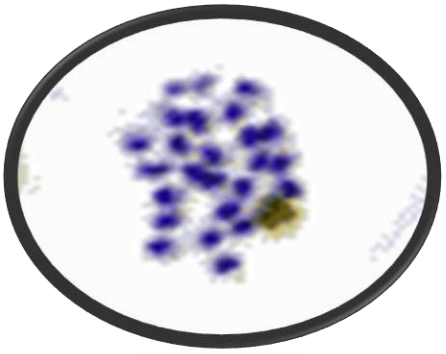
Larbi Ben M 'hidiUniversity, Oum El Bouaghi
Faculty of Exact Sciences and Natural and Life Sciences Department of Natural and Life Sciences

PROTOZOAN AND METAZOAN PARASITE COURSES



Dr. TOLBA Mounia

MCA , Speciality : Parasitology

(Intended for 3rd year students LMD Bachelor: Parasitology)

Academic year

2023-2024

CONTENTS

Course Pedagogical Plan

Title of Subject: Protozoa and Parasitic Metazoa Sector: Parasitology

Level: 3rd year Bachelor's

degree Teaching unit: Fundamental

Hourly volume: 90h00 Lessons + Practical work Material

coefficient: 3

Number of credits

General objective of the course

This material targets the knowledge of parasitic species that infest humans and animals through the study of their morpho-anatomy, their development, their life cycle and their classification.

INTRODUCTION

Parasitology is the study of parasites, their hosts, and the relationship between them. As a biological discipline, the purpose of parasitology is not determined by the organism or environment in question, but by their lifestyle. This implies that it is a synthesis of other disciplines, and develops techniques from fields such as cell biology, bioinformatics, molecular biology, immunology, genetics and ecology.

Parasitology is the science that aims to study parasites from the point of zoological, morphological, biological and nosological, diagnostic methods and means of struggle.

Human and animal diseases determined by parasites are numerous and deadly (malaria, sleeping sickness, bilharzia (human).and nosemosis, silkworm pebrine, bee ameboses.)

History

The evolution of parasitology is related to the evolution of microscopic technology. Since the discovery of the optical microscope by Antoni Van Leeuwenhoek (1632-1723). Before we know the disease but we do not know the animal responsible for this disease, especially when it is a protozoan. There are:

*-1500 years Egypt: bilharzia, Wired of Medina reported in the Bible.

*- In the 14th century: helminths are known

*- in the 17th century: Quinquina recognized as anti-malarial in Europe

*+1670: Leeuwenhoek invents the microscope

*+1700: concept of evolutionary cycles

*1835 Félix Dujardin discovers rhizopods

*1880 Evans discovers the first pathogenic Trypanosome

*in the 19th century: morphology and cycles of the main parasites

*in the 20th century: everything was discovered?

*1980s: first trials of anti-parasitic vaccination

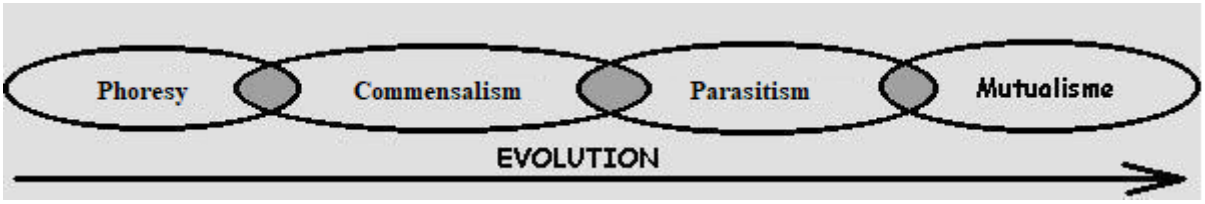
*1990s: Diagnosis by PCR.

CHAPTER I. GENERAL INFORMATION ON PARASITISM

1.1. Animal associations: definitions

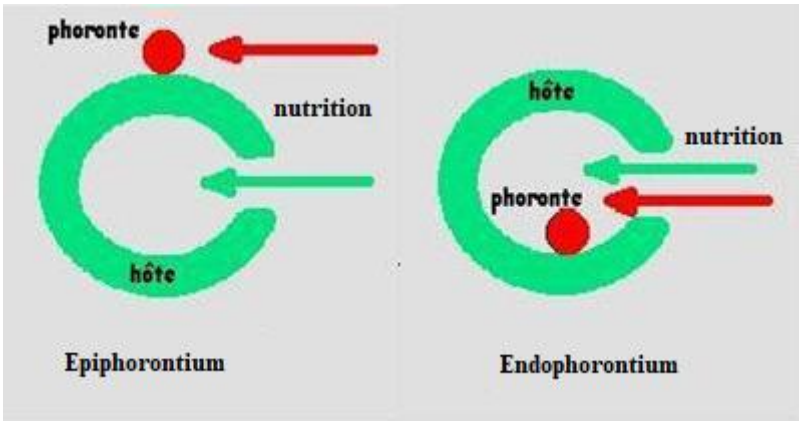
Parasites behave differently towards their host: many of them are obligate parasites, a number seem to inconvenience their host in no way and are mere phoresics, commensals**.**

An organism that uses another animal or plant as a host is often mistakenly called a parasite. It is necessary to look closely at the type of relationship that the organism has with its host to define whether it is parasitism, or phoresis, commensalism or mutualism. There are no hard boundaries between these different relationships; in nature, many cases are representative of two types of relationships. The four types of associations are linked by evolving situations:



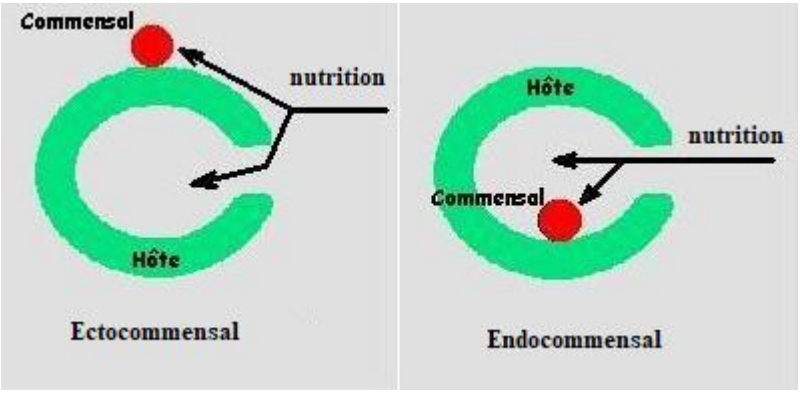
1.1.1. Phoresis

In this case, the host is only a medium or a means of transport. The organism hosted or transported is called: a "phoront". (Phoros = wear).The association is free, everyone has their own food and can live without the other. Or it is the transport of an individual by another. However, this association often resembles parasitism insofar as the carrier animal (phoront) often takes advantage of the food found on its host



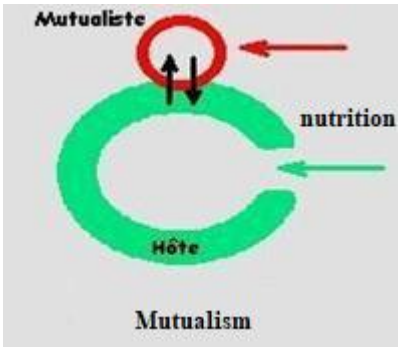
1.1.2. commensalism

This relationship concerns organisms that eat "at the same table". (Co = together) and (mensa = table). The host provides his commensal with some of his food. Only the commensal needs its host .This is the case of an animal (commensal) that lives on another animal (host) without harming it. It is a non-mandatory but beneficial relationship.



1.1.3. Mutualism

The mutualist organism (s) and the host are physiologically independent. The mutualist gets his food from the host and the host uses the mutualist to optimize the functioning of his body. It is a kind of beneficial cooperation for both partners. We also distinguish between endomutualism and ectomutualism (symbiotism)

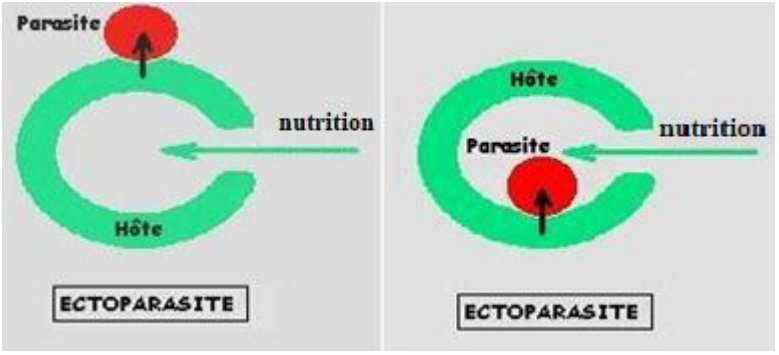


Ex: Flagellates - Termites

1.1.4. Free-riding

This is the relationship of any organism that lives at the expense of its host. (Para = from) and (sit = food). It feeds on its host. The parasite needs the host to live: habitat and resources. Usually it's a food relationship. Usually the parasite is smaller than its host. It's a negative relationship. The one who benefits is called a parasite and the one who hosts the parasite is called a host.

The parasite lives part or all of its life on its host. The most suitable parasites are those that cause a very slight disturbance in their host (s). The parasite does not have to kill its host for food



1.1.5. Predation

It is a negative relationship that exists between two members of different species, the one who benefits from this relationship is called a predator and the one who is affected by this relationship is called a prey. Generally the predator is larger than its prey. The predator is forced to kill its prey for food. The predator only hunts when hungry. (e.g. lion hare).

1.2. Reports of parasites with their hosts

1.2.1 Notions of parasitic specificity

Depending on the specificity of the parasite, it may have exclusively as host man or an animal or both at the same time (zooanthropozoonosis). Parasites have dietary requirements and consequently more or less strict locations restricting their possibilities of installation in hosts, hence a specificity (preference) that can have degrees. We distinguish several types of parasitic specificity:

* **Oligoxene parasite** : it is a parasite that is found in hosts belonging to species, genera and sometimes neighboring families (ex *Eimeria truncata* of the goose and duck).
* **Stenoxene parasite** : it is a parasite closely adapted to a single host (monoxene) (ex plasmodium)
* **Euryxene parasite** : This is a parasite that lives on several host species**(**heteroxene) most often belonging to a single class (ex *Trypanosoma brucei* which is observed in most domestic mammals)

Topographic reports of parasites with their hosts

In the host, the parasite will not settle anywhere. We are witnessing physiological and morphological adaptations of the parasite according to its location in the host. We distinguish between:

* **Ectoparasites** : are parasites that live on the external parts of their hosts (e.g. hair lice, mountain locusts, etc.). Fighting pests is easy.
* **Mesoparasites:** are parasites that live in the internal organs of their hosts but open to the outside environment (parasites that live in the intestines,

incontinence,

* **Endoparasites** : are parasites that live in the internal organs of their hosts but are not open to the outside environment. They are never found in nature (parasites that live in red blood cells). The fight against these pests is very difficult.

1.2.2. Parasitic adaptations

In general, parasitism profoundly changes the organization of animals that live at the expense of others, and parasites are generally more or less degraded (difficulty in determining the species).The most suitable parasites are those that cause little or no disturbance to their hosts.

*** Physiological adaptations**

The parasite changes physiologically to better adapt to its host, this is the case of certain parasites that encyst themselves, develop their reproductive system (*Fasciola, Taenia*), get rid of certain respiratory and circulatory systems (*Taenia*).

* **Anatomo-morphological adaptations**

There are parasites that enter their hosts get rid of their flagella (*Giardia, Trypanosoma*), their tail (Schistosoma), develop their attachment suction cups, their claws (lice, fleas)

* **Immunological adaptations (immune avoidance)**

Some parasites avoid the immune response of their host, either by changing their surface antigens (*Trypanosoma*) which can change its surface antigens several times), *Shistosoma* which as soon as it enters its host, borrow molecules from its host and surrounds itself with them and in this case it is no longer recognized as a foreign body. Other parasites secrete toxins that thwart the host's immune response (case of Tenias), other parasites use the host's macrophages as a refuge (case of *Leishmania*)

* **Ethological adaptations**

Some parasites are found in favorable environments so that they can reach their hosts, such as parasites found in water (e.g. *S. haematobium*).

1.2.2. Actions of parasites on their hosts

The action of the parasite on its host is variable, ranging from harmless (in the case of herbivorous ciliates) to those causing serious conditions (malaria, amoebiasis, etc.). We distinguish several types of action among them:

* **Spoliatrix** : Parasites feed on the host's nutrients. Ex. Destruction of a large amount of erythrocyte by *Plasmodium*.
* **Mechanical** : (rare) Embolisms of the capillaries of the viscera by *Plasmodium*, obstruction of the ductusbiliaria by *Fasciola*
* **Toxic** : By the secretion of toxins that react at low doses and can cause more or less serious disturbances. E.g. *Balantidium* has a necrotizing effect, hence the formation of abscesses, *Taenia* secretes toxins which, through blood circulation, reach the brain, causing a nervous effect.
* **Traumatic and inoculatory** : case of*Entamoeba histolytica* which invades the liver

carries bacteria from the intestine to the liver, causing abscesses in the liver

1.2.3. Host reactions and defenses (see immunology course)

Two types of reactions are observed in the host:

- **Humoral reactions:** by the production of specific antibodies (IgG, IgM, IgE.) In most parasitic organisms, there are so-called antibodies that the host secretes as a defense against the parasites that have invaded it. The products that cause antibodies to form are called antigens. Antibodies can be specific or versatile. The presence of antibodies in the body is able to determine active acquired immunity, or incomplete immunity or a state of pre-munition.

Cellular reactions: The mobile cellular elements in the parasitic organism, the phagocytes, often oppose infection in some cases phagocytosis is powerless to stop the development of the parasite and can even promote its evolution by constituting a habitat for the parasite (case of Leishmania donovani). When phagocytosis has not been able to oppose the development of the parasite, in this case we see various cellular reactions including inflammatory reactions, which are characterized by cytological changes in the blood: eosinophilia, leukopenia, anemia, these blood alterations lead to changes in various organs, including hypertrophy of the spleen, liver, lymph nodes and hyperplastic reactions which are characterized by a considerable increase in the volume of invaded cells (e.g. biliary adenomas of the liver of the rabbit affected by coccidiosis)

1.3. Biological cycles of parasites

*__Definition of the life cycle:__

It is the set of mandatory morphological transformations undergone by a parasite to pass from one generation to the next and is represented by a circle. Parasites can have a very complex (indirect) or simple (direct) development cycle.

1.3.1 Indirect life cycles

This is the case of parasites which, in order to complete their life cycle, must necessarily pass through several hosts of different species (heteroxene parasite). We distinguish between:

***HD** = definitive host (hosts the sexual form of the parasite (case of protozoa multiplying sexually and asexually: e.g. cat: Toxoplasma)

***HD** = definitive host with clinical signs usually a vertebrate (case of protozoa multiplying only asexually: Trypanosoma)

***HD** = definitive host (hosts the adult form of the parasite (case of metazoans: e.g. Dog: E. granulosus)

***HI** = intermediate host (formation of infesting forms of the parasite, asexual form of the parasite, case of protozoa multiplying sexually and asexually).

***HI** = intermediate host (non-adult (larval) metazoan case: ex sheep: Echinococcus; cattle: T. saginata)

***HI** = intermediate host of the parasite (usually an arthropod: case of protozoa multiplying asexually).

The parasite that passes through a vector is advantageous over that which passes through a free phase (encystment). Ex. Leishmania, Fasciola

Some parasites that evolve in domestic or wild animals are able to pass into humans and even cause diseases. These diseases common to

humans and animals are called Zoonoses.
Some heteroxene parasites are never found in the external environment, for example: Plasmodium, Trypanosoma, Leishmania, which involve vectors (mosquitoes). The fight against these pests is very difficult.

1.3.2 Direct Biological Cycles

These are parasites that pass through a single host (species or group of animal species) to complete their cycle (monoxenic parasites). The latter spend a large part of their lives with their host and meet in the wild in a cystic form (therefore less advantaged), and it is these cysts that infect new hosts. Ex Giardia, Ascaris.

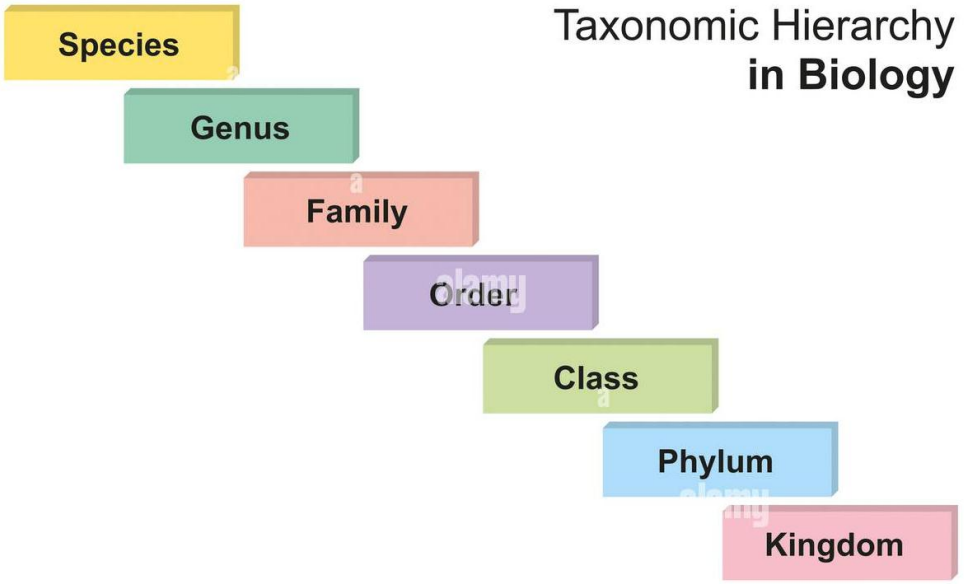
1.4. Diversity

Diversity is the rule in parasitology. Due to their morphology and biology (mobility, reproduction, metabolisms), parasites are extremely diverse, even within the same family:
Morphologically: the size of a parasite can exceed 10 meters (*Tænia*) and remain on the order of a micrometer (*microsporidia*, *leishmania*). Their search can be ensured by an examination with the naked eye (*Tænia*), conventional optical microscopy (plasmodies)*or*even electron microscopy (*microsporidia*).

Parasites can be permanent, their entire existence takes place in one or more hosts (*Taenia, trichina*), temporary dividing their lives between one free form in the environment and the other parasitic (*flukes, anguillae*), or optional having a saprophytic but occasionally parasitic life (opportunistic parasites and fungi, myiasis).

1.5. Classification: Pests are classified into 4 major groups:

1. Protozoan (unicellular being endowed with movement): depending on the case, it moves thanks to plasmopods (rhizopods), flagella, undulating membrane or eyelashes .They are in asexual form or with sexual potential, mobile or encysted , intra or extracellular.
2. *Helminth or worm* (a share of metazoans: being multicellular with differentiated tissues.). They are recognized in adult forms of both sexes in larval, embryonic or ovular form.
3. *Fungi or micromycetes,* the latter constitute a kingdom in their own right, they are microscopic fungi identified in the form of isolated or grouped spores or free or tissue filaments
4. *Arthropods, molluscs, pararthropods (porocephalus), or annelids* are
metazoan, multicellular and with differentiated tissues) Insects, mollusc and crustacean arachnids, which can come in adult (imago) male and female forms, eggs and larvae (nymphs).
Nomenclature and systematics (taxonomy) of human parasites initially morphological now uses other genetic and immunological criteria. The laws of systematics are simple but strict. Since Charles Linné all animals and plants are designated by two Latinized words (Linnean binomial) (the first: genus name, capitalized, the second without capitalization is the name of species (both in italics or underlined) followed by the name of the author who assigned it the first time and the date of
this attribution. The species is the set of individuals whose cross, made at random, always gives indefinitely fertile descendants between them, the genus grouping affine species. *E.g.: Culex pipiens Linné 1758*
Genus and species come from a series of steps:



Naturalists faced with increasing diversity had to create the subgenus, with a capital letter and in parentheses, after the name of the genus, and the sub-species which is written without a capital letter after the name of the species
Ex: *Anopheles (Maculipennia) maculipennis atroparvus van Thiel 1927*

CHAPTER II. PARASITIC PROTOZOA

2.1. General and categorisation

Parasitic protozoa are found in various living beings (humans, animals). Human and animal diseases determined by protozoa are numerous and deadly (e.g. malaria, coccidiosis, etc.). Their economic impacts require us to combat protozooses either by directly attacking the parasites in the organism of their hosts, or by reaching them during their free existence in nature (cysts), or in their intermediate hosts or their vector agents.

Protozoa are single-celled animals (a typical cell) that are heterotrophic. The size of the protozoa, which varies greatly, is generally microscopic. The structure of the nucleus varies greatly according to the groups. Most protozoa reproduce by both asexual and sexual reproduction.

The classification of protozoa is essentially based on the musculoskeletal system.The main phylla (branches) are:

- Phylum of Sarcomastigophora (e.g. Rhizoflagellates) comprising 25,000 species
    - Under the Mastigophora Phylum (ex embr. Flagellate
    - Subphylum Sarcodina (e.g. Rhizopods)
- Phylum of Apicomplexa (e.g. Sporozoa) comprising 4500 species
- Phylum of the Ciliata grouping 7500 species
- Microspora phylum with 800 species
- Myxozoa phylum with 875 species

2.2. Phylum of Sarcomastgophora (e.g. Rhizoflagellates)

Is represented by two groups which are flagellates and rhizopods. Includes species that move by pseudopods or flagella or both, multiply asexually.

2.2.1. Subphylum Mastigophora (e.g. Flagellates)

a. General morphology

Members belonging to this subphylum present in the vegetative state one or more flagella, thanks to which they move (**Fig.1**).

b. Order of Kinetoplastida

The limbs have one or two flagella and a kinetoplast in close connection with the basal body of the flagella and the infestation of the host is done using an active vector.

b.1. Trypanosomatidae family

All members of this family are parasites. Originally they are insect enteroparsitis; they also parasitize the blood and other tissues of vertebrates (mammals and birds). This family is of great medical importance. Among the genera that belong to this family, we have the genera: *Trypanosoma, Leishmania*.

They have a unique flagella inserted on a kinetoplast. The nucleus is separated from the kinetoplast. There may or may not be an undulating membrane. Reproduction of

trypanosomidae is normally carried out by binary longitudinal scission (division of the kinetoplast, follows the nucleus, then the flagella and lastly the cytoplasm). During the development cycle these parasites have different forms (Trypanomatidae *types*)(**Fig.2a, b, c, d**):

Leptomonad type (promastigote): is the simplest type, comes in the form of an elongated, flattened body, has a central nucleus, anterior kinetoplast and a free flagella. It is found in the organism of the invertebrate host. All other representatives of the family can be considered as derived from this type**(Fig.2a**).

Crithidian type (epimastigote): the kinetoplast moves backwards approaches the nucleus while remaining anterior to it, existence of a short undulating membrane and there is a free flagella. It is found in the invertebrate host except in the genus Trypanosoma or it is also found in the vertebrate host. **(Fig.2b).**

Leishmanian type (amastigote): rounded or ovoid bodies, with a nucleus and a kinetoplast, devoid of flagella, but often presenting an axonem from the kinetoplast to the wall of the body. It is found in both the invertebrate and vertebrate host. **Fig 2c**

Trypanosome type (Trypomastigote): the kinetoplast occupies a position posterior to the nucleus and is placed in the vicinity of the end of the body, the existence of a well-developed undulating membrane, and there is or is not a free flagella. It is usually found in the vertebrate host**. [Fig. 2d]**

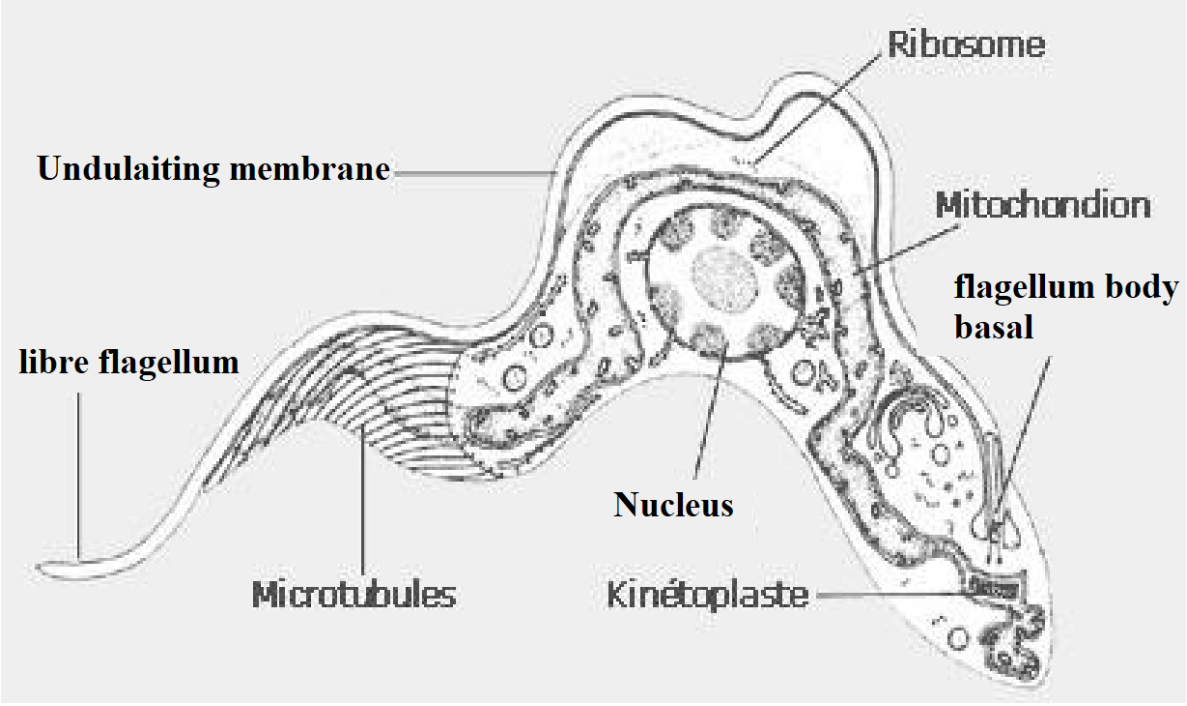


Figure 1 : General morphology of a flagellate (Trypanosoma)

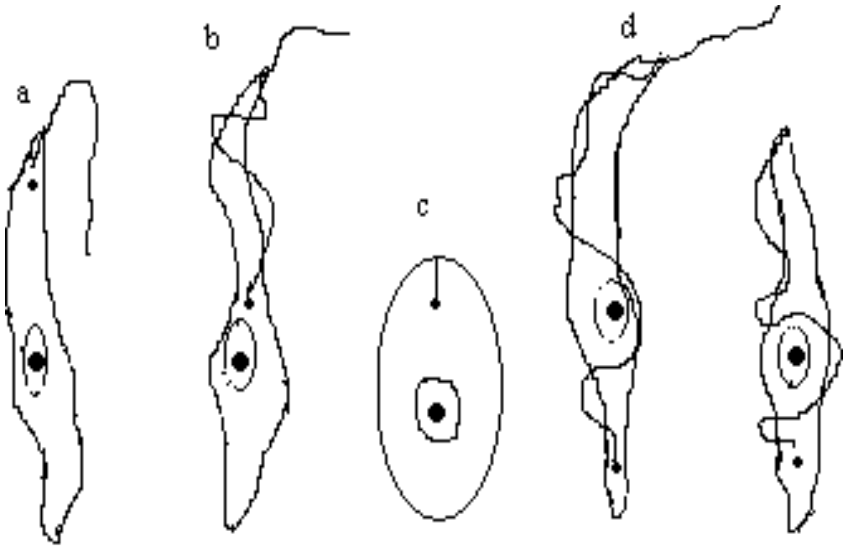


Figure 2 : différentes formes (types de *Trypanomatidae*)

***Genre Trypanosoma**

Trypanosomes are exo-erythrocytic bloodstained flagellate protozoa; they are living beings consisting of a single cell with a flagellum. Trypanosomes in their typical forms most often live in the blood, sometimes in other body fluids such as cerebrospinal fluid (CSF) or in the lymph node juice of various vertebrates and in various forms. Members of this type are characterized by a fusiform body, with a rounded posterior end and a tapered anterior end. They are parasites with two hosts, a vertebrate and an invertebrate, which transmit the infection through the intermediary of its saliva during a bite (Salivaria type) (Ex. *Trypanosoma gambiense*) or its droppings (Stercoraria type) (Ex. *Trypanosoma cruzi*). There are no cystic forms. The dimensions of trypanosomes are highly variable. Most mammalian pathogenic species have a length of 25 to 30 µ and a width of about 2 µ. Trypanosomes feed by osmosis depending on the blood serum or fluid in which they live. In the blood of the vertebrate, the trypanosomes move mainly using the flagella (forward flagella) and their undulating membrane. Multiplication is done by binary division.

There are several species belonging to this genus, we will mention among others;

- ***Trypanosoma theileri*** : H.D= Bovidae and the vector agent an insect (Tabanidae)
- ***Trypanosoma evansi*** : H.D= Horse, donkey, mule, beef, buffalo, camel, dromedary, dog, cat, and vector agent an insect (Tabanidae) and vampire bats.
- ***Trypanosoma equiperdum*** H.D= equidae and transmission is through coit (sexually).
- ***Trypanosoma(b) gambiense*** H.D= human and experimentally all mammals and the vector agent an insect (H.I) (*Glossina palpalis*: tsetse fly). Disease: West African trypanosomiasis.

- ***Trypanosoma(b) rhodesiense*** H.D= human, dog, antelopes, warthogs and the vector agent an insect (diptera) (H.I) (*Glossina morsitans* : tsetse fly). Disease: trypanosomiasis is African

- ***Trypanosoma cruzi*** H.D = human and vector agent an insect (hemiptera) (H.I) (*Triatoma* : hematophagous flea).

Study of the parasite *Trypanosoma (brucei)gambiense*

Responsible for African sleeping sickness (Trypanosomiasis).

Category

Reign of the Protists
Under Protozoa Branch of
Sarcomastigophora Under branch of
Mastigophora Order of Trypanosomatida
:
Trypanosomatous family*Gender*
:Trypanosoma
Species *: T. (b) gambiense*

Body type:

T. gambiense was discovered by RM Forde in 1902 in the blood of a European in English Gambia. It is a polymorphic trypanosome. *Trypanosoma brucei gambiense* is 20-30 microns long. We encounter: Trypomastigote (and amastigote) forms in the definitive vertebrate host Promastigote and epimastigote forms in the intermediate insect host

Evolutionary cycle (see Fig.4)

It is an indirect cycle (heteroxene).

HD : human for *T. (b) gambiense* and experimentally almost all mammals. Exocellular formestrypomastigotes in circulating blood and tissues.

T.(b)gambiense multiplies in human blood by longitudinal, binary or multiple division.

H. A : is mainly the man with sleeping sickness.

HI: dipteran insect (tsetse fly) male and female are haematophagous; genus *Glossina*: *group* G.palpalis develops in the digestive tract of the tsetse. (Fig.5)

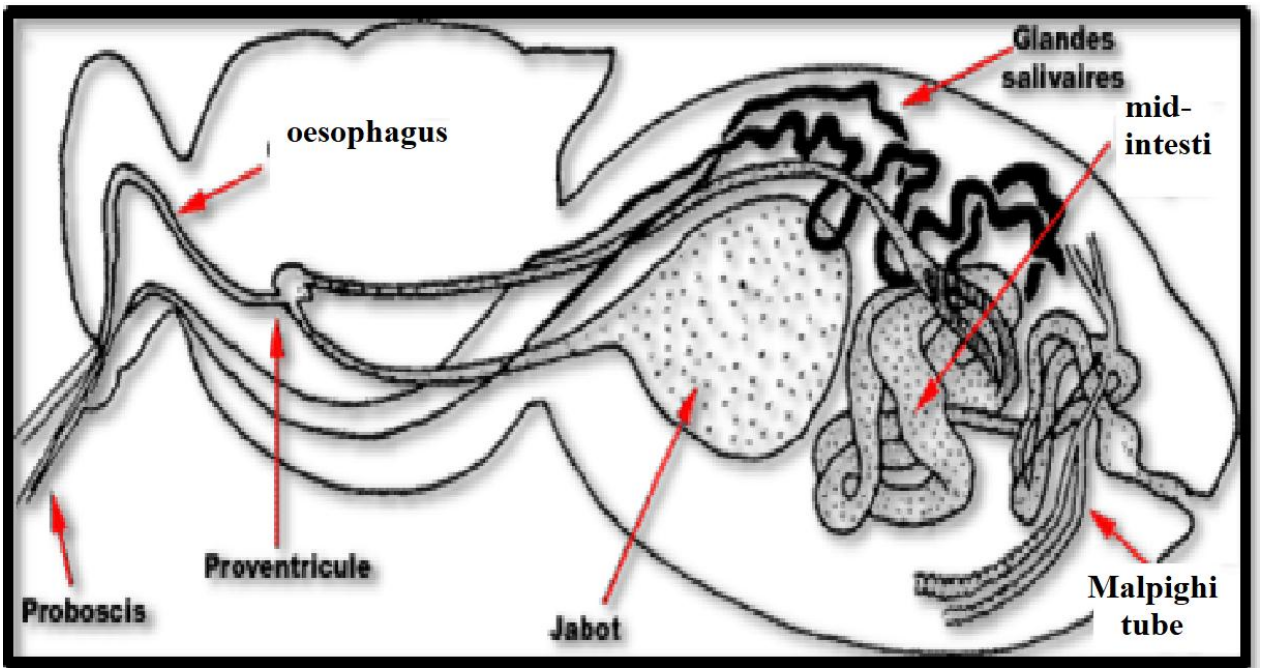
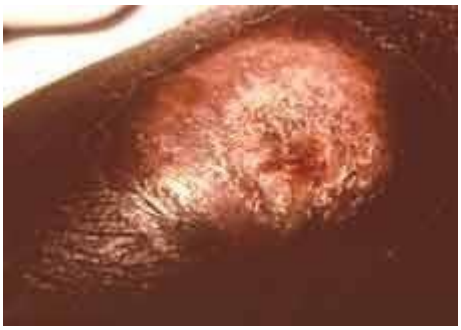


Figure 3: Digestive tube of a tsetse

At the Definitive Host

Trypanosomes are inoculated into humans during the blood meal of an infective tsetse. The trypanosomes that penetrate during the sting will first remain in the subcutaneous tissues, at the sting point, giving a kind of boil, the **inoculation chancre** (photo below)



These trypanosomes will then pass into the blood where they will multiply and disperse thanks to blood and lymphatic circulation. They will thus be found in different organs and in the **lymph nodes, especially in the cervical region** (photo below).

Trypanosomes will remain in the blood and lymphatic systems for varying lengths of time, from a few weeks to several years. One day, they will manage to cross the meningeal barrier that protects the central nervous system. At this moment, the patient will move into a new phase of the disease, the phase of cerebral polarization. This will result in a worsening of his condition with the appearance of neurological signs, but also in an almost total disappearance of trypanosomes in the blood: at this stage they are found almost exclusively in the tissues and fluids of the central nervous system.

If a tsetse feeds on a human or animal whose blood carries trypanosomes, it will swallow the parasites with the blood. The higher the parasitemia in the host, the greater the risk of infestation. The trypanosomes mature in the insect's digestive tract and then migrate and multiply in its salivary glands: from then on, the fly becomes infective, i.e. 20 to 35 days after the contaminating blood meal. It will contaminate a new healthy host by injecting its saliva which is used to anesthetize the wound caused by the sting and prevent blood clotting.

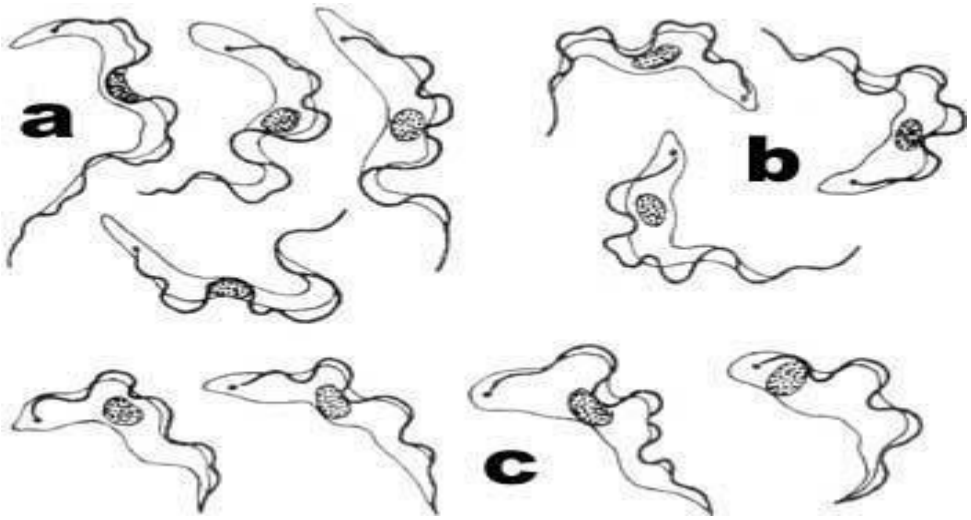
Tsetse is naturally refractory to trypanosome infection.

Forms taken by the trypanosome during its cycle

The *T. brucei gambiense* cycle takes place in the blood of the mammalian host and in the vector. During this cycle the parasite undergoes a number of changes.

In mammalian blood it is found in several forms, in particular in the long or **slender** form (**a**), in which the trypanosome multiplies, and the stocky or **stumpy** form (**c**), from which a new antigenic variant will emerge. Between these two types there are **intermediate forms** (**b**).

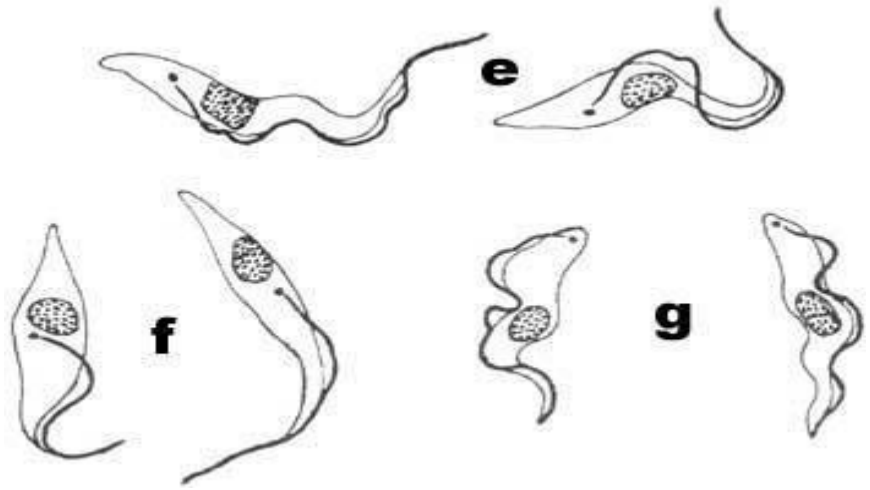
All blood forms of trypanosome are grouped under the term " **trypomastigote form** ".



