, human). Females penetrate the intestinal mucosa and release two larvae per hour during the twenty-eight days of their life.

The females are viviparous and produce larvae that reach the right side of the heart via the lymphatic fluid, then migrate to the lungs, pass into the left side of the heart, and are distributed throughout the organism via the aortic circulation. These larvae settle in striated muscles and become encysted for 12 to 18 months. The cysts calcify and are infective. Humans become infected by consuming undercooked meat.

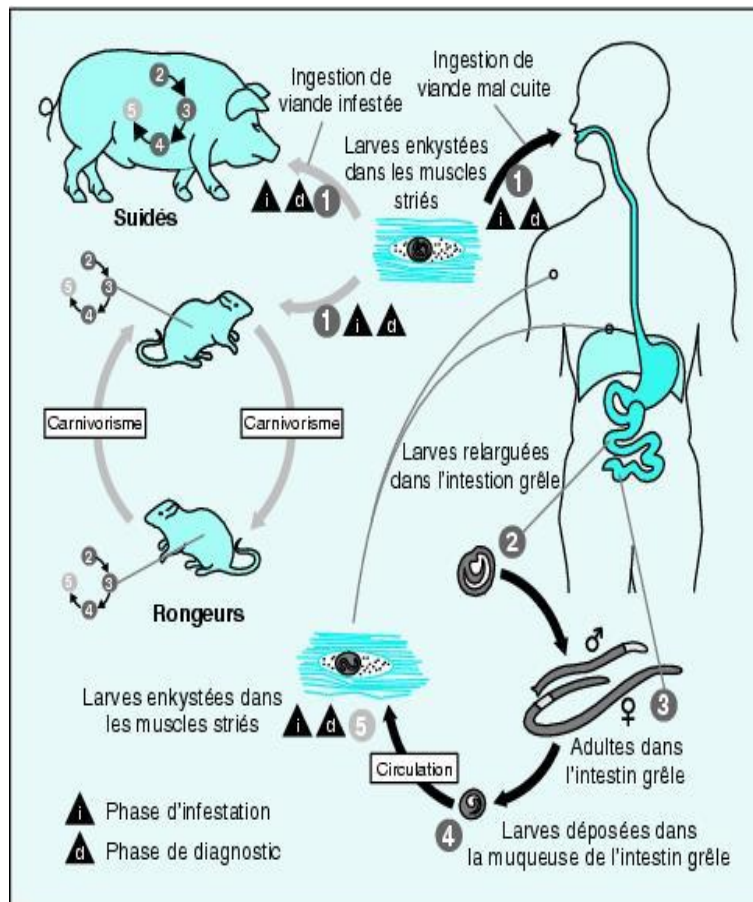


Figure 54. Life cycle of *Trichinella spiralis*.

Genus *Ancylostoma*

The genus *Ancylostoma* (from *agkylo* = hooked and *stome* = mouth) belongs to the Phylum Nematoda, Class Secernentea, Order Strongylida, Family Ancylostomidae, Subfamily Ancylostominae (*Ancylostoma duodenale*) or Subfamily Bunostominae (*Necator americanus*).

Hookworm infections (ankylostomiasis) are anthroponoses caused by *Ancylostoma duodenale* and *Necator americanus*, hematophagous nematodes of the duodenum and jejunum. This parasitosis is transmitted through the skin, and larval development depends on temperature (*Ancylostoma* > 22 °C, *Necator* > 25 °C), conditions typically found in hot and humid countries, tropical areas, or microclimate zones (mines, brickyards, etc.).

A. duodenale is distributed in Africa, Europe, and the Middle East. In Algeria, it has been reported in certain foci such as Ouenza (Tébessa), El Tarf, and Chiffa (Blida).

Necator americanus is endemic in Latin America.

a) Morphology:

These parasites possess a buccal capsule equipped with teeth or cutting plates. Observation of the buccal structure allows species diagnosis: a mouth with teeth (*Ancylostoma*) or a mouth with cutting plates (*Necator*).

Adult hookworms resemble large white pinworms (Figure 55), turning red after feeding on blood (hematophagous). Their cuticle is smooth with transverse striations.

Male: Measures 8–10 mm. It carries a copulatory caudal bursa and two spicules allowing species identification.

Female: Measures 10–15 mm and ends with a blunt posterior extremity. The vulva is located in the posterior third of the body (Y-shape in copula).

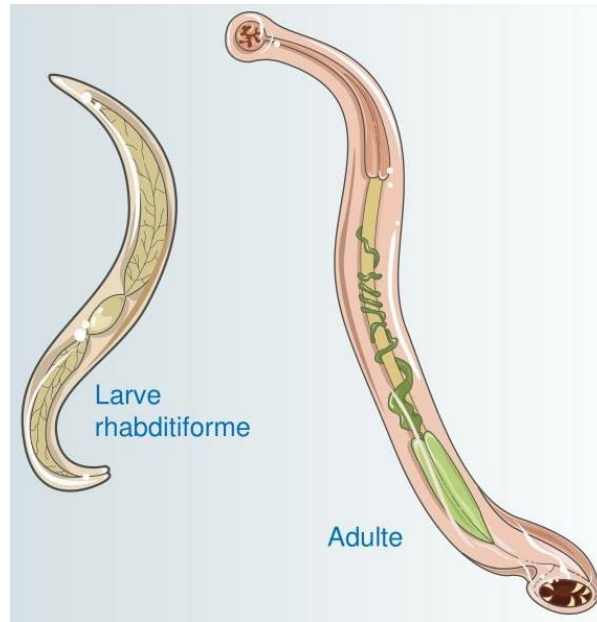
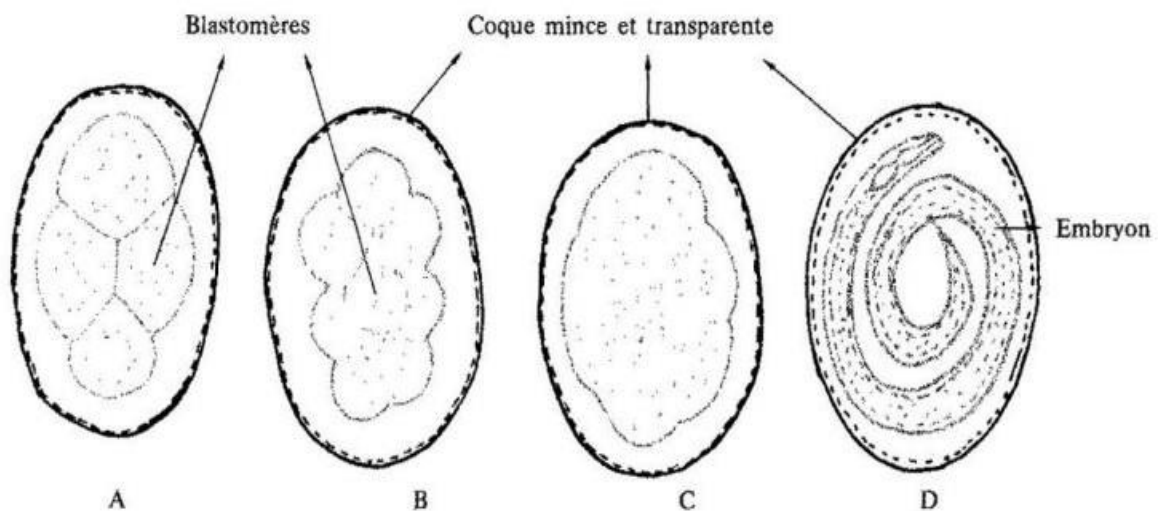


Figure 55. Morphology of *Ancylostoma duodenale*.

Egg: Ellipsoidal, measuring 60–70 $\mu\text{m} \times 40 \mu\text{m}$, and covered with a thin, transparent shell. The egg is colorless and not embryonated at deposition but contains blastomeres that multiply rapidly; the morula stage is observed at the time of coprological examination.

The eggs contain 4 blastomeres at oviposition (*Ancylostoma duodenale*) and 8 blastomeres at oviposition (*Necator americanus*) (Figure 56).



A : *A. duodenale* ; B et C : *N. americanus* ; D : oeuf embryonné

Figure 56. Eggs of Hookworms.

Larval stages: Several developmental stages can be observed:

- **L1 rhabditiform larva** (250 µm, double oesophageal swelling);
- **L2 strongyliform larva** (500 µm, single oesophageal swelling);
- **L3 infective strongyliform larva** (600 µm, single oesophageal swelling, oesophagus equal to 1/4 of the body length, enclosed in the sheath of the previous moult).

b) Life cycle:

Contamination occurs transcutaneously through direct contact with the infective encysted strongyliform larva, especially in individuals walking barefoot in muddy soil (agricultural work, gardening, mines, brickyards, etc.).

The cycle is direct, and transmission occurs via penetration of the infective L3 larva found in muddy soil, under the effects of positive thermotropism (attracted by body heat), positive histotropism (attracted by skin odor), and sufficient humidity. The definitive host is humans.

Ancylostoma duodenale and *Necator americanus* can multiply both in the small intestine and in the soil, and both species share the same parasitic cycle. They inhabit the upper small intestine: *A. duodenale* in the duodenum, and *N. americanus* in the upper jejunum.

The parasitic cycle (Figure 57) begins when fertilized females in the small intestine of an infected human lay eggs that are eliminated in the external environment with feces.

These eggs embryonate within 24 hours, provided that humidity is high, oxygenation is adequate, temperature ranges between 14-37°C (optimum 22–26°C for *A. duodenale* and 27-30°C for *N. americanus*), and light is low.

The egg hatches and releases a rhabditiform larva, which transforms on day 3 into a strongyliform larva, and on day 5 into an encysted infective strongyliform larva within its moult. At this stage, the larvae exhibit notable resistance and vitality, surviving 3 weeks in cold periods, 2 to 10 months in favorable conditions (cultivated soils or mines), and up to 18 months in water.

The Hookworm life cycle can be summarized as follows:

- **Day 0:** egg expelled in feces onto soil
- **Day 1:** hatching of egg → free L1 larva
- **Day 3:** first moult → free L2 larva

- **Day 7:** second moult → L3 larva retained within the exuviae, survival 1-2 months (or more) ;
- **L3 strongyliform larvae penetrate actively through the skin** (within 5 minutes), with possible oral ingestion for *Ancylostoma* only ;
- **Larvae migrate via the bloodstream to the lungs**, pass through alveoli, ascend the tracheo-bronchial tree, and are swallowed (entero-pulmonary migration similar to *Ascaris*) ;
- **They then reach the digestive tract**, where the final moults occur and the adult stage is reached.

