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Zoonoses

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MCA , Speciality : Parasitology

(Intended for Master 02 students, Speciality: Parasitology)
Academic year
2024-2025

CONTENTS

Course Pedagogical Plan

Title of Subject: Zoonoses

Sector: Parasitology

Level: Master 02 year

degree Teaching unit: Fundamental

Hourly volume: 90h00 Lessons + TD

coefficient: 3

Number of credits 6

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.

Preface

Parasitic zoonoses are the combination of two subjects, parasitology and

zoonoses, where knowledge of both fields comes into play for the study of zoonotic parasites. Parasitic zoonoses are the most diverse group of zoonotic diseases, representing all the classes mentioned in the classification of zoonotic diseases. They have been classified and discussed according to etiological agents, transmission cycle, reservoir hosts and major hosts involved, as well as examples. Important topics such as food-borne, vector-borne and occupation- related parasitic zoonoses were also covered in the introduction. We also discussed the factors responsible for the emergence of zoonotic parasites, namely climate change associated with global warming, increasing vector populations, global tourism, demand for livestock feedstuffs, changing socio-economic conditions, poverty, lack of drinking water, large numbers of stray animals, changes in culinary practices, outdoor defecation, poor personal hygiene and high population density in the tropics. More than 15 protozoans and 50 other parasitic diseases are zoonotic in nature, and all were discussed in detail. Each disease has been divided into the following sections: synonyms/common names, etiology, epidemiology, cycle, mode of transmission, clinical signs in humans and animals, diagnosis, prevention and control. Life- cycle diagrams color photographs of these parasites have been included wherever necessary. An alphabetical bibliography for each disease has also been included to give readers access to further information.

Few diseases are rare or their zoonotic potential still in question, but they have also been given space to update readers' knowledge. We hope that this book will be of benefit to students and others interested in the subject.

INTRODUCTION

Parasitism is essentially the association between two species: the parasite (first species) and the host (second species). Parasitic diseases cause major problems in developing countries. At present, there are over 15 protozoan and 50 other parasitic diseases of a zoonotic nature. Zoonotic parasites are important for human and animal health, food safety and economic concerns. The emergence and re-emergence of many zoonotic parasites has been reported worldwide. Contaminated food and water significantly increase the transmission of these parasites. Factors influencing the prevalence of these parasites include the resurgence of vector populations, climate change associated global warming, international food trade, poverty and lack of drinking water in non-industrialized countries, etc. Most animals living in close contact with man could harbor and transmit zoonotic parasites to humans. Livestock, pets, domestic animals, wild animals, fish and certain other animals could all transmit zoonotic parasites. An update on the current status of zoonotic parasites was provided.

The parasite lives on the host or its tissues for certain periods of time. This association can be of different types, namely symbiosis, commensalism, mutualism, phoresis, predation or parasitism. Such a relationship with the host parasite could lead to pathological disorders and diseases in the host. In both animals and humans, many major illnesses occur as a result of animal diseases. Parasitic diseases cause major problems, particularly in developing countries. Some 370 parasitic species (300 helminths and 70 protozoa) have been found in humans (Ashford and Crewe 1998).

CHAPTER I

CLASSIFICATION OF PARASITIC ZOONOSES

I. Parasite classification

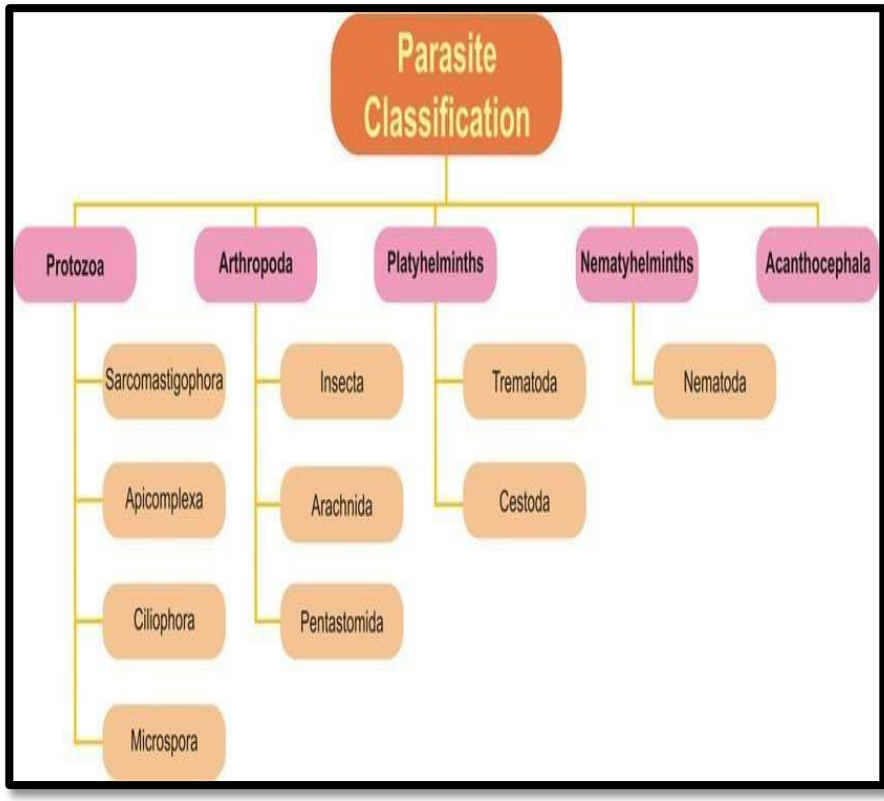


Fig 1. Parasite classification

Parasitic zoonoses can be classified into different categories, as in the case of other zoonoses (Schwabe 1984). Parasitic zoonoses are the most diverse group of zoonotic diseases, and are classified on a number of different bases.

1. Classification of parasitic zoonoses

1.1. Based on etiological agents

1. Protozoonoses: cryptosporidiosis, giardiasis, toxoplasmosis, etc.
2. Trematode zoonoses: clonorchiasis, para-gonimiasis, etc.
3. Cestodial zoonoses: hydatidosis, taeniosis, cysticercosis, etc.
4. Nematode zoonoses: larvae migrans, trichiosis, zoonotic

ancylostomiasis, etc.

5. Arthropod zoonoses: myiasis, zoonotic diseases, etc.

1.2.Based on transmission cycle

Parasitic zoonotic diseases can be classified according to the life cycle of zoonotic pathogens.

1.2.1. **Direct parasitic zoonoses**: The parasite is transmitted by direct or indirect contact. through food, e.g. scabies, trichinellosis, giardiasis, etc.

1.2.2. **Cyclozoonoses:** Diseases caused by zoonotic parasites require two or more vertebrate hosts to complete the parasite's life cycle. Cyclozoonoses are classified into two categories:

- **Compulsory cyclozoonoses**: humans must act as hosts to complete the parasite's life cycle, e.g. Taenia solium and Taenia saginata taeniosis.
- **Non-compulsory cyclozoonoses**: humans are not obliged to complete the parasite's life cycle, but could act as an accidental host, e.g. human hydatidosis.

1.2.3. **Metazoonoses**: Diseases caused by these zoonotic parasites that require both vertebrate and invertebrate species to complete the parasite's life cycle. Metazoonoses are further classified into four categories:

- **Type I metazoonoses**: Diseases caused by these zoonotic parasites that require an invertebrate and a vertebrate host, e.g. *Dipylidium caninum* dipylidiasis.
- **Type II metazoonoses**: Diseases caused by these zoonotic parasites that require two invertebrate and one vertebrate hosts, e.g. paragonimiasis.
- **Type III metazoonoses**: Diseases caused by these zoonotic parasites

that require one invertebrate and two vertebrate hosts,

e.g. clonorchiosis.

- **Type IV metazoonoses**: Diseases caused by these zoonotic parasites that require transovarial transmission, e.g. *Babesia* diverge babesiosis.

1.2.4. **Saprozoonoses**: Diseases caused by these zoonotic parasites that require non-animate material in addition to their hosts to complete their life cycle. Saprozoonoses are further classified into three categories:

- **Saproanthropozoonoses**: animals to humans via an inanimate partner,

e.g. cutaneous larva migrans, myiasis.

- **Saproamphixenoses:** Animals and humans may act as reservoir hosts, and etiological agents are transmitted by inanimate objects, e.g. probably micronemosis.
- **Saprometanthropozoonoses**: Sapromet- anthropozoonoses require a vertebrate host, an invertebrate host and an inanimate substance for disease transmission, e.g. fasciolosis.

1.3. Based on reservoir hosts

1.3.1. ***Anthropozoonoses*:** Diseases in which animals act as reservoir hosts and humans become accidentally infected, e.g. hydatido- sis, visceral larvae migrans.

1.3.2. ***Zooanthroponoses***: These diseases are present in humans but could be transmitted to animals, for example amoebosis.

1.3.3. ***Amphixenosis*:** Diseases in which humans and animals may act as reservoir hosts, e.g. clonoriasis.

1.4. Depending on the main host involved

- ✓ Parasitic Zoonoses linked to cattls : *T. saginata*

taeniosis, cryptosporidiosis.

- ✓ Parasitic zoonoses associated with sheep: fasciolosis, hydatidosis.
- ✓ Parasitic zoonoses linked to pigs: ascariosis, swine tainiosis.
- ✓ Parasitic zoonoses linked to fish: diphyllo- Bothriosis.
- ✓ Zoonoses parasitic linked to dog: ancylostomiasis, echinococcosis.
- ✓ Cat-related parasitic zoonoses: toxoplasmosis.
- ✓ Parasitic zoonoses linked raccoons: baylisa- scariosis.