In the intermediate host: tsetse

The ingested trypanosome (trypomastigote) begins by elongating in the digestive tract of the insectand loses its surface antigenic mantle (layer of variable surface glycoproteins located on the outer part of the trypanosome cell membrane and responsible for the antigenic variation). This is the " **pro-cyclical form** ". Then the trypanosome will reach the salivary glands of the insect. It shortens and its kinetoplast migrates to the back of the nucleus (**epimastigote form**) (**f**). In the next step, the kinetoplast is replaced in front of the

nucleus and the trypanosome reconstitutes its surface antigenic mantle (**metacyclic form**) (**g**). At this time it is again capable of infecting a mammalian host (**infesting metacyclic form**) in which it will recover its trypomastigote form.



In the blood, *T. brucei gambiense* can be seen in several forms. The long or hail shape called"slender" whose average size is 23 to 30 microns but which can exceed 40 microns. It has a well-marked free flagellum of about 6 microns, and a well-developed undulating membrane. The kinetoplast is sub-terminal more than 4 microns from the posterior end which is elongated. The core is oval. It is in this form that the trypanosome multiplies in the blood.
The short or stocky form, called "stumpy", measuring 12 to 26 microns is thick without free flagella (or weakly marked), with a kinetoplast more terminal than in the long form, a rounded posterior end, a rounded core and a well-developed undulating membrane.
The proportion of these different forms in the blood is dependent on the host's immunological response.

The trypomastigote forms evolve into epimastigote forms that multiply, cross the peritrophic membrane and reach the salivary glands, where they transform into infectious metacyclic trypomastigote forms, which during a bite these forms are inoculated with healthy HD. After 1 to 2 weeks, the trymastigote forms migrate via the blood to SRE and the lymph nodes, after a few months, we see a weakening of the meningeal barrier which allows the trypomastigote to pass into the central nervous system, fluctuating persistence of the blood trypomastigote forms which will be used to contaminate the IH.
The trypanosomes ingested by the vector insect during a blood meal develop in the middle intestine in the so-called procyclic trypomastigote form at this stage. They then migrate to the salivary glands where, after an intermediate epimastigote stage, they transform again into trypomastigotes. At this stage, the parasite is referred to as metacyclic. The trypanosomes are then likely to be inoculated into a mammal where they

develop in the blood. The parasite eventually invades the cerebrospinal fluid where its presence causes the characteristic disorders of sleeping sickness. Blood trypomastigotes come in two forms: a long form capable of dividing but not differentiating once ingested by a fly (" slender " form) and a stocky form without a free flagellum that does not divide but is capable of differentiating in the gut of the tsetse fly into a pro-cyclic form (" stumpy " form).

Location: blood, cerebrospinal fluid, lymph node juice and definitive host parenchyma. Stomach and salivary glands of the tsetse.

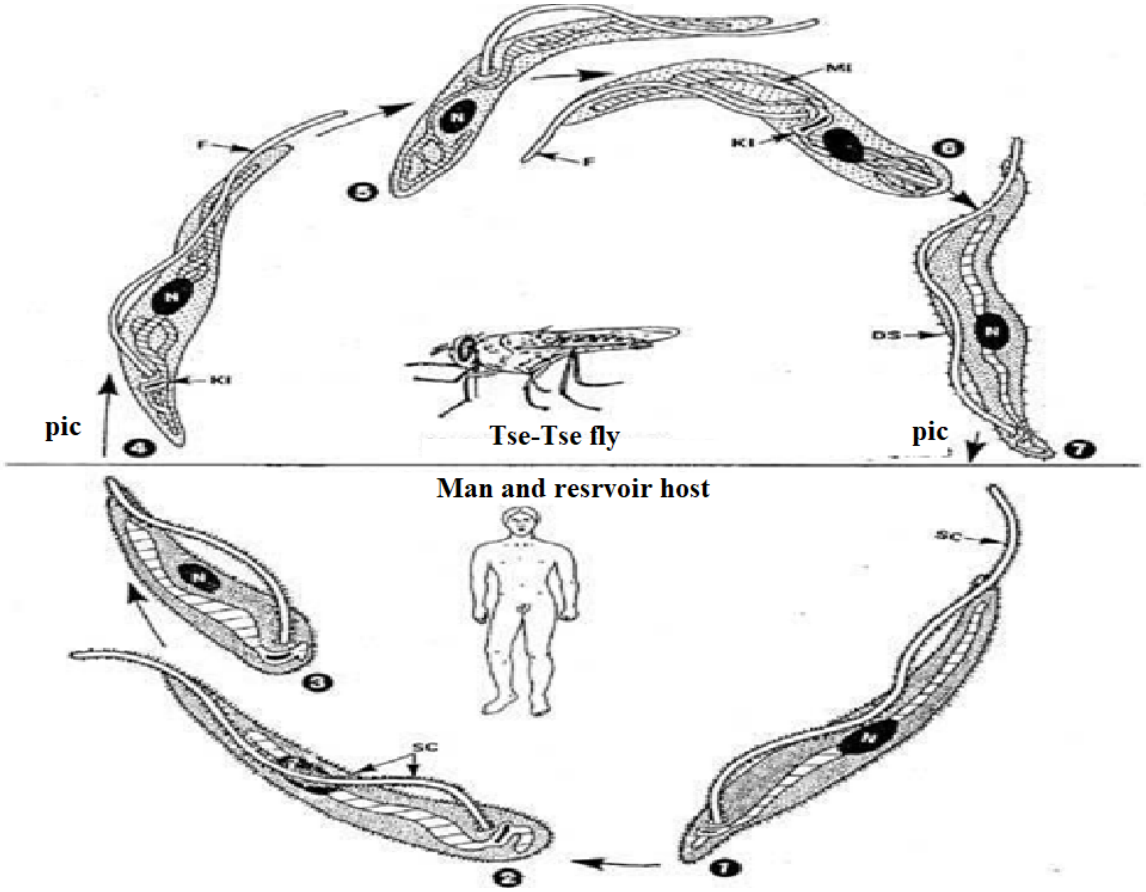


Figure 4: Development cycle of *Trypanosoma brucei rhodesiense* and *Trypanosoma b. gambiense* (after Mehlhorn & Ruthmann, 1988).

1: Bloody trypomastigotes (" slender " forms); **2:** intermediate trypomastigotes that multiply by binary division; **3:** trypomastigotes (" stumpy " forms) that develop inside the vector; **4:** procyclic trypomastigotes; **5-6:** transformation of trypomastigotes into epimastigotes that multiply by binary division; **7:** metacyclic trypomastigotes in the salivary glands of the vector. These forms are infectious to humans. **DS:** Developing Cell Coat; **F:** Flagella; **KI:** Kinetoplast; **MI:** mitochondria; **N:** nucleus; **SC:** cell mantle; **SF:** short flagella.

Epidemiology

Recall that Trypanosomes are extracellular flagellate protozoa. In the blood, they are mobile thanks to the movements of their undulating membrane. They come in two forms, short and stocky (15 µ); long and thin (40 µ). Glossines are strictly anthropophilic African diptera(tsetse flies) (between 15° north latitude and 30° south latitude), require heat (25 - 30°C) humidity and shade (trees and thickets) so sleeping sickness is strictly intertropical African. Both sexes are hematophagous.

Symptomatology: It is subdivided into 3 phases:

*** Incubation phase**

It is a period of variable duration, moreover difficult to determine generally quite short, from 8 to 10 days then appears,

* **Invasion phase:**

It is characterized by irregular febrile bouts. We first see an initial lesion (trypanosome at the point of inoculation (appearance of a boil that does not mature), nervous excitation with insomnia, fatigue, inappetence and sometimes intense headache. These different symptoms disappear after 2 to 3 weeks except for fever.

* **Status Phase:**

It begins after a few weeks or months after contamination in which two phases can be distinguished

1 - **lymphatic-sanguine phase**:Due to the presence of the parasite in the blood. It is characterized by irregular fever, adenopathies of the cervical chain (excellent clinical sign), often discrete splenomegaly, cardio-vascular disorders: palpitations, very clear return of neurological manifestations (hyperesthesia, headache), exanthemata sitting at the trunk or root of the limbs, a feeling of fatigue, muscle weakness, cramps upon awakening, tingling in the limbs, appetite is preserved and urine is normal; menstruation persists in women but abortion is frequent. It can last from a few months to a few years.

2 - **meningoencephalitis phase**: It corresponds to the crossing of the meningeal barrier by the parasite (presence of the parasite in the cerebrospinal fluid, which leads to CSF abnormalities. It results **in an increase in neurological signs** sensory disorders: hyperesthesia (sign of the key), cramps, neuralgia motor disorders: tremors, convulsions, drunken gait, incoordination, psychic disorders: irritability, depression, apathy, reversal of the nycthemera (hence the name sleeping sickness), sexual disorders: impotence, frigidity, disorders of thermal regulation, clavicles and ribs take shape under the skin and the patient has the appearance of a skeletal **(Photo2)**, various ocular manifestations. At the end of this phase, the patient takes a special attitude, with his head bowed to his chest and his eyes closed; he falls asleep wherever he is. "Without treatment" the patient falls

in a coma and dies within 2 years

Photo 1: Showing a person with sleeping sickness

Diagnostics

Diagnosis is often very delicate during the first period of the disease. In all cases, the search for trypanosomes is essential.

*** Orientation diagnosis**: A stay in intertropical Africa, polymorphic clinic (cerebral lymphadenopathies), character changes, ECG and EEG changes

Blood: anemia, ESR increased, hypergammaglobulinemia

CSF: clear, lymphocytes > 4 / mm3, presence of IgM (pathognomonic), increased proteinorachia

*** Direct diagnosis**: parasites are evidenced by direct examination of peripheral blood and cerebrospinal fluid

Blood: thin smear, thick drop and/or concentration ==> look for trypomastigote forms (their number decreases during the disease)

Nodalsuccess: trypomastigote forms during the lymphatico-blood phase

During the nervous phase of the disease: search for trypomastigote forms in postcentrifugation CSF

PCR

*** Indirect diagnosis**: Serum antibodies are tested

Treatment:

There is no known medication absolutely specific to human trypanosomiasis, but products such as arsenicals have shown encouraging results. Hospitalization of the patient is mandatory.

Prophylaxis

General and individual prophylaxis should be considered successively

*** General prophylaxis**: is intended for patients and tsetse

For patients: field screening and treatment of humans, no mass treatment possible

Tsetse control: it is difficult to achieve: use of insecticides, brush clearing around homes, trapping (blue visual appeal + fragrant appeal + deltamethrin)

*** Individual prophylaxis**: it consists of ensuring the mosquito bite and avoiding infection by wearing light clothing (make tsetse flee) to keep tsetse away and avoiding traffic at night. At present there is neither chemoprophylaxis nor vaccination (variable trypanosome antigens)

Note

* *Trypanosoma(b) rhodesiense* **H.D** = human, dog, antelopes, warthogs and the vector agent an insect (diptera) **(H.I**) (*Glossina morsitans*: tsetse fly). The geographical distribution: West Africa, same life cycle and same symptomatology as *Trypanosoma (b) gambiense.*

* **Evolutionary cycle of Trypanosoma cruzi (Fig. 5)**

The cycle of this species, more complex than that of *Trypanosoma brucei*, includes intracellular forms.

The trypomastigote forms ingested by the vector undergo several differentiation processes during their migration in the intestine: differentiation into epimastigote and then into metacyclic trypomastigote (infesting forms). These forms will be deposited with the faeces during a new blood meal about 2 weeks after the infesting meal. The trypomastigotes eliminated in the droppings of the reductees pass through the injured skin or certain healthy mucous membranes such as the conjunctiva. Carried by the bloodstream, they enter the cells of the vertebrate host, macrophages and muscle cells (in particular cardiac) where they transform into amastigote forms which by binary division lead to the formation of a pseudo cyst filled with amastigote threads. Inside the cell, these amastigote forms, after a short passage through the epimastigote stage, turn into trypomastigotes. The rupture of the cell releases the trypomastigotes which can then infest new cells or be ingested by a reduction during a new blood meal.

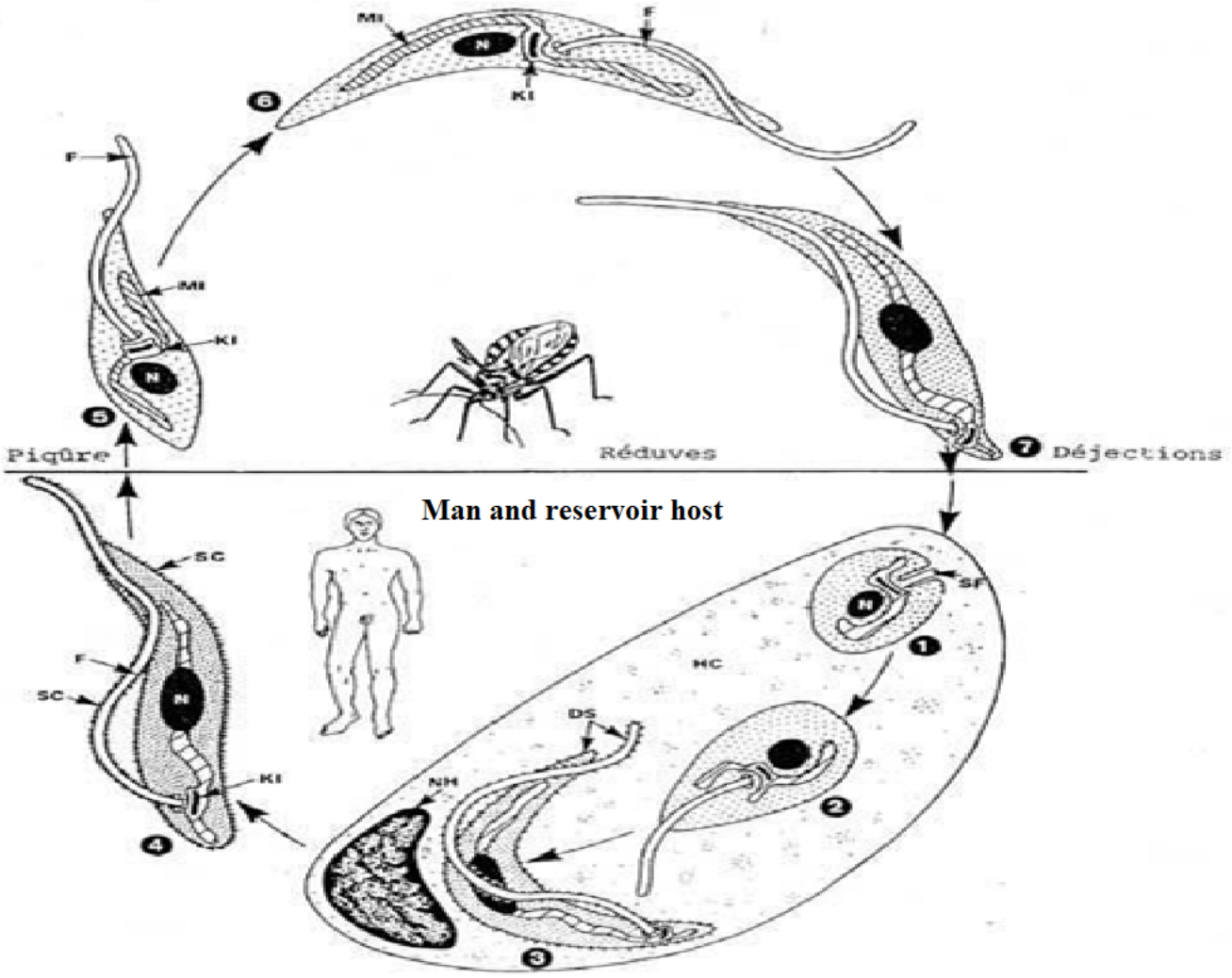


Figure 5: Development cycle of *Trypanosoma cruzi* (after Mehlhorn & Ruthmann, 1988).

1: Amastigotes multiplying by binary division inside the cytoplasm of the host cell; **2-3:** transformation into trypomastigotes (3) *via* the epimastigote form (2); **4:** appearance of trypomastigotes in the bloodstream; **5-6:** transformation of trypomastigotes into epimastigotes in the gut of the vector insect where these parasitic forms multiply by binary division; **7:** transformation of epimastigotes into infectious metacyclic trypomastigotes. **DS:** Cellular coat in development. **F:** flagella; **HC:** host cell; **KI:** kinetoplast; **MI:** mitochondria; **N:** nucleus; **NH:** host cell nucleus; **SC:** cell mantle; **SF:** short flagella.

Genus *Leishmania*

Members of this genus are characterized by an elongated body, with more or less tapered ends, having a nucleus and an anterior kinetoplast. *Leishmania* are often referred to as Leishman bodies or Donovan bodies. They are very small spherical, ovoid or fusiform organisms, hardly exceeding 2 to 6 ù long by 1 to 2 ù wide. The nucleus is more or less spherical and the karyosome is central. Multiplication is by binary division, in the Leishmanian form in the vertebrate host and in the Leptomonadian form in the invertebrate host (heteroxene parasites). The usual vector hosts of *Leishmania* are lephlebotomous and transmission is by sting. It is possible that transmission occurs by crushing the insect on the skin.

Leishmania are found in the reticuloendothelial system (RES) of their hosts, in connective and mesodermal cells rarely in peripheral blood leukocytes. All parenchyma and organs can harbor the parasite. The latter feeds at the expense of the cellit inhabits and the latter frequently erupts.

In vertebrates, during their intracellular life, the Leishmanian form exists. They are parasites with two hosts, a vertebrate and an invertebrate, which both host the Leptomonadian and Leishmanian forms. The Leishmania were first seen by Cunningham in 1885. There are several species belonging to this genus, including:**Those responsible for visceral Leishmaniasis:** *Leishmania donovani infantum* HI: *Phlebotomus,* around the Mediterranean basin. *Leishmania donovani donovani* HI: *Phlebotomus,* in Africa, Asia, Middle East, South*America.Leishmania donovani chagasi* HI:*Lutzomyia,* South America.

Leishmania caprae xhez goat?

Those responsible for cutaneous Leishmaniasis:

Leishmania (tropica) major Wet cutaneous Leishmaniasis, HI: *Phlebotomus*, North Africa,Middle East, Southern Europe.

Leishmania (tropica) minor Dry cutaneous Leishmaniasis, HI: *Phlebotomus*, North Africa, Middle East, Southern Europe,

Leishmania aethiopica HI: *Phlebotomus,* Ethiopia, Kenya.

Leishmania brasiliensis brasiliensis, HI:*Lutzomyia,* Mexico and Brazil.

Leishmania peruviana HI:*Lutzomyia,* Peru.

Study of the parasite *Leishmania donovani* infantum

Responsible for childhood visceral leishmaniasis (Splenomegaly) (kala-azar disease).

Category

Reign of the Protists

Under Protozoa Branch of *Sarcomastigophora*

Under branch of *Mastigophora*

Order of Trypanosomatida *:*

Trypanosomatidae

family*:*Genus *Leishmania*

Species *Leishmania donovani*

Subspecies *Leishmania donovani infantum*

Body type:

It was discovered by Nicolle in 1908. Comes in 2 forms:

Amastigote forms (etymologically = without flagella). They are ovoid organisms, measuring 2 to 3 µm in diameter; presenting a nucleus and a kinetoplast with or without the short root of a flagella or rhizoplast. The multiplication is done by scissiparity. They are immobile, necessarily endocellular and are present in vertebrate HD

Pro-mastigote shapes, elongated, flagellated, 8-24 x 4-5 µm long, they are very mobile, moving flagella forward. The kinetoplast is located at the base of the flagella. The multiplication is done by scissiparity. These forms are found in the digestive tract of intermediate hosts and in in vitro culture:

Evolutionary cycle (see Fig.4)

It is an indirect cycle (heteroxene).

HD : man, dog, cat

HI : Various female sandflies: *Phlebotomus perniciosus, P. sergenti, P. argentipes.*

HR : Dog

At the Definitive Host

Following a bite, the parasites are inoculated into **humans (vertebrate**) during the blood meal of an infectious female sandfly (*Phlebotomus perniciosus*) in leptomonadian form. These forms are then phagocytosed by the macrophages present in the dermis of the definitive host. After about a few minutes, these forms will evolve into endocellular amastigote (Leishmanian) forms. The parasite is surrounded by a parasitophorous vacuole that multiplies by binary division. The macrophage bursts and releases several parasites in amastigote form. After 4 to 6 months, these forms arrive at the level of **the internal organs (in particular the spleen**, liver), invade the cells of the host where they multiply by binary division and determine visceral leishmaniasis.

*** In the intermediate host (vector): the female Phlebotome**

The complete evolution of the parasite continues in the digestive tract of the sandfly where it takes the Leptomonadian form (promastigote) which abounds in the stomach, the esophagus of the insect.

The amastigote forms ingested by the vector undergo several differentiation processes during their migration in the intestine: differentiation into promastigote forms in the middle intestine which have a small form whose size varies between 10 and 20 u, there they multiply by scissiparity, then these forms migrate to the anterior end of the digestive tract (proventricle or pharynx depending on the subgenus) in infectious promastigote forms.

During a subsequent bite, the sandfly regurgitates and injects the promastigote forms into a healthy subject.

Location

In the vertebrate *L. donovani infantum* is found in amastigote form throughout the body, abundant especially in the mononuclear and endothelial cells of

blood and lymphatic vessels especially of the spleen, liver, bone marrow, lungs, testicles, kidneys **In the invertebrate** *L. donovani infantum* is observed in the digestive tract, where it takes the formpromastigote.

Epidemiology

Recall that *Leishmania* are endocellular flagellated protozoa. Sandflies are diptera, nematocerous, of the family *Psychodidae*. Only the female is hematophagous. The insect's life cycle is terrestrial (larvae on wet soil) **(Fig.7).** Sandflies have nocturnal and twilight activity. During the day the insect lives in crevices, holes, burrows in a humid environment. They are bad sailboats, are small in size (2 mm). In Algeria, *Phlebotomus perniciosus* is the vector of Kala-azar.

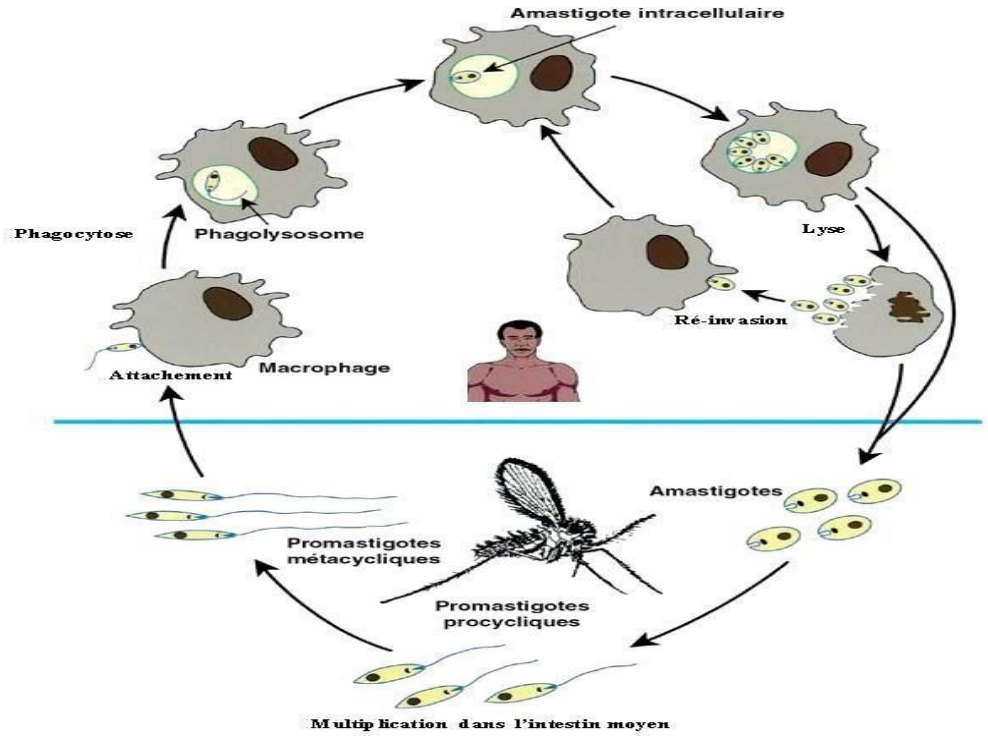


Figure 6 : Lifecycle of the genus Leishmania

Pathology: *Leismania donovani* is the pathogen of human visceral leishmaniasis and L.donovani infantum is responsible for infantile visceral (splenic) leishmaniasis. It exists only in countries bathed by the Mediterranean.

Figure 7: Phlebotomus Biology

Symptom

It is divided into 2 phases:

***Incubation phase**

The incubation period is imprecise. It is generally accepted that it lasts a few weeks to a few months.

***Phase d'état**

The dominant symptoms are a long-lasting crazy (irregular) fever (1 to 2 months), splenomegaly (the spleen is hard to palpate, painless), enlargement of the liver is less accentuated and later than that of the spleen, and anemia is blamed.

The patient is very weak (child who no longer plays), the weight loss is considerable (especially the limbs), contrasting with the volume of the abdomen distended and sensitive to pressure. Ulcerations of the digestive tract (mouth and intestine) also occur, often with renal, pulmonary and cardiac complications.

Blood has characteristic changes: decrease in red blood cells, leukopenia (especially polynuclear)

The Kala-azar ends in some cases with healing. In the majority of cases cachexia progresses, accompanied by the aforementioned disorders and the patient succumbs.

Note that gender and race have no influence on the development of the disease; on the other hand, age plays an important role and it can be said that Kala-azar is a disease of young people, especially children.

Diagnosis:

Clinical symptoms do not always allow an accurate diagnosis (confusion with other diseases: malaria, childhood leukemia, etc.). Several diagnoses can be used: Clinical and referral diagnosis:

Anemia, Accelerated sedimentation rate, hypergammaglobulinemia, anamnesis (stay in endemic region),

Direct diagnosis:

* Search for the parasite: it is done on a bone marrow puncture (or spleen, liver, lymph nodes), the blood is usually negative, PCR.
* Testing for circulating antigens sometimes yields results.

Indirect diagnosis:

Search for serum antibodies (antigens: promastigote forms) by ELISA techniques, The inconsistent response in the immunocompromised.

Prognostic

Kala-azar is a serious disease that, untreated, leads to death in 96% of cases, but early diagnosis leads to cure in 80% of cases.

Processing

It targets the parasite:

In a first line: pentavalent antimony salts (20 mg SbV/kg/d IM or slow IV, 20-day courses) are recommended who 1990 recommendation, and in a second line: Pentamidine salts (4 mg/kg/d IM, 1 day out of 2 for 2 months) are used.

Prophylaxis

General prophylaxis

* Vector control: As sandflies are the vector gents of this condition, it is first necessary to protect against the bite of these insects and to fight against sandflies by destroying unclean homes and keeping houses away from rubbish, where larvae abound.
* Fight against the reservoir of parasites: since the dog appears to be the main reservoir of the virus, it is necessary to kill useless or stray dogs in endemic areas, or to diagnose (clinically or serologically) parasitized dogs and treat them.

Individual prophylaxis

* Avoid sandfly bites: do not walk at dusk along the edge of woods and thickets,
* Use of home insecticides and fine-meshed mosquito nets given the small size of sandflies,
* Vaccines: immunization trials against visceral leishmaniasis have not yielded

No result.

Study of Parasites *Leishmania (tropica) minor and Leishmania (tropica) major*

Morphology: morphologically they are identical to *L. donovani* (see Fig.8)
Geographical distribution: are found in countries with hot and dry climates.
**** Leishmania (tropica) minor***
Disease: Dry cutaneous leishmaniasis = Biskra's pimple = urban form
Vector (H.I): ***Phlebotomus papatasi*** and *P. sergenti*
Tank host: Man, rodents, dog.

Leishmania (Leishmania) major
Disease: Wet cutaneous leishmaniasis = rural form
Vector (H.I): ***Phlebotomus caucasicus***
Tank host: rodents.

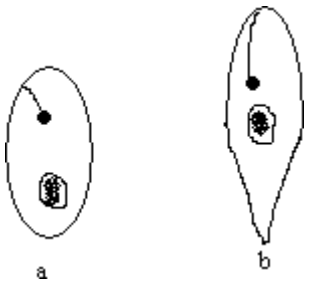


Figure 8: *Leishmania (tropica) sp* schematica: ovoid shape, b: elongated shape

Location:
*** In vertebrates:** the parasite is found in epithelial cells and in large mononuclear cells, which can be found in peripheral blood and in vessels and superficial lymph nodes.
*** In sandflies:** the parasite is located in the digestive tract in Leptomonadian form

Symptom
It is divided into 2 phases:
*** Incubation phase** : varies from a few days to a few months (3 months). We sometimes see bouts of fever.
*** Status Phase**:
This evolutionary phase can in turn be subdivided into 3 phases:
-Papular phase: begins with the appearance of a small red and itchy papule which forms at the level of the sting and evolves into a brownish crust very adherent to the underlying tissues, if it is removed, an ulceration that can reach 2 to 4 cm. of

diameter

- Ulcerative phase : wet form: ulceration covered with a crust, with inflamed border rich in parasites (Oriental button, Biskra nail, Aleppo nail, wooden yaws, Uta.),

dry form: scaly lesion, which reveals a parasite-rich serosity

- Scarring phase : the ulceration heals spontaneously and fleshy buds form under the crust.

The scar becomes indelible white, often hyper-pigmented.

Complications of Biskra's pimple are very rare; lesions are often confined to the skin. The site of the lesions is usually on the exposed parts (lower limbs, upper limbs, face).

The total duration of the disease generally lasts 1 month to one year. On average, healing is achieved after 3 months with indelible scars (state of immunity).

Diagnostics

Generally, it does not present any difficulty in countries where the disease is endemic.

Orientation or clinical diagnosis: stay in the endemic region, ulcerative lesion

Direct diagnosis: Sampling at the inflammatory border of the lesion, scraping with a vaccininostyle or curette or biopsy where we:

- Search for endocellular amastigote forms on a smear stained with Giemsa or MGG. A specialist is needed)

research success in 50% of cases.

Indirect diagnosis: - delayed hypersensitivity test (Montenegro reaction), within 48 hours

==> indurated area with a diameter greater than 5 mm in case of positivity.

- Testing for serum antibodies

Processing

The disease heals spontaneously and it is sufficient, in the mild case, to isolate the pimple by aseptic dressings to prevent secondary infection**.** Disinfection and chemotherapy**.**

Prophylaxis

General prophylaxis:

* Vector control: As sandflies are the vector gents of this condition, it is first necessary to protect against the bite of these insects and to fight against sandflies by destroying unclean homes and keeping houses away from rubbish, where larvae abound.
* Control of the pest reservoir: the rodent appears to be the main reservoir of the virus destruction of burrows by ploughing.

Individual prophylaxis

* Avoid sandfly bites: do not walk at dusk along the edge of woods and thickets,
* Use of home insecticides and close-meshed mosquito nets
* Vaccines: immunization trials against visceral leishmaniasis have not yielded

a.* Order of *Diplomonadida

Members belonging to this order have their organelles in duplicate, symmetrically arranged on each partet of a median axostyle: 2 nuclei, 2 times 4 flagella, 2 bulky parabasal apparatuses. Reproduction is by longitudinal binary division. The cysts are ovoid and most often contain two individuals. This order comprises only one family: Octomitidae *grouping* 3 genera including that of *Giardia*.

* Genre ***Giardia***

This *Octomitidae* has a piriform body, rounded anteriorly and pointed at the posterior end; the dorsal face is convex while the ventral face is flattened and has in its anterior part a circular depression in the form of a suction cup. There are 8 flagella arranged symmetrically in pairs. Two oval-shaped anterior nuclei located on either side of the midline of the body. They are intestinal parasites of many vertebrates and multiply either in the active state or under cysts that ensure contamination. There are several species of which we will mention a few:
Giardia intestinalis (man), *Giardia microti* (vole), *Giardia bovis* (South American beef), *Giardia equi* (horse in South Africa), *Giardia canis* (dog), *Giardia caprae* (goat in Holland), *Giardia cati* (domestic cat).

Study of ***Giardia (Lamblia) intestinalis*** Responsible for intestinal Giardiosis in humans.

Category

Reign of the Protists
Mastigophora Subjunction
Order of Diplomonadida:
Octomitidae family
* Genre *Giardia*
Species *Giardia intestinalis* (= *G. duodenalis*, = *G. lamblia*).

Morphology

2 forms are known:

Trophozoite (vegetative form): This flagellate is piriform "Pear cut in 2" with a very tapered posterior end and measures 10 to 20 long without counting the flagella on 6-10 µm wide and is flattened. It has 8 flagella (6 anterior flagella + 2 posterior flagella), 2 symmetrically arranged nuclei, 2 parabasal bodies (Golgi apparatus), an anterior ventral depression with an adhesive role. He is very mobile. (See **Fig 9 a**)

Cysts: They are very common in the stool of infected subjects, have a thin-shell, clear, smooth, and refractive ovoid shape, the size varies between 8 µm and 14 µm long by 6 to 10 µm wide. At emission it contains 2 nuclei with a flagellar cluster in the axis and two parabasal bodies in comma; after a stay of 24 to 48 hours in nature, the division occurs inside the cyst which then has 4 nuclei, quite resistant in the external environment, where they can live 66 days and, they are the ones who ensure the transmission of the parasite. These cysts die at a temperature of 64°C. (See **Fig.9 b**).

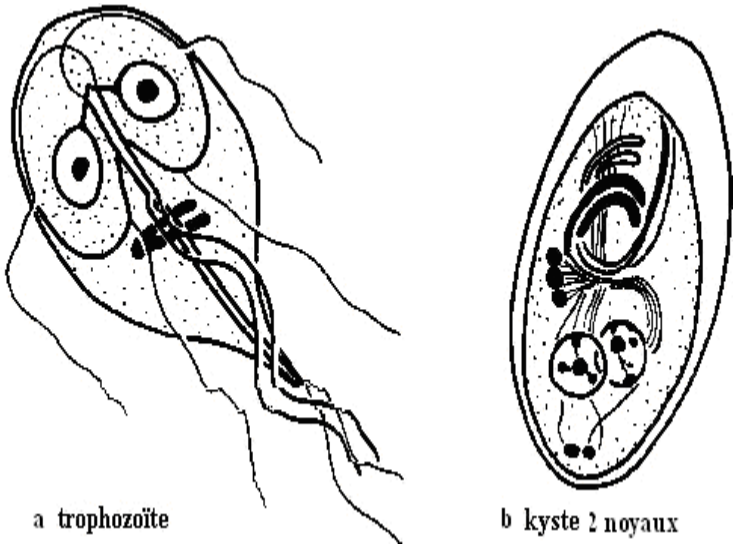
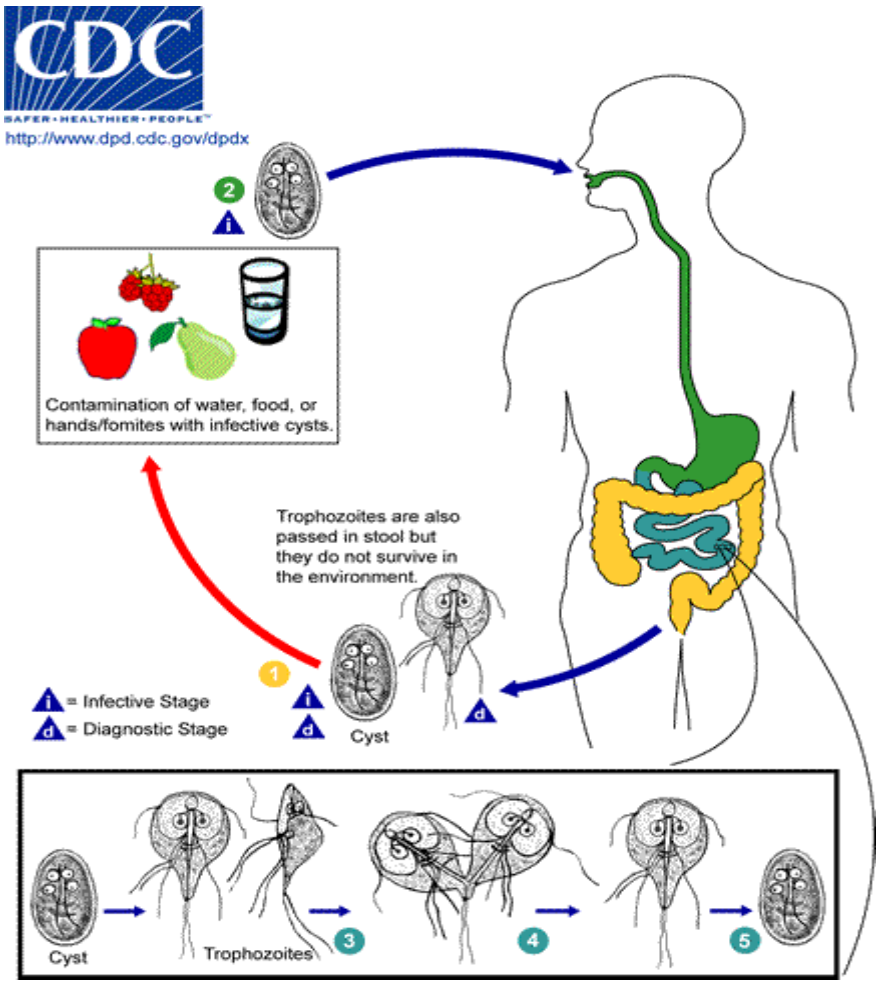


Figure 9: Both forms of *Giardia intestinalis*

Evolutionary cycle (see **Fig. 10**)

The Giradia *intestinalis cycle* is a direct (monoxene) cycle. The definitive host is the man. After ingestion of 4-core cysts spread in nature with food or drinking water soiled by unclean hands. Dékystement takes place in the duodenum. Multiplication by binary division occurs in the light of the hail (duodenum and jejunum) in flagellated form. Flagellate forms actively move in the intestinal mucus and can attach to the surface of epithelial cells. Then there is an irregular formation of cysts (with 2 nuclei) in the lumen of the large intestine which will be passively eliminated with the stool. The maturation of these cysts (with 4 nuclei) takes place in the external environment. These cysts will ensure the transmission of the parasite.

NB: Several authors have drawn attention to the role of the housefly in the spread of the disease.