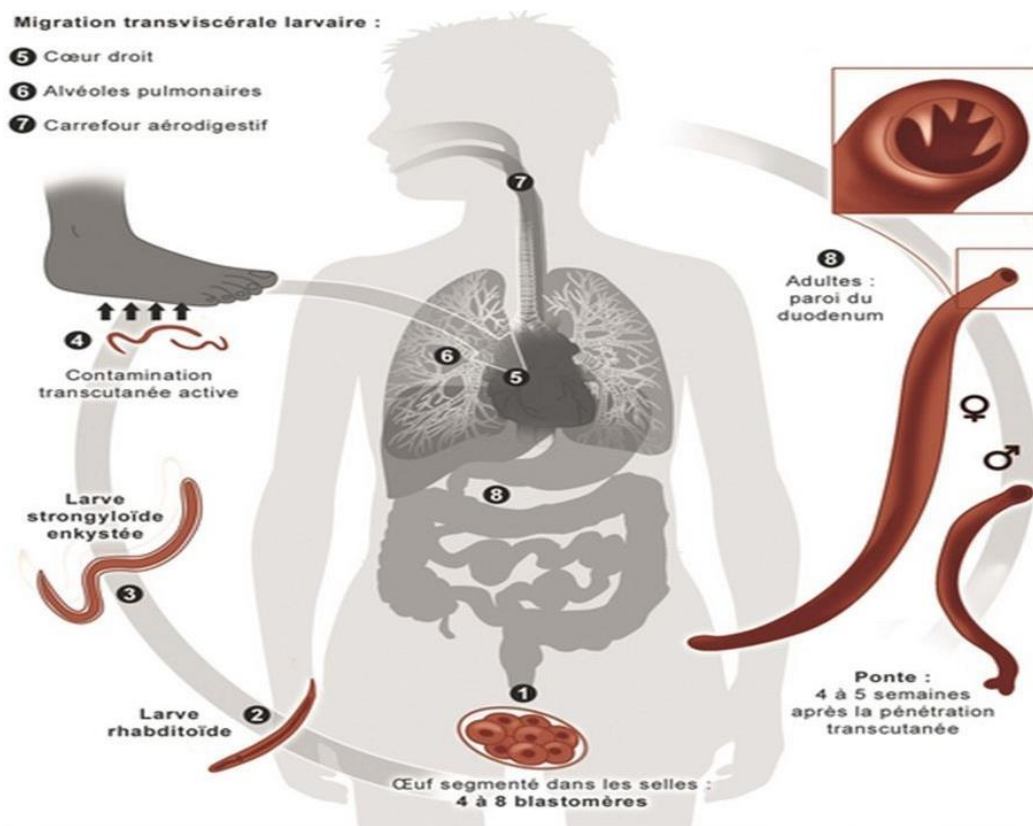


Figure 57. Evolutionary cycle of Hookworms.

The duration of the prepatent period is 6 weeks for *Ancylostoma* and 8 weeks for *Necator*. The maximum life span is 5 years.

Genus *Ascaris*

Ascariasis is a cosmopolitan helminth infection whose prevalence is closely linked to fecal–oral transmission (poor hygiene), affecting approximately one-quarter of the world’s population, particularly in developing countries.

In Europe and in industrialized nations, it has virtually disappeared.

This parasitic disease is caused by a nematode strictly restricted to humans, *Ascaris lumbricoides*, which lives and develops in the digestive tract, mainly in the small intestine.

a) Morphology:

Ascaris lumbricoides (Figure 58) is a large cylindrical worm, pinkish in color, with a finely striated cuticle. The buccal opening is surrounded by three large lips (one dorsal and two ventrolateral), each bearing sensory papillae.

- **Male:** The male (10–20 cm) has a posterior extremity curved ventrally.

The genital apparatus and the digestive tract both open into a common cloaca, which emerges externally via a subterminal cloacal pore.

Two copulatory spicules are clearly visible during mating.

There is a single filiform testis.

- **Female:** The posterior extremity of the female (15–25 cm) is straight.

A ventral genital pore is present at a slight narrowing of the body.

It contains two filiform ovaries and a large uterus.

- **Egg:** The eggs (60–70 × 30–40 μm) are oval or round.

They possess a double brown shell with an external mamillated layer and an internal hyaline layer (Figure 58).

A fertilized female releases several hundred thousand eggs per day (100,000–500,000), although egg production decreases when many females coexist in the intestine (population-regulation phenomenon).

The eggs are non-embryonated when laid.

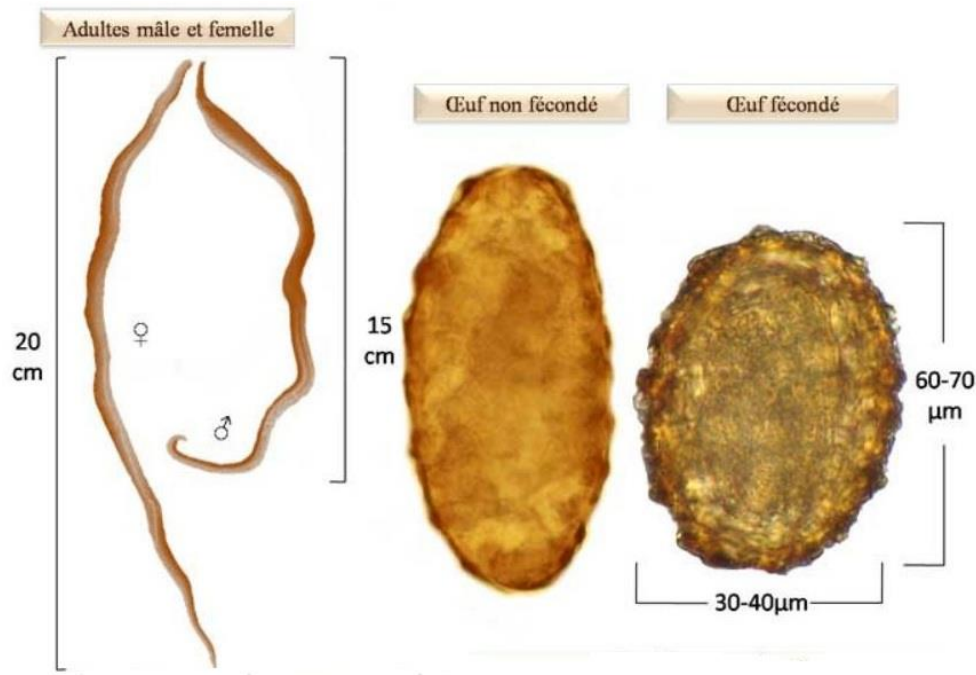


Figure 58. Morphology of *Ascaris lumbricoides*.

b) Biological cycle:

The life cycle of *Ascaris lumbricoides* is monoxenous. The adults mate and lay eggs in the small intestine. The female produces 240,000 eggs per day, which are eliminated with the feces and will develop into infective larvae after 30 to 40 days if the external temperature is around 30 degrees, with humid and shaded soil.

Contamination is oro-fecal, through ingestion of eggs (food contaminated with soil). After digestion of the shell, the larva will migrate through the organism. The larvae first cross the digestive epithelium before reaching the liver through the bloodstream. They remain there for three to four days, then reach the right heart via venous circulation, then move to the lungs through the pulmonary arteries and remain there for about one week. They undergo two molts during this phase. They then move up the respiratory tract (bronchioles, bronchi, trachea) and reach the oropharyngeal junction, after which they take the digestive route again and return to the small intestine, where they become adults.

The migration lasts approximately 15 days. After the small intestine, the larvae pass into the large intestine, then into the rectum where they reach sexual maturity (Figure 59).

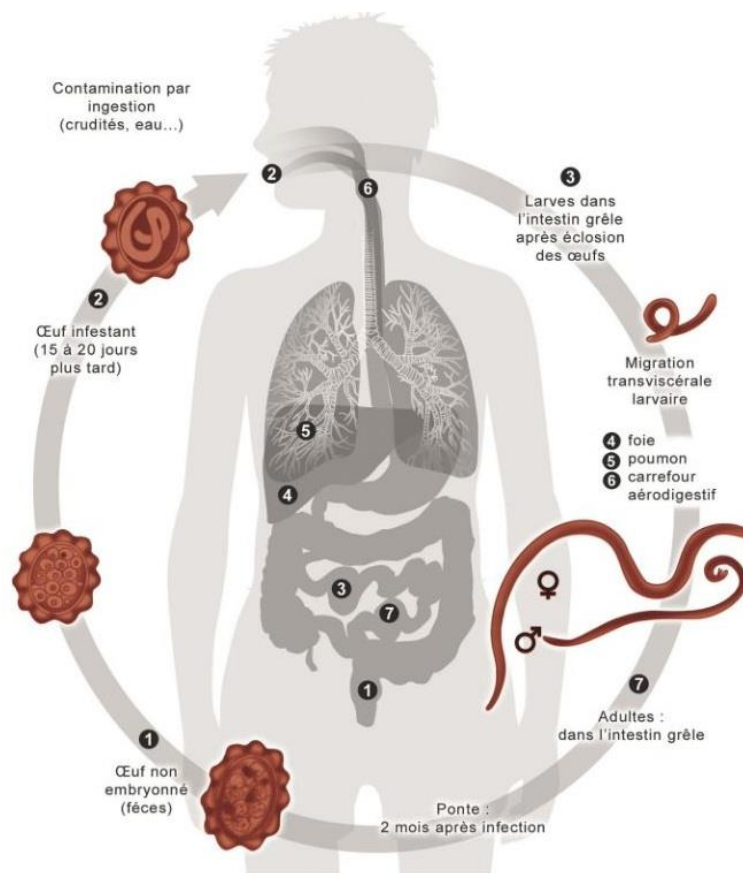


Figure 59. Biological cycle of *Ascaris lumbricoides*.

The observed symptoms include intestinal obstruction, strangulation of the digestive tract (hernias), acute peritonitis, and hepatic and pancreatic involvement.

Additionally, ascarids release toxic substances that affect the human nervous system.

Other vertebrates may be parasitized by practically the same species (horse, pig, dog, cat).

Adults can reach a lifespan of 12 to 18 months.

Genus *Dracunculus*

Phylum Nematoda, Class Secernentea, Order Camallanida, Family Dracunculidae.

Dracunculiasis is an anthroponosis caused by *Dracunculus medinensis* (also called Guinea worm, Medina filaria, or Avicenna's threadworm). The female of this nematode is very large and develops in the subcutaneous tissues of the definitive host.

It is an endemic disease with a strictly African distribution (Ethiopia, Mali, Chad, South Sudan). The intermediate host is a freshwater crustacean of the genus *Cyclops* (0.5 to 1 mm) and the definitive host is humans, dogs, and horses.

Transmission occurs orally (ingestion of infected *Cyclops* with drinking water).

a) Morphology:

The adult worms (Figure 60) have a cylindrical, filiform body of whitish color.

The cephalic extremity shows an anterior shield and a triangular buccal orifice equipped with papillae. The caudal extremity is short and conical.

Male: The male measures a few centimeters in length (2 to 3 cm × 0.4 mm).

Female: The female measures up to 1 m in length (0.7 to 1.2 m × 2 mm). It is mainly found in subcutaneous tissues. It is viviparous and its uterus contains 1 to 3 million embryos. It can live 1 year.

The larvae, or microfilariae, are elongated and measure 500 to 750 microns × 15–20 μm. Their tail is elongated but not striated, with a tapered, bifid posterior extremity and a rounded cephalic extremity. They possess a transversely striated cuticle and have no sheath. They can survive only a few days in water.