1.5. Parasitic zoonoses of animal origin

Most animals in close contact with man could harbor and transmit zoonotic parasites to humans. Livestock, pets, domestic animals, wild animals, fish and certain other animals could all transmit zoonotic parasites (Parija 1990).

1.5.1. Parasitic zoonoses associated with livestock

Livestock is an essential component of human activities, particularly for dairy farmers, veterinarians and other related professions. Foods of animal origin meat, milk and their products - are essential components of the human diet. Human and animal populations live in close association with each other. This close association and food dependence can lead to the transmission of zoonotic parasites when appropriate hygiene measures are not taken. These diseases can be transmitted via food, contaminated water or the vector population.

Parasitic zoonoses transmitted by livestock can be classified either on the basis of the species involved, or on the basis of their mode of transmission Depending on the animal species concerned, they can be classified as:

- **Parasitic zoonoses associated with livestock**: babesiosis caused by *B. divergens,* cryptosporidiosis, sarcocystosis, fascioliasis, *T. saginata* taeniosis.
- **Parasitic zoonoses associated with pigs**: balantidiosis, sarcocystosis, toxoplasmosis, gastrodiscoidosis, T. *solium* taeniosis, ascariosis, gna- thostomosis, trichinosis.
- **Parasitic zoonoses associated sheep**: toxoplasmosis, fasciolosis.
- **Parasitic zoonoses linked to goats**: toxoplasmosis.

1.5.2. Wildlife-related parasitic zoonoses

Human beings, especially hunters and forest workers, agricultural workers, military personnel, tourists and other related professional workers, may be exposed to parasitic zoonoses linked to wild animals. Wild animal reservoir hosts of certain diseases can also enter the homes of humans and domestic animals, resulting in their transmission and occurrence in humans.

Examples of parasitic zoonoses linked to wildlife Opossums (*Didelphis*

albiventris, Didelphis mar- supialis) : *Trypanosoma cruzi:* trypanosomia

1.5.3. Food-borne parasitic zoonoses

Food animals such as cattle, buffalo, sheep, goats and pigs consume forage crops and convert them into animal protein. The meat of these animals is an important source of protein and nutrients (Gracey and Collins 1992). Fish and other seafood also make a vital contribution to providing essential nutrition for the human population. Poultry is another important meat-producing species. No zoonotic parasites are present in poultry meat and it is generally free from zoonotic parasites, except that clean water must be used in slaughter operations. However, free-range chicken could serve as an important reservoir for toxoplasmosis. A recent study has shown that poultry can serve as a minor reservoir for trematodes of halieutic origin.

In addition to fish and meat, milk and raw vegetables make up a significant part of our diet. Also important components of the human diet, food and water are an important route for the transmission of parasites from one host to another. Food and water are also important for the transmission of parasites that require more than one host to complete their life cycle.