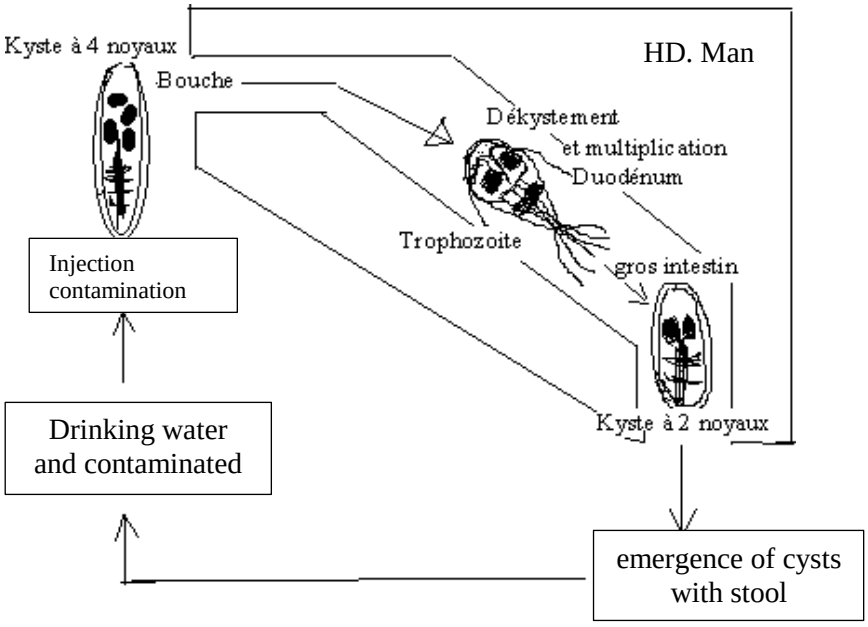


Figure 10: Life Cycle Diagram of *Giradia intestinalis*

Symptomology

He reported that 90% of carriers are healthy. We distinguish between two phases:

Phase d'incubation

For 7 to 10 days there are no symptoms (asymptomatic**)**

Status phase

The main symptom is diarrhea which, in acute cases, is manifested by the emission of 20 to 24 stools per day, with a slight rise in temperature. Most often giardiasis or lambliasis is a chronic disease and only 4 to 6 stools are observed per day with abdominal pain. We more rarely see: asthenia, anorexia, weight loss, nausea, psychic disorders. Intestinal malabsorption is possible in children. Think about giardiosis in a child who has had diarrhea for more than a week. The evolution is most often made by crises. The stools are usually abundant, almost fluid, yellow-brown, alkaline with a lot of mucus and give off a putrid smell.

Giardia can last for years and present periods of lull and relapses. It is very rare to observe symptoms of dysentery if so presence in the intestine of dysenteric amoebae.

Diagnostics

Two types of complementary diagnoses can be used: **Orientation diagnosis****Intestinal**malabsorption,Concept of epidemics Children's communities (nurseries and kindergartens)

Direct diagnosis:

It is done by stool examination. Giardia are found in vegetative form (trophozoites) in diarrheal stools and in cystic form if they are pasty (solid). Repeat the examination several times several days apart in the event of a well-founded suspicion before affirming that there are no parasites and exceptionally: testing for trophozoites at the level of an intestinal biopsy or in the fluid of the duodenal or jejunal tubing (by specialized services)

Processing

Giardia is a disease in most cases benign but some patients must treat themselves for a very long time before obtaining a cure that is sometimes spontaneous.

Uses antiparasitic chemotherapy (e.g. Metronidazole: Flagyl® 7-day cure)

Prophylaxis

- It consists above all in the care of cleanliness (Hygiene): hand washing before touching food, meticulous washing of vegetables consumed raw,
- Sterilization of healthy carriers in the environment to avoid contamination,
- Health education of the population,
- Participation in collective hygiene measures and community awareness,
- Safe soil, safe drinking water,
- Build latrines and wastewater treatment.

c. Trichomonadidae family

Members belonging to this family are characterized by the presence of 3 to 6 flagella, one of which is directed backwards and limits a wide undulating membrane. They have an axostyle that passes through the body and protrudes at the posterior end, and a single nucleus in an anterior position, in its vicinity is the basal body (see **Fig.11**).

The Trichomonadidae *family* contains about fifteen genera including the genus *Trichomonas*.

* Genus Trichomonas

Species belonging to this genus have a piriform body with 3, 4 or 5 anterior flagella and may or may not extend into a posterior flagella, an undulating membrane and an axostyle. They reproduce by binary division. Encystment is very uncommon. Parasites of many vertebrates and invertebrates. They are found mainly in the digestive tract and sometimes in the genitals. Transmission of the parasite to its definitive host takes place sexually, through the mouth, faeces and depending on the species in question.

Trichomonas are widespread parasites and have several species: *Trichomonas caviae* is a parasite of the digestive tract of guinea pigs; in humans (not very pathogenic) *T. vaginalis* (genital and urinary tract), *T. tenax* (mouth), *T. hominis* (intestine), in birds *T. gallinae* (digestive tract, liver).Many *Trichomonas* inhabit the termite digestive tract and are transmitted by cysts.

Parasite Study *Trichomonas vaginalis*

This cosmopolitan parasite is responsible for **genital Trichomoniasis in humans**

Category

Reign of the Protists
Sarcomastigophora branch Under *Mastigophora* branch *Zoomastigophora* class
Order of *Trichomonada*
Family of *Trichomonads*
Genus *Trichos*
Species ***Trichomonas vaginalis***

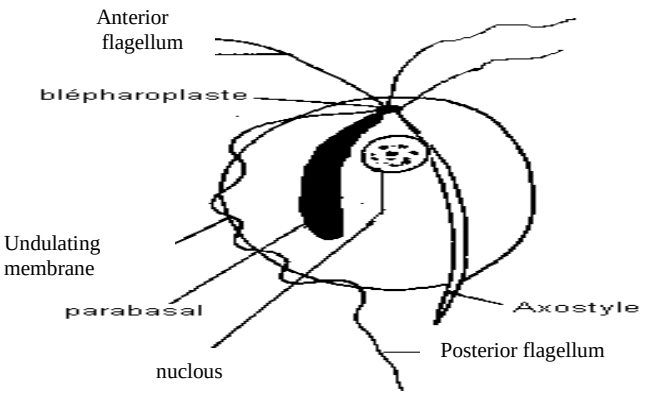


Figure 11: Species *Trichomonas vaginalis*

Morphology (Fig.11)

The shape is very variable in live animals (trophozoite): ovoid to spherical shape from 7 to 30 µm long by 5 to 12 µm wide. There are generally 4 anterior flagella and an axonema (posterior flagella attached to the body of the animal) limiting a fairly short undulating membrane, an axostyle projecting into the posterior part of the body, an oval nucleus, visible after coloration. The division is made by scissiparity, there is neither sexual reproduction nor encystment.

Figure 10: Trichomonas vaginalis: Typical form representing a trophozoite.

Evolutionary cycle (see Fig.4)

It is a direct cycle. The definitive host is the male (female in the majority of cases). The reservoir of the parasite is the human species and the direct transmission through sexual contact most often and rarely through underwear.

It is a parasite that is usually located in the vagina (vaginal mucus), sometimes urethra, the bladder. The resistance of trophozoite is very low in the external environment so rarely an extra-body phase.

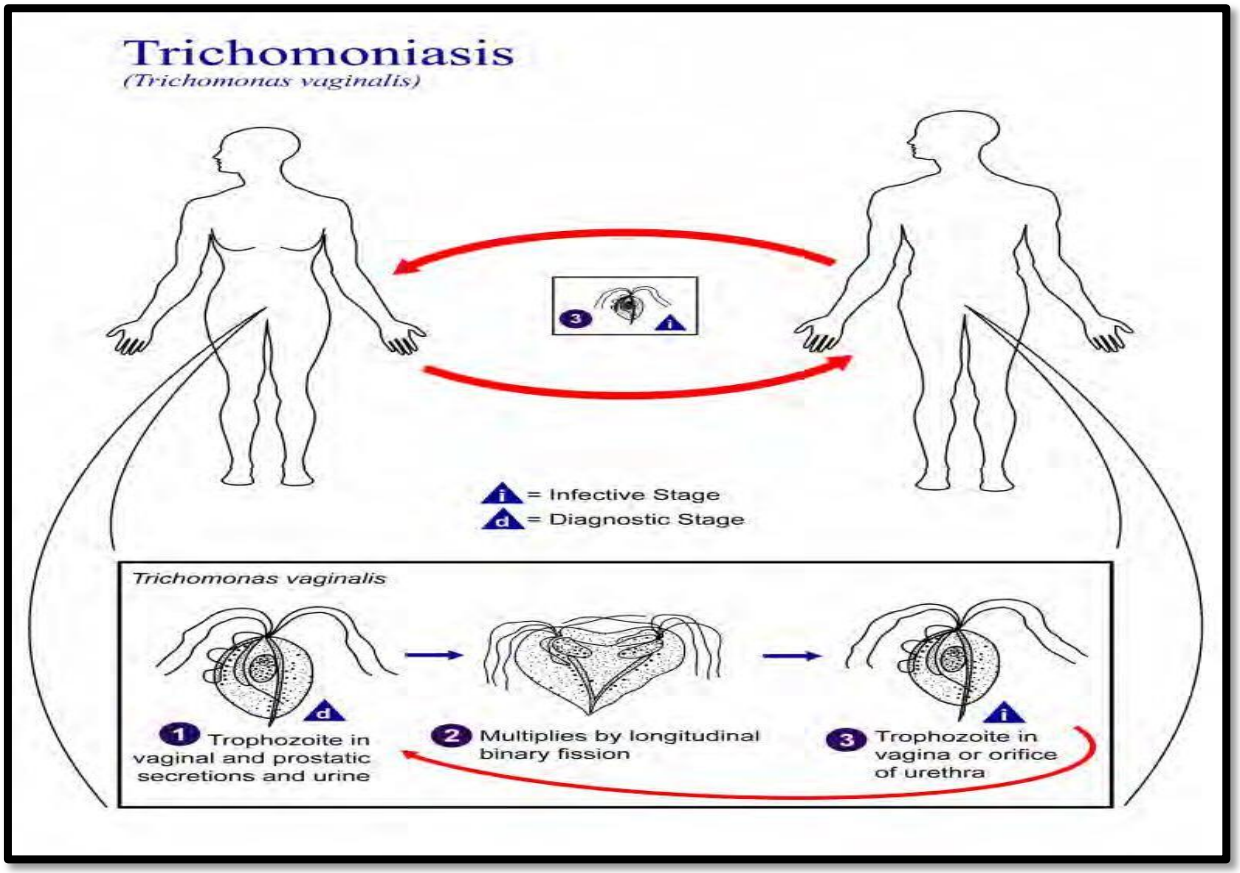


Figure 12: Life Cycle Diagram of *Trichomonas vaginalis*

Pathology

It does not appear to be pathogenic. It is frequently observed in women with conditions of the genital tract, especially in cases of vaginitis when the vaginal mucus is acidic (from pH 4-5 to pH 5-6).

Subacute vaginitis: inflammation of the edematous and sensitive vaginal mucosa, signs: vulvar pruritus, abundant leucorrhoea (whitish, foamy or mucous, sometimes greenish). In the case of acute vaginitis: sensation of vulvar burns, dyspareunia (painful sex), abundant leukorrhea. This same organism has been encountered in men where it has a localization in the urethra, prostate and seminal vesicles. The living conditions of the parasite are less favourable in humans because some of the parasites are eliminated with each urination. Most often it is a healthy (asymptomatic) carrier or: subacute urethritis: mucopurulent discharge, itching or even burning sensation when urinating. Rare complications: cystitis and prostatitis.

Diagnosis Direct

Diagnosis

Precautions must be taken during sampling due to the fragility of the parasite and its sensitivity to cold and desiccation.

*in women: the vaginal sample is taken after the installation of a speculum without lubricant. It is advisable to use curettes or pasteur pipettes (preferably do not use swabs).

*in humans: the collection is done in the morning, before the first stream of urine, it is advisable to collect the secretions using a sterile loop, possibly it is necessary to perform a prostatic massage beforehand. The parasite can be found in the centrifugation pellet of the first morning urine, but it is quickly destroyed there.

Processing

Trichomoniasis is a sexually transmitted disease, to avoid recontamination, all partners must be treated simultaneously and sexual intercourse must be avoided or protected during treatment.

Parasitic chemotherapy: Metronidazole (Flagyl®) for 10 days. In women, it should be combined with oral treatment of metronidazole eggs (Flagyl®) for 10 days

Prophylaxis

Individual prophylaxis

Sexual intercourse must be protected. It is necessary to treat if a partner has trichomoniasis.

2.2.2. Subphylum *Sarcodina* (e.g. Rhizopods)

They are microscopic unicellular beings, devoid of envelope and whose body constantly changes shape as a result of the emission or retraction of pseudopods that play a role in the movement and gripping of food. The cytoplasm is differentiated into ectoplasm and endoplasm. The nucleus is unique in the majority of rhizopods. Almost all rhizopods lead a free life very few are parasites. Multiplication is done by binary division. The rhizopods encyst themselves. After encystment the nucleus of the organism then divides into several nuclei and the multinucleate cytoplasm in turn divides thus giving rise to small organisms, resembling the individuals from which they derive. The *Sarcodina* , of which the *Rhizopoda* class is a member. The latter includes several orders including the order of *Eumoebida* which are characterized by the presence of a single nucleus, sometimes two or more. Individuals belonging to the Amoebidae family *have* a single nucleus. This family contains a large number of free forms and a number of parasitic forms, most of which are found in the intestines of humans and animals. It includes several genres including the *Entamoeba genre.*

* Gender ***Entamoeba***

Members of this genus have a cytoplasm differentiated into ectoplasm and endoplasm and a nucleus. Pseudopods are usually lobed. They multiply by simple binary division and form cysts. All representatives of this genus are parasites of humans or animals. It includes several species including:

E. histolytica (the only human pathogen)
E. coli (human large intestine lumen)
E. gingivalis (human and horse dental tartar)
E. bovis (beef stomach)
E. equi (horse intestine in South Africa)
E. caprae (goat intestine in South Africa).

* **Study of the parasite *Entamoeba histolytica***

Causes amoebiasis disease (intestinal and hepatic) in humans.

Category

Sarcomastigophora branch
Under Branch of Sarcodina (or Rhizopods)
Superclass of Rhizopoda
Class of Lobosea Order
of Amoebida Family of
Euamoebidae Genus
Entamoeba
Species *Entamoeba histolytica*

Morphology

It takes the following forms: (See Fig.13 a,b,c) Vegetative form (Fig. 13a

It is the pathogenic form or histolytica (*E. histolytica histolytica*), with a diameter of 20 to 40 µm. This is the form of multiplication and is diform. She is hematophagous.

Pre-cystic form: (Fig. 13b

Under various influences, the hematophagous form or histolytica gives smaller amoebas or minuta forms (E. histolytica minuta) which originate may be the large forms. These are mobile, non-haematophagous, non-pathogenic forms. These pre-cystic forms can be 10-15 µm in diameter. They have the same structure as large shapes but their cytoplasm is devoid of food vacuoles and erythrocytes (red blood cells). The karyosome in the nucleus is central. Shortly before encystment, a glycogen vacuole develops in the amoeba.

Encysted form: (Fig. 13c

The cyst that derives from the pre-cystic form has a spherical shape and a diameter of 10 to 14 µm. The nucleus of the cyst divides to result in a cyst with 4 nuclei (mature cyst). It is the contaminating (dissemination) and resistant form. These cysts would only be infectious for 5-6 days only.

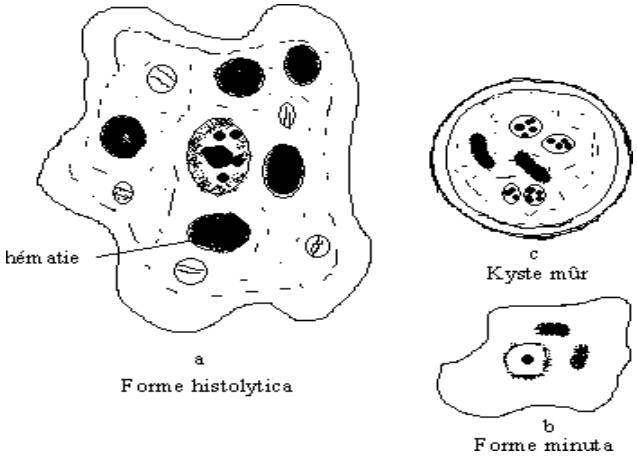


Figure 13: Morphologic forms of*E. histolytica*

Evolutionary cycle (see **Fig. 10**)

It is a direct cycle (monoxene).The definitive host is the man.

Human infestation is by ingestion of cysts either directly from contaminated stool or indirectly via contaminated food or water. Infectious cysts (with 4 nuclei) ingested by a healthy individual, under the action of pancreatic juice the cysts tear and release quadrinucleated amoebas (metacyclic amoeba). The four nuclei of the metacyclic amoeba each divide into two, resulting in 8 nuclei and the amoeba divides itself into 8 small amoebae that grow a little, resulting in 8 minuta-type amoebae, which multiply in the intestinal lumen. These minuta shapes can follow one of the following two destinies:

- either lead a saprophytic life while remaining mobile or become encysted and travel with the stool in the form of cysts, the latter pathway is called "Amoebiasis infestation". Cycle

can therefore be complete without going through the *histolytica* form: this is the **normal or non-pathogenic cycle**.

➢ Either under still poorly known influences, minuta-form amoebae grow, become more active and haematophagous and turn into pathogenic histolytica-form *amoebae.* These forms penetrate the intestinal mucosa and determine ulcerations, this pathway is called "Amebiasis disease". During amoebiasis disease, the amoeba *(E. histolytica histolytica)*either stays at the level of the colon (intestinal amoebiasis) or invades other organs like the liver (liver amoebiasis). This is the **abnormal or pathogenic cycle**. This transformation (minuta into histolytica) is especially common in countries with a hot climate.

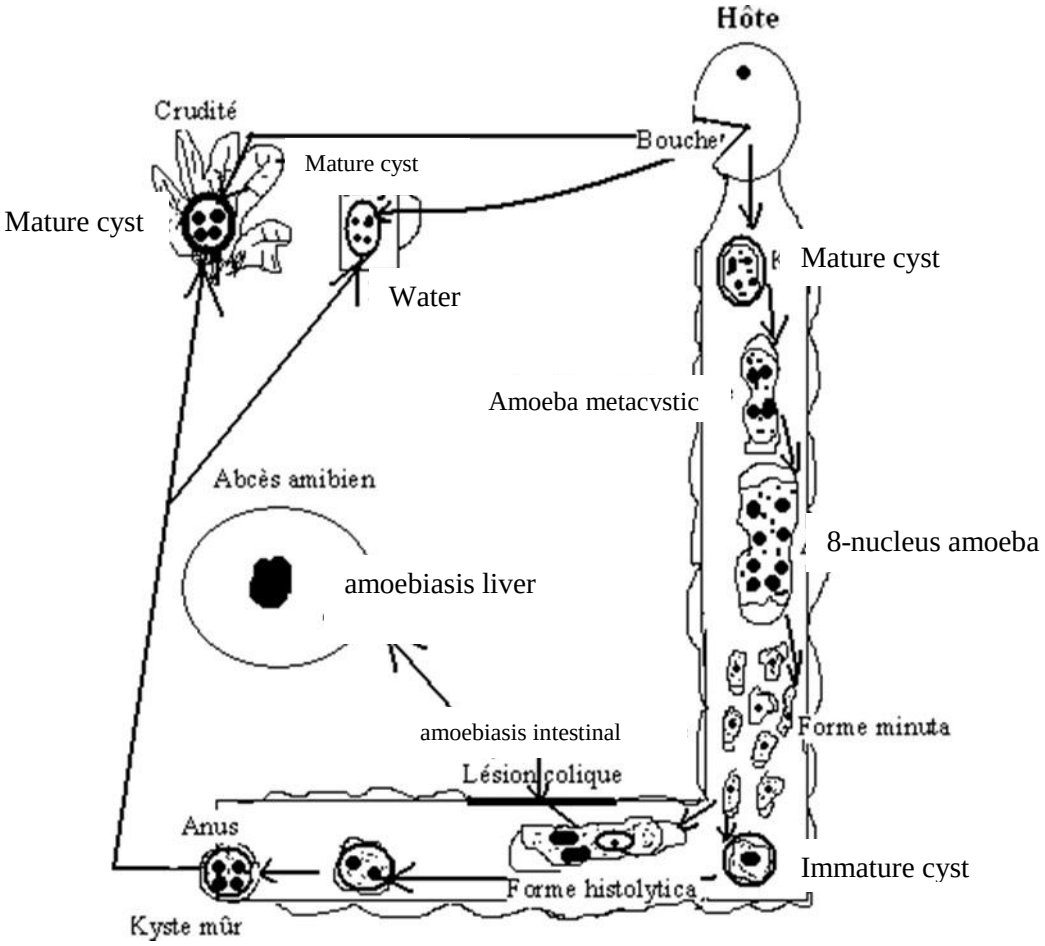


Figure 14: Evolutionary cycle of Entamoeba histolytica

Location

The hematophagous form lives in the lumen of the large intestine, in the intestinal wall, in the ulceration that it determines at the level of the large intestine. This form can pass into the blood and lymph vessels and be found in the **liver**, lungs, brain, spleen.

The minuta form is only found in the lumen of the large intestine.

Epidemiology

Amebiasis is a digestive parasitosis linked to **human fecal risk** (fecal pollution of the environment by human excreta contaminating water, raw vegetables, hands, soil. On

transport is done by flies.

The tank of the parasite **is man.** The cyst survives for at least 15 days in water at 18°C, 10 days in the stool, 24 hours dry. It is resistant to chemical agents. Vegetative forms are too fragile to survive in the wild and play an epidemiological role. Amoebas are cosmopolitan, but parasitosis develops particularly in hot and humid regions and is endemic in hot and humid countries (especially third world countries where fecal hygiene is poorly respected and where human waste serves as fertilizer). The global prevalence is about 10% or 600 million carriers, 90% of which are healthy carriers responsible for transmission.

Symptom

A number of individuals harbor dysenteric amoeba without presenting any symptoms, these are healthy carriers, but it is sufficient from any cause that the disease occurs.

As we have mentioned there may be several locations of*Entamoeba histolyticahistolytica.* Basically, there is **a distinction between intestinal amebiasis and hepatic amebiasis**.

AMEBIC DYSENTERY

The length of the incubation period is difficult to determine. The first symptom is common diarrhea, with alternating diarrheal and pasty stools, which gradually change in character and become liquid and **bloody**. In mild cases the number of stools per day varies between 4 and 5; in severe cases it varies between 20 and 30, but it is always less than in bacillary dysentery.

The patient presents in addition to digestive disorders, experiences pain at the time of defecation, is tired at the slightest effort, drowsiness especially after meals, and slims. It presents with colic pain sitting along the large intestine, complicated by very painful episodes (colic pain ending in a desire to go quickly to the stool) and tenesmus (painful contracture of the anal sphincter). There is no fever. Health in general remains good but the individual is a bit anemic.

Note that many complications can recur during intestinal amebiasis such as: rupture of a blood vessel, tissue necrosis. The most common complication is liver abscess (liver amebiasis). Other extra-digestive locations can be seen after intestinal amebiasis due to *E. histolytica*, they are pulmonary pleuro or cerebral.

ALA - Amebic liver abscess

Hepatic amoebiasis (hepatic amoebosis) is the most common visceral form. About three months after an amoebic infestation (more or less unnoticed in the case of an attenuated form), liver damage is first expressed by painful hepatomegaly and moderate fever. Then, in the event of the formation of a liver abscess, we see a polynuclear neutrophil hyperleukocytosis, an oscillating fever, an alteration in the general condition, and an inflammatory syndrome.

Diagnosis Intestinal amebiasis

Diagnosis is relatively easy during the acute period of the disease: look for hematophagous forms and immature cysts (1 to 2 nuclei) in the diarrheal stool of the

sick or wall cysts (4 nuclei) if the stool is pasty.

Conditions to be taken for a good diagnosis :

The direct diagnosis during intestinal amebiasis is made on recently emitted stool (The patient comes to defecate in the laboratory), if there is difficulty transporting the stool in the MIF (Merthiolate Iodine Formol) fixing medium. This examination must be repeated three times in order to get rid of the so-called "silent" periods during which the parasite is not emitted.

ALA - Amebic liver abscess

The search for amoebas in the stool is most often negative during visceral (e.g. hepatic) amebiasis. The biological diagnosis is then serological (ELISA, immunofluorescence, indirect haemagglutination, immunoelectrophoresis).

Processing

Amoebic dysentery is a relatively serious disease in hot and poor countries where mortality is quite high.

Intestinal amebiasis and visceral amebiasis should be systematically treated with a tissue amoebicide (FLAGYL® (Metronidazole) 2g daily for 7 days). This treatment should always be followed by treatment with a contact amoebicide (used orally to act on the minuta forms), in order to prevent distant relapses.

Medication is not everything in the treatment of intestinal amebiasis, it is necessary to keep the patient at rest,calm his pain and institute an appropriate diet.

Prophylaxis

There is no antiamoebic drug prophylaxis, prevention is based on appropriate hygiene measures, since amoebic dysentery is contacted by ingestion of cysts:

* Individual prophylaxis consists of :

Avoid ingesting cysts: drinking water is boiled, vegetables eaten raw will be carefully washed, food will be protected from insects (especially flies), wash hands before each meal, avoid direct or indirect contact with patients or amoeba carriers

* General prophylaxis consists of:

- Health education: treating patients, screening and also treating healthy carriers;
- Fecal hygiene: disinfect their stools and build latrines; Local antiseptics (iodine,chlorine) have only a partial action on amoeba cysts;
- The isolation of patients and all objects that have been in contact with them, must be disinfected;
- Prohibit amoeba carriers from all professions where humans handle food materials (especially cooking and baking).

2.3. Phylum *Apicomplexa* (e.g. sporozoa)

2.3.1. Overview

Apicomplexa (or apicomplexans) are a group of about 5,000 species that are all intracellular parasites of other eukaryotes. Each species has its own cycle, most often with several successive hosts. They have different morphologies that can be variable depending on the stage of the cycle. The infectious cell is called sporozoite.

In adulthood, they are immobile and never present pseudopods. Very frequently, they are intracellular parasites of vertebrate or invertebrate hosts. They present a characteristic cycle in three phases: **Schizogony**, **Gamogony** and **Sporogony**. Gametes are often flagellated. Morphologically, sporozoa are organisms of generally ovoid or rounded shape. The *Apicomplexa* phylum includes the class Sporozoea, which includes the order Eucoccidida (*Eucoccidiorida*) which are small protozoa. Depending on the species, they are parasites of Invertebrates or Vertebrates. They have anisogamy **:** their gametes are different and their cycle has the three typical phases. The limbs are characterized by specific parasitism, and coccidia are intracellular parasites (in the cytoplasm or in the nucleus).

It is in this group of organisms that the most devastating human parasites are found. These include *Plasmodium spp* (responsible for malaria = malaria), *Toxoplasma gondii* (responsible for toxoplasmosis) and *Cryptosporidium* (which affects AIDS patients), etc. Animals are no better off with coccidia (*Emeria* and *Theileria* which can lead to the loss of livestock in Africa), *Babesia* which affects livestock and chickens or gregarines which affect insects.

This order is divided into two suborders:

* ***Eimeriorina*** **suborder**: the evolution of macrogamonts and microgamonts is independent. We distinguish between monoxene coccidia (genus *Eimeria*) and tenene coccidia(genus Toxoplasma).
* ***Haemosporina*** **suborder**: These heteroxene Coccidia are bloodthirsty: they are **haemosporidia**. An example is *Plasmodium falciparum*, an agent of malaria. It requires two different hosts, the Human and a Bloodsucking Insect, *Anopheles*. The *Plasmodium* cycle comprises a sexual phase that takes place in the vector insect, the female anopheles, and an asexual phase observed in the vertebrate intermediate host

2.3.2. Coccidia subclass

These sporozoa are intracellular parasites throughout their growth period. Asexual reproduction occurs by repeated schizogony. Sexual reproduction or gamogony, sporogony occurs by means of male gametocytes (microgametocyte) and female gametocytes (macrogametocyte). The method of fertilization is called **anisogamy**

a**. Order Eucoccidida (*Eucoccidiorida*)**

a. **1. Under order *Eimeriorina***

*** Genus *Eimeria***

Category

Reign of the Protists (Protozoa) Branch of the
Apicomplexa (Sporozoa) Class of the *Sporozoea*
Subclass *Coccidia* Order
Eucoccidiorida Suborder
Eimeriorina Family
Eimeriina *Genus*
Eimeria
Species *Eimeria spp*

Coccidiosis disease

Includes several species of mainly veterinary interest (coccidiosis of domestic birds, rabbits). The species are characterized by the presence of 4 sporocysts and each sporocyst contains 2sporozoites.

The evolutionary cycle (See **Fig. 18**) Monoxene Coccidia

Monoxenic Coccidia, like *Emeria perforans*, live in the rabbit's intestinal epithelium. Reproduction can be asexual (schizogony**) or sexual (**gamogony**) and has schizogony**

* Asexual multiplication (**schizogony**) : this is a typical:

The vermicular sporozoite, coming out of the spore that has opened in the digestive tract, enters an epithelial cell and transforms into a small amoeba, the **oviform corpuscle** , which grows rapidly and causes the gradual degeneration of the host cell. At the end of its growth, it is stuffed with paraglycogen. The Coccidia divides its nucleus three to four times in a row; then it cuts its cytoplasm into meridian slices comparable to orange quarters, in each of which a nucleus passes: there are as many slices as schizozoites that fall into the intestinal lumen at the same time as the degenerated cell and penetrate into the unharmed cells. This phase lasts about 48 hours and can be repeated indefinitely.

* Sexual multiplication (**gamogony**)

Emeria perforans also presents a sexual reproduction that appears after some schizogonia. The female gamont resembles a large vegetative individual particularly loaded with reserves, including paraglycogen, lipids and metachromatin. It transforms directly into a female gamete. The male gamont undergoes a large number of nuclear divisions. The small nuclei are carried to its periphery, each of them lifts a droplet of cytoplasm: this is the **beading**stage. The male gametes finally break away from the voluminous cytoplasmic residue. A male gamete enters the female gamete. As soon as fertilization is completed, the copula surrounds itself with a thick resistant membrane and becomes the**Oocyst** which is dragged outside with the excrement. To continue its evolution, it needs oxygen and a temperature below 37°C. Its cytoplasmic mass shrinks and its nucleus undergoes two consecutive divisions. Four small cells isolate, each wrapped in a resistant shell and become a spore to

inside of which two sporozoites are individualized. The mature oocyst is infesting: ingested by the rabbit, it will open as well as the spores and a new cycle will begin with the released sporozoites.

It should also be noted that the Rabbit hosts other Coccidias, in particular *Eimeria stiedae* which parasitizes the epithelium of the bile ducts.

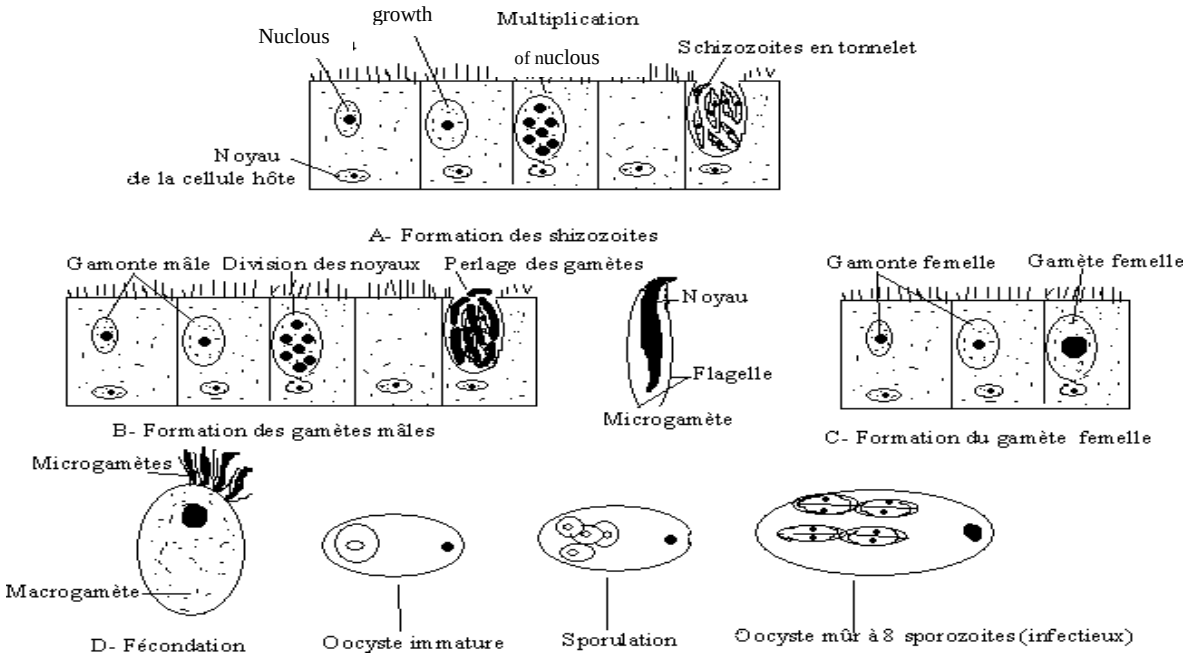


Figure 18 : The different stages of development of the genus *Eimeria*

* Gender ***Toxoplasma***

Species belonging to this genus are characterized by bisporous oocysts where each spore contains four sporozoites.

Toxoplasma gondii study

Is the agent of **Toxoplasmosis** in both its **acquired and congenital forms.**

Category

Reign of the Protists (Protozoa) Branch of the
Apicomplexa (Sporozoa) Class of the *Sporozoea*
Subclass *Coccidia* Order
Eucoccidiorida Suborder
Eimeriorina Family
Sarcocystidae Genus
Toxoplasma
Species *Toxoplasma gondii*

Morphology

Toxoplasma gondii can come in 3 forms:

- Vegetative form (**Tachyzoite) : (**See **Fig. 19)**

It can be found at the intermediary host. They are piriform organisms (*Toxoplasma*form), arched, with a tapered end. It measures 5 to 10 µm long by 3 to 4 µm. It is endocellular in the macrophages (reticulohistiocyte system) that it burst. Rapid multiplication by repeated binary division (by endodyogeny).

- The Bradyzoite **or** Cystozoite **form:**

It is found in the intermediate host, which is similar in structure to the tachyzoite. Its multiplication is slow and is found in a nerve or muscle cell, the retina which will evolve into a cyst (pseudocyst) whose size can reach 100 µm and can contain 2 to 3000 bradyzoites. Bradyzoites are more resistant than tachyzoites.

- The Oocystic **or** Sexual **form**:

This is the result of sexual reproduction in cats (definitive host). The oocyst has an ovoid shape (15 by 10 µm). Maturation (sporulation) takes place in the external environment (in the soil). The mature oocyst contains 2 sporocysts containing 4 sporozoites each. Sporozoite resembles autachyzoite. The oocyst is very resistant on the ground and is resistant to HCl.

Lifecycle (See **Fig. 20**)

The life cycle can be monoxene or heteroxene. Cat *Toxoplasma gondii* is an optional heteroxene.

H.D : It is a felid (e.g. **Cat**) and **HI** : is a **warm-blooded vertebrate** (mammal or bird),in which it determines **Toxoplasmosis**

In the intestine of the Cat, it completes its complete cycle with classic schizogony followed by sexual reproduction with microgamete and macrogamete. The cat expels with its feces the non-sporulated oocysts, the formation of spores and sporozoites takes place in the external environment. Lesoocysts (bisporated each spore contains 4 sporozoites) that the swallowed Cat arrives in the intestine of the latter and are at the origin of a new infestation with first asexual multiplication, then sexual. **This is the banal cycle** (direct or short).

If the oocysts are ingested by a host of a different species, Man, Beef, Mouse, Bird the sporozoites pass into the blood and invade various organs such as the brain, peritoneum, liver, muscles... They reproduce only and indefinitely by schizogony: This is the indirect or long cycle). In pregnant women, schizozoites in varying numbers infect the fetus, causing more or less serious lesions: at various times during pregnancy, spontaneous abortion can occur. If the infection is not massive, the fetus continues its development but the child will suffer from disorders, some of which will concern the brain, the retina.

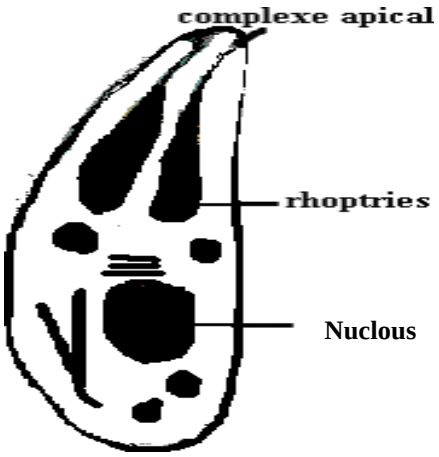


Figure 19 : Diagram of a Tachyzoite (The vegetative form of the parasite *T. gondii*)

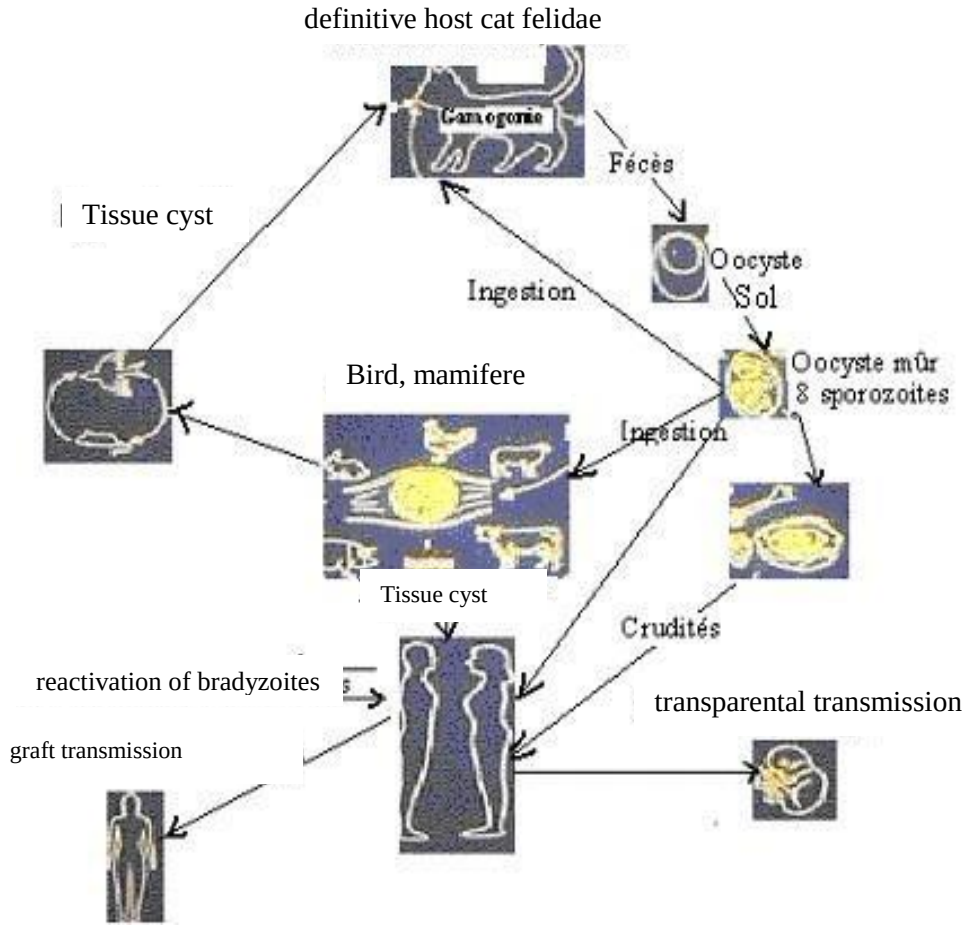


Figure 20 : *Toxoplasma gondii* life cycle

Epidemiology

- Toxoplasmosis is a cosmopolitan zoonosis. The main sources of human contamination are either:
- **by ingestion of telluric wall oocysts** (water, food, hands): 2% of cats sow oocysts, for 1 to 3 weeks (especially young cats). Sporulation conditions: 1 to 5 days if the temperature is 20°C in the presence of oxygen. They withstand several months on the ground, and are killed by heat, desiccation and freezing.
- by **ingesting live cysts** in raw or undercooked meat. Prevalence for beef: 80% of parasitized adult sheep and goats, cyst survival: several days at room temperature, several months at + 4°C. They are destroyed in 15 mm at 56°C and in 24 hours at - 20°C
- by transplacental passage: in non-immune pregnant women
- by the organ transplant

<u>Prevalence of toxoplasmosis in humans:</u>

In "immune" adults: depending on geographical location, customs, and climate

this prevalence varies between 20 and 70%. In HIV-negative pregnant women: In France about 7 per 1000 and the seroconversion rate during pregnancy is 0.5 to 1.5 per 1000 births.