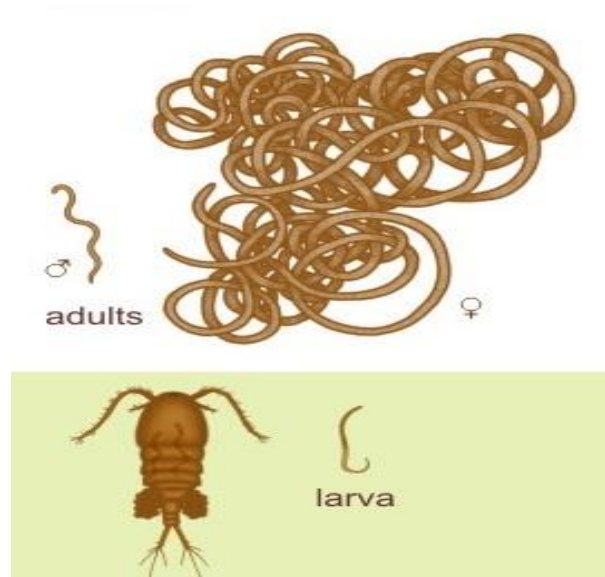


Figure 60. Morphology of *Dracunculus medinensis*.

b) Biological cycle of *Dracunculus medinensis*:

The cycle is dixenous, with humans as the definitive host and *Cyclops* as the intermediate host. The parasitic infection is acquired through ingestion of L3 larvae with unfiltered water containing *Cyclops* (Figure 61).

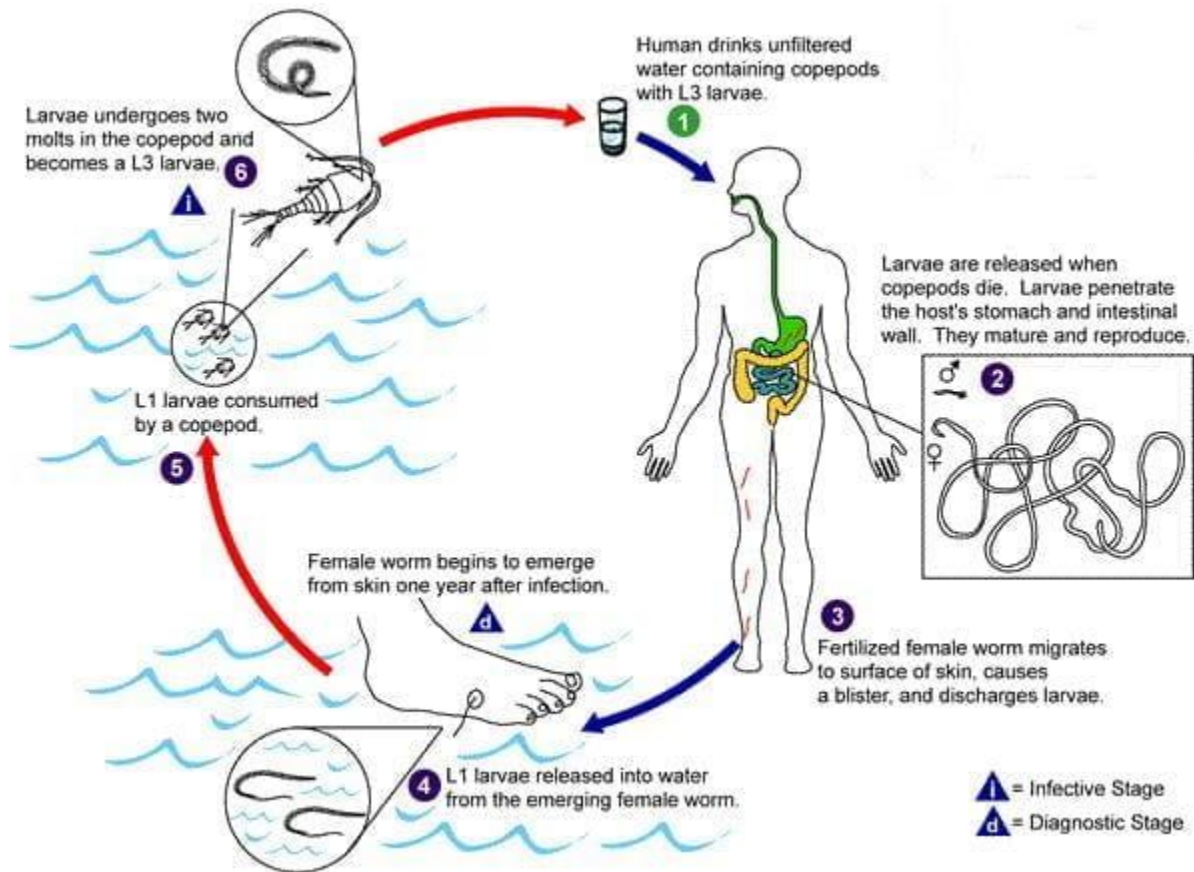


Figure 61. Biological cycle of *Dracunculus medinensis*.

Step 1. Contamination occurs by drinking water contaminated with tiny crustaceans infected with *Dracunculus* larvae.

Step 2. After being swallowed, the crustaceans die and release the larva, which crosses the wall of the stomach and intestine and enters the abdominal cavity. The larva then reaches maturity, and the adult worms reproduce.

Step 3. After reproduction, the male worms die, and the females migrate to the subcutaneous tissues, usually down to the lower leg or feet. About one year after infection (ranging from 9 to 14 months), the female worm reaches the surface of the skin and creates a blister. The blister causes an intense burning sensation and eventually ruptures.

Step 4. When the infected person tries to relieve the burning sensation by placing their leg in

water, the gravid females release larvae into the water.

Each female worm releases at least one million microscopic first-stage larvae into the water. The larvae can remain active for up to five days in stagnant water, where they die unless they are ingested by *Cyclops*.

Step 5. The larvae are ingested by other crustaceans.

Step 6. Inside the crustacean, the larvae undergo different developmental stages and become capable of causing infection. When a person ingests the crustaceans, the cycle is completed. Inside the *Cyclops*, the larvae develop to the L3 (infective) stage in 10 to 14 days. When first-stage larvae are swallowed by humans, they do not develop further and are likely killed immediately by gastric juice.

3 – Phylum Arthropoda

3.3 – Phylum Arthropoda

3.3.1 – Subphylum Hexapoda

Class Insecta, Order Anoplura.

3.3.1.1 – Order Anoplura

The Anoplura (sucking lice) constitute an order closely related to the Mallophaga (chewing lice). *Anoplura* derives from the Greek *anoplos* (“without weapons”) and *oura* (“tail”). Assigned by Linnaeus in 1758, this name refers to the fact that the abdominal extremity of sucking lice lacks cerci or visible copulatory organs.

All Anoplura are obligatory external parasites of mammals. They pierce the skin of their host and feed on its blood. Their mouthparts are highly modified into piercing-sucking structures (hematophagous) and are retracted inside the head.

Ranging from 0.5 to 5.0 mm long, the body of adult Anoplura is strongly flattened dorsoventrally; in dorsal view, it appears oval or more or less circular. Pale in color, the body may sometimes be covered with brownish or gray pigments.

The head is small, narrower than the thorax. The antennae are composed of three to five segments and are approximately the same length as the head.

The thorax bears no wings; the wings have become completely useless following the specialization of sucking lice for parasitism. The legs are short, curved inward and adapted to

grip the host's hair. At the end of each leg, a large apical claw can close around the hair shaft and ensure a solid grasp.

The oval abdomen consists of nine segments.

Anoplura undergo incomplete metamorphosis. Nymphs resemble adults and live on the same host as their parents. Lice spend their entire life — from egg to adult — and reproduce on their host.

3.1 – Genus *Pediculus*

Pediculus capitis, the head louse, and *Pediculus humanus*, the body louse.

Pediculosis of the head : Parasitic disease caused by *Pediculus capitis*.

Phylum Arthropoda, Subphylum Hexapoda, Class Insecta, Subclass Pterygota, Order Phthiraptera, Suborder Anoplura, Family Pediculidae.

Pediculus capitis is a cosmopolitan parasite, strictly human. Reservoir: humans.

Head lice infestations mainly affect preschool children attending day-care centers, primary school students, and people living with infested children.

a) Morphology

Adults: They are dorsoventrally flattened, gray in color, and equipped with strong claws. Adult individuals are hexapods and wingless (apterous) and measure about 2 mm (Figure 62).

The head is elongated and bears two eyes and a pair of five-segmented antennae. The thorax is reduced, with fused segments; the legs are strong and robust, forming a clamp with the tarsal claw and a large tooth located on the tibia.

The abdomen is composed of 9 segments, of which 7 are visible; the last segment is notched in the female and bears a conical penis in the male.

The larva resembles the adult but smaller. These parasites are beige or grayish in color and remain permanently on human skin.

All stages are hematophagous and take 1 to 2 blood meals per day (one meal lasts about 30 minutes). The female is larger than the male.

Eggs: The egg, also called nit, measures about 0.8 mm. It has the appearance of a whitish grain attached to the hair, 1 cm from the scalp (Figure 62).

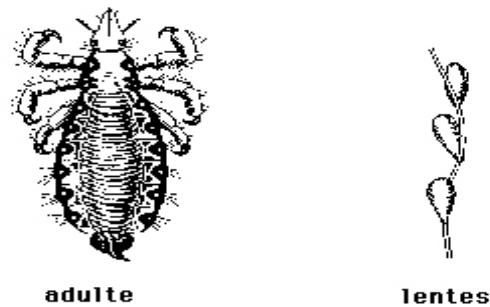


Figure 62. Morphology of *Pediculus capitis*.

b) Life cycle:

The life cycle is monoxenous and the DH = IH: human.

The life cycle of a louse is divided into three stages: the egg (or nit), the nymph, and the adult louse. The nits are laid at the base of the hair shaft, very close to the scalp. The female lays about 10 eggs per day for 1 month (300 nits per female).

The nits hatch after eight or nine days. The nymphs have the same appearance as the adult lice but are smaller. The nymphs reach their adult form in nine to twelve days.

Adult lice mate 10 hours after the final molt. The average duration of the head louse cycle is 18 days from egg to egg, and lice can live up to 30–40 days on a person's head, but if they fall off, they will die within one or two days.



Figure 63. Biological cycle of *Pediculus capitis*.

Contamination is direct (hair-to-hair contact) and/or indirect (sharing of hair accessories, combs, brushes, hats, scarves, shirt collars, etc.).

Persistent itching due to bites corresponding to blood meals is observed. There is no immunity, and secondary infection of scratching lesions is possible.

The optimal living temperature is 28-30 °C (lice leave febrile individuals and corpses).

Adult lice survive 8 to 10 days at temperatures above 10 °C, a few days at 0 °C, and only a few hours at -20 °C.

However, the egg dies in 10 minutes at 55 °C, and *Pediculus capitis* stops reproducing when humidity exceeds 85%.