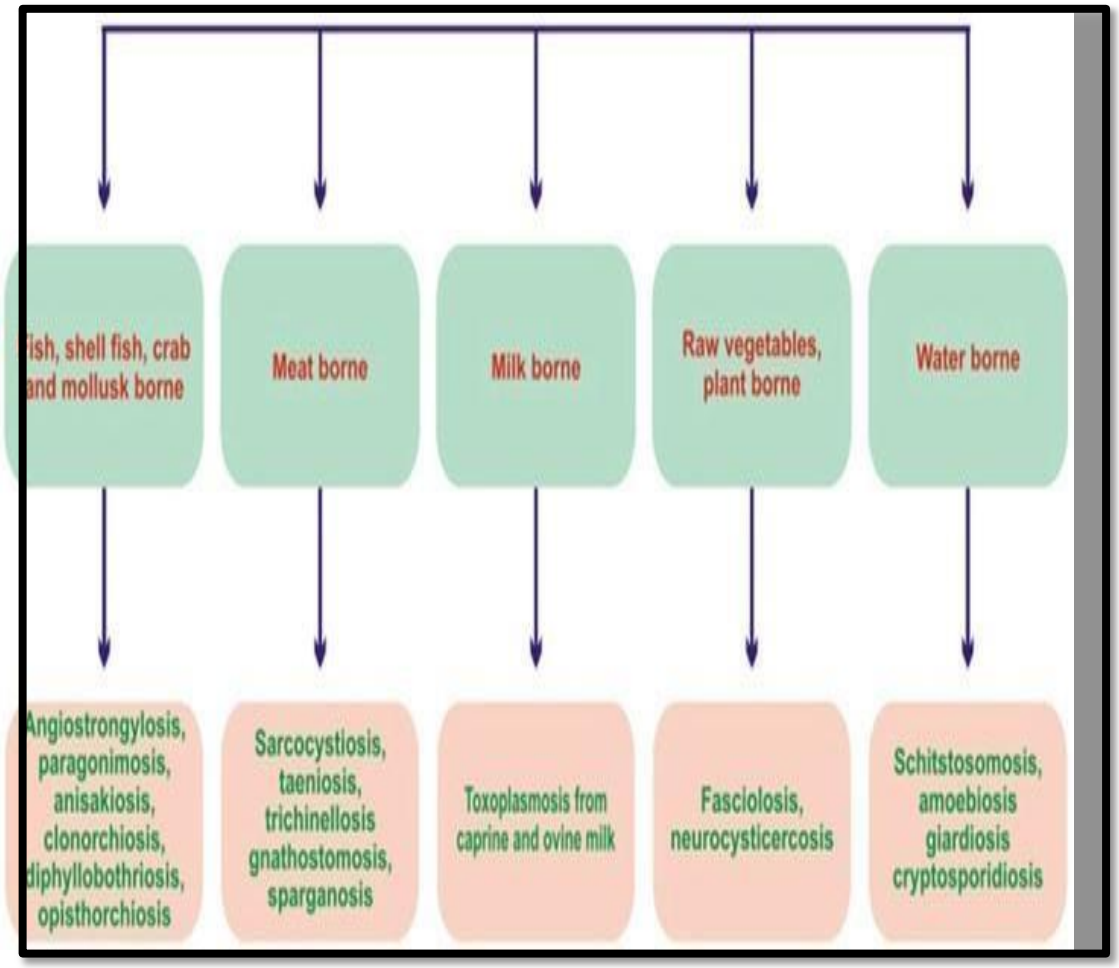


Fig 2. Parasitic zoonoses of food origin

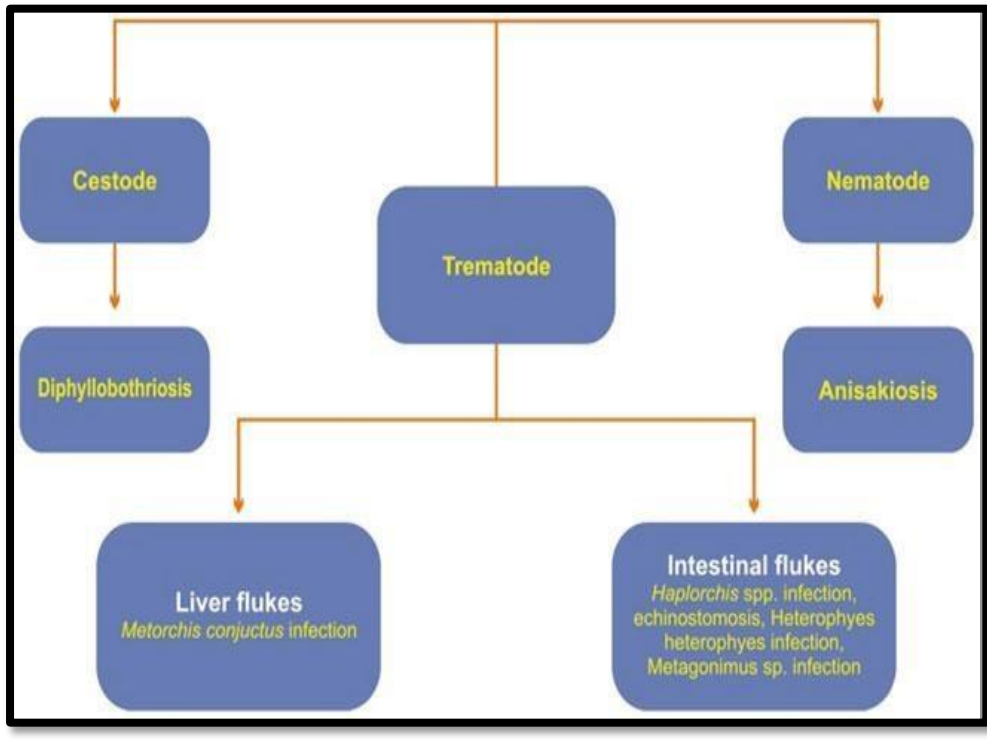


Fig 3. Parasitic zoonoses involving shellfish

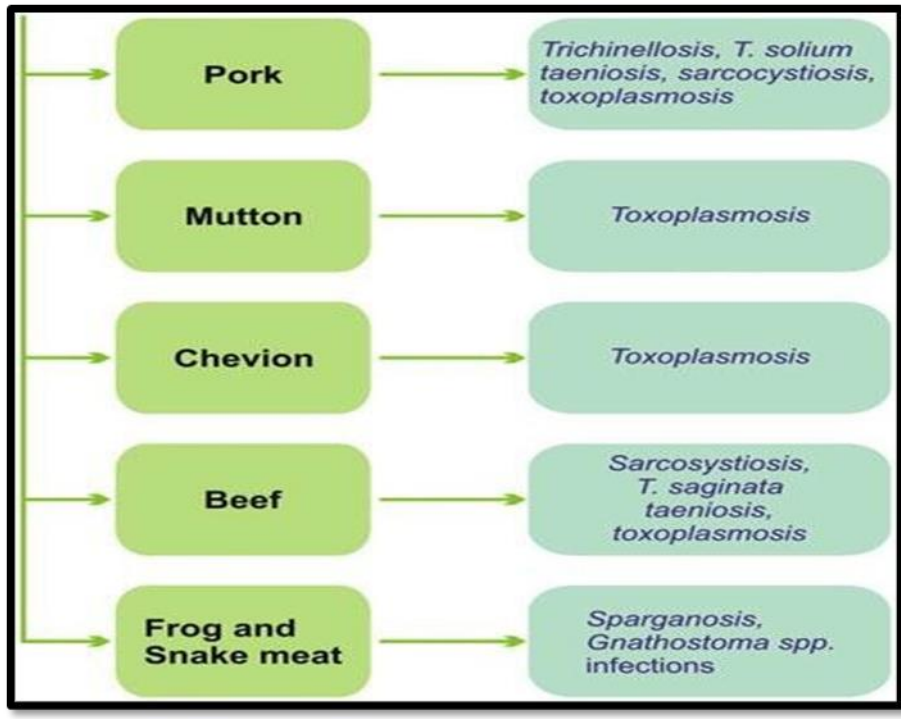


Fig 4. Parasitic zoonoses transmitted by meat

1.6. Vector-borne zoonoses

Vector-borne parasitic zoonoses (VBPZ) are diseases in which zoonotic parasites are transmitted from an infected animal to humans by an arthropod or other vector. Climate change and global warming could affect the occurrence, intensity and seasonality of many vector-borne zoonotic parasites. VBPZ are important because they affect human and animal health (WHO 2004) and cause economic losses (Bram et al. 2001). Factors responsible for the emergence of VBPZ include climate change (Lindgren and Gustaf- son 2001; Rosenthal 2009), increased tourism, the animal transport and international food trade (Weijden et al. 2007), drug resistance in parasites and vectors (Takken and Knols 2007), and changes in land use patterns (Colwell et al. 2000) ZPV can be classified either on the basis of the

vector involved, or into protozoan, cestode and nematode vector zoonoses. Protozoal zoonoses are the most important and significant, depending on the vector involved. ZPV can be classified into parasitic zoonoses transmitted by flies, mosquitoes, mites, insects, fleas and ticks.

1.7. Parasitic zoonoses transmitted by fish, crustaceans and molluscs.

Fish and related zoonoses can be classified trematode, cestode and nematode zoonoses. Trematode diseases can be further sub-classified into hepatic and intestinal flukes. According to WHO (1995), fish-borne trematodes infect more than 18 million people, and more than half a billion people worldwide are at risk from these diseases (Chai et al. 2005).

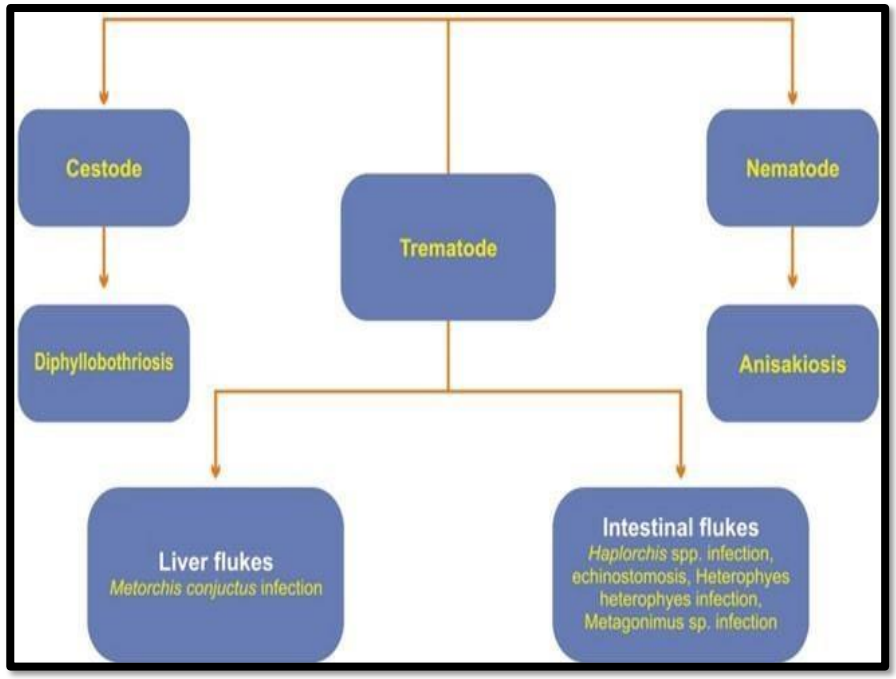


Fig 5. Classification of parasitic shellfish zoonoses according to (Chai et al. 2005).

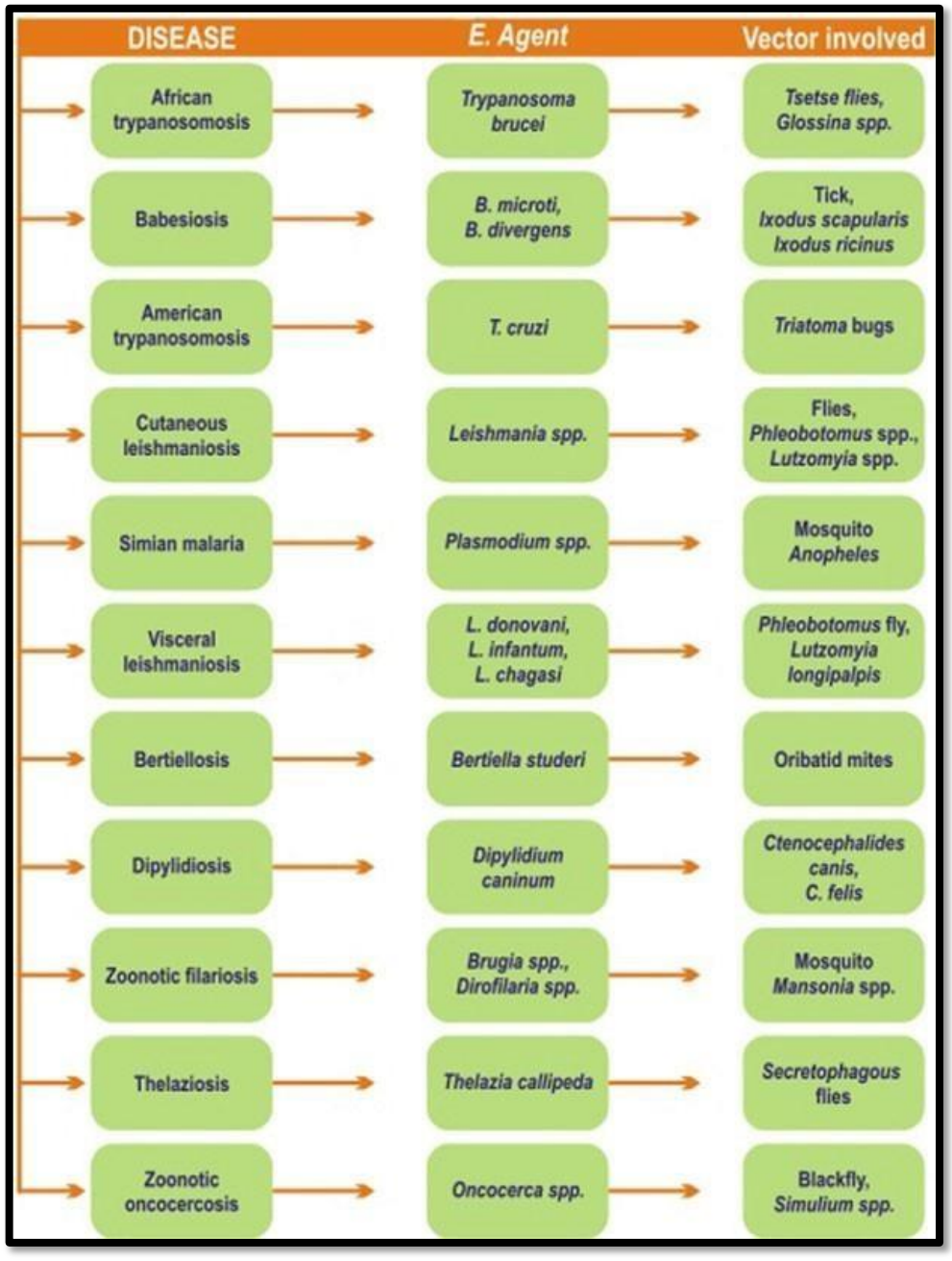


Fig 6. Classification of vector-borne parasitic zoonoses according to Colwell et al. 2000.

1.8.Waterborne parasitic zoonoses

These parasites are widespread throughout the world and pose a major threat, particularly in developing countries. The unavailability of clean, hygienic drinking water is a major risk in developing countries. Parasitic stages that can be transmitted via water range from single-celled amoebae to complex metazoans such as trematodes and cestodes. The life-cycle stages these species are well adapted to their survival in the environment and subsequent dissemination via water. Many important zoonotic parasites, such as *C. parvum* and *T. gondii*, could be transmitted via contaminated water. Transport hosts such as pigs may also play an important role in the collection and concentration of resistant exogenous stages of many parasites. Important waterborne parasitic zoonoses can be classified by protozoa, by trematodes and by these zoonoses, but protozoan zoonoses are more frequent and more important.