Symptom

Toxoplasma gondii is responsible for toxoplasmosis. This parasite has a very broad host spectrum and infects all mammalian species (some say all nucleated cells?!?). However, it is established that the **cat is the definitive host** where the **sexual phase**occurs. Transmission is most often through food (made with affected animals) but can also be through contact with soiled materials that have been in contact with cat feces.

- In humans, the infection most often leads to an asymptomatic or low amplitude condition (low fever, headache...): **Toxoplasmosis acquired from the immunocompetent**
- but in fragile subjects (especially AIDS patients), the parasite can infect all types of cells and can cause hepatitis, blindness, neurological disorders **: Toxoplasmosis of the immunocompromised**.
- In pregnant women, transmission of the parasite to the fetus through the placenta causes spontaneous abortions or very significant mental or physical delays (hence the advice to avoid cats during pregnancy; about 5 pregnant women out of 1000 have toxoplasmosis during pregnancy and only 10% have abortions and/or affected children): **Congenital toxoplasmosis.**

Noting that the risk of fetal contamination is low in early pregnancy (before the 4 th month), but if there is a transplacental passage the risk of congenital toxoplasmosis will be serious (they can lead to death *in utero*, or in the months following birth, or lead to serious psychomotor delays, changes in the appearance and volume of the skull: hyroencephaly, ocular disorders: chorioretinitis). The risk of fetal contamination is greater at the end of pregnancy (it increases from the 4 th month to the 9 th month), in this case congenital toxoplasmosis will often be benign (recognized at the birth of the child with: ocular forms: chorio-pigmentary retinitis, neurological forms: seizures, psychomotor retardation, too rapid increase in the cranial perimeter) or latent (80% of cases: the child is free at birth but it carries IgM, it may later declare toxoplasmosis; it will most often be ocular lesions appearing after a few years).

It is a very common disease because anti-toxoplasma antibodies are detected in about 50% of the population of the United States, up to 90% in other populations. The parasite may be dormant and re-emerge during passage of immune weakness**.**

Diagnostics

The search for tachyzoites would only be possible during the sepsis phase of the disease, their rarity makes their detection unlikely, hence the need to detect persistent serum antibodies, which will reveal the subject's state of "immunity" to toxoplasma. So,

It is important to know the kinetics of the specific immune response IgM, IgG and

IgE are the first antibodies synthesized after contamination, their detection usually indicates toxoplasmosis at its onset; but their persistence varies between subjects

: **IgM** appear one week after contamination, their level increases for about 1 to 2 months, and are detectable at most for 1 year,

IgA have a parallel evolution to that of IgM but are detectable for a maximum of 6 months (it is exceptional to detect them later). Undetectable in 6% of the population. In addition to their reduced time of presence there seems to be no natural non-specific antibodies, which makes them useful in the diagnosis of congenital toxoplasmosis.

IgE have a parallel evolution to IgA, their kinetics are very fast during the evolutionary phase of the disease, they are never detected in old "immunities".

IgG, the kinetics are different depending on the antigens that cause them and therefore the serological techniques used, but anti-antigen membrane (or surface) antibodies are the earliest they appear 1 to 2 weeks after contamination, their maximum is recorded around 2 months and can persist up to 6 months then slow decrease.

Negative antenatal and neonatal assessments require specific monthly IgM and IgA serological monitoring and IgG: IgG synthesis delayed by several months may be the first and only biological sign of congenital infection

Processing

In the case of Toxoplasmosis acquired during pregnancy - Practical attitude -

Spiramycin, until delivery if amniocentesis is negative Ultrasound monitoring,

Fetal infection demonstrated: replacement of spiramycin (Rovamycin) with pyrimethamine (**teratogenic**(Malocide) and sulfonamides, until delivery, if there are ultrasound abnormalities: indication of termination of pregnancy **to be discussed**

Antenatal

As soon as maternal infection is proven: Rovamycin: 9 million IU (3 g) / day, until delivery, if the antenatal diagnosis (amniocentesis) is negative.

As soon as fetal infection is demonstrated: discontinuation of Rovamycin which is replaced by Malocide:

50 mg / day, Adiazine: 3 g / day, Lederfoline or Osfolate: 50 mg / week, until delivery.

Prophylaxis

The prevention of acquired toxoplasmosis consists in avoiding the consumption of undercooked meat that may contain cysts and contact with the definitive host "e.g. cat" (possible carrier of oocysts).

Prevention of congenital toxoplasmosis consists of serological testing for specific antibodies in pregnant women from the beginning of growth and during the 3 trimesters of this pregnancy. Here we will give some hygieno-dietary advice to an HIV-negative woman (if the test for IgG and IgM is negative, the patient is at risk) so it is necessary to:

- Avoid the consumption of raw or bleeding meat; prefer very cooked or pre-frozen meat,
- Thoroughly wash fruits, vegetables and aromatic plants as well as

utensils and surfaces used for the preparation of meals,

- Always wash your hands before and after handling food.
- Regularly clean the refrigerator,

Category

Branch of Apicomplexa (Sporozoa) Class of
Sporozoea
Subclass of Coccidia Order of
Eucoccidiorida Suborder of
Haemosporina Family of
Plasmodiidae Genus
Plasmodium
Species Plasmodium spp
In humans Plasmodium vivax Plasmodium falciparum Plasmodium ovale Plasmodium malariae
Causes the disease known as Malaria= malaria

Malaria is a parasitosis caused by haematozoans of the genus Plasmodium, transmitted by haematophagous mosquitoes of the genus Anopheles (female).In 2005, malaria remained the world's leading endemic (almost half of the world's population lives in endemic areas). Each year, there are between 300 and 400 million malaria cases and 1 to 3 million deaths each year, the majority of which are children from sub-Saharan Africa, placing a financial burden on these countries.

For these reasons, the fight against malaria, along with AIDS and tuberculosis, is one of the Millennium Goals defined by the United Nations, and the Global Fund is intended to equip these countries with medicines.

In vertebrate animals Plasmodium gallinaceum(Chicken) Plasmodium caprae(Goat)

Morphology (See **Fig. 22)**

The 3 invasive forms of *P. falciparum* : Merozoite, an invasive form of *Plasmodium* for red blood cells,sporozoitis (hepatocytes) and ookynetus (the intestinal epithelium in invertebrates)

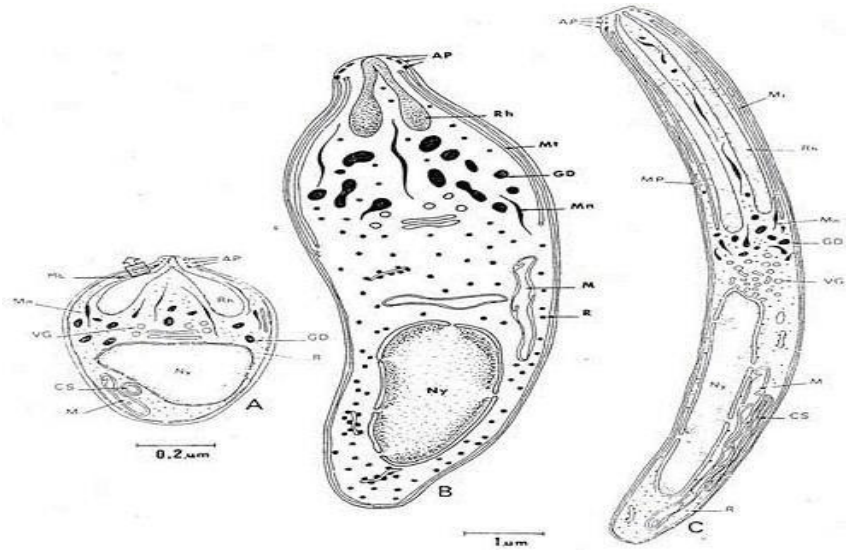


Figure 22: *Plasmodium* invasion stages**A:** Merozoite; **B:** sporozoite; **C:** ookinete

- When eating outside the home, avoid raw vegetables and prefer cooked vegetables,
- Wear gloves when gardening and wash your hands after handling dirt,
- Have another person clean the cat's litter box every day (or wear gloves),and

do not give it raw meat.

a. 2. Under the order of ***Haemosporina***

Microgametes are born abruptly from microgametocytes in the form of few flagella. The zygote or ookynet is mobile and becomes a thin membrane oocyst increasing in volume and leaking infectious sporozoites. The life cycle involves two successive hosts: the asexual cycle (schizogony) in a vertebrate; the sexual cycle (sporogony) in an invertebrate. The diagram of the evolution of hemosporidia is given in **Figure 21**. There is no free destade in nature. The schizogonic cycle in vertebrate red blood cells. It contains a single genus: the *Plasmodium*genus.

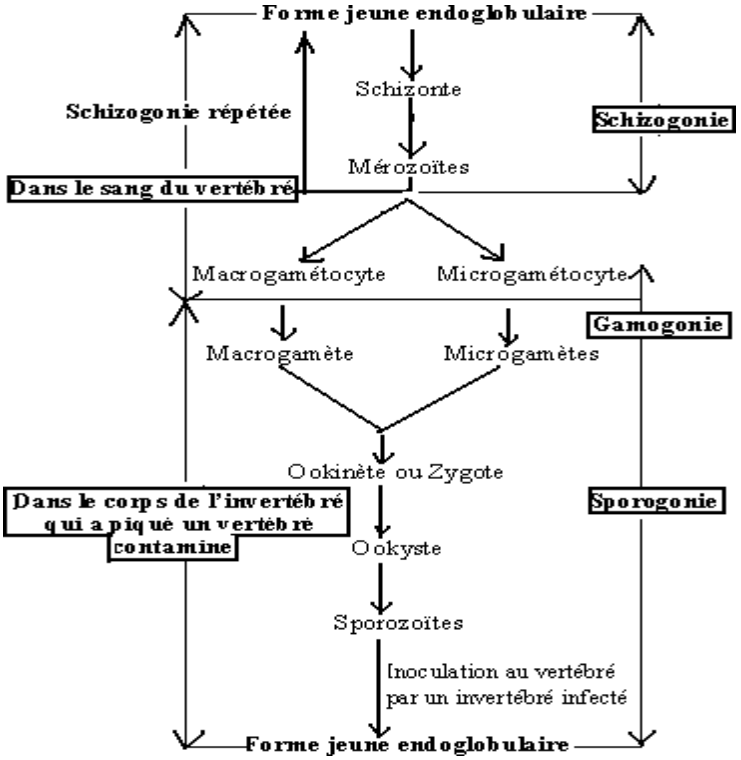


Figure 21: Diagram of the Evolution of Hemosporidia

Genus ***Plasmodium***

The cytoplasm contains grains of black or brown pigment (melanin or hemozony). The gametes are spherical or banana-shaped. They are parasites of human red blood cells and various vertebrates including birds. ***Plasmodium*life cycle**

It is a heteroxene (indirect) ring.
The *Plasmodium* cycle comprises a sexual phase that takes place in the vector insect, the female anopheles, and an asexual phase observed in the vertebrate intermediate host

*** The asexual phase**

It is characterized by the multiplication of the parasite by schizogony. There are two types of schizogony:

- exo-erythrocytic schizogony: during a bite by the contaminated mosquito, sporozoites, infectious cells characteristic of all sporozoa (Gregarines, Coccidia, Hematozoa) are injected into the bloodstream. Within a few tens of minutes they enter the hepatocytes where they transform into vegetative cells called trophozoites. Thanks to repetitive mitoses and the differentiation of new infectious single-cell germs or exo-erythrocytic merozoites, the number of parasites increases rapidly. Hepatocyte lysis releases several thousand merozoites (10,000 in *P. vivax*, 40,000 to 50,000 in *P. falciparum*, more than 15,000 in *P. ovale* and 7,500 in *P. malariae*) which will migrate via the blood pathway to red blood cells.

In some species, such as *P. vivax* and P. *ovale* in *humans or P.* cynomolgy in monkeys, sporozoites may remain dormant in hepatocytes in the form of hypnozoites and develop only later (revival periods). Exo-erythrocytic schizogonia that lasts 6 to 9 days in *P. falciparum* and 15 days in *P. malariae* is often asymptomatic.

- endo-erythrocytic schizogony: exo-erythrocytic merozoites released from hepatocytes enter the erythrocytes by a complex mechanism (See **Fig. 24**), where they evolve in the young trophozoite stage (" ring "), mature trophozoite then schizont (form of multiplication and maturation). The rupture of the red blood cell membrane by the mature schizont (rosette stage) releases the merozoites which will invade healthy red blood cells and initiate a new cycle of intracellular development or differentiate into male and female gametocytes.

The endo-erythrocyte cycle lasts 48 hours in *P. vivax*, *P. ovale* and *P. falciparum* and 72 hours in *P. malariae*. The pathological signs of malaria are generally detected during endo-erythrocytic schizogony: the bursting of red blood cells containing the mature schizont forms releases, in addition to the merozoites, haemozoin (a malarial pigment resulting from the degradation of haemoglobin) and toxic substances that induce febrile attacks characteristic of the disease.

***The sexual phase**

Gametocytes absorbed during the blood meal of a female anopheles undergo gametogenesis in the stomach of the mosquito. The male microgammon (microgametocyte) differentiates 8 haploid and flagellated microgametes in a space of about ten minutes; this is the phenomenon of exflagellation. The female macrogamont (macrogametocyte) evolves into a macrogamete without nuclear division. Fertilization gives rise to a mobile zygote (ookinete) that crosses the intestinal epithelium and encyst between the basal lamella and the epithelial cells to evolve into an ookyst. The oocyst undergoes a reductional division

followed by multiple mitoses giving rise to thousands of sporozoites. After bursting of the oocyst (oocyst), and by a poorly known tropism, sporozoites (Infectious Forms) migrate into the salivary glands where they accumulate.

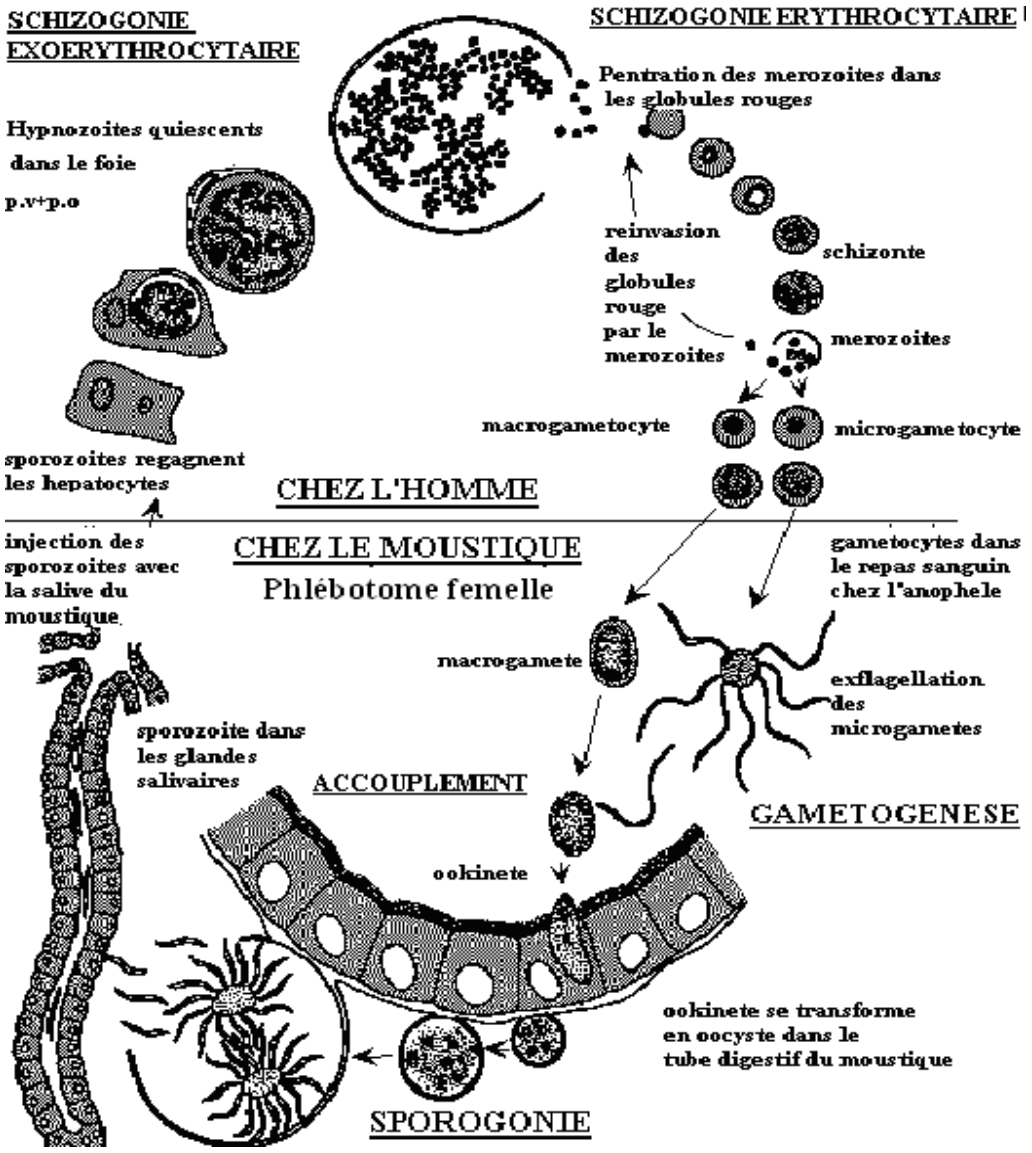


Figure 23: Development cycle of a *Plasmodium (P. vivax)*

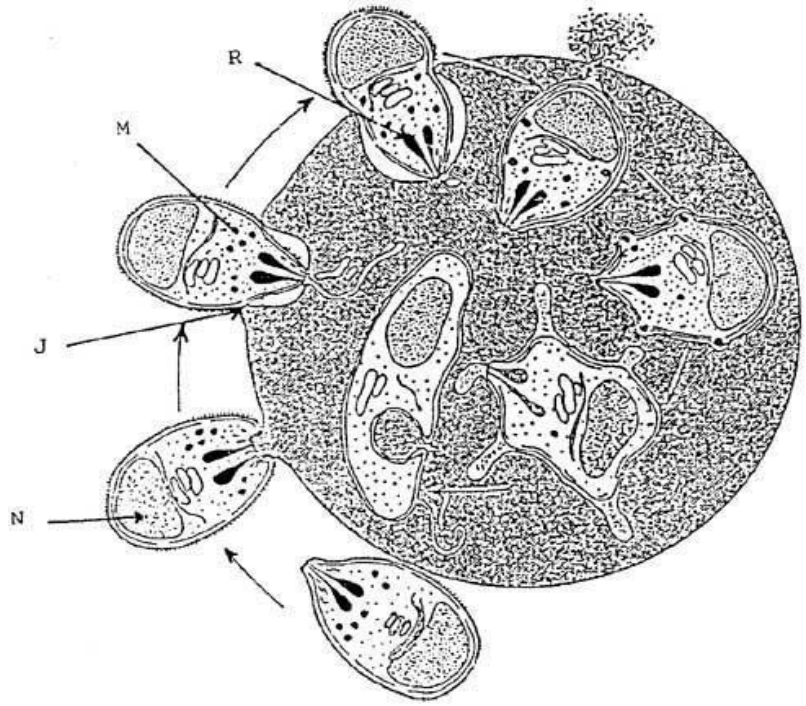


Figure 24: Invasion of the red blood cell by the merozoite of a *Plasmodium*
J: Mobile junction; **M:** micronemes; **N:** core; **R:** rhopters.

Characteristics of the different *Plasmodium*

The distinctive characteristics of the species of the genus *Plasmodium* parasites of man are reported in Tables 1 and 2.

P. falciparum

Definitive host:different species of anopheles especially *Anopheles maculipenis*
Intermediate Hosts: The Man
Location: Definitive host: General cavity and salivary glands
Intermediate hosts: in peripheral blood red blood cells and deep organs **Geographical distribution :** In equatorial regions, it is transmitted throughout the year with seasonal upsurges. In the sub-tropical regions, it occurs only in hot and humid periods. Its transmission is interrupted when the temperature drops below 18°C. This also explains why, whatever the latitude, malaria is no longer transmitted in altitude (above 1500 meters in Africa and 2500 meters in America and Asia).
Biology : *P. falciparum* is responsible for serious clinical forms, including neuropalaria. It is responsible for daily and malignant third fever: 48-hour erythrocyte cycle and summer-autumnal). Its incubation period is 7-12 days.

Plasmodium vivax malaria

Definitive host:different species of anopheles especially *Anopheles maculipenis*

Intermediate Hosts : The Man

Location: Definitive host: General cavity and salivary glands

Intermediate Hosts: In peripheral and visceral red blood cells.

Geographical distribution : Very widespread in South America and Asia, it is much more rarely observed in Africa. It appears in spring in the Mediterranean basin. Erythrocytes of the negative Duffy blood group (observed in the majority of subjects from West Africa) do not have the membrane receptor necessary for *P. vivax*infection. Its transmission stops below 15°.

Biology : Its incubation period is 11-13 days. *P. vivax* disease is classically considered benign (benign third party fever, i.e. due to a 48-hour erythrocyte cycle) but in endemic areas it can have serious repercussions on the health status of populations, in particular through anaemia in children. In addition, we are beginning to see some drug resistance to *P. vivax*.

P. ovale

It is prevalent in intertropical Central and West Africa (and parts of the Pacific) and causes mild third party fever, i.e. due to a 48-hour erythrocyte cycle, as

P. vivax to which it is very close. Its incubation is a minimum of 15 days but possibly much longer, up to 4 years. Its evolution is benign but we can observe, as with *P. vivax*, late relapses (5 years). Schematically, *P. ovale* is said to replace *P. vivax* where the latter species does not exist.

p malariae

Definitive host:different species of anopheles especially *Anopheles maculipenis*

Intermediate Hosts: The Man

Location: Definitive host: General cavity and salivary glands

Intermediate Hosts: In peripheral and visceral red blood cells.

Geographical distribution : This plasmodium is found throughout the world but is very unevenly spread; in general, it is more a parasite of temperate regions than tropical countries, it is rife in Africa, much more sporadically.

Biology : It differs from other species by a longer incubation (15 to 21 days), by a different periodicity of the fever (72-hour erythrocyte cycle responsible for a fourth fever whose outbreaks recur every 3 days; that is to say: the first day, the 4 th, the 7 th) and especially by its ability to cause very late revivals (up to 20 years after the return from the endemic area).

Symptom

Malaria is an infectious disease caused by sporozoa of the genus plasmodium parasites of red blood cells, inoculated to humans by a culicidae of the genus *Anopheles* (see **Photo** Appendix). It essentially results in feverish phenomena, most often of the intermittent type.

The manifestations of malaria are very varied;. We will insist on those of the typical malaria fever which is the intermittent fever.

the incubation period

It is highly variable and difficult to assess in most cases (see Tab1). It corresponds to hepatic schizogonia and the first erythrocyte cycles. There are; no clinical signs.

The invasion period

It corresponds to erythrocyte schizogonia. During this period the patient has headache, general tiredness, inappetence, nausea, asthenia, usually constipation. The temperature reaches 38° or 39° C and the pulse is fast. This period usually lasts only a week. Then follows a latency period with no net symptoms. Well treated during this period malaria can be stopped.

The state period (case of intermittent fever: includes a series of attacks at regular intervals)

The premonitory phase: sometimes appears several days before the attack itself. It is characterized by great weariness, headache, anorexia, vomiting and a slight rise in temperature.

The access itself comprises 3 characteristic stages:

The shivering stage: is characterized by intense cold. The patient clicks his teeth, covers himself, the temperature is high, we sometimes notice vomiting, convulsive attacks in the child. This stage lasts about an hour

The heat stage: follows the previous stage. The patient feels intense heat, discovers himself, his pulse is fast, his breathing is accelerated, his skin is dry and hot. The headache is intense, vomiting is very common and the temperature reaches 41 to 42 ° C. This stage lasts on average 3 to 4 hours.

The sweat stage: the patient is flooded with sweat. The temperature drops rapidly, headache and vomiting disappear and the patient feels a certain sense of well-being

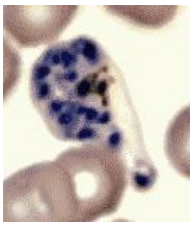
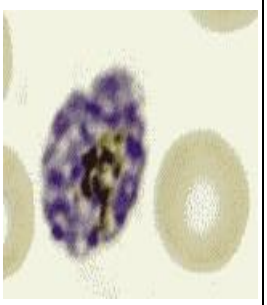
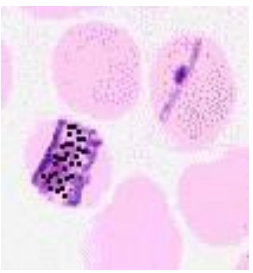
NB: When a patient has new attacks, which usually occur about two weeks after the previous ones have healed, it can be either:

- insufficient treatment;
- reinfection (new bite of infected anopheles);
- recrudescence (increase in parasitaemia to a clinically detectable level in a subject after incomplete or ineffective treatment, or in a semi-immune subject);
- relapse (exacerbation of parasitaemia from hepatic hypnozoites in the case of *P. vivax* and *P. ovale*).

Pathogenesis

Plasmodiums act in different ways on the human body: spoliative action that destroys a large number of red blood cells; mechanical action by determining embolisms; toxic...

Table 1 : Characteristics of the different *Plasmodium*

Cash	***p vivax***	***P. ovale***	***P. falciparum infection***	***p malariae***
Young living forms	Not very refractive, not very distinct contour, lively amoeboid movements	In the form of pigmented rings	net contour, very slow amoeboid movements	Refractive, net contour, very active amoeboid movements
Mature schizonts	Variable shapes, spherical larger than a red blood cell 	Quadrangular, larger than a red blood cell 	Spherical, ½ of a red blood cell 	Quadrangular, smaller than a red blood cell 
Pigment	Fine grains in sticks, light brown, very MOUNTS	Fine grains in sticks, light brown, very mobile	Small, fine and not very mobile grains	Thick grains, irregular, dark brown, little or no MOUNTS
Parasitic red blood cells	Hypertrophied, pale with Schüffner granules	Oval with jagged edges Enlarged, pale with granulations of Schüffner	Normal with Stephens and Christophers granules (or Maurer spots)	Shrunk, dark, no polychromatophilic granules
schizogony **Incub. time**	Segmentation into a spherical cap, in the shape of a blackberry, in the peripheral blood **8 days**	Segmentation into a spherical cap, in the peripheral blood **9 days**	Rosette and irregular segmentation, in the blood of the capillaries of the viscera **6 days**	Segmentation into rosette or marguerite, in the peripheral blood. **13 Days**
No. Of merozoites	16-24	4 to 16 m²	8 to 16	6 to 12

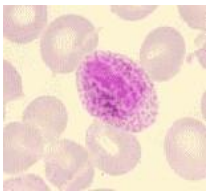
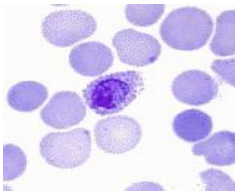
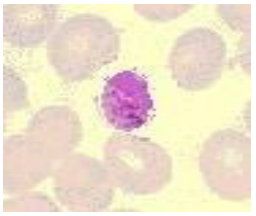
	shapes larger than a red blood cell Spherical 	shapes smaller than a red blood cell. 	Banana-shaped then ovoid and spherical Spherical 	shapes a little smaller than a red blood cell 
Spherical **Gametocytes**				
Evolution	In 48 hours	In 48 hours	24-48 Hrs	In 72 hours
Fever	Benign third party	Benign third party	malignant third party, journ.	Fourth

Table 2: Sporogonic cycle duration (in days)

Temperature	*p vivax*	*P. falciparum infection*	*p malariae*	*P. ovale*
27°C	8-9	10	14-15	12-14
-25°C...	10		15-20	16
-20°C...	16 to 17	22-23	25-30	-

In the case of acute malaria: **the blood** shows significant alterations: decrease in the number of G.R, decrease in the hemoglobin level; alteration of the basophils (polycromatophile), the blood may contain hemozoin grains. **The most affected viscera** are the hematopoietic organs: the spleen, it is voluminous, the color changes; the bone marrow is dark brown.
In the case of chronic malaria : the spleen is large and sclerosed; the liver is enlarged; the kidneys are congested and sclerosed, the adrenal capsules are enlarged

Diagnostics
Orientation diagnosis:
Stay or stay in an endemic region Clinical diagnosis:
Fever, influenza-like illness 8 days or more after arrival in endemic countries, often atypical, especially if self-medicated.
Direct diagnosis:
Microscopic examination of blood in the fresh state or after fixation and staining of a thin smear (*P. vivax*) or a thick drop (*P. vivax*) is used. The blood test will tell us about the presence of *Plasmodium* and the *Plasmodium species.* Diagnosis of the species is essential (because there is a risk of neuropalaria if *P. falciparum*).

Indirect (immunological) diagnosis :
Testing for serum antibodies Techniques used: IFI on blood plating or *in vitro* culture of *P. falciparum*, haemagglutination, ELISA, Immunotransfer; these techniques are expensive and complicated

Processing
The specific malaria drug is quinine extracted in 1820 from the bark of shrub quinquina (formula $C_{20}H_{24}Az_2O_2$). The medicines are made from quinquina salts.

- More recently discovered schizonticides: **Mefloquine** (Lariam®); **Pyrimethamine + sulfadoxine + mefloquine** combination (Fansimef®); **Halofantrine** (Halfan®);**Artemisinin** and its derivatives (Paluther®) ;**Pyronaridine** (Malaridine®) Far East only
- Gametocytocide or hypnozoitocide: Amino 8 quinoline: **Primaquine** (Anglo-Saxon countries only)

Prophylaxis
To fight malaria, we will therefore have to fight against *Plasmodium* (pathogens) and Anopheles (vectors). This control will include general prophylaxis measures, intended to destroy pathogens and their vectors, and individual prophylaxis measures, intended to preserve the individual from infection.
General prophylaxis:
(Purpose: to "control" malaria in a territory)
1) **vector**control: the aim is to limit the population of anopheles through sanitation measures, removal of large or small standing water; anti-larval control: oil, use of soluble insecticides spread on the surface of standing water, seeding of water with predators of anopheles (fish, molluscs); anti-imago control: use of residual insecticides in homes, dispersion of sterile males, genetic interventions on vector species; use of biological screens (animal species diverting anopheles from humans)
2) **antiplasmodial control** in humans: aims to destroy the pathogen in the human body, diagnosis and mass treatment of carrier subjects is impossible.
Note: ongoing research for a vaccination (efficacy and safety requirement),

2.4. Intestinal ciliates

2.4.1. General information on ciliates

Ciliates are evolved protists. Formerly called infusoria, ciliates are a unique class. They live in all aquatic environments and are characterized by the presence of a mouth. From an anus and at least at one stage of their life cycle.by vibratile eyelashes used for swimming. walking or to move the water around them so as to obtain food. Their cell contains two nuclei the macronucleus and the micronucleus .involves in sexual reproduction or conjugation. The ciliates also multiply by transverse scissiparity where many ciliates live commensally

in the thinking of the herbivores whose food they share, in a parasitic form. One of them lives in the human gut or it causes an inflammation called balantidiosis. (André Beaumont; 2010)

2.4.2 Classification

It is essentially based on the arrangement and size of the vibrating eyelashes next to simple eyelashes. There are larger organelles made by agglomerated eyelashes.

- There are two classes:

1. class of holotriches:

Ciliates whose ciliature is made of simple eyelashes and membranelles. The internal classification of this class is based on the position of the mouth.

Example: *Colpodium colpoda. Paramecium caudatum*

2. spirotrich class:

They have complex ciliary organelles such as cirres or undulating membranes forming an adoral fringe.

Example *:balantidium coli , stylonychia*

2.4.3**. Intestinal balantidiosis**

Balantidiosis, or balantidiasis, is an infectious disease caused by a parasite:

Balantidium coli It is a parasitic disease of the colon, due to the infestation of the only human parasitic ciliate protozoan (Balantidium coli). She considers it a much more occupational disease.

The colonic balantidium (Balantidium coli), is part of is a large commensal ciliated protozoan of pigs, monkeys and sometimes rats, which accidentally determines in humans an intestinal parasitosis: balantidosis. It is the largest protozoan and the only ciliate pathogenic to humans.

 Classification : Branch:

Ciliophora Class:

Litostomatea

Order: Vertibuliferida

Family: Balantidiidae

Genus: Balantidium

Species: *Balantidium coli*

- **Morphology**

A. **The vegetative form** :

In stool, Balantidium has an oval shape of 80 micrometers in general (but which can range from 30 to 300 microns). The movements of his eyelashes are coordinated and he moves in a spiral. It has 2 nuclei of which only the largest is visible without staining:

 the reniform macronucleus (or vegetative nucleus), used for vegetative functions.

 the micronucleus, located in the cavity of the previous one, which performs the reproductive functions. This protozoan has many digestive vacuoles where bacteria and cell debris are digested. In its posterior region is located a pore (the cytoprocte) from which food residues are expelled into the external medium. At each pole of the cell, a pulsatile vacuole is observed that maintains osmotic pressure by evacuating the water that permanently penetrates the cell body. (Schuster F et al; 2008)

B. Cystic form:

In the external environment, the resistance form of Balantidium is represented as a cyst surrounded by a thick wall. Spherical in shape (diameter: 50 micrometers), it is the element contaminating humans orally.

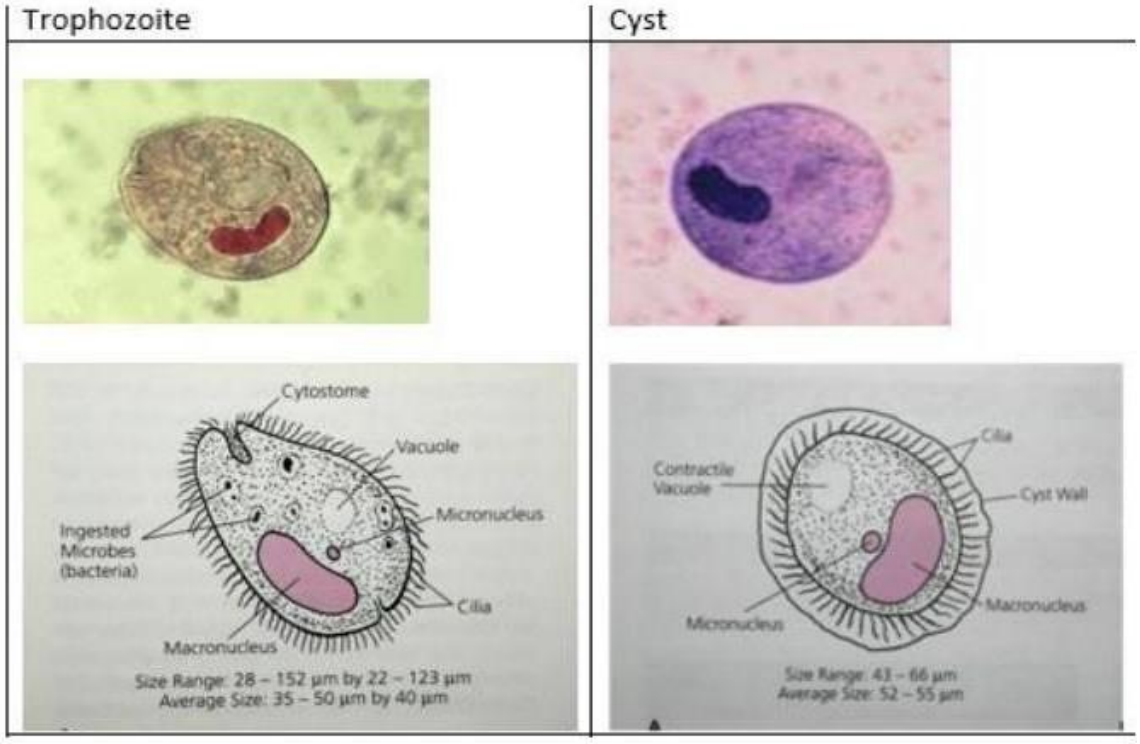


Figure 25: vegetative and cystic form of balantidosis coli.

- **Tricycle transformable to bicycle.**

Its evolutionary cycle has two stages: a ciliated trophozoite stage and a cystic stage with environmental resistance. Cysts are the infectious stage. After being ingested, the cysts lose their protective shell in the small intestine and the trophozoites attach themselves to the mucosa of the terminal ileum and the harsh colon. There will be an emission of cysts and then oral transmission.