Death occurs by immersion in fresh water (within 20 hours) and in salt water (within 48 hours); mineral oils and kerosene cause asphyxiation of lice.

Body Pediculosis : Parasitic infestation caused by *Pediculus humanus*

Phylum Arthropoda, Subphylum Hexapoda, Class Insecta, Subclass Pterygota, Order Phthiraptera, Suborder Anoplura, Family Pediculidae.

Pediculus humanus is a cosmopolitan parasite strictly limited to humans. Reservoir: humans.

This pediculosis emerges when hygiene conditions are poor: population displacement, wars, refugee situations, natural disasters, etc.

Transmission may occur directly or indirectly via clothing (in one night, a louse can travel 1 meter).

High temperature (60 °C for 15 minutes) is sufficient to kill the lice.

a) Morphology

Pediculus humanus is larger than the head louse and measures approximately 2 to 4 mm in length (Figure 64). The female is also larger than the male.

Aside from size, the other morphological characteristics are similar.



Figure 64. Morphology of *Pediculus humanus*

b) Life cycle

The life cycle is similar to that of *Pediculus capitis*.

The main difference is that the adults and eggs (nits) of *Pediculus humanus* are located on clothing. Adults move onto the body only during the blood meal.

The body louse can transmit diseases such as epidemic typhus (*Rickettsia prowazekii*) through louse feces, trench fever (five-day fever) through louse feces, and louse-borne relapsing fever (*Borrelia recurrentis*) through crushing of the louse.

3.2 – Genus *Phthirus*

Phylum Arthropoda, Subphylum Hexapoda, Class Insecta, Subclass Pterygota, Order Phthiraptera, Suborder Anoplura, Family Phthiridae.

The pubic louse (*Phthirus pubis*), commonly called the crab louse, is a cosmopolitan hematophagous ectoparasite that lives and develops on the surface of the skin. In humans, it causes phthiriasis, a bothersome but benign skin condition. It differs from standard lice (*Pediculus capitis* and *Pediculus humanus*) in morphology and habitat.

This louse is usually transmitted through sexual contact. The pubic louse is found in pubic hair and sometimes in the armpits. It may rarely be observed on the eyebrows, beard, or even chest hair in very hairy individuals. Like all Anoplura, pubic lice cling only to a specific type of hair based on its diameter.

Bites from the crab louse - whose mouthparts remain almost permanently inserted into the host's skin - cause small bluish lesions and itching that worsens at night, and sometimes severe allergic reactions. However, some individuals experience no symptoms. The crab louse blends in with skin color. Like the head louse, the female lays nits at the base of the hairs.

a) Morphology

The crab louse is a small, stout insect measuring 2 to 3 mm long and resembling a crab. It has a very broad thorax bearing powerful legs with large pseudo-claws (larger than those of the head louse) and a short, narrow abdomen. It is found in the pubic and perianal regions.

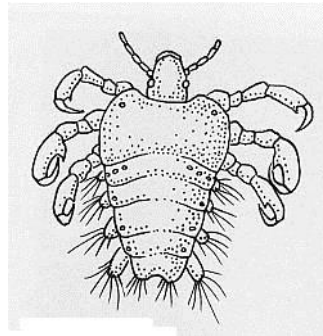


Figure 65. Morphology of *Phthirus pubis*

b) Life cycle:

The developmental cycle of *Phthirus* is comparable to that of *Pediculus*. Pubic lice go through three successive stages: egg, nymph, and adult.

The female lays and attaches her eggs (only about thirty during her lifetime, which lasts 3 to 4 weeks) glued to the hairs. Incubation lasts 7 to 8 days, and the eggs hatch into nymphs that resemble adults but are smaller.

The larva undergoes 3 molts in 12 to 17 days before transforming into an adult. Adults cannot survive more than 24 hours without contact with humans, and the eggs no more than eight days (Figure 66).

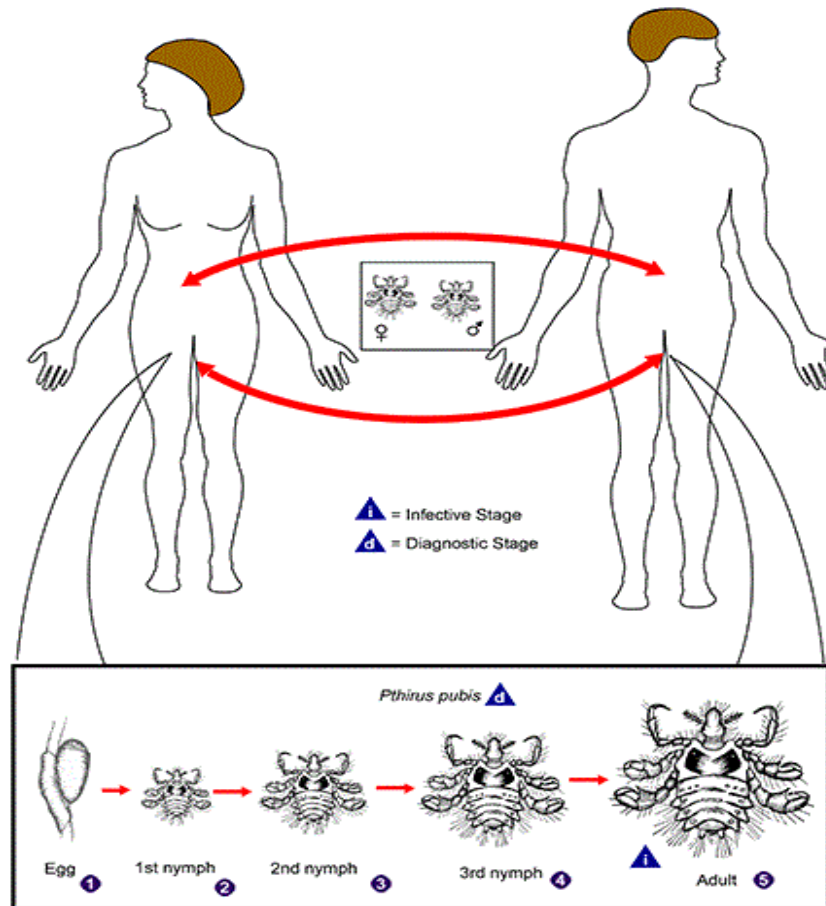


Figure 66. Biological cycle of *Phthirus pubis*

3.3.1.2 – Order Siphonaptera

Genus *Ctenocephalides*

Phylum Arthropoda, Class Insecta, Subclass Pterygota, Order Siphonaptera, Family Pulicidae.

The genus *Ctenocephalides* includes ectoparasitic insects that attack humans, livestock, domestic animals such as the cat flea, the dog flea, the guinea pig, as well as pet rabbits and, to a lesser extent, certain rodents (mainly the rat).

Ctenocephalides represent the “floor fleas.” These fleas are arthropods without wings (apterous).

Both males and females are hematophagous. Fleas feed on the blood of their host, which they suck, and the female can absorb up to 13 μ l of blood per day.

The flea life cycle lasts on average one month. Considering an average laying rate of 20 eggs per day and a hatching percentage of 50%, a single adult flea can give rise to 20,000 fleas in 2 months.

They take many very short blood meals from their host but are able to fast for several weeks. In the absence of their preferred host, the flea can feed on a host of a different species. In the presence of a blood-feeding host, the adult flea can survive for up to one year. Fasting reduces its longevity by 2 months.

After a blood meal, the females mate and lay their eggs on the host (an average of 30 eggs per day for 50 days). The blood meal is essential for the reproductive process, and egg laying does not begin until at least 48 hours after the blood meal.

a) Morphology:

The adult flea is a yellow or dark brown ectoparasitic insect with 3 pairs of legs adapted for jumping, equipped with claws. It measures between 1 and 8 mm long. Its body, covered with bristles, is flattened laterally.

The eggs, whitish and about 1 mm long, are not adherent to the hairs.

They are round or oval, with a smooth, pearly white shell. Their texture is sticky, but the egg easily falls off the animal.

Larvae: The newly hatched larva measures approximately 1.5 mm long (Figure 67).

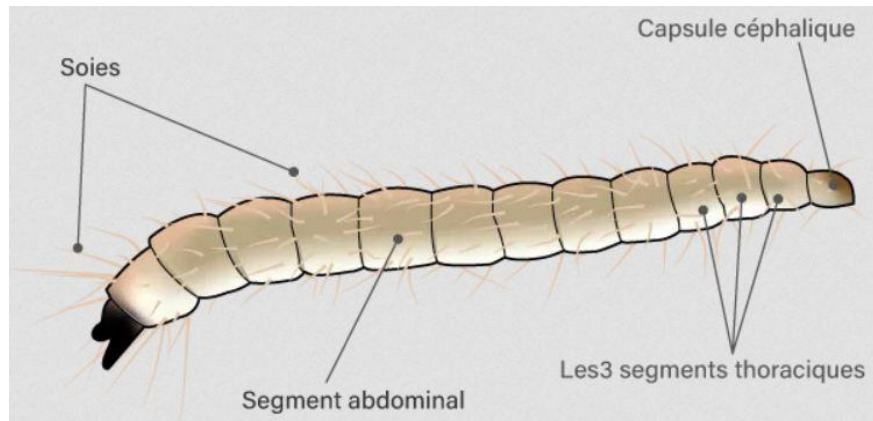


Figure 67. Morphology of a *Ctenocephalides* larva

b) Life cycle:

The biological cycle of the flea is defined by 4 stages: egg, larva, pupa and adult.

The flea takes its first (blood) meal within 20 minutes after reaching a host. Twenty-four to forty-eight hours later, the female flea lays eggs. A flea can lay 20 to 30 eggs per day until death. The egg hatches after 2 to 10 days.

A detritivorous larva emerges (feeds on remnants of the host's droppings if the larva is still on the animal, or on organic debris). It is lucifugous (dislikes light). Therefore, the larva is often hidden and usually covered with dust, fibers, sand grains or organic debris. After a period of 7 to 18 days the larva transforms. It spins a cocoon around itself using silk produced from its labial glands. The larva then reaches the pupal stage.

After about ten days the pupa darkens and becomes an adult. It can remain in its cocoon for up to 150 days, waiting for favorable conditions to emerge.

The newly emerged flea can live 1 to 6 weeks before finding a host. After taking its first blood meal, it cannot survive more than 2 days without the host (Figure 68).