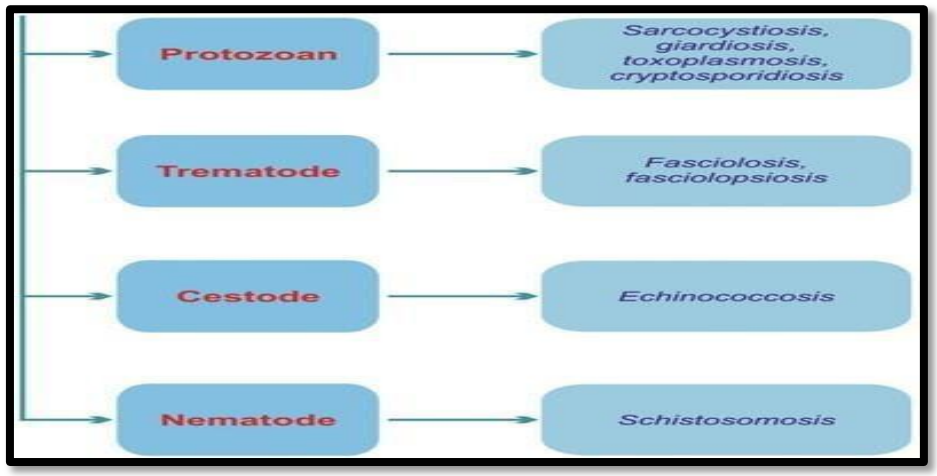


Fig 7. Classification of waterborne parasitic zoonoses according to Gaja-dhar and Allen 2004.

1.9. Raw vegetables/parasitic zoonoses of plant origin

The role of raw/salad vegetables in the transmission of zoonotic parasites is attracting particular attention in cases of neurocysticercosis in humans, where human wastewater has been used to irrigate farmland used to grow vegetables, and has been found to pose a significant risk of disease transmission. Fasciolosis is another important disease that can be transmitted by ingesting contaminated plants. Contamination of vegetables may also help to transmit parasites such as *Ascaris suum* and *Toxocara* species.

1.10. Soil-borne parasitic zoonoses

Soil-borne zoonotic parasites include helminth infections, namely ascariasis due to Ascaris lumbricoides, hookworm infections due to Ancylostoma spp. and toxocariasis due to Toxocara canis and Toxocara cati under humid conditions. Parasite eggs are generally found in the soils of tropical and subtropical countries (Bethony et al. 2006).

2. Prevention and Control

Prevention and control strategies should include the use of advanced serological and molecular diagnostic techniques, continuous surveillance of zoonotic parasites, health education, social and economic development, particularly in developing countries, and rapid treatment of cases.

Important parasites shed their eggs in the faeces, which is a major source of infection, leading to contamination of the environment, particularly water and food. One or more stages in the parasite's life cycle can transmit infection. For example, ingestion of infective oocysts alone leads to cryptosporidiosis, while *T. gondii* oocysts and tissue cysts can cause infection in humans.

- Avoid eating raw or undercooked fish.
- Interruption of the parasite's transmission cycle.
- Health education and environmental sanitation.
- Use clean water in slaughter to contamination by other waterborne zoonotic parasites.
- Avoid eating raw or undercooked meat.
- Provide safe food and water for livestock.
- Use of best hygiene practices during slaughtering operations to avoid contamination by other waterborne zoonotic parasites.
- Use properly filtered water for drinking purposes.
- Monitoring drinking water quality; Regular deworming of reservoir and animal hosts.
- Avoid surface contamination of swimming pools.

CHAPTER II

PARASITIC ZOONOSES

Protozoal zoonoses

Introduction

Protozoal zoonoses could be defined as "those protozoan diseases that are naturally transmitted between (other) vertebrate animals and man". Diseases such as toxoplasmosis and cryptosporidiosis are found worldwide. *Toxoplasma gondii, Cryptosporidium parvum* and *Sarcocys-tis suihomi*nis are the most important coccidian parasites affecting animals and humans. Immunocompromised people are always at greater risk of being infected by zoonotic parasites such as *C. parvum, T. gondii,* etc. Cryptosporidiosis is an emerging waterborne protozoan disease of public health importance. The *Sarcocystis suihominis* parasite is widespread in pigs in Asian countries such as India and China. African trypanosomiasis, Chagas , leishmaniasis and zoonotic babesiosis are important vector-borne protozoan zoonotic diseases. African trypanosomiasis remains a priority zoonosis for populations in sub-Saharan Africa. The wild rodent P. leucopus acts as a major reservoir for human infections. Chagas disease is a major medical and economic concern in Latin America. Leishmaniasis has been reported in over 80 countries.

1. Animal trypanosomiasis , African Trypanosomiasis

Order: Kinestoplastorida

Family: Trypanosomatidae

1.1. Common name: Nagana disease (which means impotent/unhelpful) (Winkle 2005) is the common name of the disease in animals (African Animal Trypanosomiasis) and sleeping sickness is the

common name for this disease in humans.

1.2. A brief history

The parasite has been present and infecting populations in sub-Saharan Africa for many centuries. *T. brucei* was first discovered by David Bruce (1855-1931, Scottish microbiologist and pathologist) as the cause of bovine trypanosomiasis (bovine nagana) in 1895. After 6 years, trypanosomes were observed in human blood for the first time.

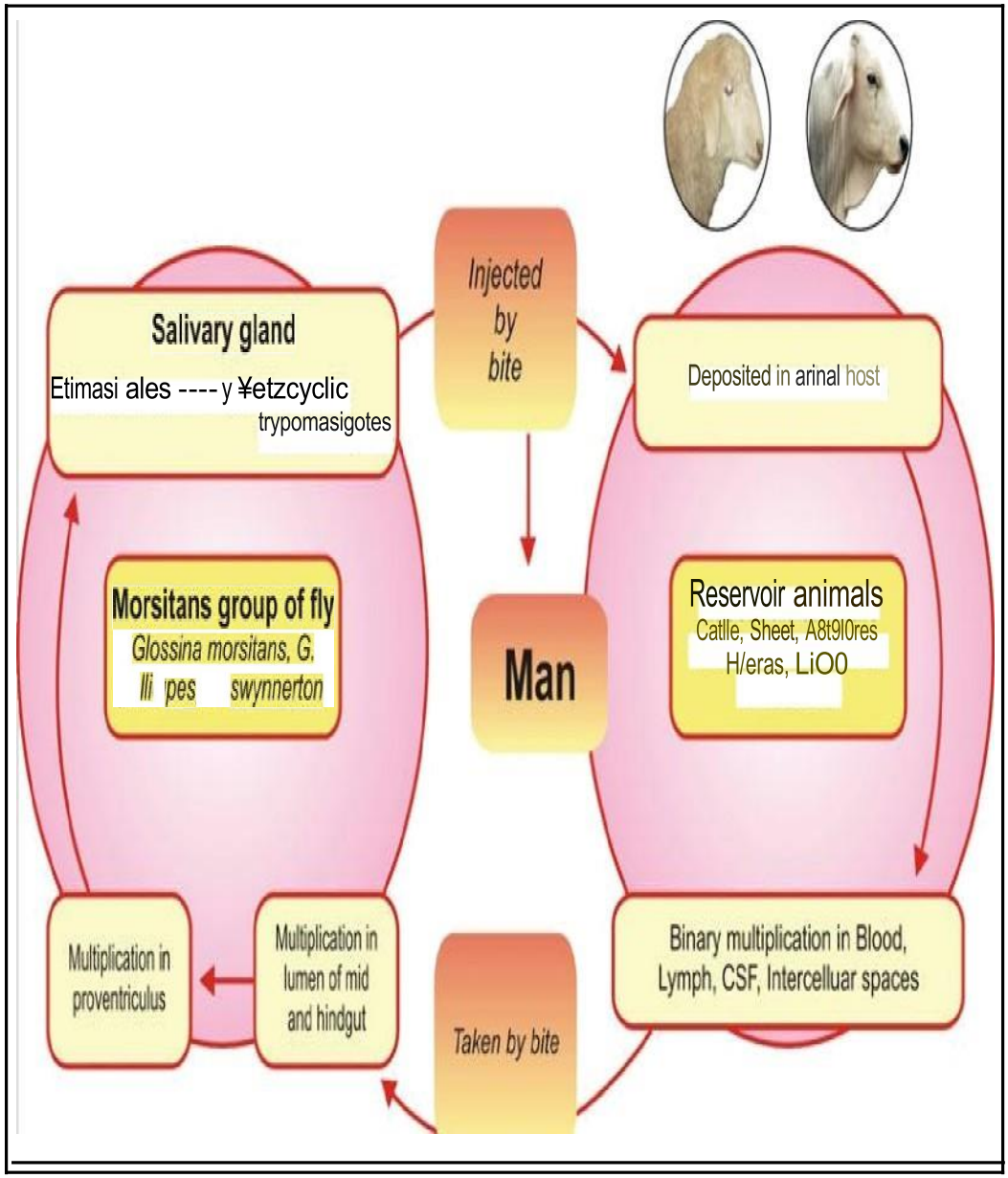
In 1901. The parasite species were subsequently identified and proposed as Trypanosoma gambiense (now *T. b. gambiense*). The second trypanosome species pathogenic to humans, T. rhodesiense (now *T. b. rhodesiense*), recovered later in 1910.