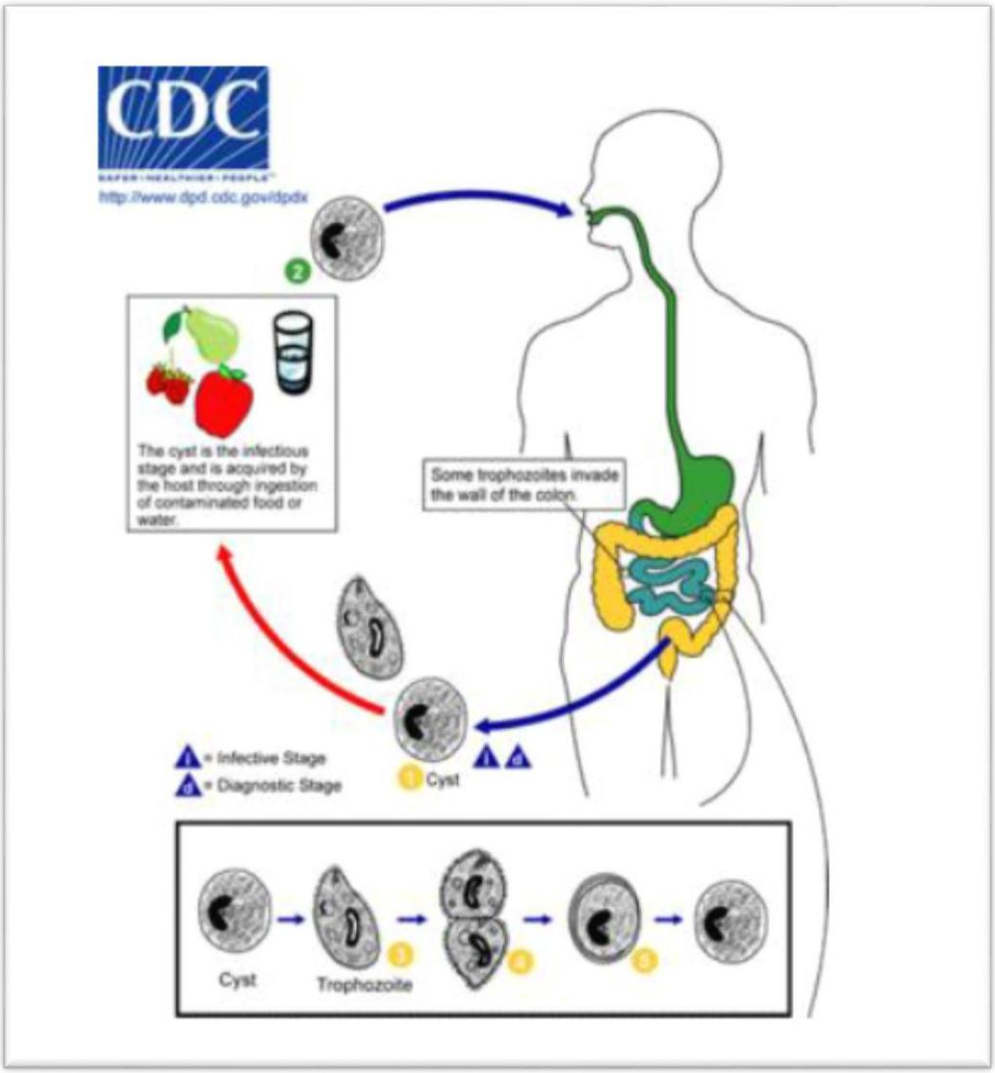


Figure 26: The evolutionary cycle of a *Balantidium coli*

Pathogenicity and toxicity

The most common foci of infection (> 95%) of B. coli are the terminal ileum and colon, especially the rectosigmoid region. B. coli is the only ciliated protozoan that is pathogenic in humans; infection of the gastrointestinal tract is often mild and may be asymptomatic. B. coli infections are similar to amoebiasis in that a silent carrier state may be present.

However, it is possible to observe symptoms of dysentery in malnourished, alcoholic or immunocompromised patients. In the acute phase, fulminant balantidiasis is characterized by intense diarrhea and mucosal bloody stools, nausea, vomiting, headache; it has a mortality rate of 30%. Chronic infections are manifested by non-bloody diarrhea, cramps, halitosis and abdominal pain following invasion of the large intestine by trophozoites Protozoa that infiltrate the submucosa can cause abscesses and hemorrhagic lesions that can ultimately lead to bacterial appendicitis and secondary infections, such as uterine infections, vaginitis or cystitis .

- **Symptoms and complications** :

Balantidiosis is a rare intestinal infection caused by the bacterium, Balantidium coli, a single unicellular parasite (ciliated protozoa) that frequently infects pigs, but sometimes (rarely) infects humans. Some infected people have no symptoms or only mild diarrhea and abdominal pain, but others may experience more severe symptoms that are reminiscent of acute inflammation of the bowel.

The symptoms of Balantidiosis may be similar to those of other infections that cause intestinal inflammation, for example, amoebic dysentery.

- Dysentery, accompanied by mucous stools, and blood with abdominal pain
- Intestinal bleeding associated with peritonitis, and chronic (long-term) colitis (inflammation of the colon)

- **Diagnostics**

Balantidiasis is diagnosed by microscopic examination of a patient's stool. A stool sample is collected and a wet mount is ready. Trophozoites or cysts can be detected in the stool. *Balantidium coli* is transmitted periodically, therefore stool samples should be collected and examined frequently immediately in order to make a definitive diagnosis.

Trophozoites can also be detected in tissues. In order to collect a tissue sample from the intestine, a sigmoidoscopy procedure is used. A thin, hollow instrument called a sigmoidoscope is used to visually inspect the sections of the large intestine: the rectum and the sigmoid colon. A doctor may look for bleeding, ulcers, and inflammation in order to diagnose the cause of diarrhea and other gastrointestinal complaints, and may take a tissue biopsy for inspection

- **Prophylaxis**

Prevention and based on hygiene measures as well as sanitation will prevent the spread of the disease because transmission is through the ingestion of contaminated food and water. Thus, the purification of drinking water is essential. Handling food and cooking can also help prevent infection. Reducing exposure to pig or pork feces will reduce the risk of infection.

CHAPTER III. Parasitic metazoa

3.1. Les Plathelminthes

3.1.1. General and categorisation

a. Overview

Plathelminths are acoelomate triploblasts. They have a dorso-ventral flattening with, obviously, the existence of the dorsal and ventral faces. The body is organized into two different territories: The anterior cephalic region (sensory functions) and the posterior caudal region. There is no true circulatory system: therefore, no blood. However, there is a liquid present in the space of the parenchyma (the hemolymph).The respiratory system is absent: gas exchange takes place through the animal's integument, or, as in parasites, there will be no gas exchange.

The digestive tract is either incomplete or absent. If it is incomplete, it is because it lacks the anus to "device in cul de sac". If it is absent, exchanges will be possible by osmosis (for parasites). The reproductive system is complex in these basically Hermaphrodites animals. Most are parasites except for a few species belonging to the Turbullaria class. The development cycle can be direct in Turbullaria or indirect requiring one, two or more hosts, as is the case in Monogenea, Digenea and Cestoda. Plathelminths include about 50,000 species and can be free or parasitic. There are six classes but we will only see 2 here: Trematodes (moat) and Cestodes (tapeworm).

b. **Category**

Three categories can be distinguished:

Turbellarians are free, aquatic and their epidermis is ciliated.

The Trematodes evolved into a parasitic lifestyle. They are called Monogenous in the case where their reproduction cycle is simple, Digenic in the case of a complex reproduction cycle.

Cestodes are ribboned and segmented pests.

3.1.2. Class of Trematodes

It includes moats, bilharzies and schistosomes. These worms have complex life cycles and are either ecto or endoparasites. They have a resistant cuticle that protects them from the digestive enzymes of their hosts

a. **General organisation**

Adults are flatworms, generally foliaceous in shape. Growth is limited and has a size of the order of a centimeter. There are hooking and adhesion devices with suction cups and hooks (Hooks exist only in direct-development Trematodes and are implanted around the posterior suction cups. The type of Trematodes is Fasciola hepatica. The nervous system is reduced. There are no sense organs. The digestive tract is represented by an anterior mouth surrounded by a suction cup opens into a muscular pharynx extended by a short and branched esophagus in two intestinal cords themselves very branched and ending in cecums. The excretory apparatus consists of protonephridia which unite into two longitudinal channels which unite themselves into a single channel opening into a contractile bladder opening to the outside through an excretory pore. Trematodes are hermaphroditic, except for species belonging to the family Schistosomidae where the sexes are separated.

***Classification of Trematodes**

- The Polystomian order: In addition to the buccal suction cup, they have several posterior suction cups usually armed with hooks. The members belonging to this order are ectoparasites of poecilothermic vertebrates (heterotherms). They stay for example in the bladder. They only need one host for their development and are therefore monoxene. There is no asexual larval multiplication: they are therefore monogenic. Ex. Polystomum integerrimum: a bladder parasite

de Grenouille, wears a buccal suction cup, six posterior suction cups and hooks. Its development cycle is typically monoxene.

- The Distomian Order **:** They have only two suction cups, one buccal and the other ventral or terminal. Sometimes they disappear (case of bloody Trematodes). The species are parasites of higher vertebrates. Their development is **digene** and **heteroxene**. The larval phases **always take place in a mollusc**. A waiting stage becomes encysted either in a temporary host or on a foreign body. The adult lives in a vertebrate viscera (especially in the intestine and related organs). Ex. *Dicrocoelium dendriticum* or Small Liver Flute has a characteristic three-host cycle. The eggs laid in the bile ducts of the **sheep** are discarded, swallowed by a Snail. It hatch in the digestive tract and develops directly into a sporocyst, then passes into the cercariae and metacercariae state which, enclosed in mucus, is rejected by the**snail's**pneumostoma. It can be swallowed by an **Ant** and becomes encysted in the general cavity. If the Ant is eaten by a sheep, the animal will settle in its bile ducts.

***Diseases caused by Trematodes**

The fluke : *Fasciola hepatica* is responsible for hepato-biliary fasciolosis (sheep, child,etc.) ; *Fasciola gigantica* : hepatobiliary fasciolosis of ruminants; *Dicrocoelium dentriticum*, parasitizes the bile ducts of sheep and other mammals; *Metagonimus yokogawai* : parasitizes the intestine of many animals (dog, cat, etc.) ; *Paragonimus africanus*
: parasites the bronchi of animals and accidentally man (in Africa); *Paragonimus ringeri is* the agent of bloody bronchitis in the Far East.

Schistosomes : *Schistosoma haematobium* causes urogenital bilharzia with frequent complications; *S. mansoni* : causes intestinal and hepatosplenic bilharzia; *Schistosoma japonicum* : causes arteriovenous bilharzia; *Schistosoma intercalatum* : responsible for rectal bilharzia.

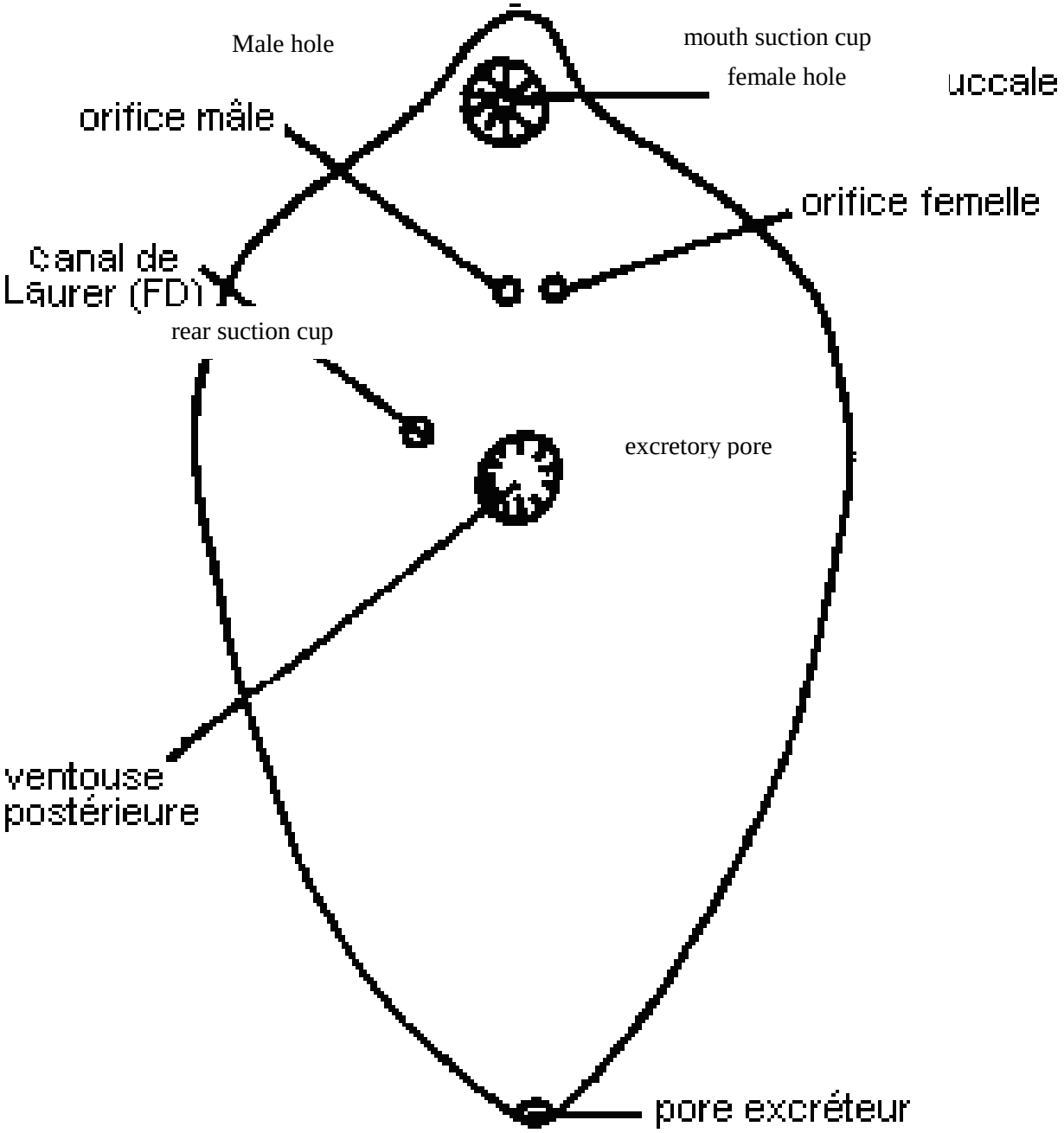


Figure 25 : General morphology of a trematode (*Fasciola hepatica*)

b. Study of some parasites

b.1. Genus Fasciola

Study of *Fasciola hepatica* commonly referred to as large liver fluke.
Disease: Distomatosis or fasciolosis is a zoonosis caused by a trematode, *Fasciola hepatica*, located as an adult in the bile ducts of many herbivores and occasionally humans. Contamination occurs through the consumption of raw plants (watercress, dandelion, etc.). It classically results in hepato-biliary signs associated with hypereosinophilia. A significant economic loss can be recorded in livestock regions (pastoral regions)

Category

Reign of Metazoans Branch of
Plathelminths, Class of Trematoda
Subclass of Digenea Order
of Prosostomata
Sub-Order of Paramphistomata –
Distomian group
Fasciolidae genus Fasciola
family
Species *Fasciola hepatica*

Morphology (see Fig. 25)

Adult: is a flat worm in the shape of a small pink-colored leaf, measuring 2 to 3 cm long by about 1 cm in its greatest width. He is a hermaphrodite. It has at its anterior end two suction cups that allow it to attach to the epithelium of the bile ducts of its host. He is hematophagous. The cuticle is thick and covered with thorns. Digestive caeca are branched.
Location: in the bile ducts of many herbivores and occasionally humans.

Egg: it has an ovoid shape, elongated, presenting the appearance of a rugby ball. It measures 140 µm long by 75 µm. It is sealed and not laid.

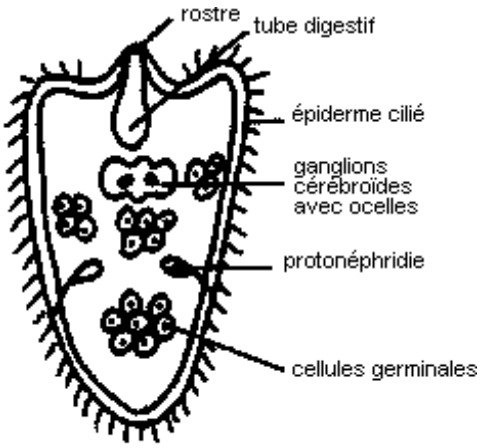
The miracidium : It has a ciliated epithelium making it suitable for swimming. Two cerebroid nodes exist, two dorsal ocelli, two protonephridia. There is a rudimentary non-functional digestive tract, masses of cells that remain embryonic and germ cells. Its lifespan is 8 hours.

The sporocyst : This is the phase following the magnification of the miracidium. There are no sense organs. The general cavities divide and each give a new type of larva. Each sporocyst gives 5 to 8 redies.

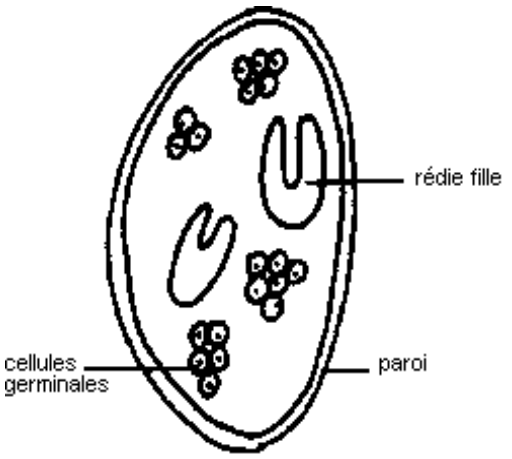
The redies : each redie measures between 1 and 3 mm, it is an elongated bag carrying a mouth, a muscular pharynx, a simple digestive tract and a laying orifice at the front. It still contains germ cells.

The cercaria : It has the organization of the adult Douve: two suction cups, a two-branched digestive tract, an excretory apparatus, cerebroid nodes but no differentiated genitals. Its tail is muscular, the larva has many kystogenic glands. The life of the free cercarium is a few hours. The body of the cercaria measures between 0.25 and 0.35 mm.

Metacercaria: this is an infesting encysted form and is attached to an aquatic plant. Its diameter is about 0.2 mm.

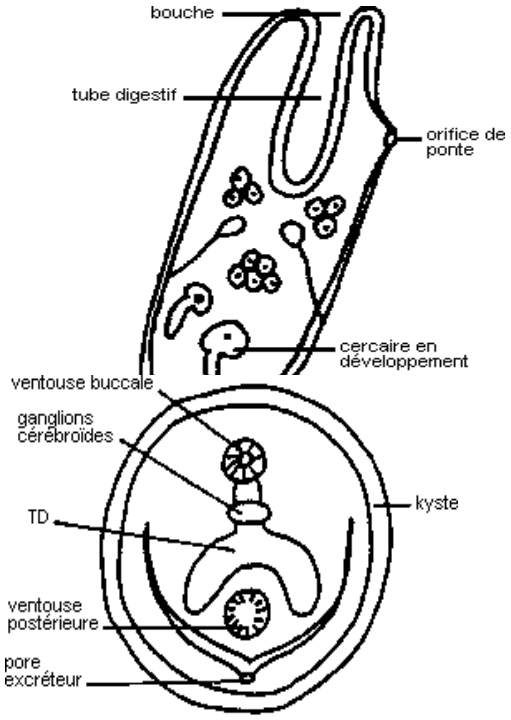


Larve *Miracidium* de *Fasciola hepatica*



Sporocyste de *Fasciola hepatica*

Adult *Fasciola hepatica*



Métacercaire de *Fasciola hepatica*

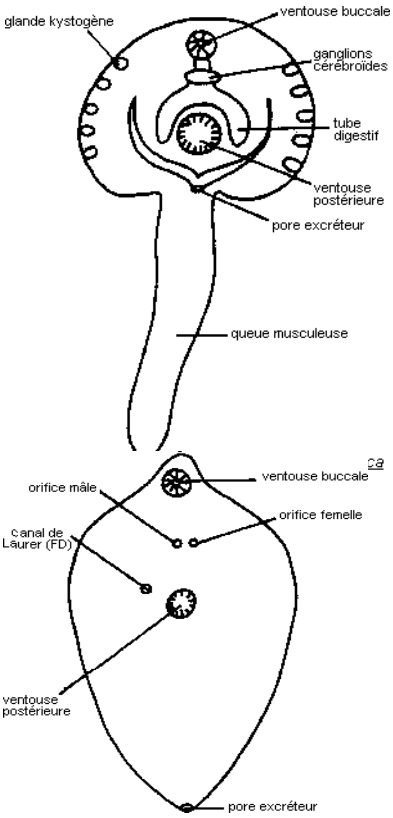


Figure 25 : Morphology of different *Fasciola* forms

Hepatica

Evolutionary cycle (see Fig**.** 10)

The ring is heteroxene. The definitive host is a ruminant mammalian vertebrate (sheep, cattle, goats, etc.) and man. The invertebrate host is a freshwater pulmonate mollusc of *the genus Limnea*.

The adult (about 3 cm) lives wrapped in a horn in the hepatic ducts of **Ruminants** (mainly Sheep). Fertilization is reciprocal during mating. The eggs fall into the intestine and are expelled with the animal's faeces. If they arrive in the water, they continue their evolution. The larva they generate escapes by lifting the polar operculum and swims vigorously thanks to its ciliated tegument: it is called **miracidium**. It is attracted by almost all species of **Limnea** (Pulmonary**gastropod molluscs**), mainly ***Limnea trunculata***. It enters the lung of the Mollusc and transforms into a **sporocyst** by degeneration of all its organs except the integuments (which lose their eyelashes) and reproductive cells. In this bag, the germ cells come into action and form larvae or redies that **have** a rough draft of the digestive tract and clusters of seed cells. By bursting the sporocyst, the redies are released and migrate into the hepatopancreas of their host. If the temperature is low, they generate other redies, the daughter **redies**. If the temperature rises, they give rise to a new type of larva, the **cercaria** in which a young Trematode with suction cups, digestive tract and locomotor tail is sketched. The cercaria comes out of the Limnea, making its way through the viscera (evolution time in molluscs under optimal conditions (20-22°C) is 40 days). After a short period of free life, it attaches to the underside of a **semi-aquatic plant** (near the surface of the water), loses its tail and secretes a thick cystic envelope (allowing them to resist in the outdoor environment for several weeks), thus becoming the infesting **metacercaria** . The definitive host becomes contaminated by ingesting the plants on which the metacercariae are fixed. The cystic wall is digested in the duodenum; the pupa crosses the hepatic parenchyma creating lesions before settling in the bile ducts (10 to 20 days after contamination) where it becomes an adult 3 months after contamination. The longevity of adult moats is approximately 3 to 5 years.

Noting that contamination is often seasonal, beginning in late summer or autumn. Rainy summers that are conducive to limnea increase the risk of contamination.

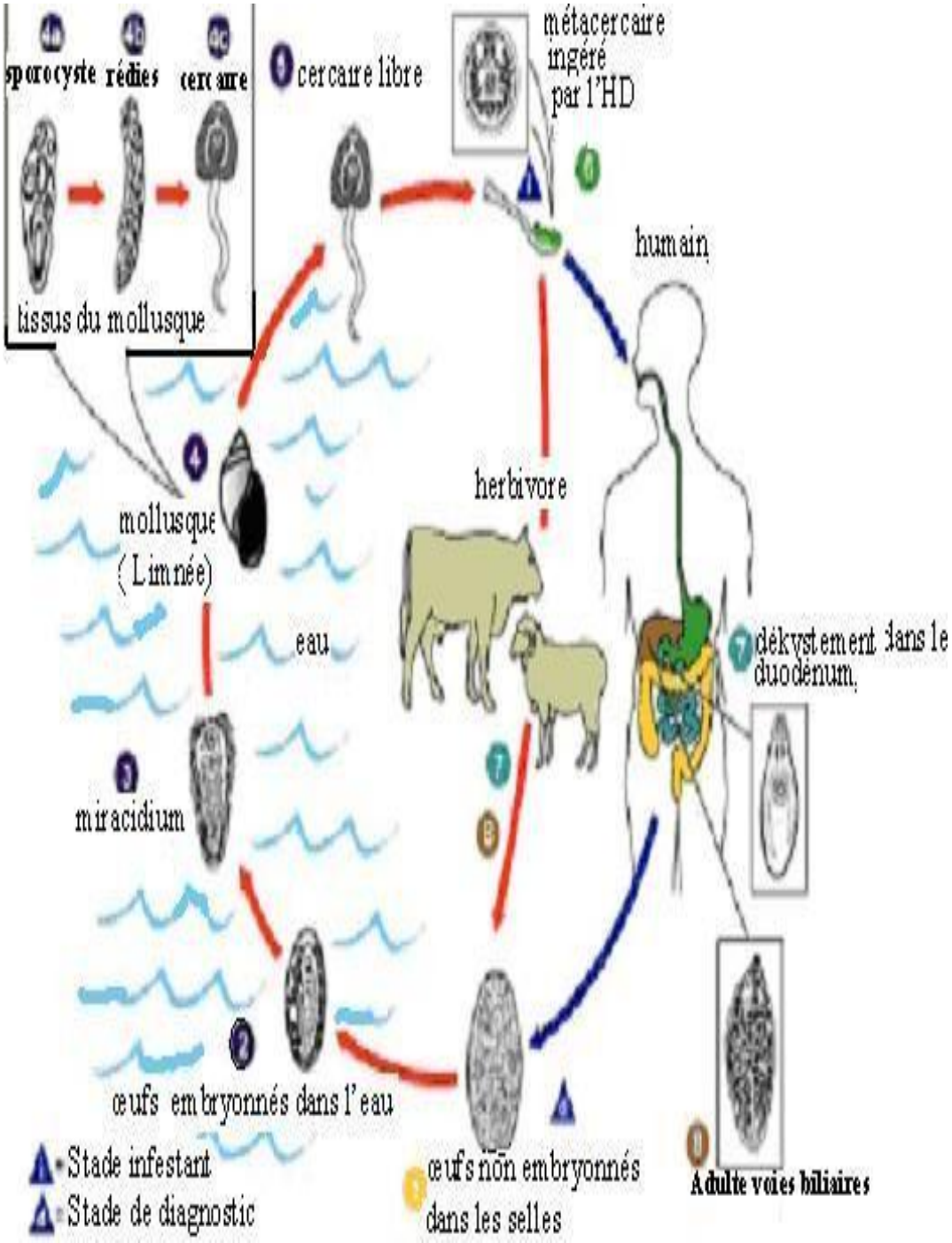


Figure 26: *Fasciola hepatica* life cycle

- Ecological changes: the control is currently directed against vector molluscs, the most vulnerable point in the epidemiological chain. The use of molluscicides is a technique that can be applied systematically. Some molluscicides do not spare fish, an important base of the diet. Ecological methods can be used such as the periodic dewatering of irrigation canals, the destruction of the plants on which molluscs feed. The use of molluscs competing with intermediate hosts.

The use of predators is currently being tested: *Anatidae* (ducks) and carnivorous molluscs. Individual prophylaxis: avoid swimming in stagnant fresh or brackish waters, even for very short and very partial immersions, swimming should only be accepted in sea water or in a duly controlled swimming pool.

b.2. Genus Schistosoma

Metacercary stage does not exist. Species of this genus live in the circulatory system of mammals. The sexes are separated and are haematophagous. Several of them parasitize humans. Adults live in veins. The male lodges the female, very filiform, in a groove on its ventral face (gynecophoric canal). The two suction cups are very close and are located in the anterior part of the animal's body. It should be noted that there is no pharynx. Bilharzia or schistosomiasis are parasitic diseases due to trematodes, flatworms, separated sexes, hematophagous, living in the adult stage in the circulatory system of mammals and evolving in the larval stage in a freshwater mollusc. There are 200 million cases of schistosomiasis worldwide and five species are pathogenic to humans and endemic on three continents

Category
Plathelminthes branch, Trematoda
class
Subclass of Digenea Order of
Prosostomata Suborder of
Strigaeta Family of
Schistosomatidae Genus
Schistosoma

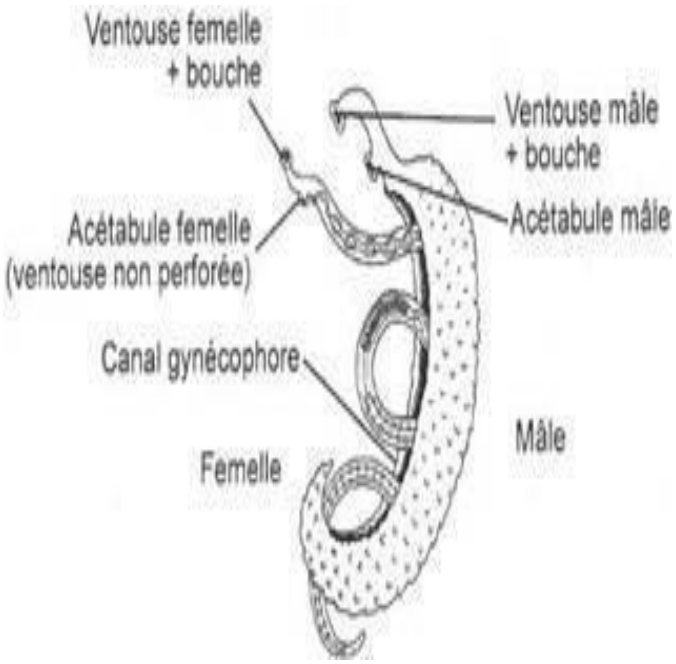
Species: 5 species that can parasitize humans:
Schistosoma haematobium : responsible for urogenital schistosis
Schistosoma mansoni: responsible for intestinal and hepatosplenic bilharzia
Schistosoma intercalatum : responsible for rectal bilharzia *Schistosoma japonicum* : responsible for arteriovenous bilharzia *Schistosoma mekongi*: responsible for arteriovenous bilharzia

Schistosoma haematobium study

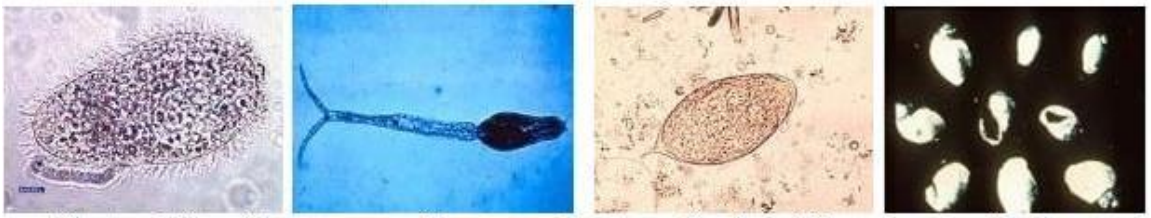
Morphology

We distinguish the different stages except the redi and metacercary stages (see Photos) Adult: present in the definitive host (the man); the sexes are separated. The 2 suction cups

occupy a very anterior position. The integument is warty and thorny facilitating the attachment of the animal to the blood vessels. The male: measures 10 to 15 mm long by 0.7 to 1 mm wide, has a leafy appearance; the lateral edges are folded into a gutter constituting the gynecophoric canal. The genital pore is located under the ventral suction cup, or 4 to 6 testicles. Female: measures 15 to 20 mm long by 0.1 mm wide. It has a cylindrical shape. The ovary is located in half of the body of the animal the number of eggs visible in the uterus is 20 to 30.



Eggs: have an ovoid shape, clear, and have a terminal spur of 15 µm. The size of the eggs is on average 140 µm long by 60 µm wide. Noting that the eggs are not embryonated during laying by the female but embryonated during externalization with urine.
They are the ones who determine the disease. The lifespan of adults is 10 years.
The cercaria: It has a bifurcated tail, hence the **name** furcocercaria given to the Schistosoma larva*which* is eliminated by the mollusk. This is the infesting form (of man).



Photos : Miracidium **Furcocercaire** **Oeufs de S.h** **Bulinus**

Tricycle transformable to bicycle.

The cycle is indirect. **HD** = human, **HI** = Bulin, 1 cm high sinus freshwater conoid mollusk (genus *Bulinus*); **RP=** human.

Adult worms are located mated in the large hepatic and mesenteric venous trunks, fertilized females ascend the network of bladder capillaries and lay non-embryonated eggs there. The eggs, by their spur and their lytic enzymes leave the capillary and break through the tissues until falling into the bladder lumen (bladder). The eggs are rejected into the external environment by urination (S.h) and if the conditions are favorable (pH close to neutrality and temperature between 18°C and 33°C), in contact with fresh water, they release a ciliated larval form: the miracidium (whose lifespan is short: a few hours) which must swim in search of the specific mollusk of the schistosome species: genus *Bulinus* for S.h. At the level of the hepatopancreas of the mollusk, when the temperature is adequate (30°C), the larval forms will give the sporocysts of stages I and II. Larval evolution in molluscs takes 1 month. From the mollusc comes the ultimate form of larval evolution: the cercaria, which measures 0.5 mm, has a bifid "tail" (furcocercaria). By polyembryonic phenomenon a miracidium gives thousands of cercariae (10,000). This Furcocercaria circulates in water, ready to penetrate transcutaneously in a few minutes into any immersed part of the human body (oral contamination is a very rare eventuality). The survival time of furcocercariae is short (24 to 72 hours) and it is by chemotaxis that they are attracted and break into the integuments of the definitive host (by the action of enzymes secreted by the head of the furcocercariae; duration of the transcutaneous passage: a few minutes). As soon as the furcocercariae are fixed, they separate from their “tail” and the anterior part or schistosomula is carried by the lymphatic route into the large circulation. From the 48th hour, and for several days, they are in the pulmonary capillaries and then reach the heart, and through the abdominal circulation and the veins of the portal system, the parasites reach the liver (large hepatic vessels) where they become adults around the 2nd month.

After mating, the worms move up the back door circulation. The fertilized females then separate from the males and engage, according to a particular tropism, in the fine branches of the bladder, where they deposit their eggs.

Symptom

Apart from the irritant action of cercariae penetrating through the skin and the toxic phenomena due to the migration of schistosomules and adults, it is essentially the eggs of the parasites that are at the origin of the anatomical lesions and consequently of the clinical disorders observed.

Phase d’invasion

It corresponds to the penetration of furcocercariae. It often goes unnoticed, but can lead to a picture of swimmer's dermatitis (allergic skin erythema) occurring 15 to 30 minutes after the infesting bath: Swimmer's dermatitis.

Toxemic phase

It is contemporary with the migration and maturation of schistosomules in the bloodstream and in the intrahepatic portal vessels. It can be marked with

general feelings of illness,

: asthenia, fever, headache, anorexia (safari fever) accompanied by anaphylactic disorders: pruritus, arthralgia, myalgia, urticaria flare-up...

It corresponds to the laying of females. In the case of urogenital bilharziosis, the master symptom is hematuria. This one is painless and of capricious evolution. It can be microscopic and incidental or macroscopic, discrete and terminal or abundant and total with clots.

Phase of complications

It corresponds to the retention of eggs. For *S. haematobium* , the entire urinary tree may be affected: urethral fistula, urethral stenosis, ureterohydronephrosis, superinfection (cystitis, pyelonephritis, pyonephrosis,etc.), bladder lithiasis, glomerulonephritis. The genital system of both sexes can be affected: urethritis, epididymitis, spermato-cystitis, prostatitis, salpingitis, endometritis, vaginitis, cervicometritis which can lead to impotence and sterility. The prognosis for urogenital bilharzia is mainly at the renal level.

Epidemiology

The disease is related to human urinary peril, bulls and furcocercariae in fresh and warm water. Contamination of humans occurs through transcutaneous penetration. It is a disease endemic to tropical regions: Africa, North Africa, Nile Valley, South Africa), Near and Middle East. There are about 100 million parasitic subjects.

Diagnostics

Orientation diagnosis:

- Epidemiological: stay in a bilharzian endemic area
- Clinics: urinary haematuria
- Biological: hypereosinophilia

Noting that diagnostic methods will be different during the schistosome cycle:

- During the invasion phase: the host reaction leads to significant hypereosinophilia.(Rarely diagnosed at this stage)
- During the maturation phase (**direct method**): there is emission of eggs that can possibly be found in the urine; hence the detection of eggs provides indisputable proof of parasitosis. The best test is to examine the 24-hour urine sedimentation pellet after removing the chemicals that interfere with reading (mainly phosphates and blood). These 24-hour urine should be collected, if possible, after premictural physical exertion (walking, climbing stairs, pelvic gymnastics, jumping...), which improves the sensitivity of the examination **Indirect methods**

These are serological techniques

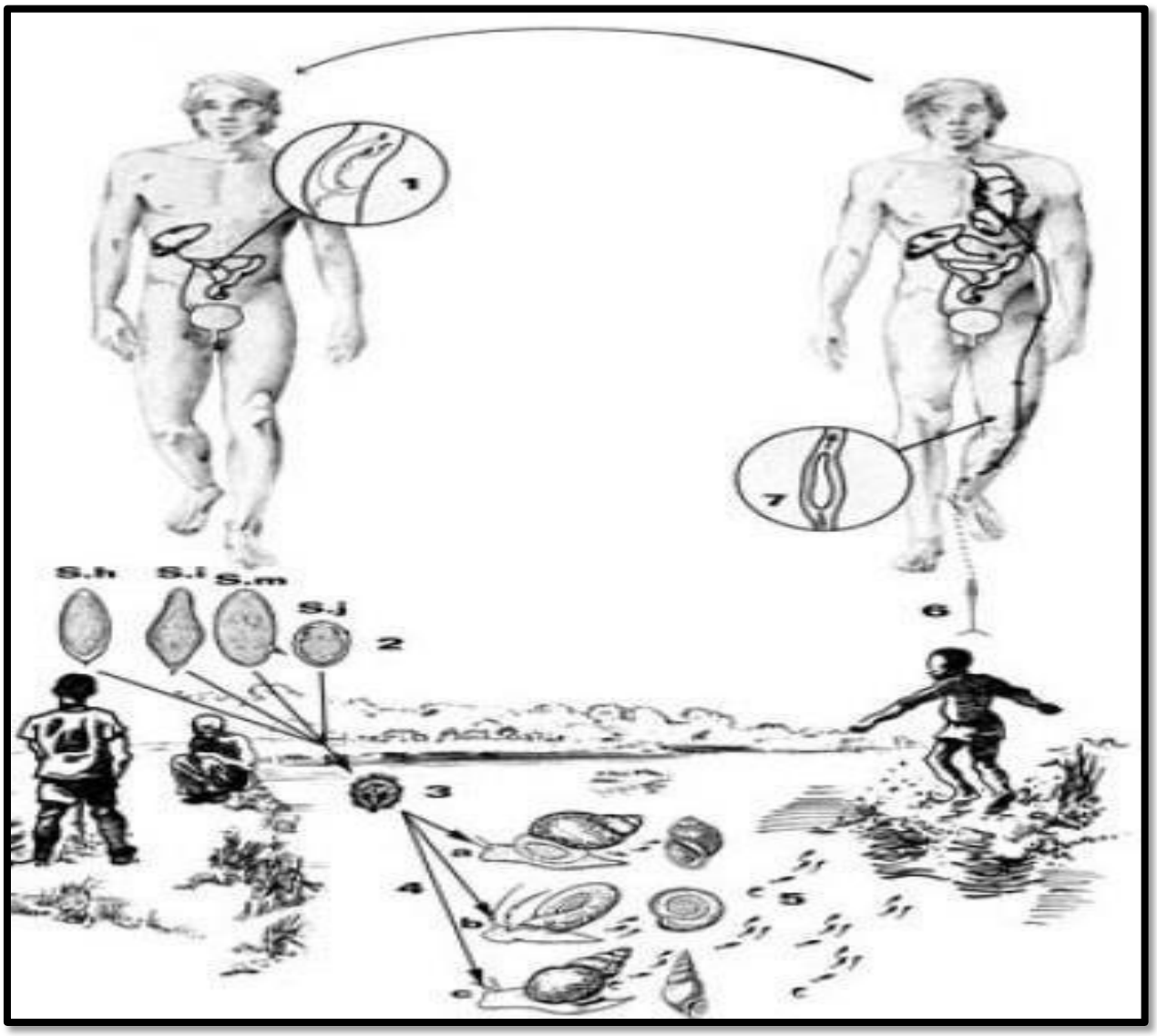


Figure 28: Life cycle of Schistosomes

1. Adults living in the abdominal arteriovenous plexuses 2.The eggs are eliminated in the external environment by urine: *S. haematobium (S.* h.) or by stool: *S. mansoni (S.* m.), S. intercalatum (S. i.) and S. japonicum (S. j.) 3. Miracidium released by hatching eggs in freshwater 4. Compulsory intermediate host mollusc a) Bulin (S. h., S. i.); b) Planorbe (S. m.); c) Oncomelania (S. j.) 5. Infesting furcocercariae obtained after transformation of miracidiums into sporocytes and multiplication of the latter 6. Furcocercariae infestation of humans in contact with contaminated water

7. Schistosomules migrating to the portal system where they become adults before gaining the abdominal arteriovenous plexuses

Processing

Praziquantel (Biltricide®) is effective on all species. After treatment, the eggs can be disposed of for several months

Prophylaxis

General prophylaxis: It is based on:

Health education and prevention of contamination of water bodies by urine, but the protection of individuals against parasitic water remains dependent on the improvement of living standards

3.1.1. Class of Cestodes

a. **General organisation (see Fig. 29**)

They are all internal heteroxene parasites, most often of the intestine or liver of vertebrates. TheCestodes are ribboned. The tapeworm is formed of successive segments or Proglottis **,** the whole of which is called **Strobile**. The proglottids are budded by a zone of proliferation or **Neck**, located immediately behind the anterior part named **Scolex** and which carries the fixing organs. In *Taenia* , the suction cups are called **Inermes**. They can differentiate into **Bothridia** in bothriocephalus. there is no mouth or digestive tract. There are two welded cerebroid nodes. **The excretory system** consists of protonephridia. Cestodes are hermaphroditic. In each proglottis, a male apparatus and a female apparatus develop in different times.