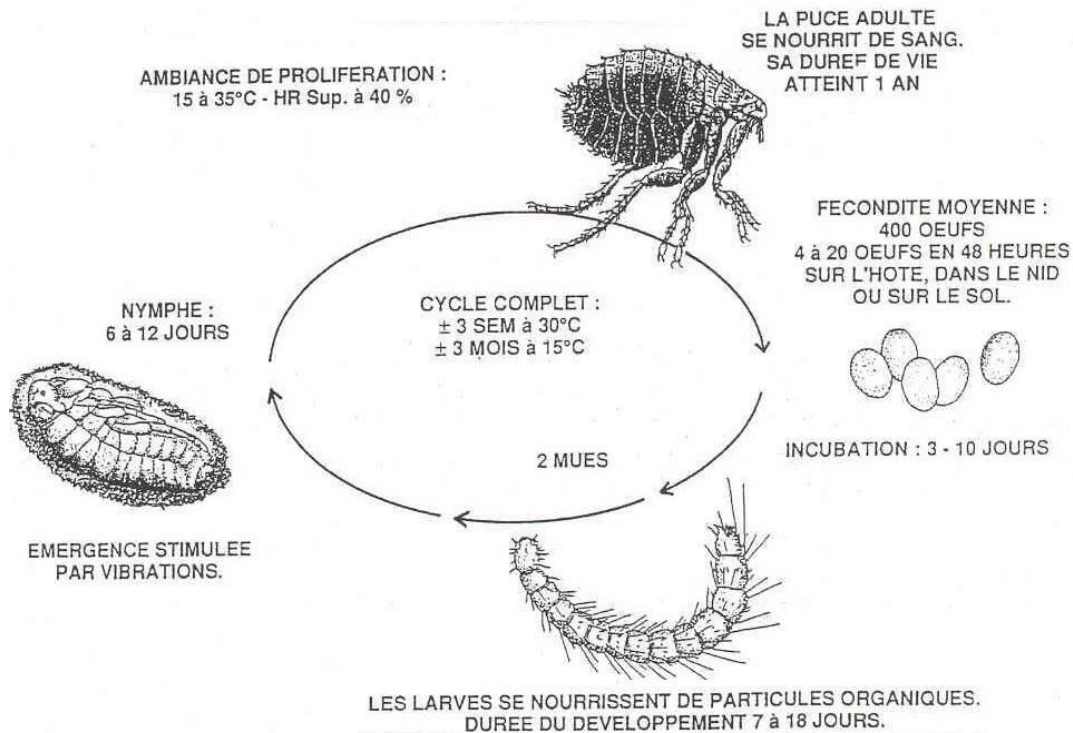


Figure 68. Biological cycle of a flea

The first larval stages occupy the same biotopes as the eggs. The larvae exhibit positive geotropism (they are attracted to the ground) and negative phototropism (they avoid light). They are also attracted to wood. Therefore, they are mainly found in hidden places (behind baseboards, between floorboard gaps, under staircases, under carpets and rugs, tile cracks, etc.).

Genus *Pulex*

Phylum Arthropoda, Class Insecta, Subclass Pterygota, Order Siphonaptera, Family Pulicidae.

The genus *Pulex* groups ectoparasitic insects of the order Siphonaptera, family Pulicidae.

Less common than the genus *Ctenocephalides*, *Pulex irritans* is commonly called the human flea. It is found on humans and on hunting dogs or semi-free roaming outdoor dogs. It may also be found on homeless individuals living in extreme poverty. *Pulex irritans* has a cosmopolitan distribution; its origin is American and it is thought to have spread to Africa.

Pulex irritans (Figure 69) has a rounded forehead, a well-developed eye, a genal comb reduced to a single spine (preocular spine), and a prothorax without a comb. A post-cephalic spine can nevertheless be observed. This allows easy differentiation from *Ctenocephalides* spp.

They live permanently on their host and only leave it exceptionally when disturbed or when the host's body temperature drops (anesthesia, death). These fleas, in general, have strong jumping ability.

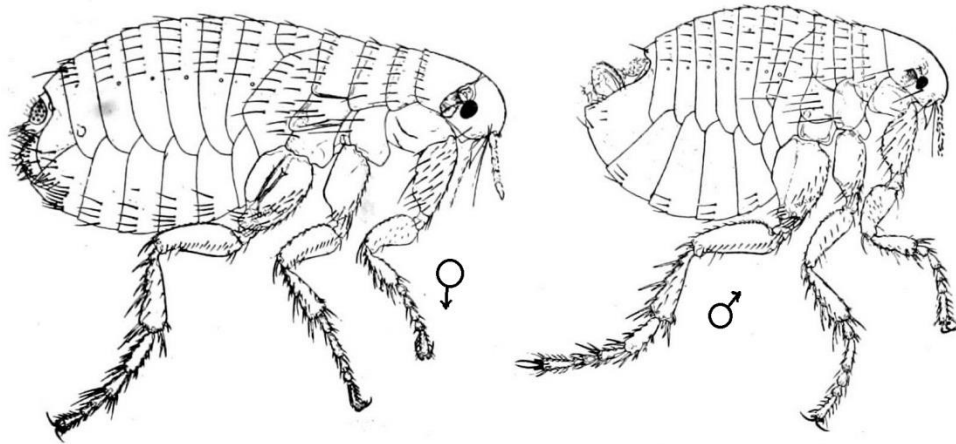


Figure 69. Morphology of *Pulex irritans*.

3.3.1.3 – Order Heteroptera

Genus *Cimex*

Phylum Arthropoda, Class Insecta, Subclass Pterygota, Order Hemiptera, Family Cimicidae.

Both sexes of *Cimex lectularius* are hematophagous. The feeding lasts between 10 and 20 minutes. These insects are gregarious; their resting, egg-laying, and mating sites are usually difficult to access: mattress seams, bed structures, cracks in wooden frames, picture frames, curtain rods, etc.

a) Morphology:

Adults: Adult *Cimex lectularius* measure between 5 and 8 mm in length and are reddish-brown in color, with an oval and flattened body. They are wingless (apterous) and possess microscopic hairs, giving them a striped appearance (Figure 70). Adult bed bugs do not move quickly enough to escape the sight of an attentive observer.

Eggs: Measuring 1 to 3 mm, whitish, the eggs are operculated and laid in small clusters of 5 to 15 units. A female lays 200 to 500 eggs over her life (Figure 70). They are laid 3 to 10 days after

mating, for temperatures between 14 and 27 °C. A blood meal is essential for their maturation (trophic cycle).

Nymphs: The young or immature stages (referred to as nymphs in English literature) are pale-colored when unfed and not easily visible. The nymphs are translucent and lighter in color. As they reach adulthood, they become progressively darker and more opaque. Their size ranges from 1.5 mm to 4.5 mm (Figure 70).

The bed bug avoids light, hiding in bedding and all dark areas of a home, and becomes active at night.



Figure 70. Morphology of the developmental stages of *Cimex lectularius*.

b) Biological cycle:

A bed bug can survive without feeding for up to 1.5 years, or even 2 years if conditions are favorable (temperature, shelter, etc.).

During its lifetime, a bed bug goes through several successive stages:

- **Eggs (1 mm).**

The young or immature stages (or nymphs according to Anglo-Saxon authors) develop through five instars before becoming adults (Figure 71). A blood meal is essential to reach the next stage, and each stage lasts from 3 to 15 days.

- **1st nymphal instar (1.5 mm)**
- **2nd nymphal instar (2 mm)**

- **3rd nymphal instar** (2.5 mm)
- **4th nymphal instar** (3 mm)
- **5th nymphal instar** (4.5 mm)

And finally:

- **adult stage.**

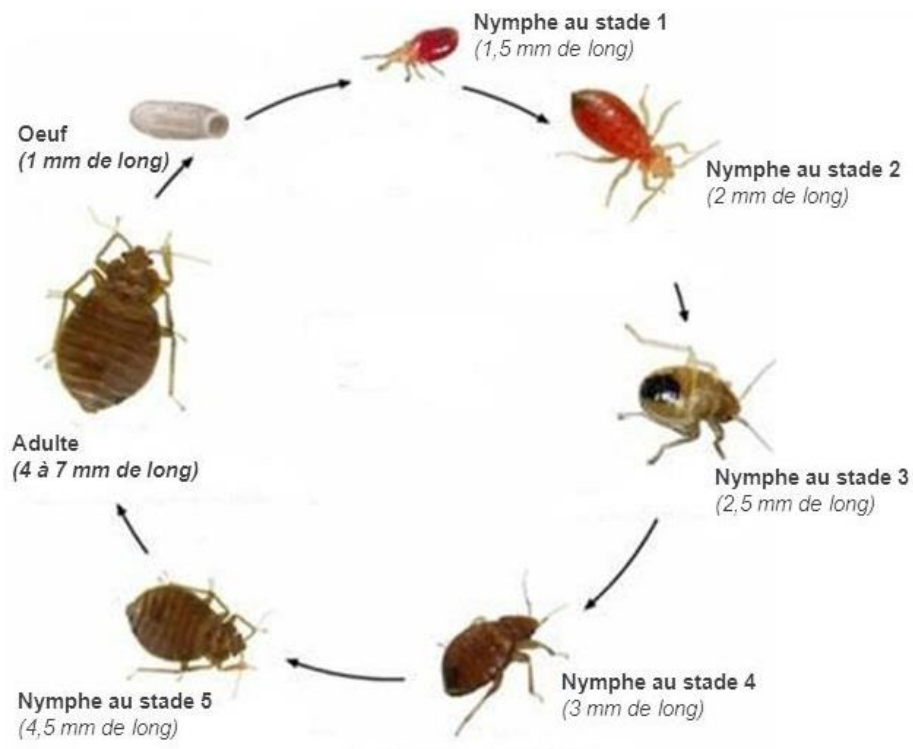


Figure 71. Biological cycle of *Cimex lectularius*.

Adults and juveniles are active at night and avoid daylight or artificial light (bedside lamp or flashlight), which makes their detection difficult.

After the blood meal, the bed bug returns to an identical or new resting place to digest, molt, or lay eggs. This new hiding place becomes a new contaminated site (pyjama hem, luggage, clothes at the foot of the bed, picture frame, dresser, etc.).

Able to move or be transported by humans, they preferentially infest places with high human density.

Genus *Triatoma*

Phylum Arthropoda, Class Insecta, Subclass Pterygota, Order Hemiptera, Family Reduviidae, Subfamily Triatominae.

Triatoma is a genus of heteropteran insects (bugs) of the subfamily Triatominae. Members of this genus (like all members of the Triatominae subfamily) are hematophagous and can transmit serious diseases, such as Chagas disease.

Triatominae (Hemiptera, Reduviidae) are bugs that attack a wide range of vertebrate hosts, both during the larval stages and in the imaginal stage. These insects may contain in their digestive tract a hemoflagellate parasite, *Trypanosoma cruzi*, the causative agent of American Trypanosomiasis or Chagas disease. This parasitosis is endemic from the southern United States to southern Argentina.

Vector morphology:

Adults: Belonging to the class of Insects, triatominae are composed of an exoskeleton, a body divided into three segments (head, thorax, and abdomen), a pair of segmented antennae, two eyes, three pairs of legs, and two pairs of wings (Figure 72). Triatominae also have piercing–sucking mouthparts.

The rostrum or proboscis is relatively straight, slender, and composed of 3 segments (in predators these 3 segments are curved, while in phytophagous insects only 4 segments are found).

The head is almost twice as long as it is wide (or at least as long).

The thorax is divided into 3 parts: the prothorax, mesothorax, and metathorax.

Two salivary glands are found on each side of the anterior part of the digestive tract. They secrete various anticoagulant, antihistamine, and antiserotonin substances to facilitate the digestion of blood, the circulation of blood through the digestive tract, to guide the maxillae into the host's blood vessels, and to make the bite painless.