1.3. Epidemiology:

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). Tsetse flies are strictly African anthropophilic (between 15° north latitude and 30° south latitude), requiring heat (25 - 30°C), humidity and shade (trees and thickets), so sleeping sickness is strictly African intertropical. Both sexes are hematophagous.

Tank

Human beings act important reservoirs *of T. b. gambiense*. Wild and domestic animals could also act as important parasite reservoirs for human trypanosomiasis.


Salivary gland
Etimasi ales ---- y ¥etzcyclic
trypomasigotes
Injected by bite
Deposited in arinal host
Morsitans group of fly
Glossina morsitans, G. lli pes swynnerton
Man
Reservoir animals
Catlle, Sheet, A8t9l0res
H/eras, LiO0
Multiplication in proventriculus
Multiplication in lumen of mid and hindgut
Taken by bite
Binary multiplication in Blood, Lymph, CSF, Intercelluar spaces

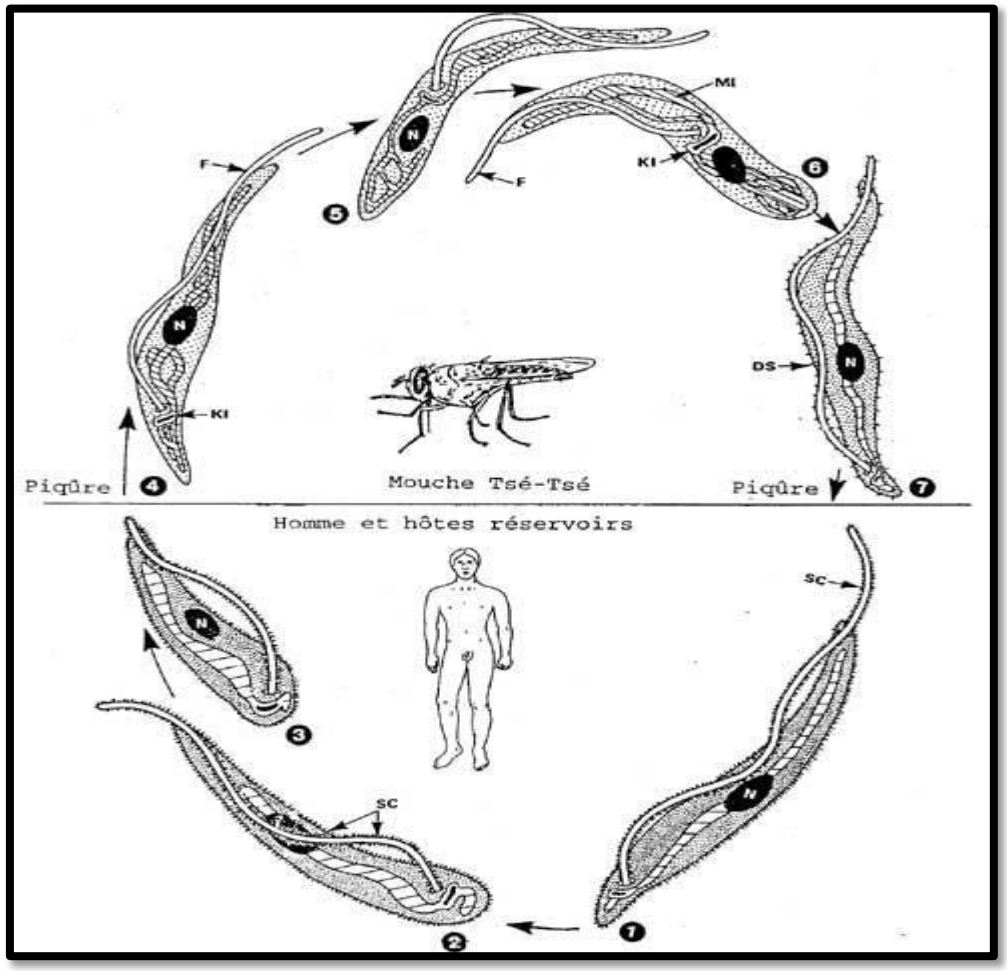


Fig 8. **Development cycle of *Trypanosoma brucei rhodesiense* and *Trypanosoma b.gambiense* according to (Mehlhorn & Ruthmann, 1988).**
1: blood-borne trypomastigotes ("slender" forms); **2:** intermediate trypomastigotes that multiply by binary division; **3:** trypomastigotes ("stumpy" forms) that develop inside the vector; **4:** procyclic trypomastigotes; **5-6:** transformation of trypomastigotes into epimastigotes that multiply by binary division; **7:** metacyclic trypomastigotes in the vector's salivary glands. These forms are infectious for humans. **DS:** developing cell coat; **F:** flagellum; **KI**: kinetoplast; **MI:** mitochondria; **N:** nucleus; **SC:** cell coat; **SF:** short flagellum.

Symptomatology: This is subdivided into 3 phases:

***Incubation**

This period varies in length and is difficult to determine, but is generally quite short, lasting from 8 to 10 days,

*** Invasion :**

It is characterized by irregular febrile attacks. The initial lesion is a trypanosome at the point of inoculation (like a furuncle that doesn't mature), followed by nervous excitement and insomnia, lassitude, inappetence and a headache that is sometimes intense. These symptoms disappear after 2 to 3 weeks, except for the fever.

*** Status phase:**

It begins a few weeks or months after infection and can be divided into two phases

1 - **lymphatico-sanguineous phase**: due to the presence of the parasite in the blood. It is characterized by irregular fever, cervical adenopathy (excellent clinical sign), splenomegaly (often discreet), cardiovascular disorders: palpitations, marked return of neurological symptoms (hyperesthesia, headaches), exanthems on the trunk or limbs, fatigue, muscular weakness, cramps on 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 - **meningoencephalitic phase**: This corresponds to the crossing of the meningeal barrier by the parasite (presence of the parasite in the cerebrospinal fluid, leading to CSF abnormalities). It leads to **an increase in neurological signs** Sensory disorders:

hyperesthesia (key sign), cramps, neuralgia Motor disorders: tremors, convulsions, shaky gait, incoordination Psychological disorders: irritability, depression, apathy, nycthemeral inversion (hence the name "sleeping sickness"), sexual disorders: impotence, frigidity, disturbances in thermal regulation, the clavicles and ribs are outlined under the skin and the patient has a skeletal appearance, various ocular manifestations. At the end of this phase, the patient assumes a special posture, with head tilted to the chest and eyes closed; he falls asleep wherever he is.

"Without treatment, the patient falls into a coma and dies within 2 years.

Diagnosis

Diagnosis is often very delicate during the early stages of the disease. In all cases, trypanosome testing is essential.

***Referral diagnosis**: A stay in intertropical Africa, polymorphous clinical features (cervical adenopathy), character changes, ECG and EEG changes.

Blood: anemia, increased ESR, hypergammaglobulinemia

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

***Direct diagnosis**: parasites are detected by direct examination of peripheral blood and cerebrospinal fluid Blood: thin smear, thick drop and/or concentration ==> search for trypomastigote forms (their number decreases during the course of the disease).

Node Suc: trypomastigote forms during the lymphatic/blood phase During the nervous phase of the disease: search for trypomastigote forms in the CSF after centrifugation

PCR

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

Treatment:

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

Prophylaxis:

General and individual prophylaxis should be considered successively

***General prophylaxis**: is intended for patients and tsetse flies For patients: field screening and treatment of humans, no mass treatment possible

Tsetse control: difficult to achieve: use of insecticides, clearing brush around dwellings, trapping (blue visual attractant + odorant attractant + deltamethrin).

***Individual prophylaxis**: this consists of protecting oneself from mosquito bites and escaping infection by wearing light-colored clothing (to scare away tsetse flies) and avoiding night-time traffic. At present, there is no chemoprophylaxis or vaccination (variable trypanosome antigens).

Note

* For *Trypanosoma(b) rhodesiense***, H.D=** man, dog, antelope, warthog, and the vector is an insect (diptera) **(H.I**) (*Glossina morsitans*: tsetse fly). Geographical distribution: West Africa, same life cycle and symptomatology as *Trypanosoma*.
(b) gambiense.

2. Intestinal giardiasis

Responsible for *Giardia intestinalis (= G. duodenalis, = G. lamblia).*

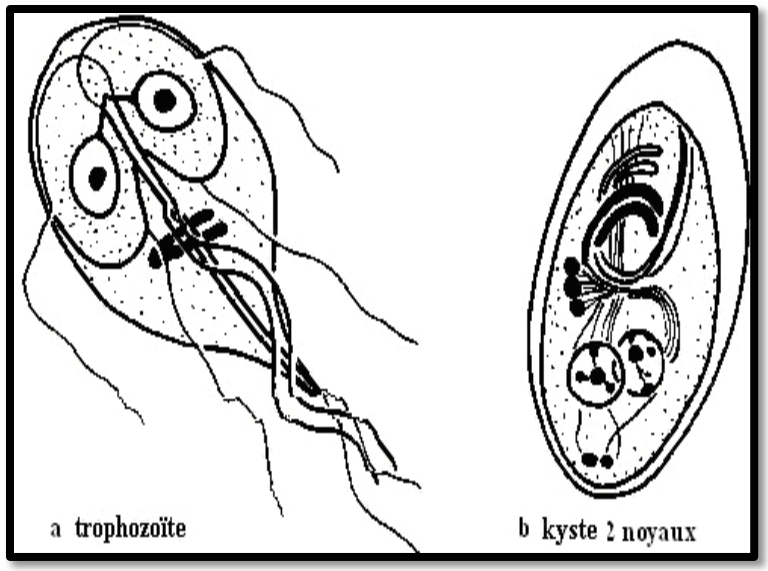


Fig 9. The two forms of *Giardia intestinalis*

2.2. Evolutionary cycle

The *Giradia intestinalis* cycle is direct (monoxenous). The definitive host is man.