The classification of Cestodes is based on the shape of the scolex and the type of attachment. There are two important orders: The **Cyclophyllidians,** have a rostrum with hooks (Ex. *Taenia solium*or armed Ténia, *Taenia saginata* or Ténia inerme, *Echinococcus granulosus or* Echinococcus tenia) and the **Pseudophyllidians,** have bothridia instead of suction cups (Ex. *Diphyllobothrium latum* or Bothriocephalus

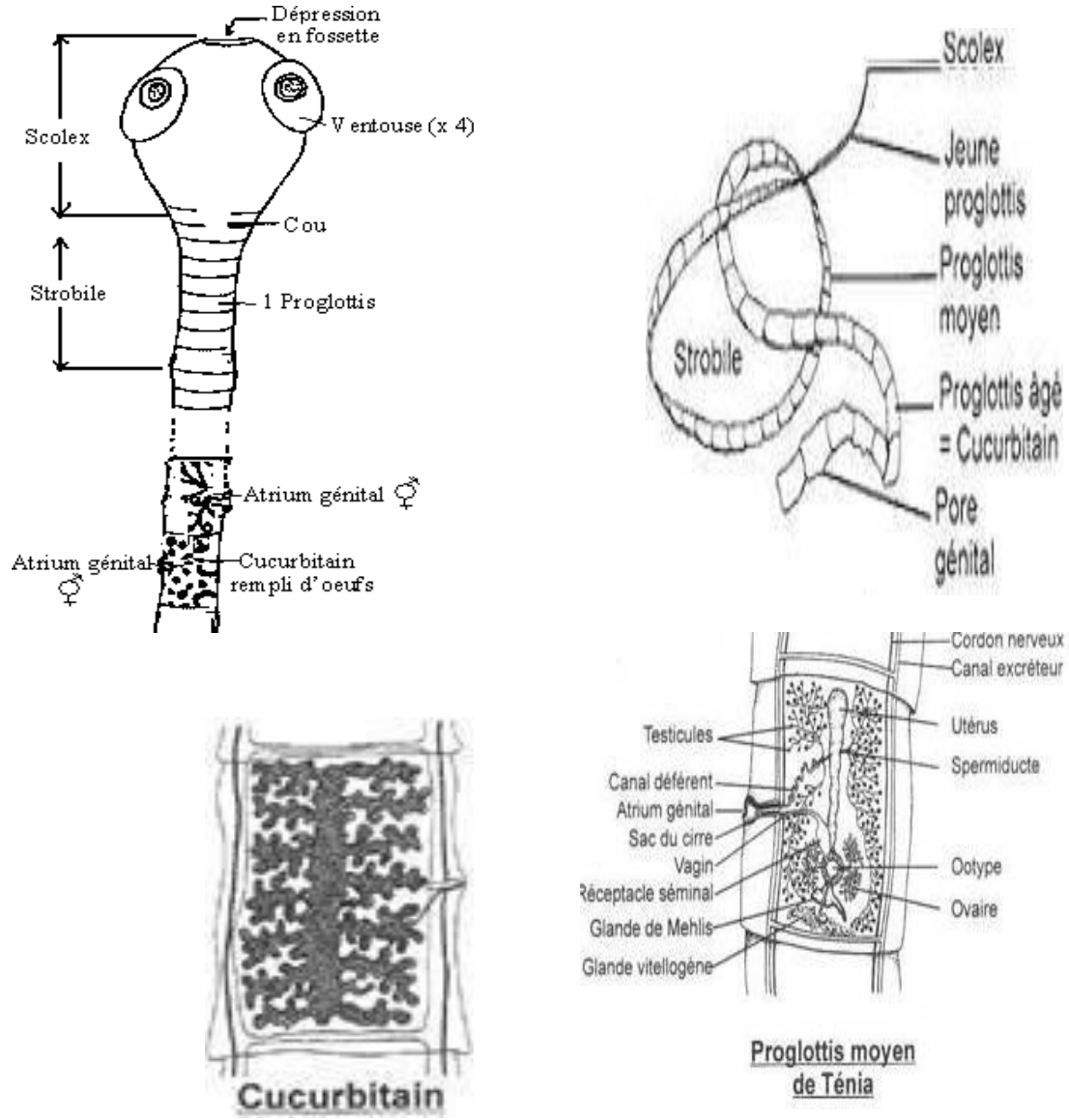


Figure 29: Morphology of a Cestode (*Taenia saginata*adult)

b. Study of some parasites

b.1. Genus *Taenia*

Category

Branch of Plathelminths, Class of Cestodes,

Order of Cyclophyllidae

Family of *Taeniidae*

Genus: *Taenia*

Species: There are several species belonging to the genus *Taenia*

Men

There are two species of flatworms in the class of cestodes belonging to the genus Taenia, which live in the small intestine of humans responsible for Taeniasis in humans: *Taenia saginata*, also called beef tapeworm, and *Taenia solium*, also called pig tapeworm.

For the animals..:

*Tænia sp.*des Carnivores: *Tænia pisiformis, Tænia hydatigena, Tænia tæniæformis*
Adults live in the small intestine of carnivores (tapeworm agents), larvae in the liver and peritoneum of Ruminants and Rodents (hepatoperitoneal cysticercosis agents).

Taenia saginata study

Morphology
Adult : This is a large Cyclophyllid cestode, up to 10 m, consisting of a chain of 500 to 2000 rings, originating from a small scolex (1 mm) with 4 suction cups. The ripe segment contains more than 50,000 eggs, about half of which are ripe. The survival time is longer than 15 years.
The egg has a very thin wall that is easily torn and often absent, it contains a 35 µm diameter spherical embryophore with a thick, brown and radiated shell containing a "hexacanth" embryo (with 6 hooks)

Cysticercus: **is an ovoid mass 5 to 7 mm in diameter called a cystic vesicle filled with an albuminous liquid. The host's tissues react by surrounding themselves with a cyst. The Epidemiology wall**
Hepatic distomatosis is a cosmopolitan disease except in cold regions with a predominance in breeding regions, for animal parasitosis. Human disease is closely linked to eating habits: consumption of raw semi-aquatic plants. It is a disease at animal faecal risk.
The intermediate host is a freshwater herbivorous mollusk called *Limnea truncatula*.
Evolution in molluscs: optimal temperature 20°C (summer in temperate regions), duration inversely proportional to temperature, inhibition of development below 10
°C
The reservoir of the parasite are sheep and cattle. Egg resistance can last for several months in wet stool, and are killed by desiccation and freezing. There is no development below 10°C. Survival of metacercariae on plants can take several months, and are killed by drought in 40 days, and high temperatures
; they are well resistant to diluted bleach and vinegar.

Symptomatology
Invasion phase
It begins 2 to 4 weeks after the contaminating meal. Then, there is migration of the douvules to the bile ducts (traumatic action).
Status Phase:
It begins 3 to 4 months after contamination. The severity of the disease is related to the number of metacercariae ingested by the definitive host. The main symptoms are in the liver and bile ducts. Hepatic distomatosis can be acute or chronic (most common).
It begins 3 months after contamination and coincides with spawning.
In humans in case of severe contamination: We are witnessing digestive disorders:

diarrhea, vomiting, hepatic colic, jaundice, fatigue, pain, poor general condition, sometimes anemia,decrease in eosinophil levels after being elevated during the invasion phase.
In animals (cattle and sheep): inflammation of the bile ducts, liver is fibrous, very clear drop in weight resulting in economic losses for farmers.

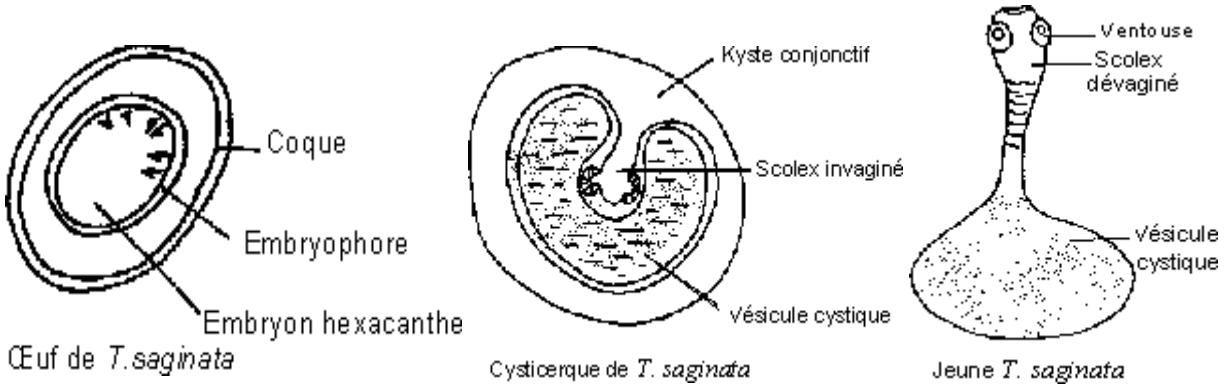
Diagnostics
During the invasive phase
Orientation diagnosis:
Hyperleukocytosis, hypereosinophilia, notion of contaminating meal (wild watercress)
During the status phase
Direct diagnosis: test for eggs in stool (repeat examination after 3 days) Indirect diagnosis: mandatory test for serum antibodies.

Processing
Antiparasitic chemotherapy: **Triclabendazole** (Fascinex®) a veterinary drug currently being evaluated for humans, seems promising because it is effective in all phases of the disease.
Niclofolan (Bilevon®) veterinary medicinal product not used in humansOther therapeutic: Surgery in case of retentional jaundice

Prophylaxis General
prophylaxis
Livestock monitoring (asymptomatic), pasture management, delimitation of risk areas (fence), preventive treatment, approved watercresses
Control of molluscs (chemicals, predators, biological means: dewatering, grazing,

Individual prophylaxis
Watercressand aquatic plants
monitored or cooked

cysticercus invaginates at one pole and buds a scolex. The structure can then remain for two to three years in slowed life. It is the contaminating human form by carnivorism. The cysticercus has a lifespan of about 2 years after which it dies and calcifies in situ.



Life cycle (see Fig. 30)

It is an indirect cycle: **HD** = human and **HI** = bovine

Adult **tapeworms** are found specifically in humans. A single worm parasitizes a host's digestive tract. The terminal rings are ripe (cucurbitans) and detach from the body to be excreted with the stool. Fragile, they release embryonated eggs into the environment whose fragile outer shell disappears. A resistant embryophore remains. Ingested by a species-specific intermediate host (**cattle**), the embryos released into the digestive tract pass through its wall and are disseminated throughout the body at the level of muscles, subcutaneous tissue and sometimes the central nervous system. They then encyst themselves in the form of cysticercae, vesicles 5 to 10 mm in diameter containing the future scolex (protoscolex).

The man ingests these cysticercus when eating undercooked meat, the scolex is then released, attaches to the intestinal wall and begins to bud. The first ripe rings are released from the third month after infestation and the worm may persist in the intestine for several years. Upon accidental ingestion of *T. saginata* embryophore by humans, the larval form can reach the muscles and central nervous system determining the **Teniasis**.

Epidemiology

Tapeworms are cosmopolitan. With beef as a reservoir, *T. saginata* is common in Algeria and is found in subjects consuming undercooked beef. Le réservoir de parasite est l'homme. Embryophores are very resistant (more than 6 months in soil) and withstand cold better than heat. Cysticercia survive a few days at + 4°C, they are killed by heat (56°C) and freezing (10 days at - 10°C). Brine is less effective (need for brine with 20% salt for 5 days). There is no faecal peril.

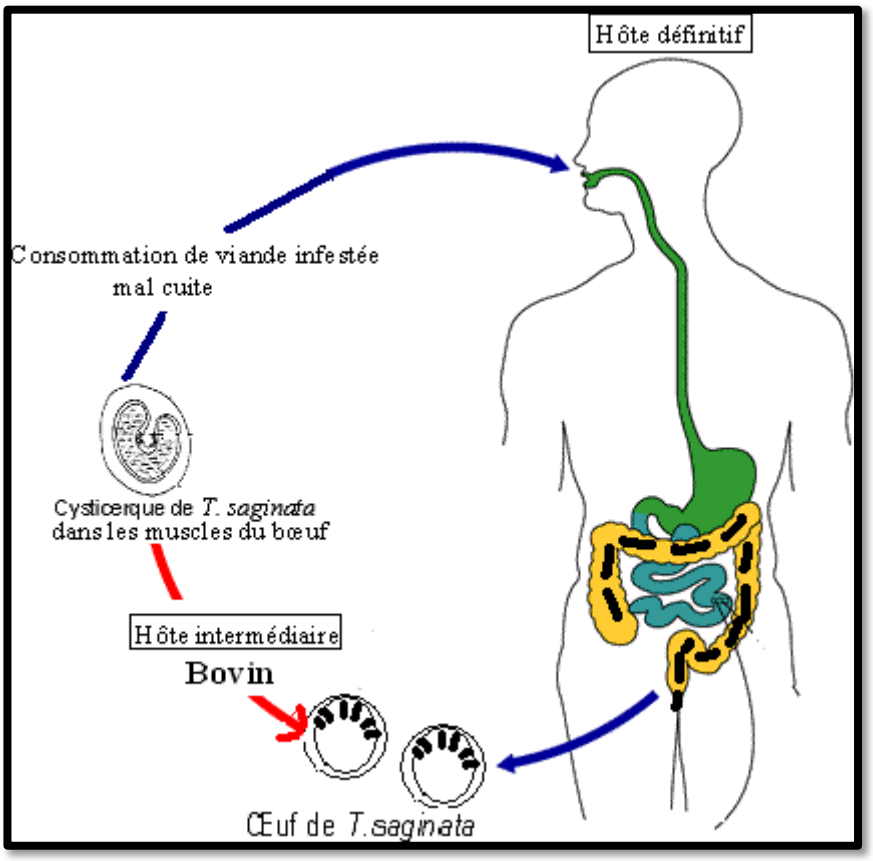


Figure 30: *Taenia saginata* life cycle

Symptom

Incubation phase: Most often it is asymptomatic (silent) with, however, hypereosinophilia for 1 month.

Invasion phase: it can be described by abdominal pain, nausea and appetite disorders (anorexia or bulimia), neuro-vegetative disorders, diarrhea. She is sometimes silent.

Status phase: it is often asymptomatic, with anal pruritus at the time of exit from the segments. Various symptomatic forms: Nervosis: 3.5%, diarrhea: 6%, dizziness: 8 %, constipation: 10%, headache: 15%, bulimia: 17%, weight loss: 21%, asthenia: 25 %, nausea: 34%, abdominal pain: 35%, sometimes anaemia.

Complications: are very rare.

Diagnostics

Clinical diagnosis:

Clinical symptomatology is absent. The expulsion of tapeworm rings is sometimes reported by the patient or parents finding rings in their child's bed.

Biological diagnosis

The diagnosis of certainty is based on the detection of tapeworm rings or tapeworm eggs (embryophores). The rings are found in stool and laundry. Eggs

are found in the stool or on the anal margin ("scotch test") when the egg-filled rings are broken at the time of active passage of the anus.
Indirect diagnosis:
Its interest is restricted and therefore not used.

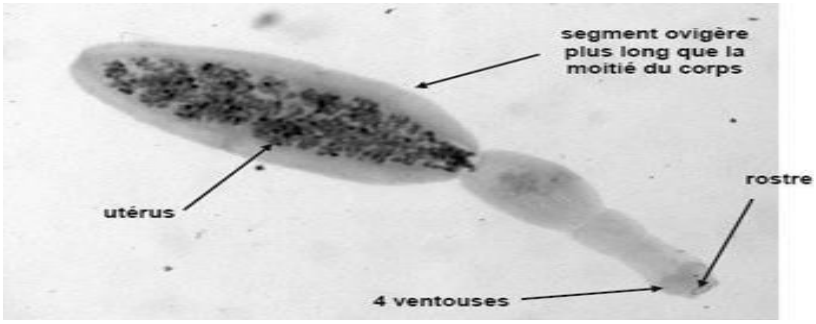
Processing
Chemotherapy: Niclosamide (Trédémine®); Praziquantel (Biltricide®)
Phytotherapy: Fresh squash seeds (30g crushed seeds with 30g honey) then castor oil purgation (for pregnant woman and child)

Prophylaxis
General prophylaxis: Veterinary control of slaughterhouses (search for cysticaria in butcher's meat), and faecal hygiene advice for farmers
Individual prophylaxis: Avoid eating raw or undercooked cattle meat and manual hygiene after handling meat

b. 2. Genus *Echinococcus*
Category
Branch of Plathelminths, Class of
Cestodes,
Order Cyclophyllidae
Family *Taeniidae* Genus
Echinococcus Species *E.*
granulosus
E. multilocularis
Sub-species *E.granulosus granulosus;E.granulosus equinus; E.granulosus borealis*
E.granulosus canadensis

Study ***E.granulosus granulosus***

Morphology
The **adult form is found (**see **Fig. 31 a**) in canids (small intestine). It can measure 4 to 8 millimeters long. The scolex has 4 suction cups and a rostrum with a double crown of hooks. The strobile consists of 3 to 5 proglottis. The first two proglottis are maturing; the third, longer than half the body, has formed genitals (oviger).The last two contain fertilized eggs. Cucurbitains each contain 300-800 eggs.
The compound vesicular type larva (hydatid) **(**see **Fig. 31b**) develops in humans, cattle, sheep, camelids, goats and determines hydatic**echinococcosis (** hydatic cyst). The vesicles are numerous and bulky. Hydatic larvae develop in the liver and lungs of sheep, cattle, camelids, goats and humans.



a

b

Figure 31: Photos showing an adult
(a) and a Hydatide (b) of *E. granulosus*

Evolutionary cycle (see. **Fig. 32**.
This Tenia whose reproductive cycle has multiplication phases in the larval state has a heteroxene cycle (indirect cycle). **HD** = canids (Adults are numerous and live a few months in the digestive tract). The externalization of the wall segments is done with the faeces; **HI** = herbivores including man, the larva is located at the level of the viscera (survival of the larva a few years). There is no externalization of the larva.
Cucurbitains each contain 300-800 eggs, are released with the dog's feces, and then ingested by herbivores. Man can be accidentally contaminated through soiled crudities or, if he touches a dog carrying the parasite (anal pruritus).
The worm crosses the intestinal wall and joins the liver of the herbivore and there, differentiates into a ball 20 centimeters in diameter: the hydatid stage. This stage develops slowly: 16 months in sheep and cattle; 18 months to 30 years in humans. In hydatid, there is a budding of scolex from the inner membrane (proliger): we can reach 400,000 scolex per cubic centimeter.

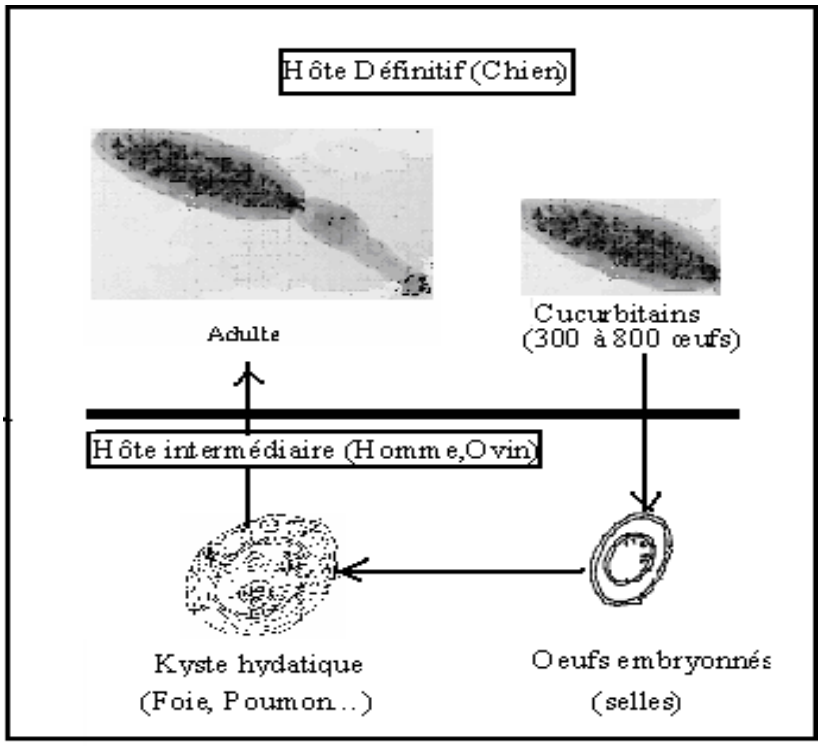


Figure 32 : Life Cycle of Echinococcal Tenia (*E.granulosus*)

Epidemiology

Hydatidosis, or hydatic echinococcosis or hydatic cyst is a zoonosis. Hydatidosis is a cosmopolitan parasitosis. It is widespread in regions where sheep (the sheep: the main intermediate host) and shepherd or vagrant dogs (the dog: the definitive host and reservoir of the parasite) coexist: mainly in South America, Australia and the Mediterranean basin, particularly in the Maghreb. In Algeria, a hydric index of 7 per 100,000 inhabitants is given. Human contamination is by ingestion of embryophores (contact with carrier dog) or water and/or food soiled by embryophores. The latter are very resistant in the external environment. Cysts (protoscolex) are also resistant (2 months at + 4°C and a few days at + 20°C, even in rotting meat).

Symptom

In dogs: asymptomatic and welltolerated.In humans: **primary echinococcosis**

The incubation and invasion phases are long (several years) and asymptomatic. The state phase: the symptoms are reminiscent of a cancerization of the infected organ.

From where we see signs of tumor pathology depending on the larval location:

Liver (60 to 80% of cases): jaundice, hepatomegaly, **lung** pain (20 to 30% of cases): cough, late hemoptysis.Kidney, spleen, nervous system, bone...

For peritoneal **secondary echinococcosis** is observed following spontaneous or induced rupture of the larva, where the scolex released by bursting form vesicles and reform as many hydatic cysts that can then appear anywhere in

the organism.

Evolution is slow and spontaneous healing is rare

Diagnostics

Clinical Diagnosis

* **Hepatic hydatidosis:** The latency period can last up to fifteen years. Clinical examination may reveal non-painful hepatomegaly and hypersensitivity reactions (urticaria, angioedema). The diagnosis can also be discussed during an abdominal imaging examination.
* **Pulmonary hydatidosis:** The clinical latency period is shorter. Single or multiple, rounded, opaque or hydroaeric pulmonary parenchymal opacities reveal the diagnosis on a chest radiograph sometimes performed systematically. Cough, dyspnea and hemoptysis complete the picture.

Biological diagnosis

Puncture biopsy of the liver should be avoided (risk of secondary echinococcosis by dissemination of protoscolex during puncture). The hypereosinophilia in this helminthiasis is inconstant and can be demonstrated more particularly on the occasion of a cyst rupture. Serological reactions (ELISA, immunoprecipitation, indirect immunofluorescence) make it possible to guide the diagnosis but latent cysts sometimes remain silent. The diagnosis of certainty is sometimes made by a parasitological examination of puncture fluid despite the prohibition to puncture, surgically extracted cysts, vomiting (sputum after rupture of a cyst in a bronchus). It then highlights the head of the adult worm (or scolex), or the scolex hooks.

Processing

In humans, there is a need for surgery: block extirpation of the hydatic cyst or hydatidectomy. During this operation, any risk of swarming must be avoided by not breaking the wall of the cyst. Complementary drug treatment in inoperable patients or to reduce the risk of secondary echinococcosis during the procedure can be carried out with ESKAZOLE ® (albendazole) Monitoring of the effectiveness of the treatment can be carried out by serology every three months.

Prophylaxis

General prophylaxis : It is based on the veterinary supervision of flocks of sheep and dogs (deworming treatment of domestic dogs, elimination of stray dogs, regulation of slaughterhouses).

Individual prophylaxis : the man must protect himself by individual measures (hygiene measures in case of promiscuity with dogs: hand washing, avoid contact of food with dogs). Education and health information have a very important role.

Genus *Diphyllobothrium latum*

- **Morphology:** *Diphyllobothrium* is a ribbon flatworm (class Cestodes, order Diphyllobothriidae, family Diphyllobothriidae) of about ten meters that can live several years in the digestive tract of man. It is responsible for a parasitic digestive infection called diphyllobothriosis.

Figure:Genus *Diphyllobothrium latum*

- **Life cycle** : The parasite's life cycle includes a definitive host
: humans (and other piscivorous mammals) and at least two intermediate hosts3: a planktonic crustacean and one or more freshwater fish. Under favorable environmental conditions, the eggs (45 x 65 µm) emitted in freshwater with the feces of the definitive host complete their maturation in 8 to 12 days and then hatch and release a ciliated embryo, the coracidium. It is ingested by a microscopic crustacean of the genus Cyclops or Eudiaptomus and transforms into a larva (called procercoid) within the general cavity. When a carnivorous fish ingests this planktonic crustacean, this larva transforms into a second type of larva (called plerocercoid**),** a few millimeters long. It becomes encysted in the musculature or viscera of the fish. Man and other piscivorous mammals then become contaminated by ingesting the raw or undercooked flesh of these freshwater fish. Once in the definitive host intestine, the plerocercoid larva grows several centimeters per day and the first eggs are released with the stool, about a month after the infestation. There are several species of this parasite, pathogenic to humans, but only the species D. latum can be contracted from metropolitan freshwater fish. However, cases of D. nihonkaiense (Pacific species) have been observed in consumers of salmon (Onchorynchus sp.) imported from the Pacific (Canada)

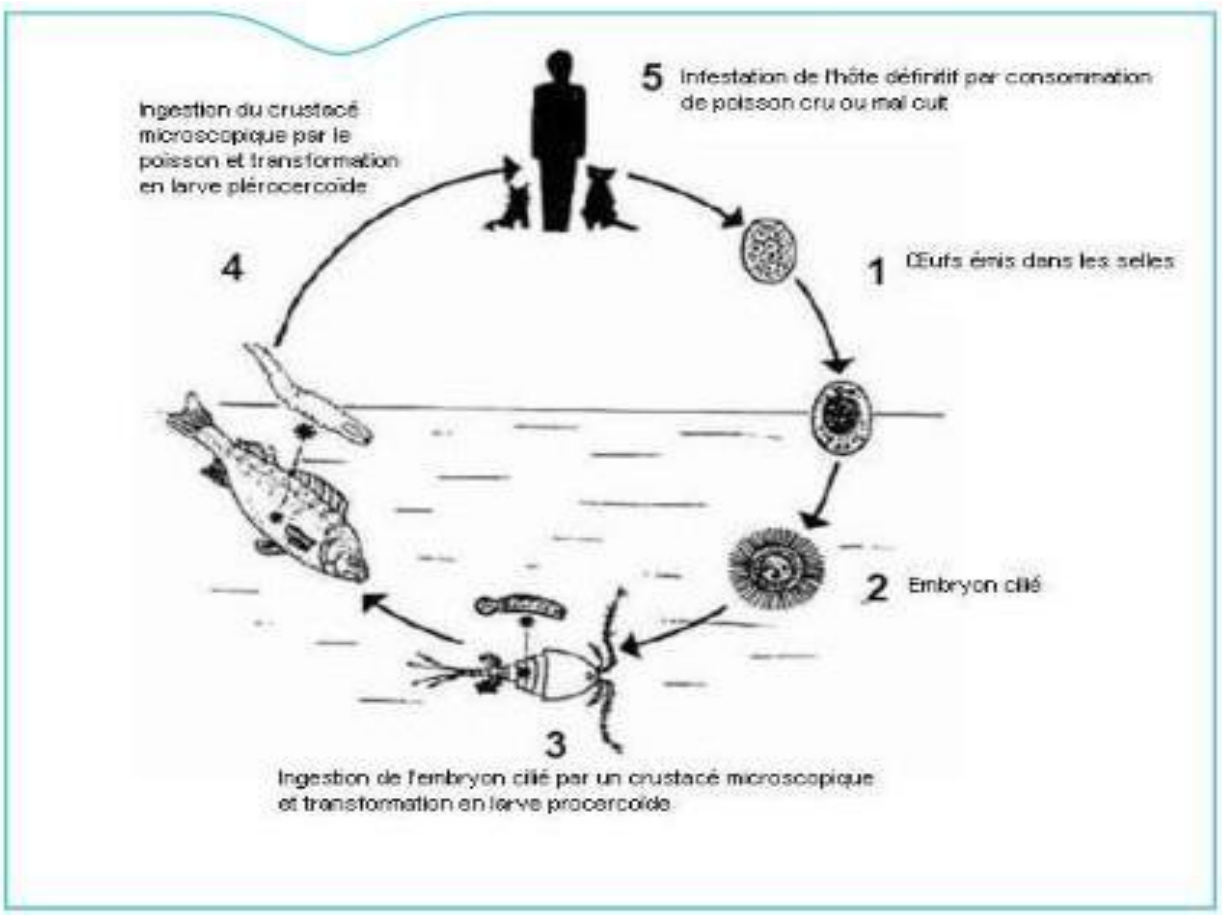


Figure: *Diphyllobothrium latum* life cycle (Rickard Ballweber. 2001)

- **Hazard sources**

The source of the hazard is freshwater or anadromous carnivorous fish (i.e. living in both freshwater and saltwater). The tank consists of the definitive hosts of the parasite (human, cat, dog, fox, etc.) and its intermediate hosts (carnivorous fish).

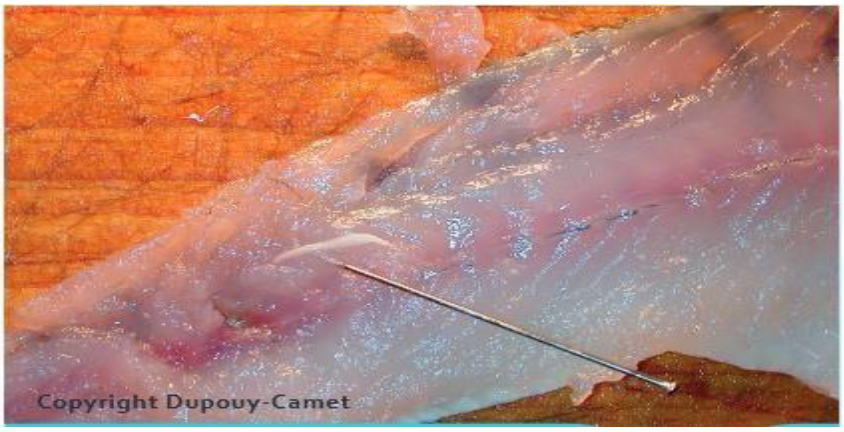


Figure: Plerocercoid larva on the surface of a pole net

- **Transmission channels**

This parasite affects piscivorous mammals and fish and is therefore the cause of zoonosis4. Human contamination is exclusively by ingestion of meat or eggs of fish eaten raw or undercooked. Eggs emitted with human feces are not directly contaminating

- **Epidemiology**

There is no surveillance system for diphyllobothriosis in France and Europe (except in Poland and the Baltic States).
Hazard sources
The source of the hazard is freshwater or anadromous carnivorous fish (i.e. living in both freshwater and saltwater). The tank consists of the definitive hosts of the parasite (human, cat, dog, fox, etc.) and its intermediate hosts (carnivorous fish).

Table 1. Disease characteristics

Durée moyenne d'incubation	Principaux symptômes	Durée des symptômes	Durée de la période contaminante	Complications
Un mois	Manifestations digestives : douleurs abdominales, diarrhées Signes généraux : asthénie, sensations vertigineuses, hyperéosinophilie	Toute la durée de vie du parasite dans l'intestin	Toute la durée de vie du parasite dans l'intestin	Anémie mégaloblastique exceptionnelle[6] Létalité nulle

Diphyllobothriosis is a cosmopolitan parasitosis, still present in Western Europe. It is decreasing in the Baltic or Scandinavian countries, which were the historical hotbeds of parasitosis. On the other hand, it seems to be emerging in the French- and Italian-speaking areas of the peri-alpine lakes where a professional fishery often exists.

- **Household Hygiene**

Recommendation for Consumer Action:
• The inactivation of the parasite is ensured by:
- baking throughout (60°C for ten minutes or 65°C for one minute). Pink cooking at the edge is insufficient to inactivate the potentially present larvae.
- freezing (-20°C for 24h) the fish in a domestic freezer.
• Wild fish should be eaten after cooking or freezing (-20°C for 24h).

C- Classs des Monogenea (or Heterocotyles) that develops on a single host. They are mostly ectoparasites and attach to the skin or gills of various aquatic organisms such as Pisces and Batrachians. They can however become secondarily endoparasitic and live in the urinary bladder of the frog.

Branch of Nemathelminths (Nematoda) General

Digestive parasitosis due to nematodes (or nemathelminths or roundworms) have a high prevalence in developing countries.

Digestive parasitoses due to roundworms, whipworms and hookworms are geohelminthiases transmitted as a result of soil contamination by infested human excreta. The groups most at risk of geohelminthiasis are those with particularly high micronutrient needs, namely preschoolers, school-age children and women of reproductive age. Geohelminthiasis is a neglected tropical disease.

Table: Zoological classification of nematodes and nematodoses

Nématodes (némathelminthes ou vers ronds)	Nématodoses
Cosmopolites à transmission orale : *Ascaris lumbricoïdes* *Enterobius vermicularis* *Trichiuris trichiura* *Trichinella spiralis* *Anisakis spp.*	 Ascaridiose Oxyurose Trichocéphalose Trichinose Anisakidose
Tropicaux à transmission transcutanée : *Strongyloïdes stercoralis* *Ankylostoma duodenale, Necator americanus*	 Strongyloïdose ou Anguillulose Ankylostomose

1. **General characters** :

- Metazoa, triploblasts, pseudocoelomata.
- Nemathelminths are present in almost all biotopes (marine, freshwater, terrestrial).
- Free or pests of plants and animals
- Fusioform body with bilateral symmetry and devoid of appendages.
- Non-segmented body.
- Cylindrical body (round worms) covered with a thick and impermeable cuticle involving growth by successive moults. This cuticle secreted by the epidermis has 4 thickenings, the epidermal fields. The dorsal field and the ventral field each contain a nerve cord, the 2 lateral fields each contain an excretory channel Under the epidermis is a layer of muscle cells with longitudinal myofibrils, the myoepithelial cells. The myoepithelial cells are in contact with the pseudocoele which contains the genital tract and which is limited on the internal side by the digestive tract.

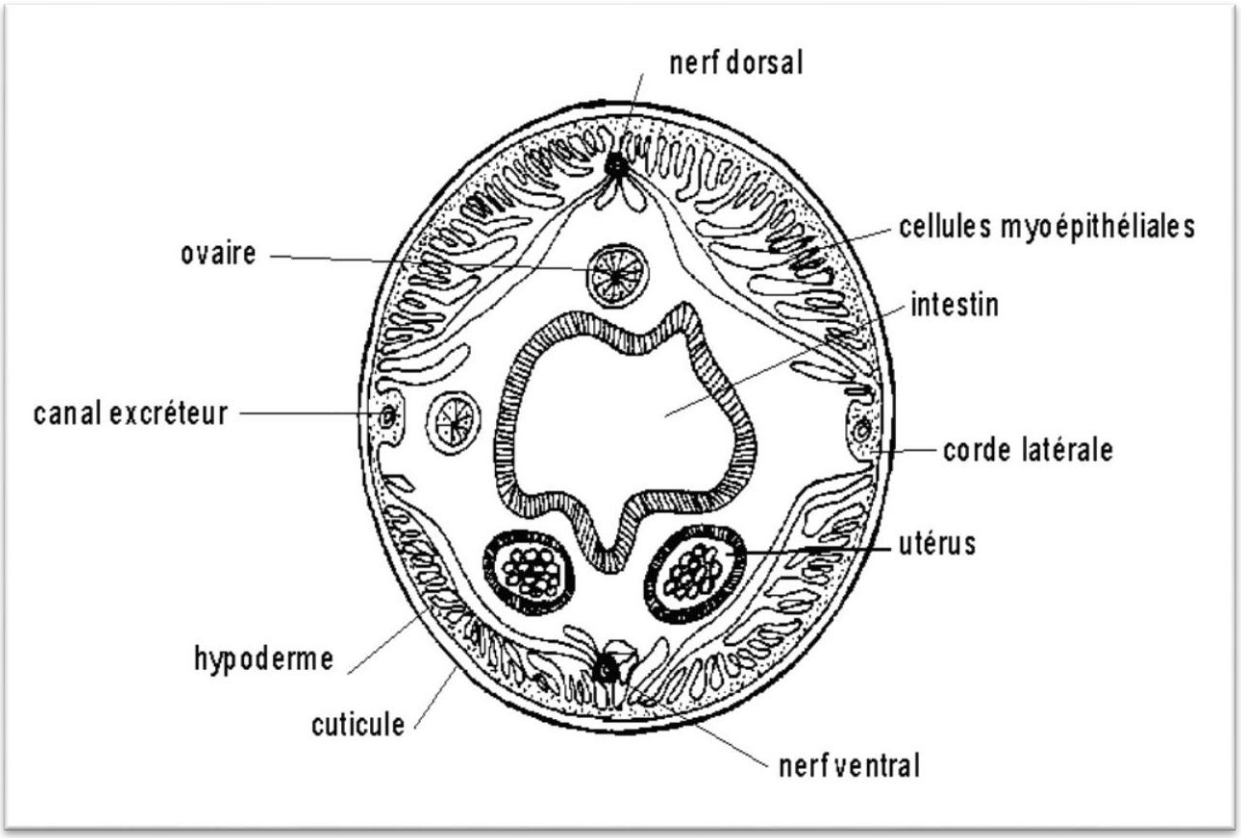


Figure Female Nematode Cross Section

- Respiratory and circulatory system are absent. Absence of eyelashes, locomotor organs and sense organs.
- The males are between 12 and 15 centimetres in size while the females are much larger.
- The digestive system is complete and rectilinear, from the mouth to the anus it is composed successively of the buccal capsule, the pharynx, the intestine and the rectum. - The female genital tract includes two filiform ovaries, also very long, two oviducts that converge into a uterus and a vagina that leads to the female orifice. The male genital tract consists of a filiform testicle, wound on itself and around the intestine.