Adult triatominae differ from nymphs (stage preceding the adult form) by the presence of wings, ocelli, and well-developed genital organs. In general, size, colour, body shape and particularly the shape of the head are sufficient to identify the different species.

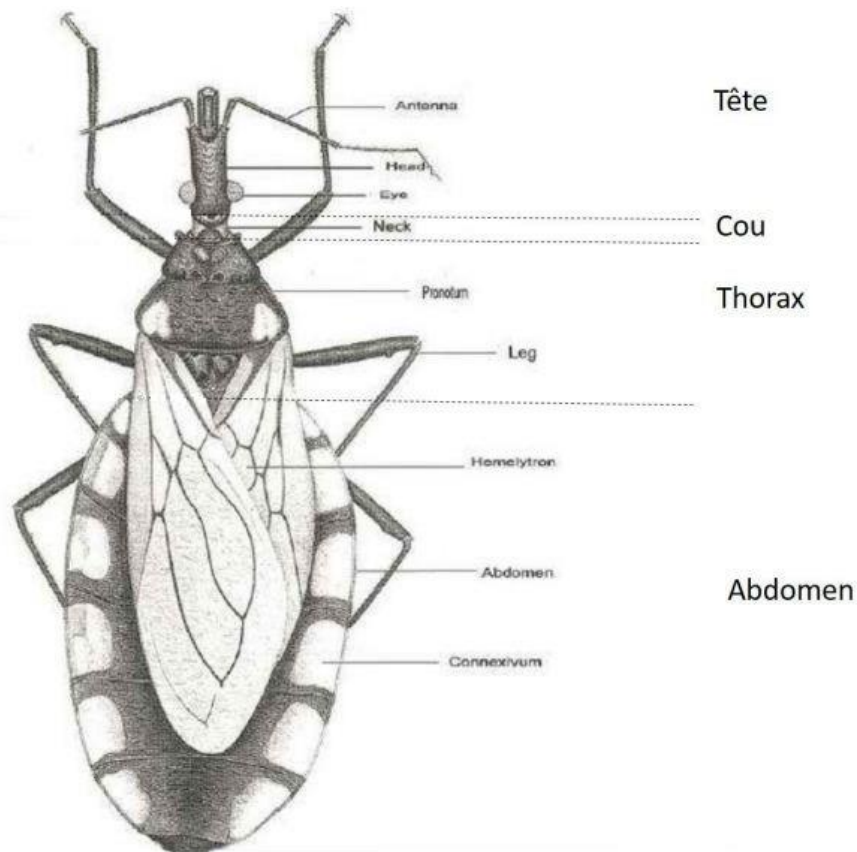


Figure 72. General external morphology of a triatomine.

Biological cycle of the vector:

The biological cycle of Triatominae is divided into three parts: the egg, 5 nymphal stages (stages named I to V) and the adult.

Copulation between a male and a female takes place with the male in a dorso-lateral position on the female and lasts between 5 and 15 minutes.

Eggs are laid between 10 and 30 days after mating and hatching occurs 10 to 40 days after oviposition. They are white and darken as the embryo develops. The female lays on average 600 eggs during her lifetime, but this number varies depending on the species, available food, temperature and humidity of the external environment.

Nymphs have a rather similar morphology between them. Hematophagy is present at all stages. The transition between the different nymphal stages up to the adult requires a complete blood

meal. Feeding lasts several minutes (about 20 minutes for an adult or a stage V larva).

The feeding frequency is relatively high, between 4 and 9 days, but the insect can fast for months if conditions are unfavourable.

The duration of the cycle varies from a few months (*Rhodnius prolixus*) to 1 or 2 years (*Triatoma dimidiata*, *Panstrongylus megistus*) and the lifespan of an adult can exceed 2 years. Similar to the variation in the number of eggs laid, the duration of the cycle depends on several factors (species, humidity, temperature, etc.).

The speed of the cycle also depends on the ability of the triatomine to take a complete blood meal without interruption. The chances of success are increased because the triatomine most often bites humans or animals while they are sleeping and because its bite is painless.

Human dwellings offer domestic species several advantages, including stable shelter and a nearby source of blood.

The triatomine has two peaks of activity:

- at dusk for food searching, and
- at dawn to look for a resting place.

It remains motionless during the day (ataxic phase) in its hiding places such as wall cracks, between frames and the wall, in blankets, or in palm-leaf roofs of precarious houses.

At night, it leaves its hiding place to feed and orients itself via its chemoreceptors attracted by carbon dioxide and components released by human breath.

Sylvatic and peri-domestic species live in various habitats such as chicken coops, small mammal burrows, bird nests or tree hollows.

Transmission of the disease:

Chagas disease is caused by the flagellated protozoan *Trypanosoma cruzi*. Its reservoir includes not only humans, but also many wild and domestic mammals (cats, dogs, guinea pigs, rodents,

armadillos, marsupials, etc.).

Acquisition is mainly vector-borne but can also be oral (food can be contaminated by bug feces) or vertical (risk of mother-to-foetus transmission). Transmission through blood transfusion or organ transplantation has also been reported.

Transmission of *Trypanosoma cruzi* does not occur through the bite but through the feces, which may contain the infective form of the parasite if the insect has fed.

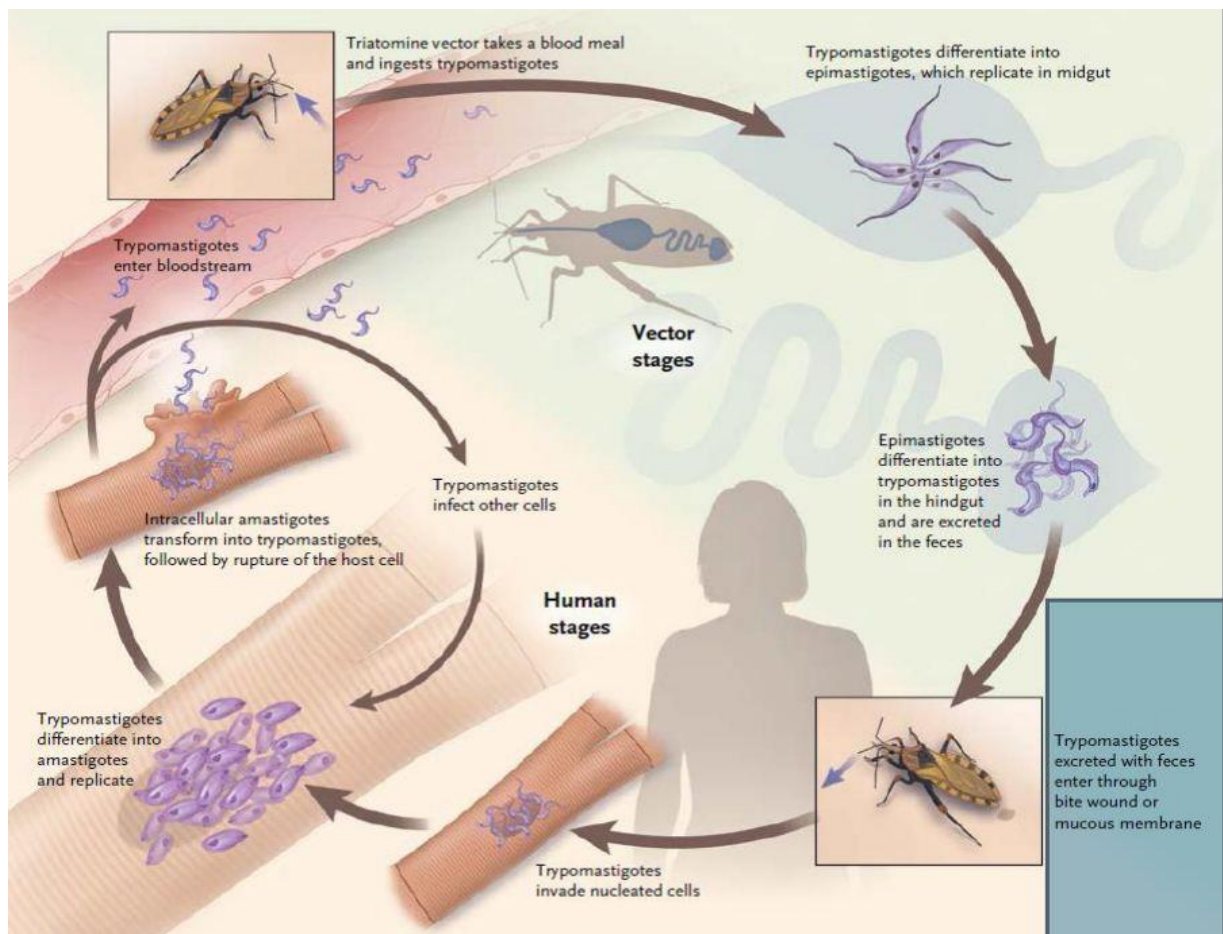


Figure 73. Life cycle of *Trypanosoma cruzi*.

15 to 20 days earlier on a vertebrate host carrying the disease. The trypanosome can enter the host through skin lesions, mucous membranes, or contamination of the bite site with nearby feces during scratching of the wound. The insect operates at night when its host is asleep. The blood meal lasts about twenty minutes.

However, once contracted, the parasite remains for life in the reduviid bug, and it is even capable of surviving in a dead triatomine; for example, in *Triatoma infestans*, 8 days at 26°C and up to 60 days at 5°C.

This disease is incurable; there is no effective chemotherapy, vaccine, or chemoprophylaxis. The only way to limit the risk of human infection at present is to reduce vector populations by chemical treatment of dwellings. It is certain that low socioeconomic level, type of housing, and some customs - such as in Mexico the ingestion of triatomines for their supposed aphrodisiac power, or treatment of warts with triatomine feces - are predisposing factors to the disease.

3.3.1.4 – Order Diptera

Two groups of dipterans are distinguished: the nematocera (mosquitoes) and the brachycerans (flies). All feed on liquid substances: their mouthparts are of the piercing type (notably mosquitoes) or sucking type (flies).

Like many blood-feeding animals, certain dipterans can transmit diseases. This is the case for anophelines (*Anopheles*), which transmit malaria, and stegomyias or aedes (*Aedes*), vectors of yellow fever. Simuliids (*Simulium*) which, in Africa, transmit onchocerciasis or river blindness.

Myiasis derive from the Greek words “myia” meaning fly and “iasis” meaning disease. They are therefore dermatological conditions due to the subcutaneous penetration of fly larvae. These parasites cause boils or sebaceous cysts.

Myiasis are dermatoses whose pathogenic agents are parasitic flies belonging to different families (Calliphoridae, Sarcophagidae and Muscidae).

Myiasis-producing species cause myiasis, that is to say disorders due to the presence of parasitic larvae in a living body, human or animal.

The causes of myiasis are attributed to ectoparasitic larvae laid directly by certain flies on the skin, on a secondary vector, on a substrate (soil, fabric) or even accidentally ingested. These larvae are elongated, conical, whitish, and resemble worms.