After ingestion of 4-core cysts spread in nature with food or drinking water contaminated by unclean hands. De-cysting takes place in the duodenum. Multiplication occurs by binary division in the lumen of the small intestine (duodenum and jejunum) under flagellated form. The flagellated forms move actively in the intestinal mucus and can attach themselves to the surface of epithelial cells. This is followed by the irregular formation of cysts (with 2 nuclei) in the lumen of the large intestine, which are passively eliminated with the stool. Maturation of these cysts (with 4 nuclei) takes

place in the external environment. These cysts ensure transmission of the parasite.

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

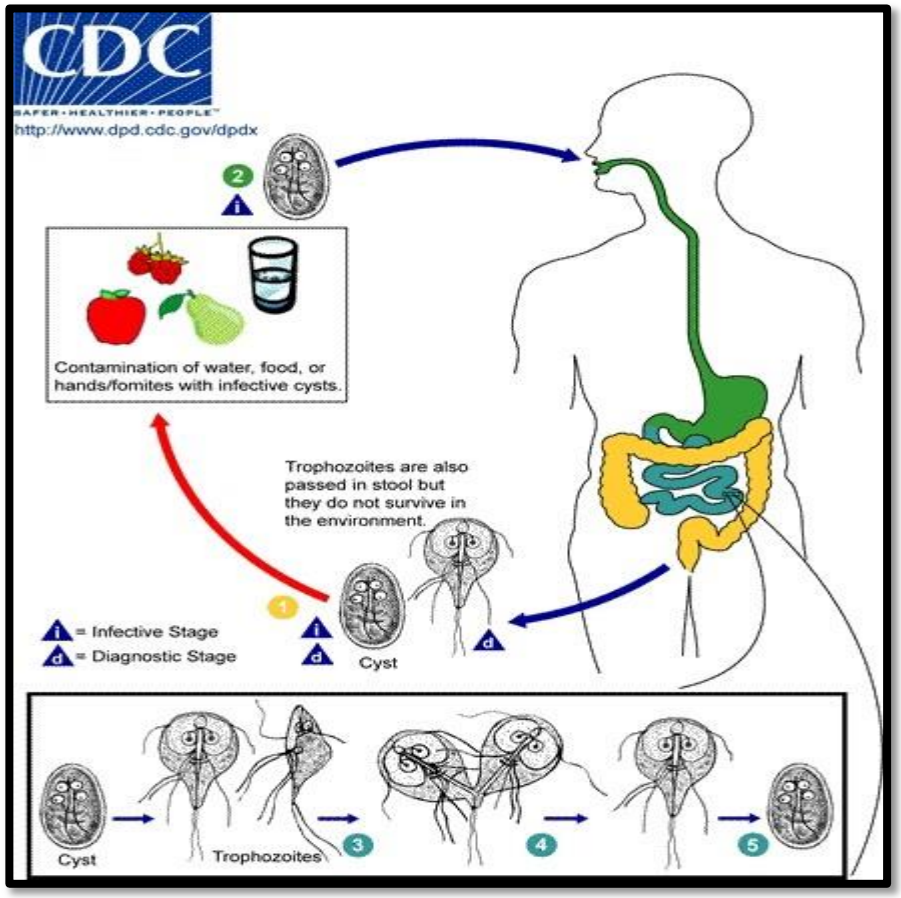


Fig 10. The *Giradia intestinalis* cycle

2.3.Symptomathology

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

Incubation

No symptoms for 7 to 10 days (asymptomatic**) State phase**

The main symptom is diarrhea, which in acute cases manifests itself as 20 to 24 stools a day, with a slight rise in temperature. More often than not, giardiasis or lambliasis is a chronic disease, with only 4 to 6 bowel movements a day and abdominal pain. More rarely, we see asthenia, anorexia, weight loss, nausea and psychological disorders. Intestinal malabsorption is possible in children. A child presenting with diarrhea for more than a week should be considered for giardiosis. Diarrhea usually progresses in fits and starts. Stools are generally abundant, almost fluid, yellow-brown, alkaline with a lot of mucus and a putrid odour.

Giardiosis can last for years, with lulls and relapses. Symptoms of dysentery are very rare, if dysenteric amoebae are present in the intestine.

Diagnosis

Two types complementary diagnosis can be used: **Orientation** diagnosisIntestinal malabsorption,Notion epidemics Children's communities (nurseries and pre-schools)

Direct diagnosis:

This is done by examining stools. Giardia are found in vegetative form (trophozoites) in diarrheal stools, and in cystic form if they are pasty (solid). Repeat the examination several times at intervals of several days in the event of well-founded suspicion, before confirming that no parasites are present. In exceptional cases, trophozoites may be found in an intestinal biopsy or in duodenal or jejunal tubing fluid (by specialized services).

Treatment

In most cases, giardiosis is a benign disease, but there are some patients have to spend a long time in treatment before they are cured, sometimes spontaneously.

Antiparasitic chemotherapy (e.g. Metronidazole: Flagyl® 7-day course)

Prophylaxis

- It consists above all in the care cleanliness (Hygiene): washing the hands before touching the food, meticulous washing of the hands, etc.

vegetables eaten raw,

- Sterilization of healthy carriers to avoid contamination,
- Public health education,
- Participation in collective hygiene and raising community awareness,
- Soil and drinking water health,
- Construction of latrines and wastewater treatment.

3. Amoebiase

Subphylum: Sarcodina

Order: Amoebidorida

Family: Endamoebidae

3.1. Common name/synonyms

The disease is known as amoebiasis, amoebic dysentery and enterobiasis.

3.2. A brief history

Amoebae were first found in faecal samples by Feder Losch in 1875 in St. Petersburg, but he did not consider them a cause of dysentery. Later, *E. histolytica* was reported to be a species complex by Emile Brumpt in 1925,

comprising two morphologically similar species, *E.* dysenteríae *and E. dispar* found in symptomatic and asymptomatic carriers, respectively.

3.3. Morphology

It occurs in the following forms:

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

Pre-cystic form Under various influences, the hematophagous or histolytica form gives rise to smaller amoebae or minuta forms (E. histolytica minuta), which may have originated from the larger forms. These are mobile, non-haematophagous, non-pathogenic forms. These pre-cystic forms may be 10 to 15 µm in diameter. They have the same structure as the larger forms, but their cytoplasm is devoid of feeding vacuoles.

and red blood cells. The karyosome in the nucleus is central. Shortly before encystment, the amoeba develops a glycogen vacuole.

Encysted form: The cyst derived from the pre-cystic form is spherical in shape and 10 to 14 µm in diameter. The cyst nucleus divides to form a 4-nucleus cyst (mature cyst). This is the contaminating (disseminating) and resistant form. These cysts are infectious for only 5 to 6 days.

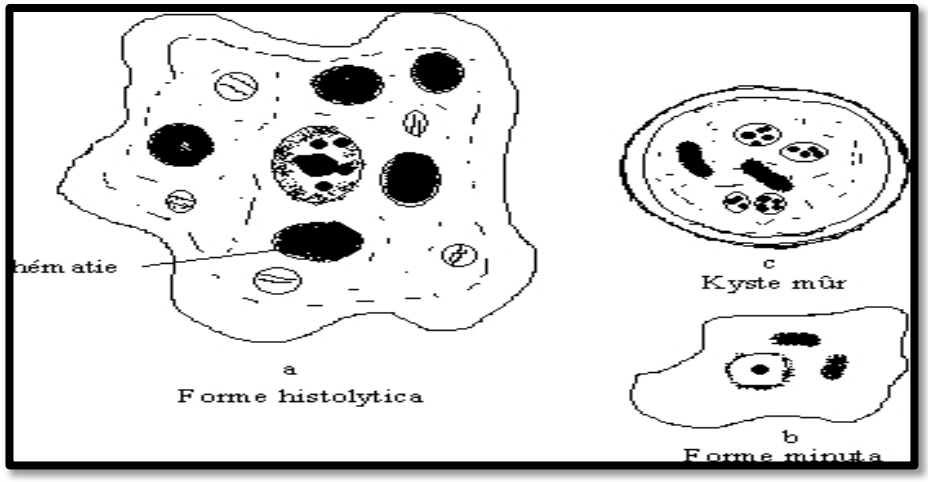


Fig 11. Morphological forms of *E. histolytica*

3.4. Evolutionary cycle (see **Fig. 12**) The host man.

Human infestation occurs through the ingestion of cysts, either directly from contaminated stools, or indirectly via contaminated food or water. Infectious cysts (with 4 nuclei) ingested by a healthy individual, under the action of pancreatic juice, rupture and release quadrinucleate amoebae (metacyclic amoeba). The four nuclei of the metacyclic amoeba each divide into two, giving 8 nuclei, and the amoeba itself divides into 8 smaller amoebae which grow a little, giving 8 minuta-type amoebae, which multiply in the intestinal lumen. These minuta forms can follow one of two destinies:

- either lead a saprophytic life, remaining mobile, or become encysted and cysts in the stool, the latter route is known as "amoebiasis infestation". The

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

- Either under influences that are still poorly understood, amoebae of the shape minuta grow, become more active and

hematophagous, and transform into

pathogenic *histolytica* amoebae. These forms penetrate the intestinal mucosa and cause ulceration, a process known as "amebiasis disease". During amoebiasis disease, the amoeba *(E. histolytica histolytica)* either remains in the colon (intestinal amoebiasis) or invades other organs such as the liver (hepatic amoebiasis). This is the **abnormal or pathogenic cycle**. This transformation (minuta into histolytica) is particularly common in countries with warm climates.