It bulges in its terminal portion into a seminal vesicle, and emerges at the level of the cloaca.

- The excretory apparatus comprises two large lateral longitudinal channels, which join at their anterior end to lead to a single orifice placed behind the mouth.

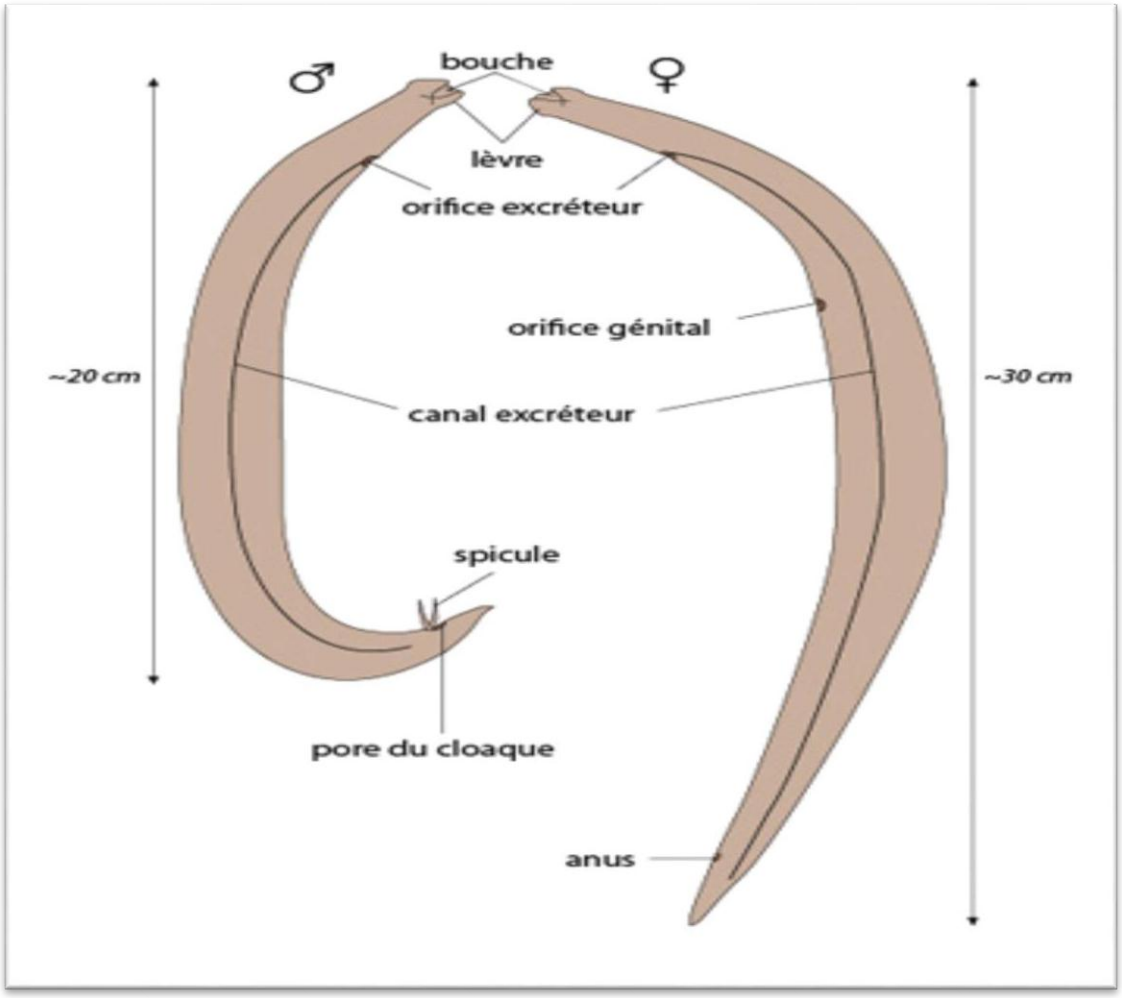


Figure Anatomy of a Nematode: Digestive, Excretory and Reproductive System

Systematic

According to the structure and organization of the digestive tract. There are two classes:

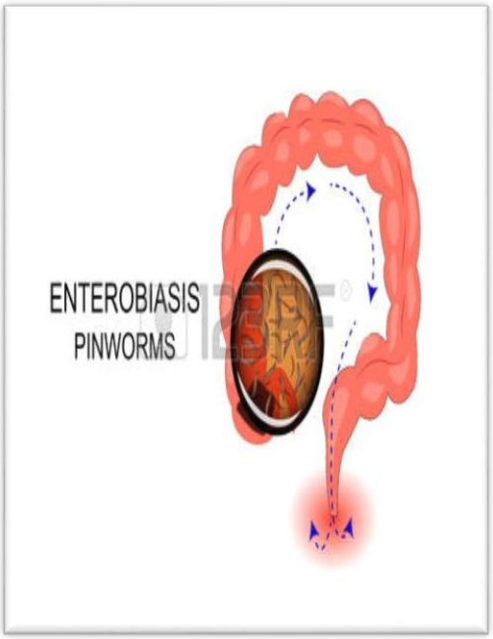
A. Class of Gordians very elongated roundworms with atrophied digestive tract. ex: Vinegar eel.

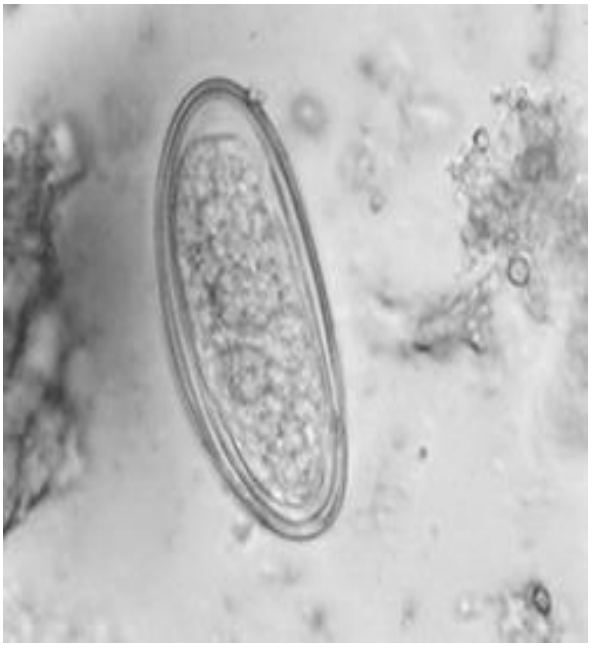
B. Class of Nematodes Complete digestive tract, this is the most representative. Parasitic nematodes of animals (e.g. Ascaris) and plants (e.g. Globodera).

b.1. Genus Enterobius (Oxyurosis)

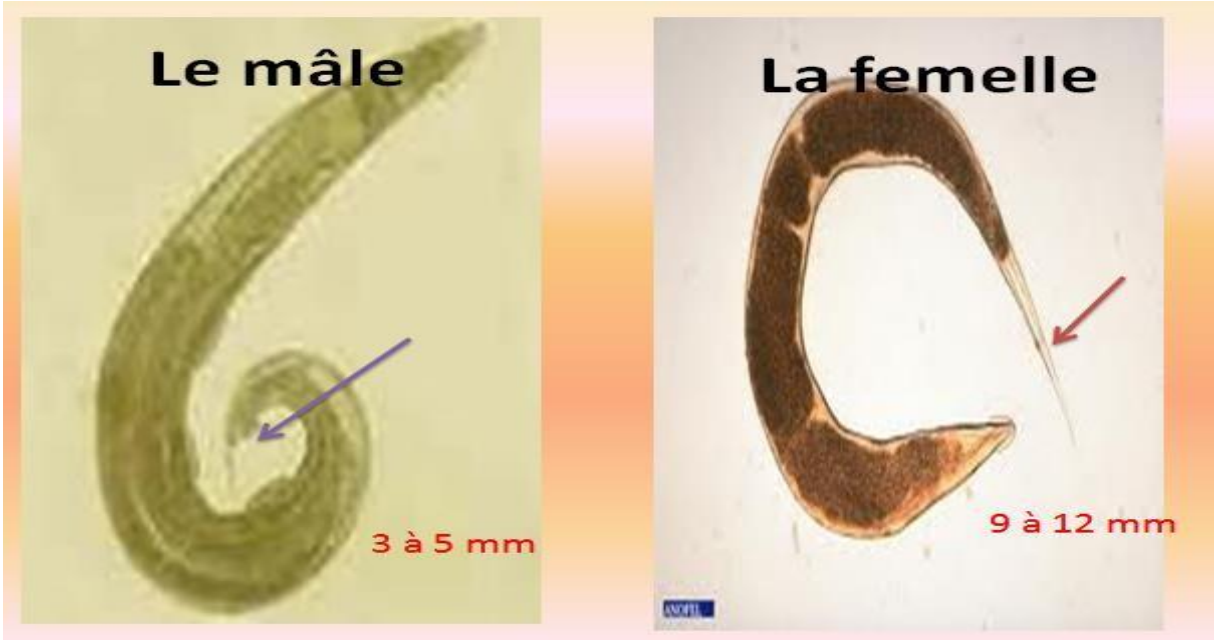
- **Overview**

Oxyurosis is due to a nemathelminth, *Enterobius vermicularis*. It is a cosmopolitan digestive parasitosis, reaching one billion individuals, very common both in temperate and tropical areas.





- **Epidemiology**



Life cycle

The cycle of oxyurosis is simple: swallowed eggs hatch in the stomach and duodenum, giving rise to larvae that reach the ileum and caecum. The adult larvae mate and the females migrate to the anus and lay eggs at the anal margin, which embryo within a few hours.

The parasite tank is the man alone. Transmission is direct from the anus to the mouth through the fingers, especially in children; indirect through objects and food. Inhalation transmission of embryonated eggs in dust is possible. Oxyurus is a parasitosis of life in the family, in the community, especially in children.

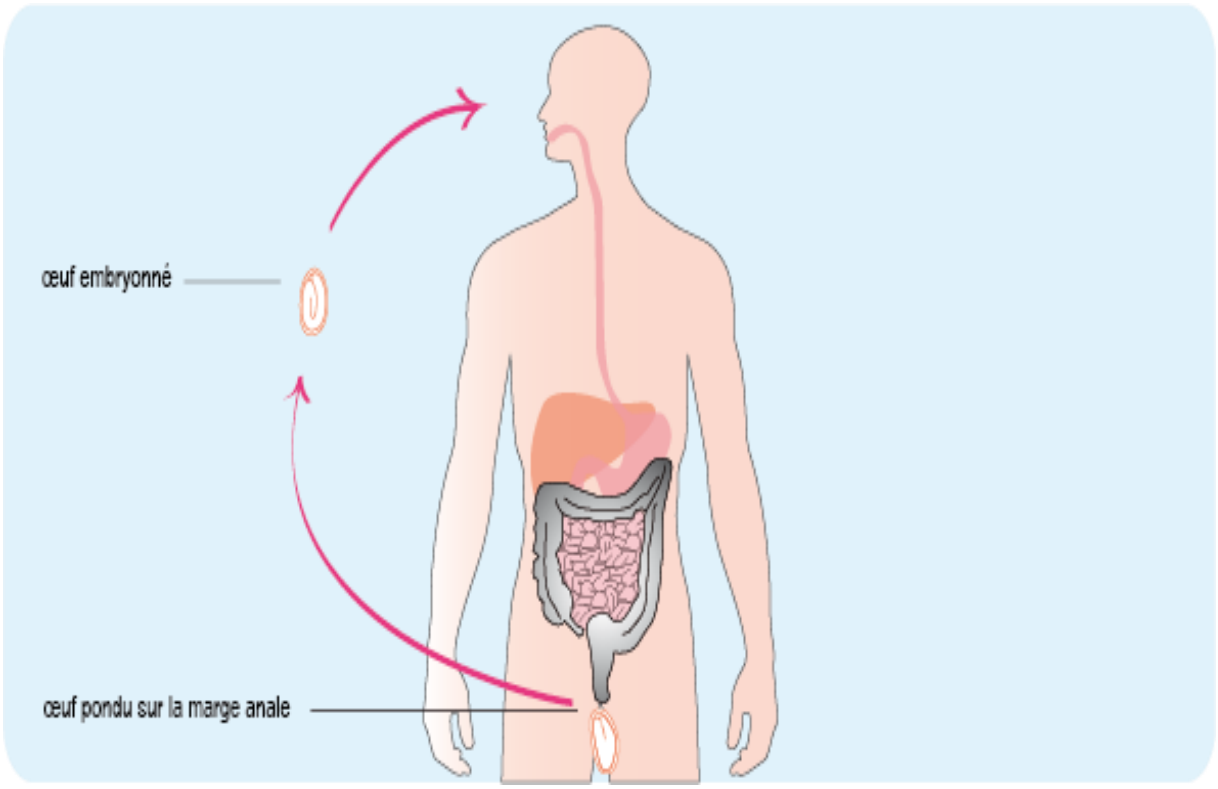


Figure: The oxyurosis cycle

- **Clinic**

Oxyurosis is often asymptomatic. The essential sign, if symptomatic, is anal pruritus especially in the evening and at night. It causes scratching lesions. Abdominal pain, sometimes diarrhea made of soft stools, wrapped in mucus, sometimes streaked with blood can accompany pruritus. Character changes in children are described (verminous syndrome).
Complications include genital and urinary manifestations in girls: vulvovaginitis, urinary colibacillus infections. Subacute appendicitis with intra-luminal pinworms is not exceptional.

- **Diagnostics**

It is parasitological:
- visualization of adult worms on the margin of the anus, in stool or underwear: presence of female pinworms, white worms, round, 5 to 12 mm long, - above all, search for eggs by the Graham scotch test method: presence of smooth, thick-walled, asymmetrical eggs, 50 to 60 µm long by 30 µm in diameter. There is no immunological diagnosis. Hypeosinophilia is possible, but still moderate.

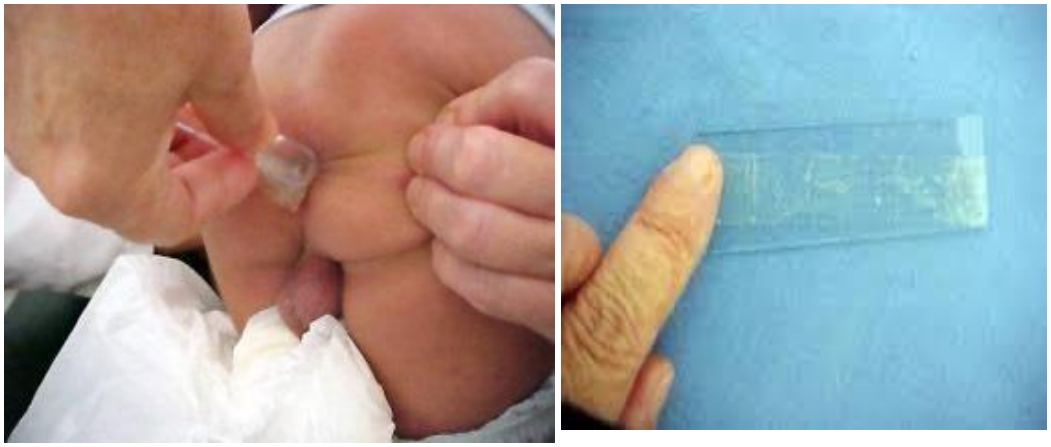


Figure: Graham's Scotch Test Method selon Rickard Ballweber. 2001

- **Processing**

It uses benzimidazoles: VERMOX® or FLUVERMAL® 100 mg/ 1 day, ZENTEL ® 400 mg/1 day. Alternative: pyrantel pamoate (COMBANTRIN®) 11 mg/kg/ 1 dose Pryvinium emboate (POVANYL ®) at a dose of 5 mg/d/3 days with the disadvantage of colouring the stool red. Regardless of the drug prescribed, a 2nd course must be administered on D15. In case of recurrence, the whole community should be treated with VERMOX® or FLUVERMAL®, 2 tablets at 100 mg for 3 days, then 1 tablet every 15 days for 3 months.

- **Prevention**

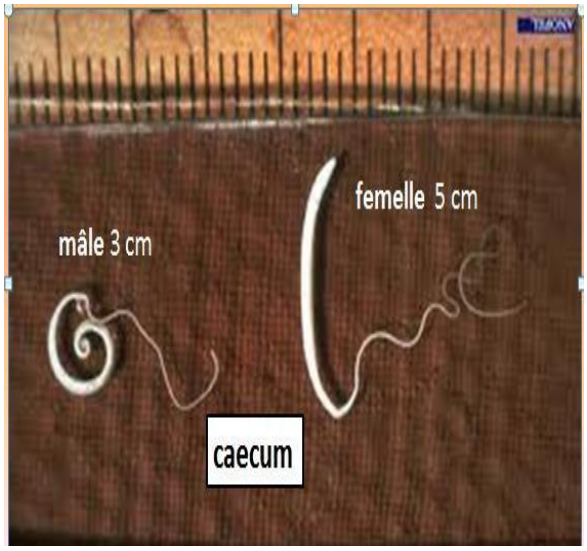
The essential fight is against direct transmission (self-infection): it consists of regular hand washing and nail cleaning, wearing closed pajamas for the night, maintenance of bedding, underwear, child's objects (especially toys). The fight against fecal danger is secondary here.

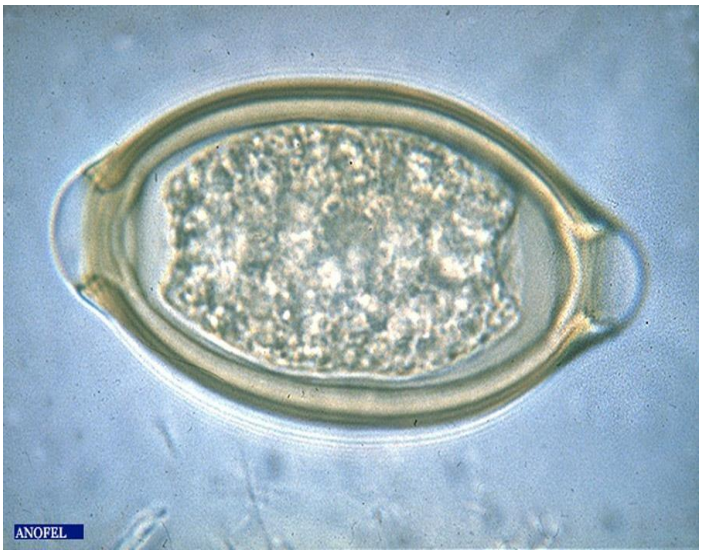
- **Genus *Trichiuris trichiura* (Trichocephalosis)**
  - **Overview**

Trichocephalosis is caused by a nematode, *Trichiuris trichiura*. It is a cosmopolitan intestinal parasitosis most often asymptomatic, but which, in case of massive infection, can lead to serious manifestations in young children.

  - **Epidemiology**

Adult worms live at the colon. They are stuck by their filiform anterior end in the intestinal mucosa, especially at the level of the cecum, due to the relative stercoral stagnation at this level. They feed on blood. The eggs are eliminated in the stool and embryogenize in the external environment. Man becomes contaminated by ingesting embryonated eggs with food or by hands soiled by the earth. The embryo gives in the small intestine a larva that will settle in the colon where it becomes an adult in 4 to 5 weeks. An adult worm absorbs 0.005 ml of blood per day. Massive infections (more than 1000 worms) lead to anemia.
According to the who, there are 500 to 800 million affected subjects. It is disadvantaged populations, and in particular children aged 2 to 7 living in poor hygiene conditions, who are mainly affected.





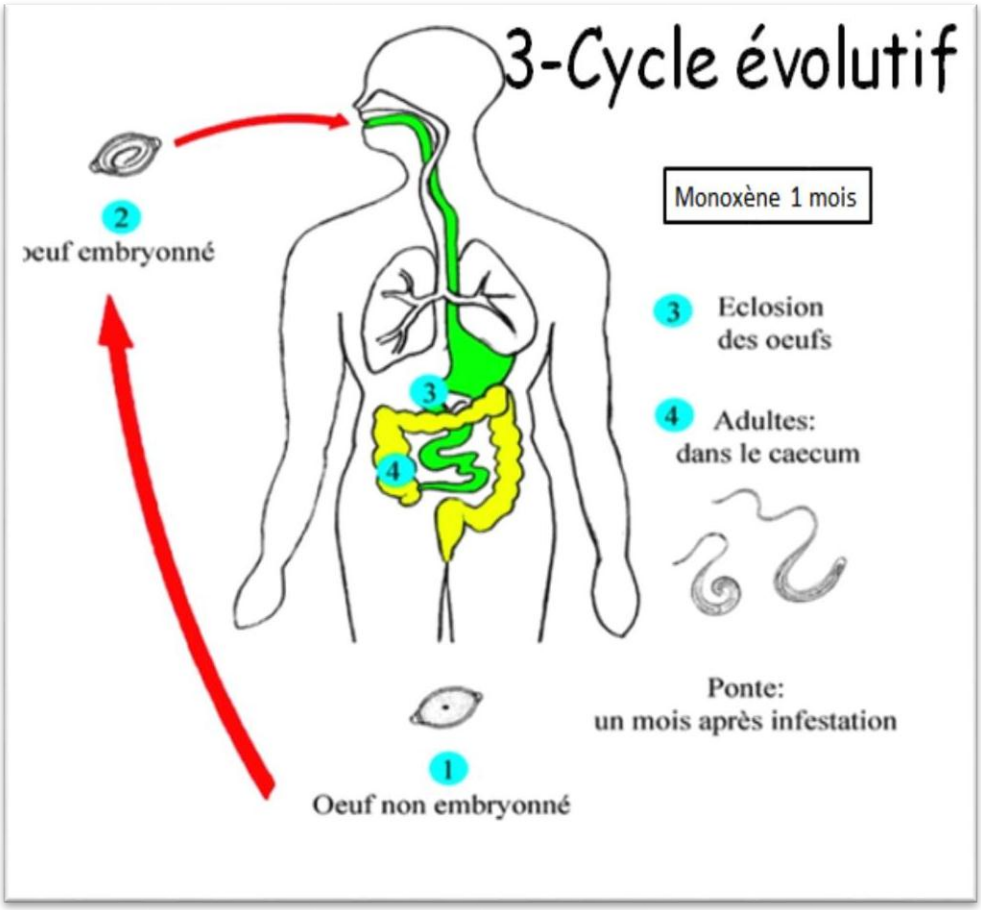
- **Clinic**

There are 2 phases:

- **A still asymptomatic invasion phase** , with blood hypereosinophilia as the only stigma.
- **A state phase or intestinal phase** where clinical expression is a function of parasite load:
  - ✓ **Asymptomatic**, the most common form,
  - ✓ **Symptomatic or trichocephalosis disease**, uncommon: it is mainly found in children or immunocompromised adults. There are two forms:

    - the minor form is characterized by dyspeptic syndrome with nausea and flatulence, often associated with constipation, weight loss and "verminous syndrome",

    - the major form is **infantile massive trichocephalosis,** linked to massive involvement of the colon, from the caecum to the rectum. It reaches children from 2 to 7 years old and produces several clinical pictures: trichocephalic enteritis with abdominal pain and diarrhea leading to dehydration; trichocephalic appendicitis; trichocephalic recto-colitis producing a dysenteric syndrome with tenesmus and perforations. Rectoscopy makes the diagnosis by showing many worms plugged into a hyperhemic mucosa with a hemorrhagic sting and sometimes ulcerations. This rectocolitis can be complicated by profuse rectal haemorrhages and rectal prolapse lined with white filaments (adult worms) stuck in a haemorrhagic mucosa. This severe form is accompanied by hypochromic, microcytic, hyposideremic anemia.



- **Diagnostics**

The diagnosis is parasitological: presence of characteristic eggs, ovoid in shape, provided at their two poles with translucent mucous plugs. They are surrounded by a thick dark brown shell. They measure 50 µm by 22 µm. Their 'little lemons' look makes them easily recognisable under the microscope. The diagnosis is also endoscopic by highlighting adult worms in the rectum and/or caecum.

- **Treatment** It uses benzimidazole: mebendazole or flubendazole 200 mg/d x 3 days regardless of age, or albendazole 400 mg/d x 1 day in adults and children over 2 years, 200 mg/d x 1 day in children under 2 years. A second course of treatment is prescribed on D15. Nitazoxamide is a therapeutic alternative.
- **Prevention:** Fecal peril disease, prophylaxis is identical to that of ascariasis.

➢ **Genus *Trichinella spiralis* (Trichinosis)**

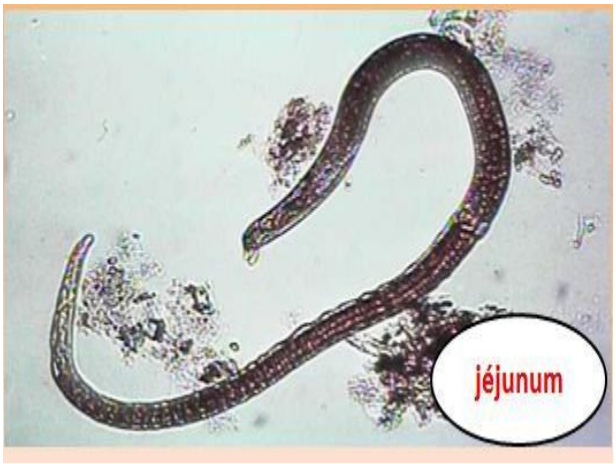
➢ **Overview**

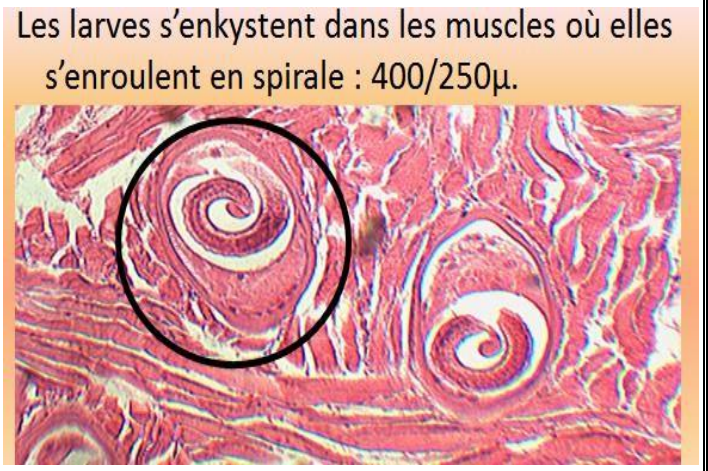
Trichinosis is a very particular cosmopolitan nematode due to *Trichinella spiralis (T. nelsoni* in East Africa): it is a parasitic stalemate due to the ingestion of contaminated meat. Only the migration phase of adult females leads to digestive symptomatology.

➢ **Epidemiology**

Trichinella spiralis is a small round worm, 3 mm long for the female and 1.5 mm for the male. The main reservoir of parasites is the pig, but many domestic or wild carnivores can be infected (bears, warthogs, wild boars, seals, rodents). Contamination occurs through the ingestion of meat infested with larvae of

trichinae, consumed undercooked. The larvae are released into the stomach after lysis of their wall by the gastric juices. They become adults in the small intestine. After mating, the fertilized females sink into the intestinal mucosa and lay expelled eggs in the form of larvae which pass into the lymphatic and then blood circulation, reach the right heart, the pulmonary circulation, the left heart, then are disseminated in the body where they will fixate and encyst in the muscles 10 to 20 days after contamination. The disease is rife in Central Europe, North America, Southeast Asia, Africa (consumption of "bush" meat), in the polar regions (consumption of bear or seal meat). In metropolitan France, epidemics are regularly reported, due to the consumption of imported meat; there are still some indigenous cases due to wild boar meat.

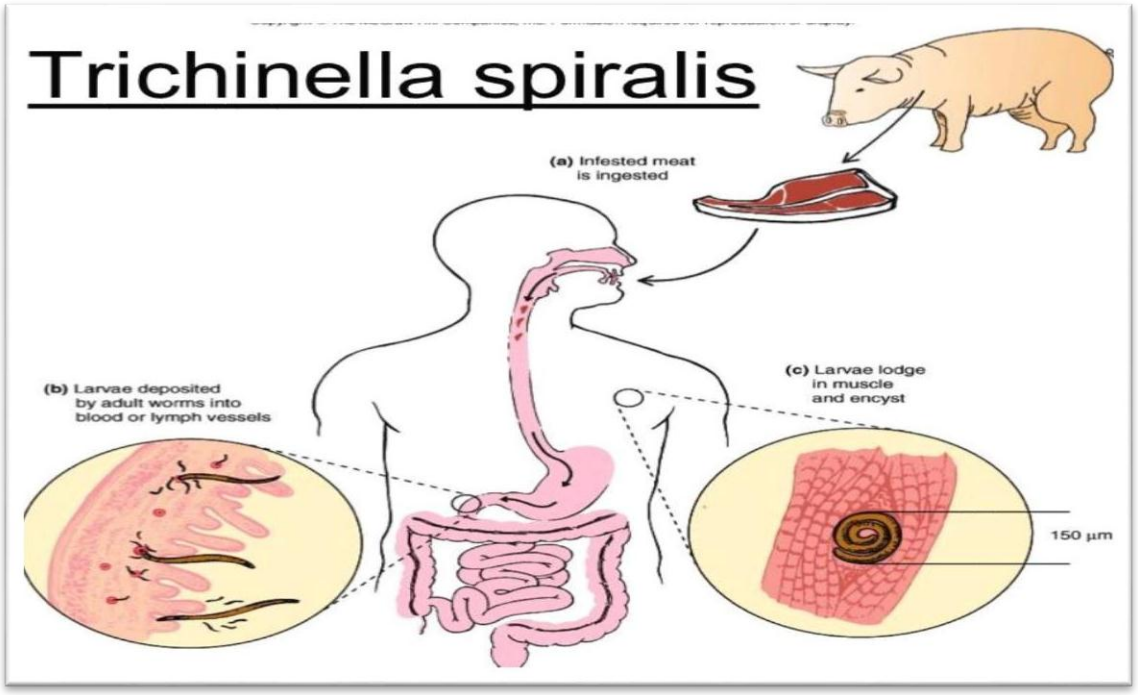




- **Clinic**

The symptomatology of trichinosis varies according to the stage of the parasite's evolution. - In the migration phase of adult females (intestinal phase), it leads to digestive disorders: violent abdominal pain, diarrhea, vomiting, with a high fever at 39 - 40°C.

- In the larval dissemination phase, between the 10th and 20th day, it is characterized by a marked impairment of the general state with plateau fever, very painful myalgia, edema of the face (disease of the large heads), bilateral conjunctivitis. It is at this stage that serious, sometimes fatal complications can occur: myocarditis, encephalitis, glomerulonephritis, acute pulmonary edema. Thereafter, muscle pain persists for several months.



- **Diagnostics**

The diagnosis is evoked on hypereosinophilia (50 to 65% of leukocytes), a biological inflammatory syndrome and an elevation of muscle enzymes (LDH, CPK). The diagnosis of certainty is provided by immunology: ELISA and Western blot which detect specific antibodies 3 weeks after the onset of the disorders.

- **Processing**

It uses albendazole (ZENTEL ®), 800 mg/d in 2 doses in adults, 15 mg/kg in 2 doses in children, for 10 days, under cover of corticosteroids (prednisone) 1 mg/kg for 48 hours, then decreasing doses for 10 days. **7.6. Prevention.** It is based on veterinary checks and food hygiene: consumption of well-cooked meats.

- **Genus *Ankylostoma duodenale* and *Necator americanus* (Ankylostomosis)**
  - **Overview**

Hookworm or hookworm is a cosmopolitan parasitosis that has become tropical. One billion individuals are infected. Two nematodes cause human hookworm: *Necator americanus and Ankylostoma duodenale*.

Necator americanus affects tropical regions: Sub-Saharan Africa, Indian Ocean, India, China, Southeast Asia, Central and South America.

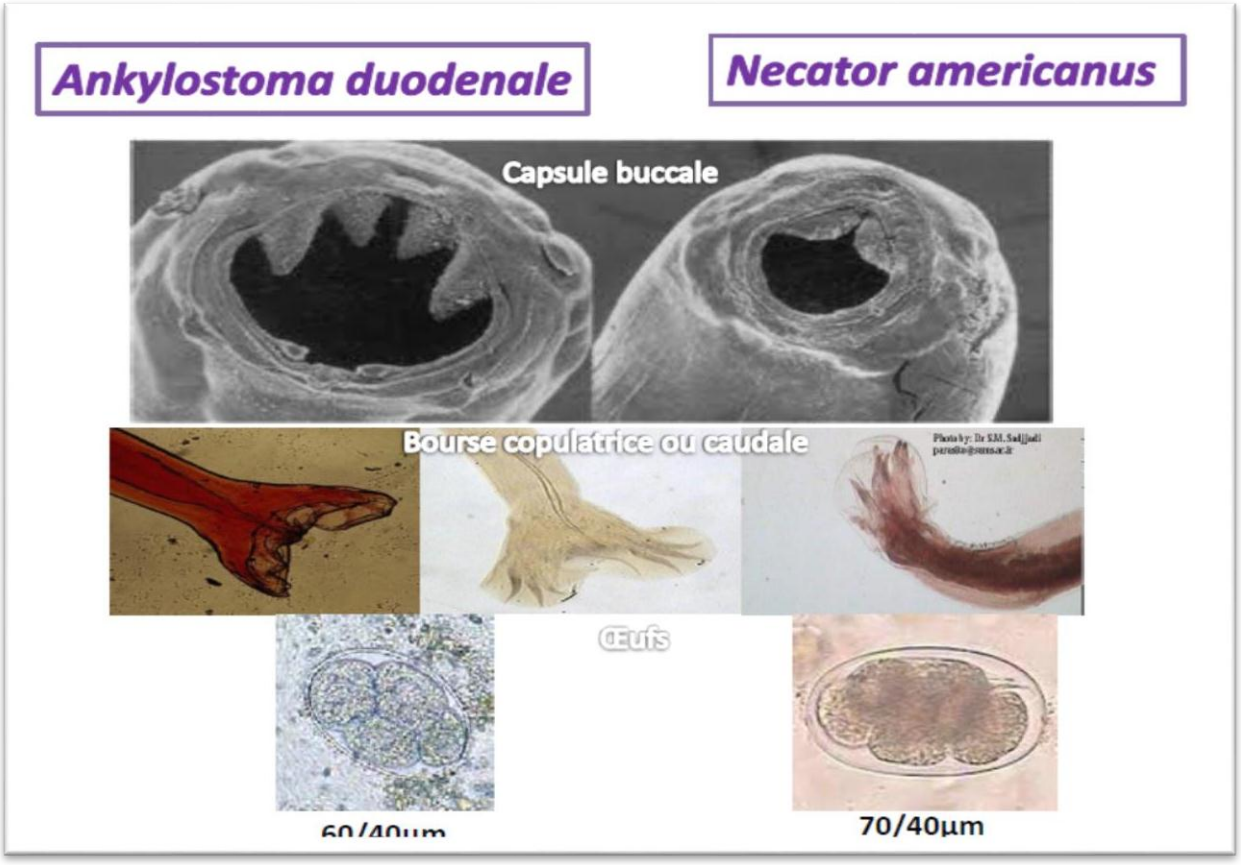
Ankylostoma duodenale interests the same tropical regions, but also temperate regions: North Africa, southern Europe, northern India and China.

  - **Epidemiology**

Hookworms are roundworms that are about 10 mm long. Adults live in the human duodeno-jejunum. They are attached to the intestinal mucosa by the sharp blades and hooks of their oral capsule with which they graze on the mucosa and cause bleeding. They therefore result in calculated blood losses of 0.01 to 0.04 ml/worm/day, i.e. 30 ml/day for Necator americanus and 0.05 to 0.3 ml/worm/day, i.e. 140 to 400 ml/ day for Ankylostoma duodenale.

Hookworms are exclusively human parasites, with no intermediate host. Eggs emitted in the stool are non-embryonic. They will embryonate in the external environment under certain temperature conditions: 22 to 26°C for Ankylostoma

duodenal, 27 to 30°C for Necator americanus, which explains the geographical distribution and cases of hookworm described previously in temperate regions (mines, tunnels).
The embryos transform into infected L1, then strongyloid L2, then L3 rhabditoid larvae. They are very resistant in the outdoor environment: 2 to 10 months on the ground, 18 months in water. Contamination occurs in soil, sludge and fecalized fresh water, most often at the feet. The L3 larvae penetrate the skin, reach the heart, lung and trachea by blood or lymph before being swallowed in the digestive tract. In the duodenum, the larva transforms into an adult worm. The cycle lasts between 50
and 60 days.
Transmission is exclusively cutaneous for Necator americanus, cutaneous, but also possible via buccal, transplacental or during breastfeeding for Ankylostoma duodenale.

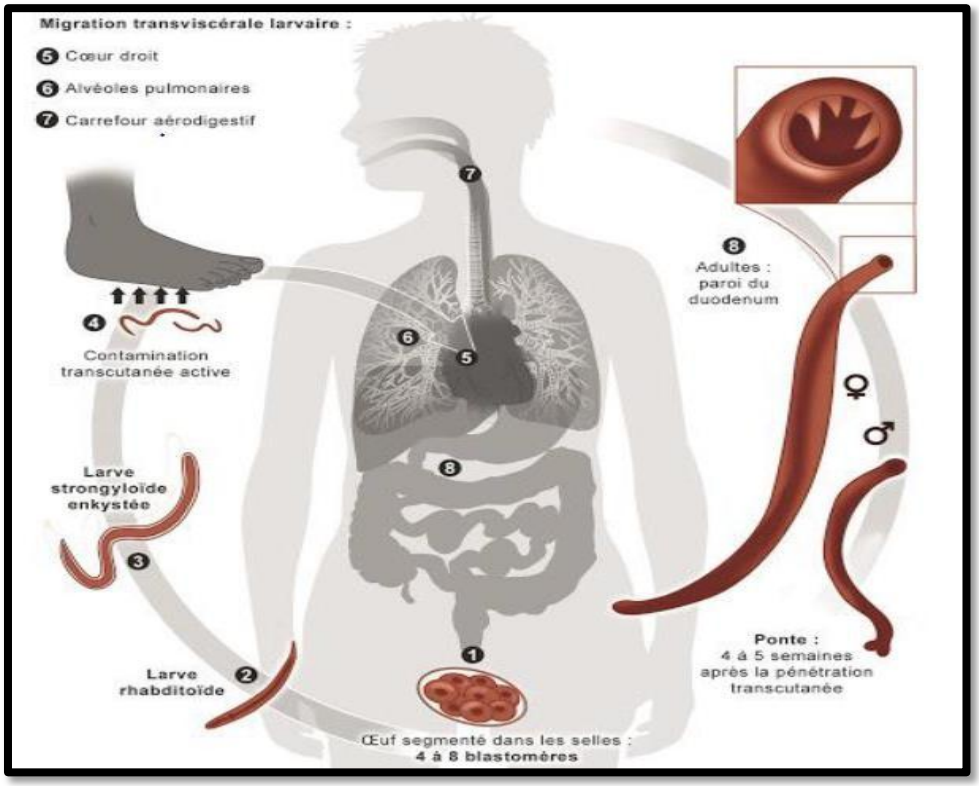


- **Clinic**

Three clinical phases related to the cycle are described: skin penetration, larval migration and action of adult worms at the intestinal level

- ✓ Skin penetration phase: it is pruritic dermatitis, a fleeting maculopruritic erythema that is seen during the primary invasion (expatriates in tropical areas, minors in temperate areas). A chronic infection leads to the "gourm" of minors.