Several varieties of larvae exist, but the best known are the “Cayor worm,” very widespread in Africa, and that of America called the “macaque worm.”

The “macaque worm” originates from eggs laid by the fly *Dermatobia hominis* on the abdomen of mosquitoes, which then deposit them on the host’s skin.

The “Cayor worm” is laid by the fly *Condylobia anthropophaga* on a substrate (soil, clothing). Upon skin contact, the larva penetrates the host’s skin painlessly. A pruritic, sometimes painful papule then appears at the penetration site.

Myiases manifest as boils or cysts caused by penetration of the larva under the skin. These cysts are reddish papules with a central opening allowing the larva to breathe.

The treatment of myiases consists of spontaneous extraction of the larva by asphyxiation with petroleum jelly or bialfine or even with a piece of lard (pork fat) or by surgical extraction at any stage of the infection.

Family Calliphoridae

The family Calliphoridae, commonly known as “green and blue flies,” contains species whose larvae live either in decomposing meat (such as the blue meat fly) or as parasites under the skin of various mammals. The most important species is the screwworm fly (*Callitroga hominivorax*), whose larvae live under the skin of cattle (Figure 74).

Although the majority of Calliphoridae are coprophagous and necrophagous, some species have medical or veterinary importance because they generate myiases.

Calliphoridae are robust-bodied flies measuring 4 to 16 mm with metallic colors: black-blue, violet-blue, blue-green, or green for most species.

Most Calliphoridae are necrophagous, coprophagous or detritivorous, sometimes predatory or parasitoids of snails or earthworms.



Figure 74. Adult screwworm fly, *Cochliomyia hominivorax*.

Calliphoridae are usually the very first insects to reach a corpse, where the development of their larval stages takes place, preceding the larvae of Sarcophagidae, Muscidae or those of other necrophagous families.

Females are anautogenous, meaning that they need a protein supply to bring their eggs to maturity, which they obtain from consuming carcasses. The eggs are white or yellow, 0.6 to 1.5 mm long, and resemble grains of rice. A female generally lays 150 to 200 eggs per batch and around 2,000 throughout her life.

Most species are oviparous, but a few are viviparous.

One species of this family, *Lucilia sericata*, has been traditionally - and in recent years also clinically - used in medicine to treat wounds through its maggots (maggot therapy). However, it should not be confused with other members of the same family that can consume living flesh (e.g., *Lucilia bufonivora*).

The “Cayor worm” or *Cordylobia anthropophaga* is an obligatory parasite of the skin, forming boils that cause “furuncular myiasis” (Figure 75). It mainly parasitizes dogs and humans, but also cats, rats and monkeys. It occurs only in sub-Saharan Africa.

In the adult stage, it is a fly 6 to 12 mm long, brown-yellow in color, with blackish bands on the abdomen. The eyes are close together in the male and separated in the female.

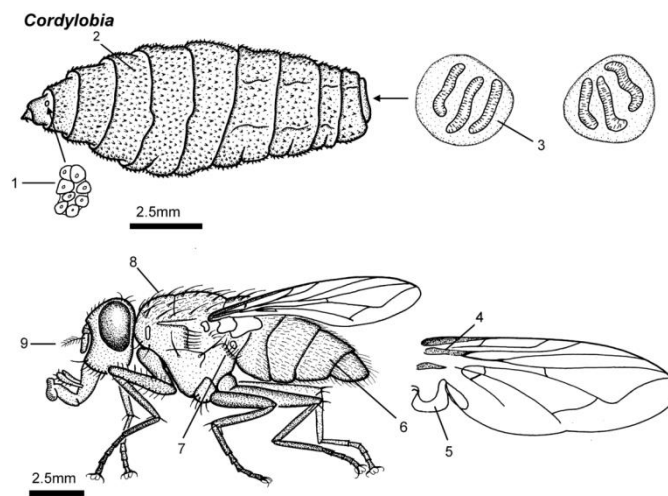


Figure 75. *Cordylobia* — larva and adult (lateral view).

Adult females are active in the early morning and late afternoon. They remain for the rest of the day and night in dark places under house roofs. Adults feed on the sugary juices of fruits, excrement, and decomposition fluids from animal carcasses.

The female lives 15 days to three weeks. She lays about 150 to 300 eggs, 0.8 mm long, on a dry and shaded surface previously soiled with urine or feces, such as sand, baby diapers, or clothes left to dry. She never lays her eggs directly on the skin, nor on hair or fur.

Hatching occurs after three days. The newly hatched larva, about 1 mm long, moves in search of a host, which may take up to ten days. Once the larva reaches the skin, it burrows into it. It settles in the dermis, leaving a small external opening for respiration. Penetration sites are mainly the feet, fingers, genital organs and, in animals sleeping on the ground, the body parts in contact with the soil. Humans usually feel mild itching during the first 48 hours. The papule becomes red and the pruritus generally disappears. Molting occurs two to three days after penetration. The second-stage larva, 2.5 to 4 mm long, is equipped with backward-pointing spines that prevent any exit through the entry orifice.

The larva reaches the third stage 5 to 6 days after invasion. The host develops a firm boil, with a small dry crust at the apex (composed of yellowish serous fluids produced by the organism and

the larval waste). At this stage, the victim suffers from intense pruritus. The larva may die, and its cuticle can trigger an abscess. When boils are very close together on the limbs, edema may occur, sometimes followed by gangrene.

Moderate pressure at the base of the boil is enough to expel the larva, which can reach 15 mm in length. Very active and cylindrical in shape, it rapidly crawls across the ground toward the darkest places. Pupation occurs within 24 to 48 hours, and the emergence of the adult takes place 8 to 15 days later.

Cochliomyia hominivorax is an obligatory parasitic “flesh-eating fly” of mammals, and more rarely of birds. Females do not lay eggs on carcasses, but on living individuals, at the level of wounds or natural orifices.

Family Sarcophagidae

Larval development of the Sarcophagidae, and especially of the subfamily Sarcophaginae, occurs on numerous nutritive substrates. Most species are saprophagous, contributing to the decomposition of organic matter. In general, the larvae are polyphagous, developing on decomposing animal and plant substances, on animal and human excrement, and on the carcasses of various vertebrate and invertebrate animals.

The fetid odors of fermentation or putrefaction attract the females and stimulate oviposition or larviposition. Thus, certain species, being attracted to the shelters of sick animals or to the nests of birds and small mammals dead in various states of decomposition, deposit their eggs on them and their larvae become zoophilous.

The larvae of the subfamily Sarcophaginae are very similar to those of the family Calliphoridae, but they present well-defined differentiating characteristics. In the third instar, the larva is acephalous, apodous, and amphipneustic (Figure 76).

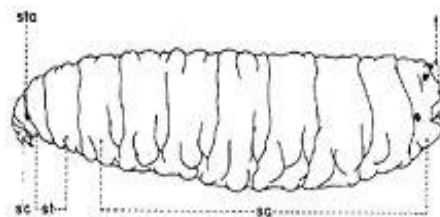


Figure 76. Third-instar larva of Sarcophaginae.

Sarcophagids are larviparous or ovoviviparous, meaning that they deposit larvae or eggs from which larvae hatch immediately.

Family Muscidae

This family includes flies ranging from small (2 mm) to large size (18 mm).

The larvae (maggots) are thinner anteriorly and rounded posteriorly, with fused mouth hooks. Adults are generally dull in coloration, but may vary from orange-yellow, grey, or brown, and very rarely exhibit a metallic sheen.

Selected model species, *Stomoxys calcitrans*, commonly called the stable fly, biting fly, dog fly or “charbon fly”, is so named because it can mechanically transmit anthrax (a highly feared infectious disease among livestock breeders), as well as pernicious anemias.

It is a common fly in rural areas — in stables, sheepfolds or barns — where it finds abundant manure and dung.

The female lays her eggs in these substrates and the larvae (maggots), which are coprophagous, develop over 10 to 80 days, depending on temperature.

Morphology :

- *Stomoxys* resembles the common housefly, but it bites and feeds on blood in the adult stage (its bite is painful to both humans and animals) (Figure 77).

- 6 - 8 mm long.
- Bears a black, shiny proboscis, slightly longer than the head, very pointed, and permanently directed forward.

- Thorax with four dorsal black stripes.
- Checkerboard pattern of brown and grey spots on the dorsal surface of the abdomen.
- Clear wings, longer than the abdomen and not crossed at rest.
- Unlike most other haematophagous insects, both sexes bite.

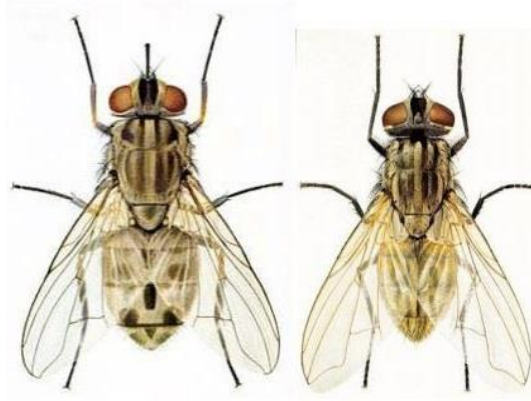


Figure 77. *Stomoxys calcitrans* and *Musca domestica*.

Parasites (*Trypanosoma* spp., *Besnoitia* spp.) are also transmitted by stable flies (*Stomoxys*). Stable flies are intermediate hosts of the parasitic nematode *Habronema microstoma*.

3.3.2 – Subphylum Cheliceriformes

Order Acari

Mites are chelicerates belonging to the class Arachnida. In higher species, the cephalothorax and abdomen are fused, giving them an ovoid body shape, whereas primitive species may appear segmented and articulated.

The ecological niches of terrestrial mites are extremely diverse. Some are detritivores and are common in household dust, soil, bedding, flour or cheese crusts (*Dermatophagoides* sp.), and they play an important role in the development of certain allergies. Others are predators, mainly of small invertebrates.

The vast majority are parasites of plants or animals.

This is the case, for example, of ixodid ticks, which are primarily haematophagous and are formidable vectors of viral (babesiosis), rickettsial (Rocky Mountain spotted fever), bacterial (Lyme disease) or parasitic diseases, as well as *Sarcoptes*, responsible for scabies.

The classification of mites is based on the number and arrangement of the respiratory openings or stigmata.

Although a few forms of mites are free-living, the vast majority are parasitic, either throughout their entire life or during specific ecological phases.

Groups	Characteristics
Ixodoids	Temporary parasites of vertebrates, on which they feed by sucking blood. <i>Ixodes, Argas, Ornithodoros, etc.</i>
Sarcoptoids	They are the causative agents of scabies in humans and various other mammals (<i>Sarcoptes, Psoroptes</i>). Others parasitize birds (<i>Cytodites</i>). Some are strictly detritivorous (<i>Tyroglyphus, Glycophagus</i>).

3.3.2.1 – Suborder Metastigmata: Ixodidae (The Hematophagous Ticks)

Genus *Ixodes*

The Metastigmata possess a pair of spiracles located behind the 4th pair of legs.