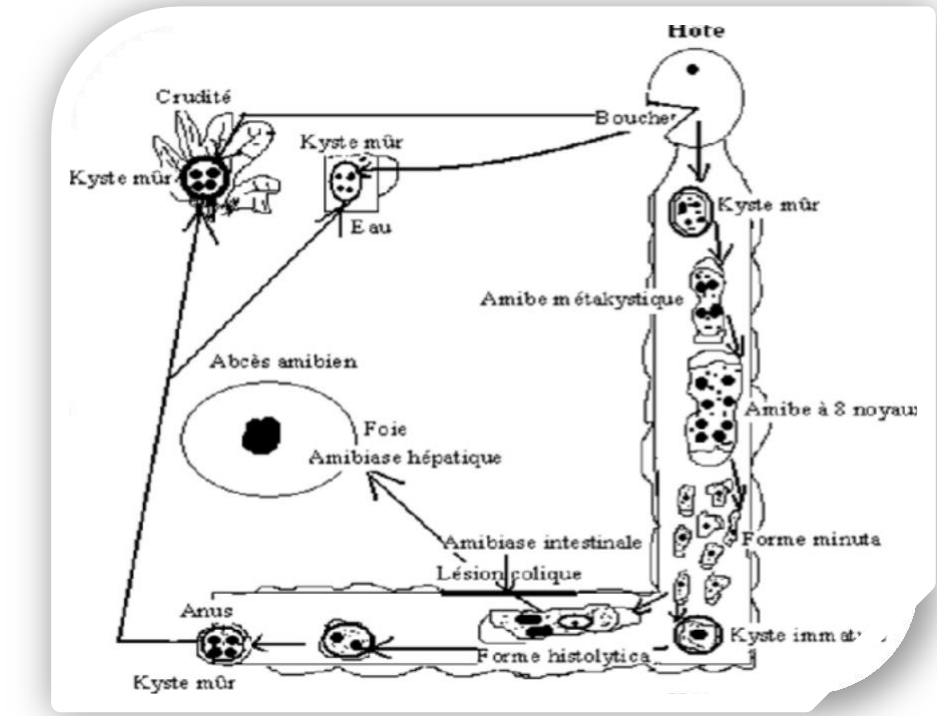


Fig 12. Evolutionary cycle of *Entamoeba histolytica* Location

The hematophagous form lives in the lumen of the large intestine, in the

intestinal wall, in the ulceration it determines in the large intestine. This form can pass through blood and lymphatic vessels and can be found in the **liver**, lungs, brain and spleen.

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

3.5. Epidemiology

Amoebiasis is a digestive parasitosis linked to **human faecal peril** (faecal pollution of the environment by human dejecta contaminating water, raw vegetables, hands and soil). It is carried by flies.

The parasite's reservoir **is man.** The cyst survives a minimum of 15 days in water at 18°C, 10 days in feces, 24 hours dry. It is highly resistant to chemical agents. Vegetative forms are too fragile to survive in the wild and play an epidemiological role. Amoebae are Although cosmopolitan, the parasite thrives particularly in hot, humid regions and is endemic in hot, humid countries (especially Third World countries where fecal hygiene is poorly respected and human excreta are used as fertilizer). Worldwide prevalence is around 10%, i.e. 600 million carriers, 90% of whom are healthy carriers responsible for transmission.

Symptomatology

A certain number of individuals harbor the dysenteric amoeba without showing any symptoms - these are healthy carriers - but all it takes is one cause or another for the disease to break out.

As mentioned above, *Entamoeba histolytica histolytica* can be found in a number of different locations. Broadly speaking, a distinction is made between **intestinal amoebiasis** and **hepatic amoebiasis**.

Intestinal amebiasis

The length of the incubation period is difficult to determine. The first

symptom is ordinary diarrhea, with alternately diarrheic and pasty stools, which gradually change 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 is always less than in bacillary dysentery. In addition to digestive problems, the patient experiences pain at the time of defecation, is tired at the slightest effort, drowses especially after meals, and loses weight. He presents with colicky pains along the large intestine, complicated by very painful epreintes (colicky pains ending in a desire to have a quick bowel movement) and ténesme (painful contracture of the anal sphincter). There is no fever. Overall health remains good, but the individual is a little anemic.

Intestinal amebiasis can lead a number of complications, including blood vessel rupture and tissue necrosis. The most frequent complication is liver abscess (hepatic amebiasis). Other extra-digestive localizations may occur after intestinal amebiasis due to *E. histolytica*: pleuropulmonary or cerebral.

Hepatic amebiasis

Hepatic amoebiasis is the most frequent visceral form. Approximately three months after amoebic infestation (more or less unnoticed in the case of an attenuated form), hepatic involvement initially manifests itself as painful hepatomegaly and moderate fever. Then, in the event of liver abscess formation, we see neutrophil hyperleukocytosis, fluctuating fever, altered general condition and an inflammatory syndrome.

Diagnosis Intestinal amebiasis

Diagnosis is relatively easy during the acute phase of the disease.

Search for hematophagous forms and immature cysts (1-2 nuclei) in the patient's diarrheic stools, or mature cysts (4 nuclei) if the stools are pasty.

Requirements for a good diagnosis :

Direct diagnosis of intestinal amebiasis is carried out on recently emitted

stools (the patient defecates in the laboratory), if it difficult to transport the stool in MIF (Merthiolate Iodine Formol) fixing medium. This test must be repeated three times to avoid the so-called "silent" periods when the parasite is not shed.

Hepatic amebiasis

Amebic stool tests are usually negative in cases of visceral amebiasis (e.g. hepatic amebiasis). Biological diagnosis is then based on serology (ELISA, immunofluorescence, indirect hemagglutination, immunoelectrophoresis).

Treatment

Amebic dysentery is a relatively serious disease in hot, poor countries, where mortality is fairly high.

Intestinal and visceral amoebiasis should always be 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 per os, acting on minuta forms), to prevent distant relapses.

Medication is not everything in the treatment of intestinal amebiasis.

keep the patient at rest, relieve pain and institute an appropriate diet.

Prophylaxis

There is no drug prophylaxis against amoebic dysentery; prevention relies on appropriate hygiene measures, since amoebic dysentery is caused by ingestion of cysts:

* **Individual prophylaxis consists of :**

Avoid ingesting cysts: boil drinking water, carefully wash raw vegetables, protect food from insects (especially flies), wash hands before each meal, avoid direct or indirect contact with patients or amoeba carriers.

* **General prophylaxis consists :**

- Health education: treating the sick, and screening and treating healthy carriers;
- Fecal hygiene: disinfect their feces 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 occupations involving the handling of foodstuffs (especially cooks and bakers).

Nematodes zoonoses

1. Species *Ankylostoma duodenale* and *Necator americanus* (Ankylostomosis)

1.1.General

Hookworm is a cosmopolitan parasite that has become tropical. One billion people are infected. Two nematodes cause human hookworm disease: *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 is found in the same tropical regions, but also in temperate zones: North Africa, southern Europe, northern India and China.

1.2.Epidemiology

Hookworms are round worms measuring around 10 mm in length. Adults live in the duodeno-jejunum of humans. They are attached to the intestinal mucosa by the sharp blades and hooks of their mouth capsule, with which they graze the mucosa and cause bleeding. They cause calculated blood losses of 0.01 to 0.04 ml/worm/day, or 30 ml/day for *Necator americanus* and 0.05 to 0.3 ml/worm/day, or 140 to 400 ml/day for *Ankylostoma duodenale.*

Hookworms are exclusively human parasites, with no intermediate host. Eggs emitted in the stool are non-embryonated. 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 hookworm disease previously described in temperate regions (mines, tunnels).

Embryos develop into rhabditoid L1 larvae, then strongyloid L2, then infective L3. They are highly resistant in the external environment: 2 to 10 months in soil, 18 months in water. Infection occurs in soil, sludge and stained freshwater, most often on the feet. L3 larvae penetrate the skin and enter the heart, lung and trachea via the bloodstream or lymphatic system, 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 the buccal, transplacental or breast-feeding route for Ankylostoma duodenale.

1.3. Clinic

Three clinical phases are described in relation to the cycle: skin penetration, larval migration and adult worm action in the intestine.

✓ Skin penetration phase: pruritic dermatitis, a fleeting maculopruritic erythema seen during the

- ✓ first-time invaders (expatriates in tropical zones, miners in temperate zones). Chronic infection leads to "strangles" in miners. Larval invasion phase: larvae cause airway irritation or catarrh gourmes, allergic manifestations: asthmatic dyspnea, Loëffler syndrome.
- ✓ Intestinal phase with digestive disorders and anemia - digestive disorders: epigastric pain, heaviness or bloating abdominal, painful hunger, pseudo-ulcer pain with pyrosis leading to geophagia (ingestion of earth), diarrhea with 5 to 10 stools per day,
- ✓ anemia with asthenia, exertional dyspnea, tachycardia, palpitations, systolic murmur, pallor mucocutaneous, dizziness, changes in soft, flattened nails (onychomalacia). When anemia is severe, hypoprotidemia with hypoalbuminemia leads to soft, cup-shaped edemas.