- ✓ Larval invasion phase: larvae cause irritation of the upper airways or catarrh of gourms, allergic manifestations: dyspnea asthmatiform, Loëffler syndrome.
- ✓ Intestinal phase with digestive disorders and anemia - digestive disorders: epigastric pain, abdominal heaviness or bloating, painful feeling of hunger, pseudo-ulcer pain with pyrosis resulting in geophagy (ingestion of soil), diarrhea made of 5 to 10 stools per day,
- ✓ anemia with asthenia, exertional dyspnoea, tachycardia, palpitations, systolic murmur, mucocutaneous pallor, dizziness, soft nail changes, flattened (onychomalacia).
  When the anemia is severe, soft edema, taking the bucket, due to hypoprotidemia with hypoalbuminemia, appears.



- **Diagnostics**
  - **Guidance Elements**

- Complete blood count;
- microcytic anemia, hypochromic, hyposideremic, aregenerative, hemoglobin level may be less than 3 g/dl,
- hyperleukocytosis due to maximum hypereosinophilia in the 3rd month (60 to 80% of the leukocyte formula), then the curve bends and slowly returns to normal (1 to 2 years) - Hypoprotidemia, hypoalbuminemia.
- Serum zinc falls.

  - **Elements of certainty:**

Parasitological examination of stool (direct examination, concentration techniques)
- Evidence of eggs with characteristic appearance: ellipsoids, symmetrical, thin and transparent shell, 60 µm by 40 µm, containing 4 blastomers (Ankylostoma

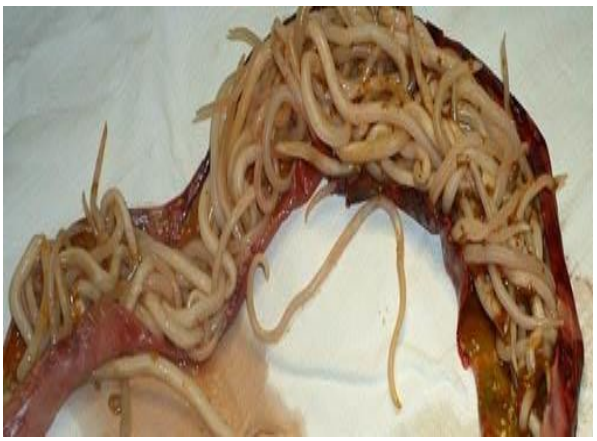
duodenal) or 8 blastomers (Necator americanus) - Egg count: it makes it possible to judge the extent of the infection (moderate infection: 2,000 eggs/g stool, average infection: 2,000 to 10,000, severe infection >
10,000 - The stool examination must be done quickly within 3 hours, otherwise there is continued segmentation of the blastomers and the 2 hookworms cannot be differentiated from each other; within 24 hours, otherwise there is transformation into rhabditoid larvae, then strongyloids and ankylostomosis and anguillulose cannot be differentiated.
Note that upper digestive endoscopy shows edematous, purpuric duodenitis and in a few rare cases adult worms stuck on the mucosa. 3.5. Processing
The antiparasitic treatment uses benzimidazoles: mebendazole (VERMOX®) or albendazole (ZENTEL ®) at the same doses as for ascaridiosis).
Pyrantel, in the form of emboate (HELMINTOX®) or pyrantel pamoate (COMBANTRIN®) is active at a dose of 20 mg/kg in 1 or 2 doses for 3 days. A few cases of treatment resistance have been reported with mebendazole for N.
americanus and with pyrantel for A. duodenale. Alternative therapies could include combination therapies such as mebendazole 500 mg + levamisole (SOLASKIL®) 80 mg once or albendazole 400 mg + ivermectin 200 µg/kg once.
Treatment of anaemia involves oral ferrous sulphate: 200 mg/d in adults, 10 mg/d in children under 30 kilos for 3 months and folic acid, 20 mg/d for 3 weeks. If the haemoglobin level is < 5 g/dl, globular pellets are prescribed.

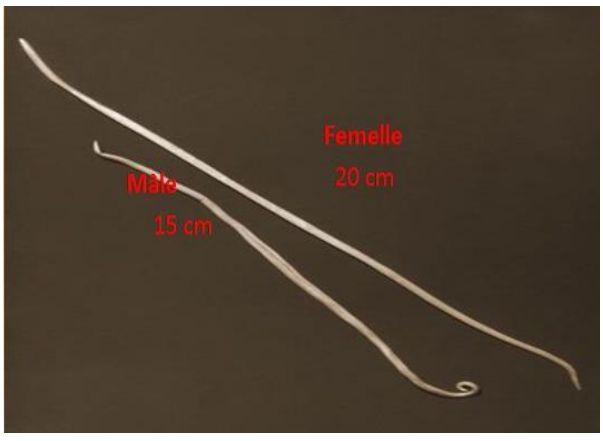
- **Prophylaxis**

Fecal peril disease, the prophylaxis of hookworm is identical to that of ascariasis. In addition, individual prevention requires walking with shoes.

- **Genus *Ascaris lumbricoids***

It parasitizes the large intestine of humans by causing ascariasis, the general symptoms of which are gastroenteritis disorders (inflammation of the gastric and intestinal mucous membranes), slimming and sometimes nerve attacks. It is not uncommon to see intestinal obstruction due to the accumulation of roundworms.



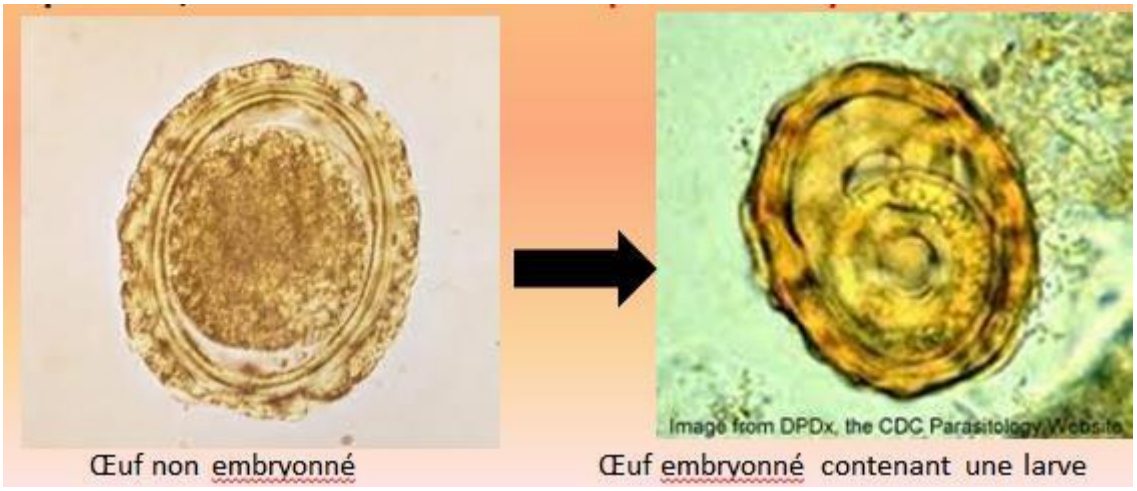


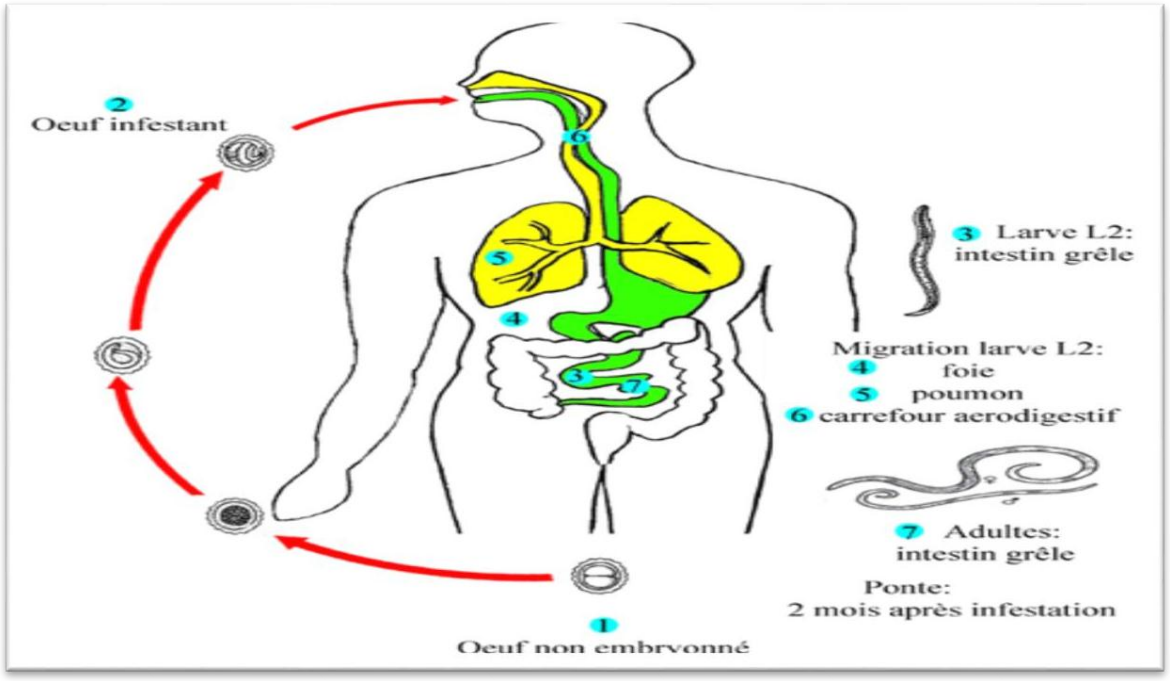
- **Epidemiology**

Ascaris lumbricoids is a round worm, 12 to 30 cm long by 2 to 4 mm in diameter for the male, 20 to 35 cm by 3 to 6 mm for the female. He lives in the jejunum of man. The number of hosted worms is variable, possibly exceeding one hundred. The prevalence is at its maximum in children between 4 and 14 years of age.

The evolutionary cycle is simple, direct, with no intermediate host. Fertilized females lay discarded eggs in the outdoor environment where they embryo under conditions of high temperature (28 to 32°C) and high humidity.

Eggs ingested, with drinking water, vegetables, soiled fruits, soil, even inhaled mixed with dust, release larvae that pass through the intestinal wall, reach the liver through the portal vein, then the right heart, artery and pulmonary capillaries in 3 to 4 days. Then, after a week, they cross the alveolo-capillary wall, pass into the tracheobronchial tree, are swallowed and arrive at the jejunum and transform towards adults. Six to eight weeks later, the females begin to lay eggs. The cycle lasts a total of 60 to 90 days.





- **Clinic**

Ascariasis is frequently asymptomatic. When it is symptomatic, it takes place in 2 phases:

- **The invasion phase:** it is characterized by immunoallergic manifestations due to the passage of the larva. **Loëffler syndrome is** the most classic manifestation. It results in dry cough, dyspnea, mucosal sputum and a subfebrile state. Chest radiography shows labile opacities, varying in size and number, disappearing without sequelae within a few days. Hypeosinophilia (HE) draws a "bow stroke" curve, called **the Lavier curve.** EH is maximum at the end of larval migration (eosinophils then represent between 30 and 50% of leukocytes).
- **The state phase** : it is due to the presence of adult worms in the intestinal lumen. It causes pseudo-ulcerous abdominal pain, nausea, vomiting, diarrhea or dyspeptic syndrome. The rejection of adult worms during vomiting can then provide the diagnosis. In children, a "verminous syndrome" is described, mainly due to irritability.
- **Digestive complications** are the severity of ascariasis:
- **intestinal occlusion** due to a mechanical action by the presence of bundles of roundworm in the small intestine. There are two types of occlusion: subacute occlusion and acute occlusion. Subacute occlusion is the most common. It causes abdominal pain, vomiting, fever. The abdomen is distended and sensitive. Radiography of the abdomen without preparation shows a zebra-like appearance characteristic of the presence of many roundworms in the small intestine. The abdominal ultrasound shows a swirling appearance in a small loop dilated by the presence of many roundworms. First-line treatment is medical. Acute occlusion results in severe abdominal pain with vomiting and fever. The abdomen is weathered, very painful. There is a risk of perforation. Surgery is required.

- **acute appendicitis** due to the presence of an adult roundworm in the appendix,
- **ascarid peritonitis**, complication of an occlusion or appendicitis or due to another cause: typhoid fever, amoebiasis, tuberculosis.
- **biliary and pancreatic**ascariosis, cause of hepatic colic, acute angiocholitis, acute cholecystitis, acute pancreatitis. Hepatic colic is due to the presence of eggs or adult worms in the bile ducts, which promotes the formation of a calcium-pigment lithiasis with the presence of intra- and extra-hepatic stones, not of interest to the gallbladder. Acute angiocholitis is due to the intra-papillary isolation of an adult worm or its migration into the bile ducts. Acute cholecystitis is due to the migration of an adult worm into the cystic duct. Acute pancreatitis is due to the presence of an adult worm in Vater's papilla or in the Wirsung canal. In addition, there is an impact of ascariasis on **malnutrition in school-aged children** with stunted growth.

- **Diagnostics**

Diagnosis in the invasion phase is a presumptive diagnosis, based on hypereosinophilia and serology, the interest of which is very limited by cross-reactions with

other parasitoses. In the state phase, 2 to 3 months after contamination, the diagnosis is parasitological:
- Parasitological examination of stool in the fresh state and after concentration (Ritchie, Bailenger, Junod, Kato techniques): presence of oval and symmetrical eggs from 50 to 80 µm x 35 to 55 µm, thick outer shell and dark brown nipple, smooth inner shell, colorless and very thick, when fertilized.
- Adult worms rejected in vomiting
- Upper digestive endoscopy: presence of adult worms in the duodeno-jejunum, sometimes enclosed in Vater's papilla
- Abdominal ultrasound in case of biliary obstruction or ascarisiosis: shows linear echogenic structures in the biliary tree, without a shadow cone. At the level of the pancreas, the sign of the "four lines" is described, consisting of 4 echogenic lines, the 2 outer lines corresponding to the walls of the duct and the 2 inner lines at the limits of the body of the ascaris. - Retrograde Cholangiopancreatography (CPRE). It aims in case of acute angiocholitis of adult worms in the bile and pancreatic ducts and allows their extraction.

- **Processing**

It uses benzimidazoles: mebendazole (VERMOX®), flubendazole (FLUVERMAL®) albendazole (ZENTEL®). They are active in the intestinal lumen. The reference drug in developing countries is mebendazole (VERMOX®) at a dose of 200 mg/d x 3 days. Pregnant women are prescribed pyrantel pamoate (COMBANTRIN®) or pyrantel emboate (HELMINTOX ®), 11 mg/kg 1 dose x 3 days.

- **In case of complications:**

- intestinal obstruction is treated in the first line by: nasogastric tube, hydroelectrolytic rehydration, antihelminthics and antibiotics. If the signs persist at the 24th hour, it is necessary to operate.
- biliary and pancreatic ascariosis is treated as a first-line treatment with antispasmodics, anthelmintics and antibiotics. ERCP, if available, with sphincterotomy and naso-biliary drainage is performed or, failing that, surgery: cholecystectomy, choledocotomy with sphincterotomy, extraction of worms and, if present, stones.

- **Prevention**

Ascariasis is a fecal peril disease. Prevention is collective: construction of latrines, wastewater treatment, prohibition of the use of human fertilizers

3.2. Phylum the arthropods

3.2.1. General data

Arthropods refer to all animals with locomotor appendages formed of several articulated parts and whose segmented body (metameres) has a rigid substance called chitin. This invertebrate branch encompasses by far the largest number of species and individuals in the animal kingdom (more than half of known species). It is also the most diverse on the planet: their shapes and sizes vary considerably from one group to another but they share a number of common traits. By colonizing all ecosystems - aquatic, terrestrial and aerial - arthropods have demonstrated a formidable ability to adapt. Namely, the term arthropod comes from the Greek arthron (joint) and podos (foot).

3.3.2. Hexapoda subphylum

- **General Anatomy**

Hexapods are composed of three parts: the head, thorax and abdomen. They get their name from the fact that they all have three pairs of legs located on their thorax. The second pair of antennas has disappeared and they present on the thorax 2 pairs of wings which may however be absent.

The thorax is composed of three segments, each of which bears a single pair of legs. The abdomen is formed by a maximum of eleven segments. In general, the abdomen does not have appendages but some remain (diploma styles, springtail furcas, etc.).

Breathing uses a trachea system, which is done through the stigmas, small orifices placed on the sides of the abdomen and thorax.

Excretion is done by ectodermal derivatives, Malpighi tubes.

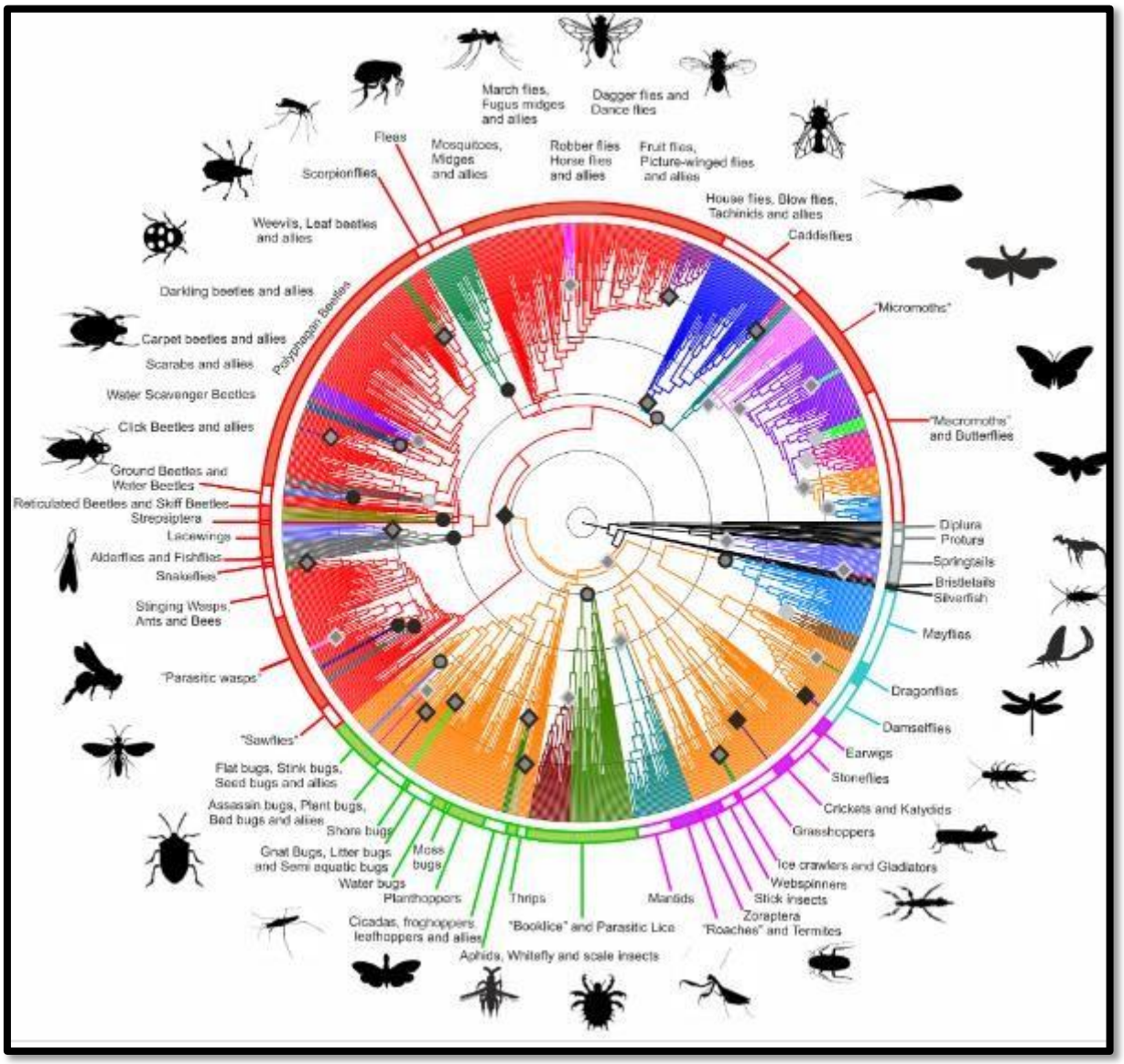


Figure: Hexapod phylogenetic tree.

- **Anatomy of the head**

Insects have, as an upper lip, a lava that is composed of two fused metamers. This lube covers the mandibles, maxillae and labium that are used to bring food to the mouth. The labium is formed by the second pair of fused maxilla. Beneath all these mouthpieces is the lypopharynx, the equivalent of the mouth.

The eyes are composed of subunits, the ommatidia. Each of them is followed by a nerve cable that extends to the brain. The visual pathways have a double chiasma, thanks to the lateral position of the eyes on the head. Colour perception in insects is displaced in ultraviolet compared to human vision; they thus perceive UV but not red. They can also perceive polarized light.

Thanks to the three ocelli forming a triangle between their two eyes, they are extremely sensitive to movement. Indeed, these complementary photosensitive organs of the eyes

perceive variations in light intensity. They are, like the eyes, organs related to theft.

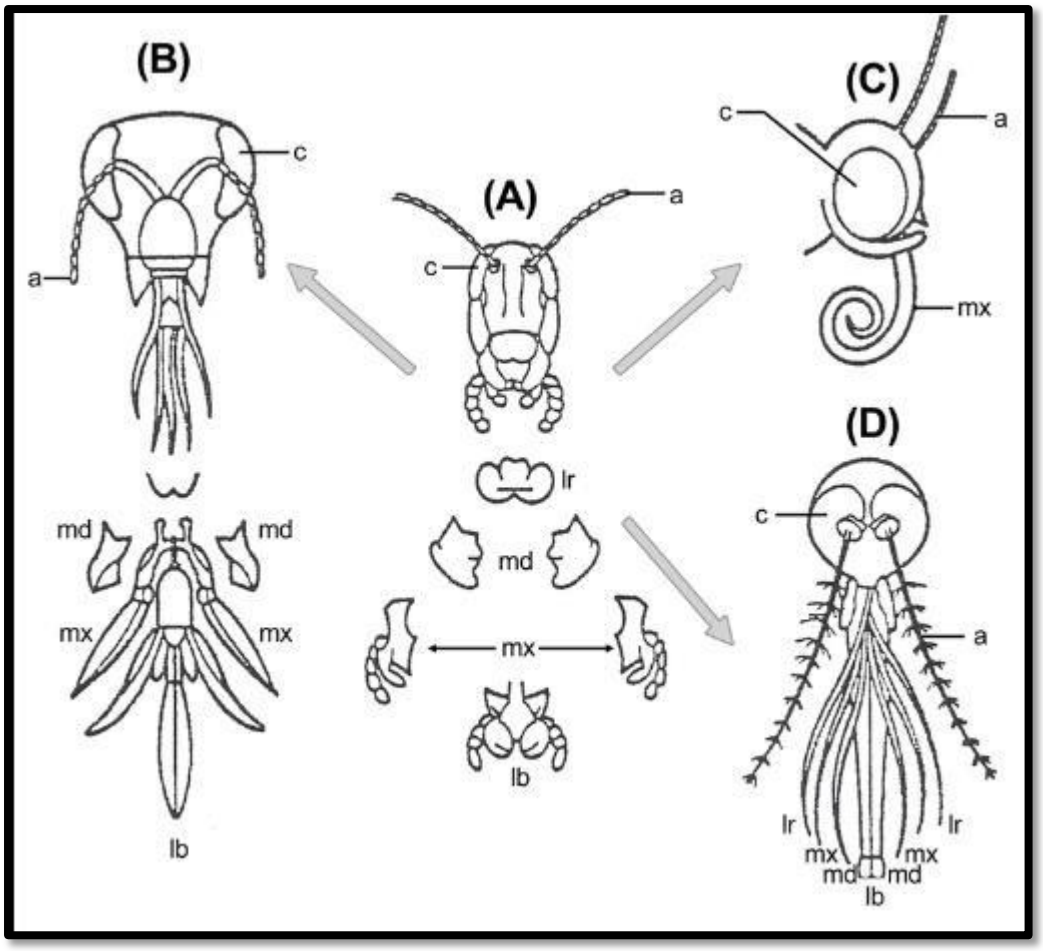


Figure: Main types of insect mouthpieces

Anoplura Order

The Anoploures order includes nearly 500 species grouped into about fifteen families.

All Anoploures have a number of morphological characteristics in common: they are small greyish or brownish insects, airless, with a flattened dorso-ventral body. Their mouthpieces form a short, retractable trunk.

They are insects with incomplete metamorphosis (heterometabolisms).

The only family of medical importance is the Pediculidae with 2 main genera: Pediculus and Phthirus both ectoparasitic and specific to humans.

Genus Pediculis

Pediculus humanus is the louse of humans, a parasitic insect. The lice infestation is called "pediculosis".

Pediculus humanus comprises two parasitic subspecies of humans: *Pediculus humanus humanus* , *P. h. corporis* (body lice) and Pediculus humanus capitis (head lice). Pubic lice belong to another species.

Figure: Gender: Pediculus
Selon Mehlhorn H. 2001

Human Louse - Pediculus humanus

Pediculus humanus humanus is 2 to 4 mm long; Pediculus humanus capitis is smaller: 1 to 2.5 mm. In these 2 species, the female is larger than the male. Apart from the size, the other morphological characteristics are similar.

The head is elongated and has 2 eyes as well as a pair of 5-item antennas. The thorax is reduced and the segments fused; the legs are strong and stocky, they form a clamp with the claw of the tarsus and a strong tooth located on the tibia (the "thumb"). The abdomen is formed of 9 segments of which 7 are visible, the last one is indented in the female and provided with a conical penis in the male. The larva resembles the smaller adult.

Category

Embranchement	*Arthropoda*
Classe	*Insecta*
Sous-classe	*Pterygota*
Ordre	*Phthiraptera*
Sous-ordre	*Anoplura*
Famille	*Pediculidae*
Genre	*Pediculus*
Nom binominal *Pediculus humanus* Linnaeus, 1758	

Development cycle

Lice reproduce very quickly. The cycle takes place entirely on the host, and lasts a total of about 18 days.

The female louse mates several times during its adult life, which can vary from ten to forty days.

The female lays ovoid eggs (4 to 10 eggs per day) for 3 to 5 weeks (200 to 300 during her life) 1 mm long, with a punctured shell and provided with a lid: the nits, which are stuck on a support (hair for P. humanus capitis and textile fibres of clothing in contact with the skin for P. humanus humanus).

Incubation lasts 6-9 days depending on temperature, and hatching releases a small

larva, a pupa that measures 1 to 2 mm long, which migrates little and is not very contaminating. It performs three moults in 8 to 12 days on the surface of the skin. The adult (imago) can mate 10 hours after the last moult.

Habitat and nutrition

The louse is a permanent and specific parasite (*P. humanus humanus* is located on the body, *P. humanus capitis* on the scalp), its entire life is carried out in a stable atmosphere with regard to temperature and humidity: that of the human integument, under clothing or in the hair. He does not willingly leave his host because he cannot go far and dies in a few days if he does not meet another "victim". Dissemination is carried out by contact between hair or with a contaminated object (comb, cap, pillow) for the head louse, and by contact with parasitized clothing for the body louse.

Pediculus humanus is strictly hematophagous at all stages and in both sexes, its bite is solenophagea; adults perform 1 blood meal each day, their fasting resistance is very low (1 to 2 days). They can live up to 40 days.

Medical aspect

Scalp pediculosis is due to the subspecies capitis, and body pediculosis is due to the subtype corporis.

Body lice are also vectors of three pathogenic bacteria: *Rickettsia prowazekii, Borrelia recurrentis and Bartonella quintana*, which are responsible for exanthematic typhus, recurrent ragged fever and trench fever, respectively.

The bite of lice (body and head) causes pruritus which leads to scratching lesions that can become superinfected.

Lice are also very important vectors of disease. All pathogen transmissions known to date are due to body lice: P. humanus humanus.

These diseases include:

- exanthematous typhus (Rickettsia prowazeki)
- trench fever (Bartonella quintana) Both transmitted through feces.
- cosmopolitan recurrent fever (Borrelia recurrentis) Transmitted by the crushing of the louse. .

Genus Phthirus

The snout: (*Phtirius pubis*)

Category

Embranchement	*Arthropoda*
Classe	*Insecta*
Ordre	*Phthiraptera*
Sous-ordre	*Anoplura*
Famille	*Pthiridae*
Genre	*Pthirus*
Nom binominal *Pthirus pubis* (Linnaeus, 1758)	

Body type:

Phthirus pubis measures 1.5 to 2 mm, its body is globular; it differs from Pedicles in its very wide thorax (wider than the abdomen) and even stronger legs (especially the claws) in the 2nd and 3rd pairs. The shorter abdomen has 5 visible segments. The larva resembles the smaller adult.

Development cycle

The developmental cycle of Phtirius is comparable to that of Pedicles. The female lays smaller eggs (only about thirty in her lifetime) which are stuck on the hairs. Incubation lasts 7-8 days. The larva performs 3 moults in 12 to 17 days.

Medical significance:

Apart from pruritus due to the bite and scratching lesions that can superinfect, no disease is transmitted by Phtirius pubis.

B-Order**Siphonaptera**

Siphonaptera live in close contact with their host, their attachment to the latter is intermittent. They are insects with complete metamorphosis (holometaboles).

Adults are distinguished by their ability to jump.

***Genus Ctenocephalides**

Fleas: (Pulex,Ctenocephalides)

Body type:

The adult flea is 1 to 4 mm long, it is airy and very flattened laterally. its colour is uniform and ranges from yellow to very dark brown; it has a pair of 3-item antennas housed in antenna dimples as well as 2 single eyes.

Irritant pulexes have only one silk under the eye while Ctenocephalides has 2 silk combs (ctenidia): one on the head and one on the prothorax. The thorax is formed of 3 segments and carries 3 pairs of clawed legs, the 3rd pair is highly developed and suitable for jumping. The abdomen is formed of 10 segments of which 7 are visible, the 9th and 10th segments are transformed into genitalia.

Development cycle

The fertilized female lays in small series of 2 to 6 eggs (up to 800 during her lifetime) in the litter and coat of infested animals, but also on the floor of homes.

The whitish eggs are about 0.5 mm long and are ovoid; the duration of incubation depends on the external conditions (temperature and humidity), it lasts from 2 to 15 days.

The whitish colored larvae measure about 2.5 mm and make 3 moults. They are vermiform, apodic and have 13 segments. The latter are detritivorous; then the larvae metamorphose into nymphs or pupae (immobile stage).

Under favorable conditions, the adult is trained 10 to 12 days later and lives from 8 to 17 months depending on the environment and the species.

Fleas are capable at all stages of very significant metabolic slowdowns allowing quiescence until favorable conditions return; the duration of the cycle can therefore vary from 20 days to 16 months in the same species. This explains why a habitat can be infested by fleas for a long time.

Medical importance: The direct pathogenic role of fleas is linked to the bite: pruritic papules, urticaria, bruising with edema, blood spoliation with risk of anemia if the bites are numerous and allergic reactions to the saliva of the insect. Fleas are also responsible for the transmission of various pathogens responsible for human pathologies: Bacterial infections:

-Murine typhus (Rickettsia moose ri)

- Transmitted by flea droppings. Helminthiasis

-Dipylidium canine (dog and cat tapeworm) Transmitted by ingestion of parasitic fleas.

Fleas are also important in veterinary medicine.

C- Order Diptera:

The order of Diptera has about 80,000 species; it is divided into two suborders:

• Nematoceras: slender-bodied insects with antennae with more than 6 items.

• Brachycera: stout-bodied insects with antennae of 3 items or less.

• Adults have only one pair of wings, the second being transformed into dumbbells (or pendulums). Larvae are apodic; nymphs do not feed.

They are holometabole insects. All the Diptera belonging to the Nematoceres are orthorrhaphic: the adult is freed from the pupal exuvia by a rectilinear slit. The Brachycera suborder includes orthorrhaphic and cyclorrhaphic insects.

Mosquitoes

All mosquitoes constitute among the orthorrhaphic Nematocere Diptera, the Culicidae family. It is a homogeneous family that includes about 3000 species.

Classification

Order: Diptera Family:

Culicidae

Morphology : The size of Culicidae varies from 5 to 20 mm; the body is thin and slender. The antennae, long and filiform, are feathery in the male and short-bristled in the female. The mouthpieces form a tube consisting of 6 styli enclosed in a sheath: labium. The wings are long, narrow and membranous, covered with scales attached to the ribs and the posterior edge. The long and small legs are made up of 5 items and have 2 claws. The abdomen, meanwhile, is formed of 9 segments ending in 2 circles; the female has a laying orifice and the male an external genitalia (copulatory appendages and penis). The larva, vermiform, measures from 1 mm to 1.5 cm depending on the stage; it has a head with antennas, a thorax and a 9-segment abdomen. Its body is covered with bristles characteristic of the different species. The nymph has a curved abdomen, by transparency we can distinguish the outline of the eyes and the different appendages (trunk, legs, wings).

Development cycle: After mating, the female lays 100-400 eggs (0.5 mm) on the surface of the water; each female can lay 5-6 times in her lifetime. Under favorable thermal conditions, hatching takes place 2 to 3 days later. The larva, saprophyte develops in 1 to 3 weeks and undergoes 3 moults. This is done in an aquatic environment but with air breathing, it is mobile The nymph is aquatic and does not feed, it becomes

an adult in 2-6 days. The total duration of the cycle varies from 2 to 3 weeks on average.

Medical significance: The mosquito bite plays a direct pathogenic role through the toxic and antigenic actions of saliva which can lead to the formation of pruritic papules, and significant edematous reactions in sensitized subjects. Mosquitoes are also biological vectors of many pathogens depending on the genus to which they belong: protozoa, viruses and helminths. Culicidae are the most important group in human public health because they are involved in the transmission of diseases that are among the main causes of morbidity and mortality in humans (malaria, yellow fever, dengue, arbovirosis, filariasis).

***Gender Aedes**

Aèdes sp *Aèdes albopictus*

Genre très important, il compte environ 870 espèces dans le monde

Category

Règne	*Animalia*
Embranchement	*Arthropoda*
Classe	*Insecta*
Sous-classe	*Pterygota*
Ordre	*Diptera*
Sous-ordre	*Nematocera*
Famille	*Culicidae*
Sous-famille	*Culicinae*
Genre	*Aedes*
Nom binominal *Aedes (Stegomyia) albopictus* Linnaeus, 1758	

Caractéristiques morphologiques : Semblable au genre Culex, le genre Aèdes présente également des palpes maxillaires plus courts que la trompe chez la femelle et plus long chez le mâle. En outre, il se différencie par des pattes ayant 2 griffes denticulées et par l'absence de pulville. Sa coloration est très sombre avec des marques blanches ou colorées.

Les larves possèdent un siphon respiratoire (position verticale sous la surface de l'eau).

Importance médicale :

Aedes are biological vectors of 2 tropical arboviroses: dengue and yellow fever which can be found around the Mediterranean. They are also implicated in the transmission of the West Nile virus (Mediterranean basin and Camargue).

Transmission is through the saliva of the insect during the bite.

Gender Culex

Culex quinquefasciatus

The genus Culex has nearly 800 species worldwide.

Category

Règne	*Animalia*
Embranchement	*Arthropoda*
Classe	*Insecta*
Sous-classe	*Pterygota*
Super-ordre	*Endopterygota*
Ordre	*Diptera*
Sous-ordre	*Nematocera*
Famille	*Culicidae*
Sous-famille	*Culicinae*
Genre*Culex* Linnaeus, 1758	

-Morphological characteristics : Culex is dull in colour. The maxillary palps are about three times shorter than the trunk in females and longer in males. The trunk and abdomen form an obtuse angle. The legs have 2 simple claws and 2 wings. The larvae have stigmas at the top of a respiratory siphon (vertical position below the surface of the water) and the 8th segment silk comb is almost always present.

Development cycle : Culex eggs are differentiated by the presence of a Corolla at the lower pole and measure 0.7 to 1 mm.

Medical significance : Culex can be a vector of the following diseases:

• Arbovirosis: - Virus encephalitis - West Nile virus (Mediterranean basin)

• Filariasis due to Wuchereria and Brugia in the tropics. These diseases are transmitted by the insect's saliva during the bite

Bibliographic References

(Books and handouts, websites, etc.) :

1. Cassier P, Brugerolle G, Combes C, Grain J, Raibaut A. 1998. Free-riding dynamic equilibrium Ed. Masson.
2. Cheng T.1986. General Parasitology. 2nd Edition, Academic Press. ISBN 0-12-170755-5
3. Combes C. and Jourdane J. 2003. Taxonomy, Ecology and Evolution of Metazoan Parasites. Tomes 1 and 2. Publishers Presses Universitaires de Perpignan. ISBN 2914518- 36-6.
4. England P.T. and Asher A. 1988.The biology of parasitism. Alan R. Liss, Inc. New York. ISBN 08451-2208-8.
5. Golvan YJ. 1983. Elements of Medical Parasitology. Ed.Flammarion. ISBN 2-257-12589- 4.
6. Mehlhorn H. 2001. Encyclopedic reference of Parasitology. Biology Service Function 2nd Ed. Springer. ISBN 3-540-66829-2.
7. Mehlhorn H. 2008. Encyclopedia of Parasitology. 2nd Ed. Springer. ISBN:978-3-540-48994-8.
8. Nozais J.P., Datry A. and Danis M. 1996. Treatise on Medical Parasitology. Ed. Padel ISBN 2- 9075-16-64-7
9. Poulin R. and Morand S. 2004. Biodiversity parasite. Smithsonian Institution. ISBN 1-58834- 170- 4.
10. Rickard Ballweber. 2001. Veterinary Parasitplogy . Butterworth Heinemann Edition.
11. Wall R, Shearer D. 2001. Veterinary ectoparasits: Biology, pathology and control. 2nd Ed. Blackweel Sciences.

Numerous websites made by specialists in different parasites are available, they concern both parasites that are of interest to human health and those that have an impact on animal health.