The name “*Ixodes*” comes from the Greek *ixodès*, meaning “sticky”; glue was a natural adhesive extracted from mistletoe berries (called *ixos* by the Greeks).

Ixodes is a genus of ticks belonging to the family Ixodidae. Some species of the genus *Ixodes* attack humans. In particular, *Ixodes ricinus* (Figure 78) is among the ticks that most frequently transmit certain diseases (parasitoses) to humans in Europe, including Lyme disease, tick-borne meningoencephalitis (or vernal-summer meningoencephalitis), and ovine viral encephalitis (louping ill) in sheep.

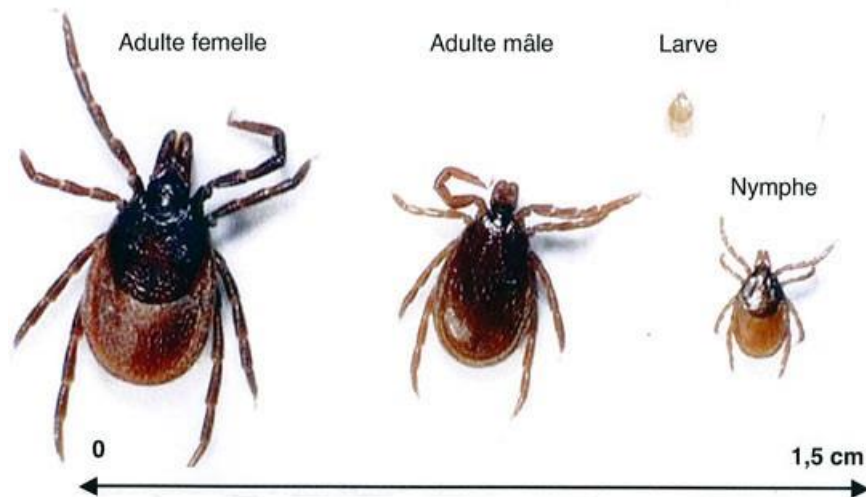


Figure 78. *Ixodes ricinus* (adult, nymph, larva).

These parasitic mites are eyeless, but they possess cells acting similarly to olfactory receptors (located on one leg) and photosensitive cells on both sides of the body. In this way, the animal

knows whether it is day or night and can detect the movement of animals if they interrupt the incoming light.

All *Ixodes* live on or near the ground (often within the first 50 to 60 centimeters and in the litter when they are not waiting for a host, positioned motionless on grasses, and sometimes on low tree branches or bushes). Their environments are most often forests and woodlands, but they are also found in hedges and wooded areas, or anywhere they may have been transported by animals such as deer or wild boar. They are more numerous in valleys and rarer or absent at higher altitudes.

Ixodes, like all ticks, develop through several successive stages. They must feed on blood. Individuals of each stage therefore search for a host to parasitize. Host-seeking occurs during the warm season, from May to September, with variations depending on latitude and altitude. *Ixodes* appear to be very sensitive to climate, particularly to mild winter temperatures and nighttime temperatures during the warm season.

The movements of *Ixodes ricinus* nymphs are mainly nocturnal and are strongly influenced by thermohygro-metric conditions.

Lyme disease appears to be emerging and increasing sharply, which seems to be explained by a proliferation of ticks in many wooded or forested regions of the Northern Hemisphere. This proliferation may be facilitated by global warming and by certain environmental changes.

Life cycle:

The developmental cycle of the tick *Ixodes ricinus* consists of three stages and ranges from 2 to 6 years in duration.

The stages are as follows: from the egg emerges a larva, which transforms into a nymph (2 mm) and then into an adult (3–4 mm). A blood meal on a host is required for the laying of eggs by the adult female tick and at every developmental stage.

The tick *Ixodes ricinus* becomes infected by feeding on the blood of hosts contaminated by *Borrelia burgdorferi sensu lato* bacteria. The main reservoir hosts of *Borrelia burgdorferi sensu lato* are small wild mammals (voles, mice, squirrels, etc.) (Figure 79).

Some species of birds or reptiles are also reservoirs. Large mammals such as cervids are hosts for adult ticks but are incompetent reservoirs of *Borrelia burgdorferi*, meaning they are unable to ensure transmission of the bacterium to an uninfected tick.

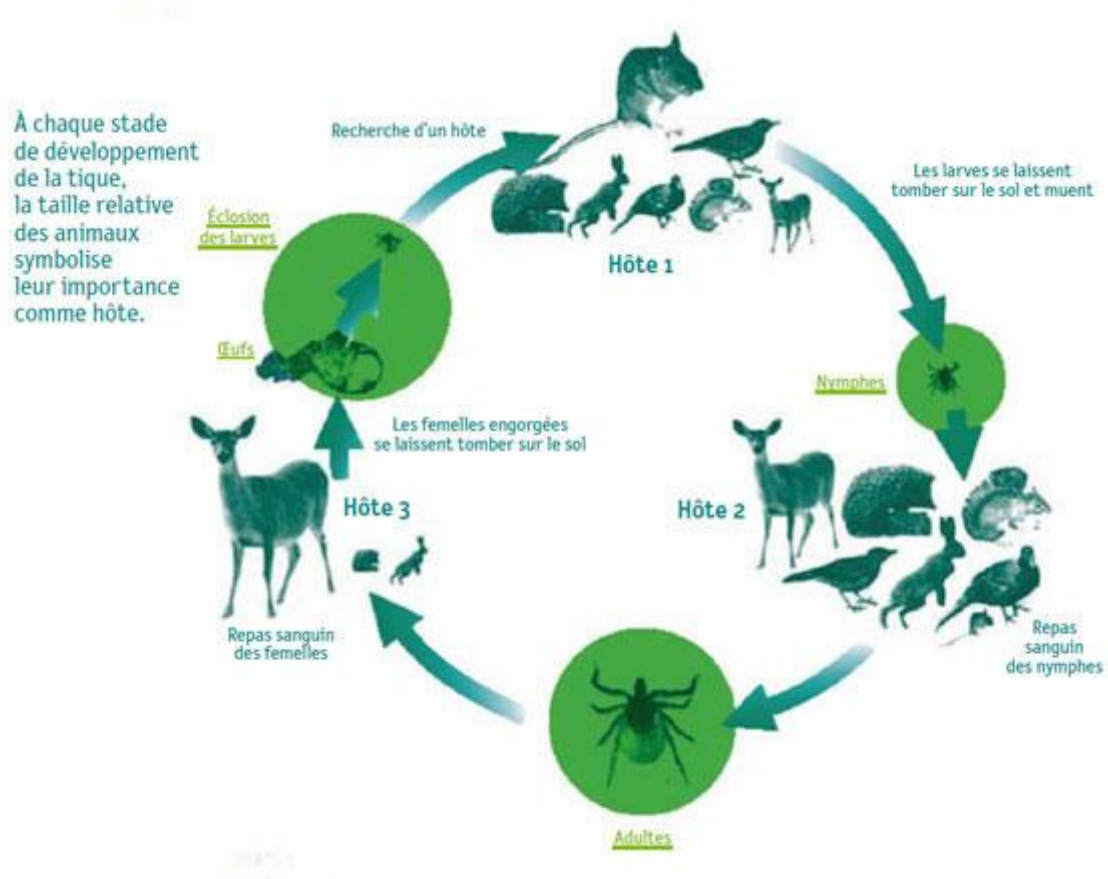


Figure 79. Life cycle of development of *Ixodes ricinus* and its different hosts.

The blood meal lasts from 3 to 7 days depending on the stage. At the end of the blood meal, the tick detaches from its host and drops into nearby vegetation close to the ground. It requires a minimum humidity level to survive (80%) and can wait several months before molting to the next stage of the cycle or laying its eggs if it is an adult female.

3.3.2.2 – Suborder Astigmata (Lymphophagous mites)

Genus *Sarcoptes*

Astigmatid mites do not possess spiracles. Respiration occurs through the tegument.

Scabies is a cosmopolitan cutaneous parasitosis caused by the colonization of the stratum corneum of the epidermis by the mite *Sarcoptes scabiei* (Figure 80). It affects individuals of both sexes, of all ages, from all social backgrounds, and on all continents.

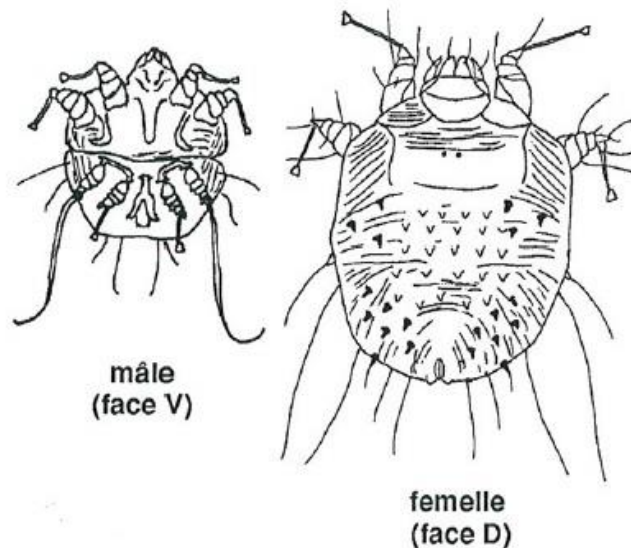


Figure 80. Morphology of *Sarcoptes scabiei*.

Transmission is mainly ensured by fertilized adult females and only very rarely by larval forms. It is primarily human-to-human, through direct “skin-to-skin” contact. It requires close and prolonged contact, particularly during sexual intercourse, which is why scabies is also considered a sexually transmitted infection (STI).

Contamination can also occur indirectly, through the environment, mainly via clothing and bedding. The survival of the mite outside the host varies according to ambient temperature and humidity: low temperature and high humidity favor survival, whereas high temperature and low humidity lead rapidly to its death (Figure 81).

In general, the survival of the mite in the environment outside its host is brief—around two days, or up to four days if conditions are favorable (temperature, humidity). Survival is longer for larvae or eggs (up to ten days), but their involvement in the spread of scabies is only possible when they are present in very large numbers.

Factors favoring transmission include frequent close physical contact (children), community living, and social precariousness.



Figure 81. Parasitic cycle of *Sarcoptes scabiei*

The human scabies mite cannot develop on companion animals such as dogs or cats. However, they may be considered occasional vectors, just like bedding or clothing.

Sarcoptes mate on the host. After fertilization, the male dies and the female digs a burrow in the stratum corneum of the epidermis. She progresses 1 to 2 mm per day and lays about three to five eggs per day for one to two months before dying. The eggs hatch after three to four days, each giving rise to a larva that emerges from the burrow.

The larvae, after several molts, develop into nymphs and then into adult mites. The maturation process to reach the adult stage takes about fifteen days.

In common scabies, there are generally between 5 and 15 female mites lodged simultaneously in the burrows. This number can be much higher (hundreds or even thousands or millions) in the case of crusted scabies.

Pruritus is attributed to immunological reactions triggered by the parasite's excretions (saliva, feces) and eggs.



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