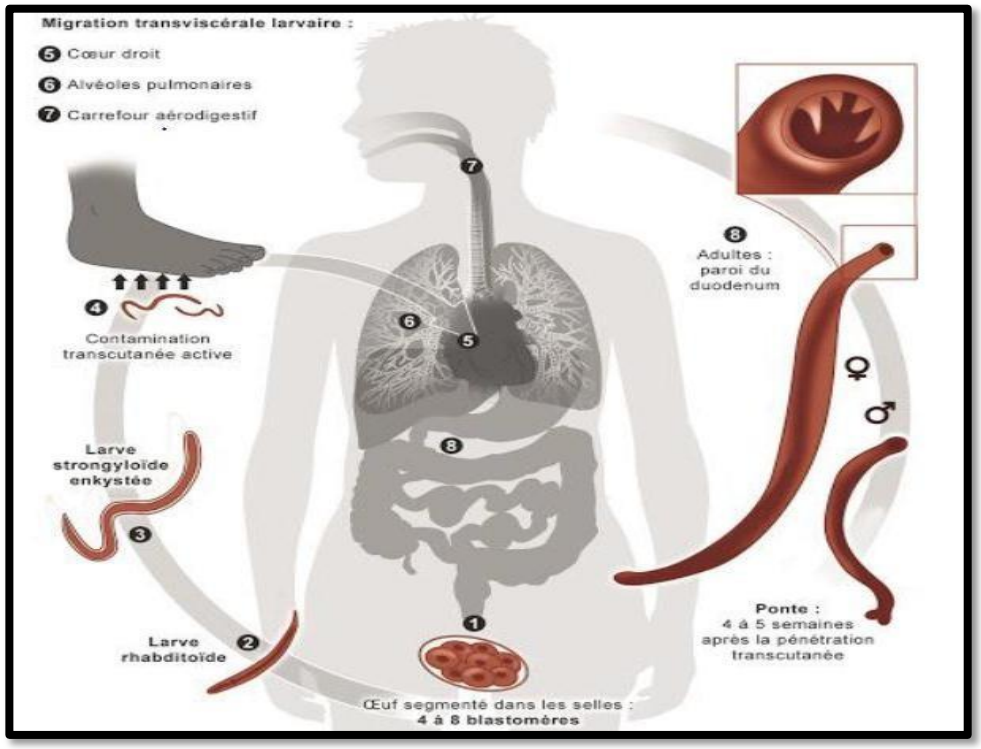


Fig 13. Life cycle of *Ankylostoma duodenale*

Diagnosis

- ✓ **Elements orientation**

- A blood count :

- microcytic, hypochromic, hyposideremic, aregenerative anemia, hemoglobin level may be below 3 g/dl,
- hyperleukocytosis due to hypereosinophilia peaks 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.
- Fall in serum zinc.

✓ **Elements of certainty :**

Examination parasitological of stool (directdirect, techniques concentration)

- Characteristic eggs: ellipsoid, symmetrical, thin-shelled and transparent, 60 μm by 40 μm, containing 4 blastomeres (Ankylostoma duodenal) or 8 blastomeres (Necator americanus) - Egg count

This is used to judge the extent of the infection (moderate infection: 2,000 eggs/g of stool, moderate infection: 2,000 to 10,000, severe infection > 1,000 eggs/g of stool).

10 000). - Stool examination must be carried out rapidly, within 3 hours, otherwise blastomere segmentation will continue and the 2 hookworms will be indistinguishable from each other; within 24 hours, otherwise transformation into rhabditoid larvae, then strongyloid larvae, and hookworm and anguillosis will be indistinguishable.

Note that upper digestive endoscopy shows edematous, purpuric duodenitis and, in rare cases, adult worms embedded in the mucosa. 3.5. Treatment Antiparasitic treatment involves benzimidazoles: mebendazole (VERMOX®) or albendazole (ZENTEL®) at the same doses as for ascariasis).

Pyrantel, as 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 pyrantel for A. duodenale. Therapeutic alternatives could include combination therapies such as mebendazole 500 mg+ levamisole (SOLASKIL®) 80 mg as a single dose, or albendazole 400 mg + ivermectin 200 μg/kg as a single dose.

Anemia is treated with oral ferrous sulfate: 200 mg/d for adults, 10 mg/d for children under 30 kg for 3 months, and folic acid 20 mg/d for 3 weeks. If the hemoglobin level is

< 5 g/dl, packed red blood cells are prescribed.

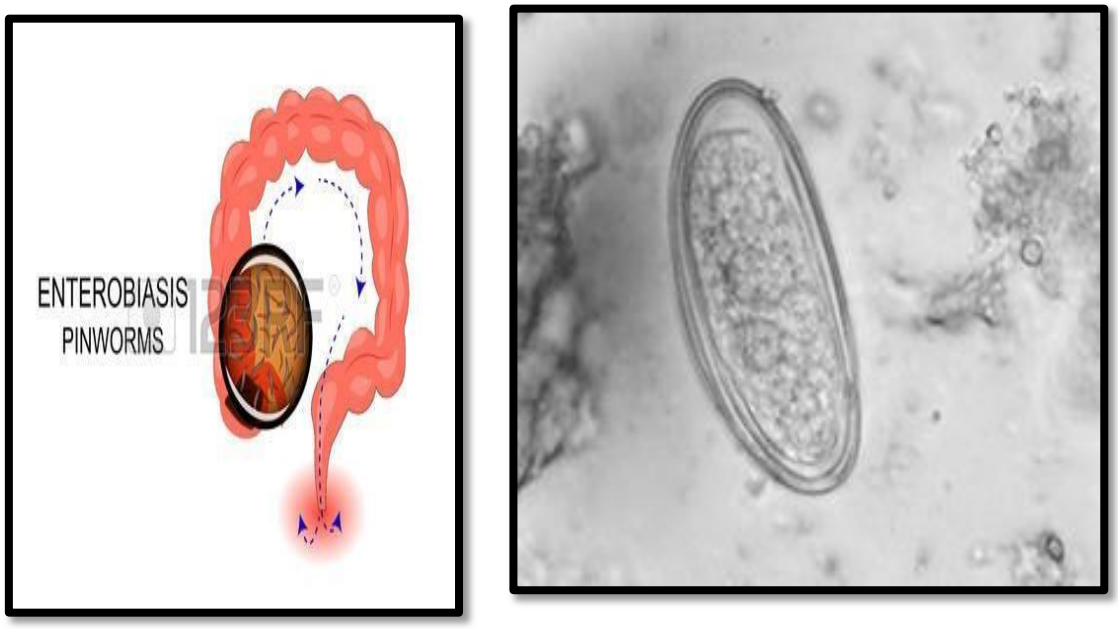
- ✓ **Prophylaxis**

A fecal peril disease, hookworm prophylaxis is identical to that for ascariasis. In addition, individual prevention requires walking with shoes.

2. Genus Enterobius (Oxyurosis)

2.1.General

Pinworm disease is caused by a nemathelminth, *Enterobius vermicularis*. It is a cosmopolitan digestive parasitosis, affecting up to a billion people, and very common in both temperate and tropical zones.



2.2. Epidemiology

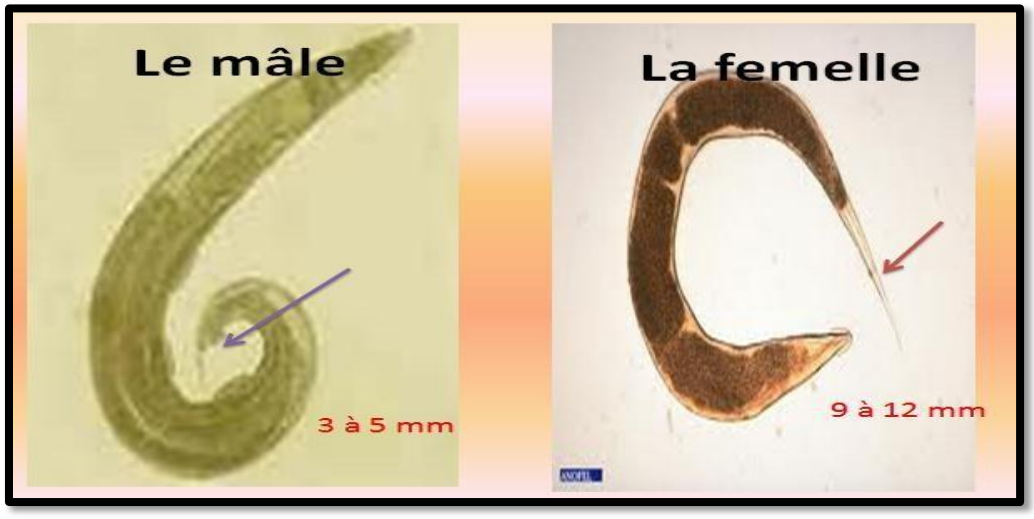


Fig 14. Morphology of nemathelminth, *Enterobius vermicularis.*

2.3. Lifecycle

The pinworm cycle is simple: swallowed eggs hatch in the stomach and duodenum, giving rise to larvae that move into the ileum and cecum. The adult larvae mate, and the females migrate to the anus, where they lay eggs in the anal margin, embryonating within a few hours.

The reservoir of parasites is man alone. Transmission is direct from the anus to the mouth via the fingers, particularly in children; indirect via objects and food. Transmission by inhalation of embryonated eggs in dust is also possible. Pinworm is a parasitosis of family life and communities, especially among children.

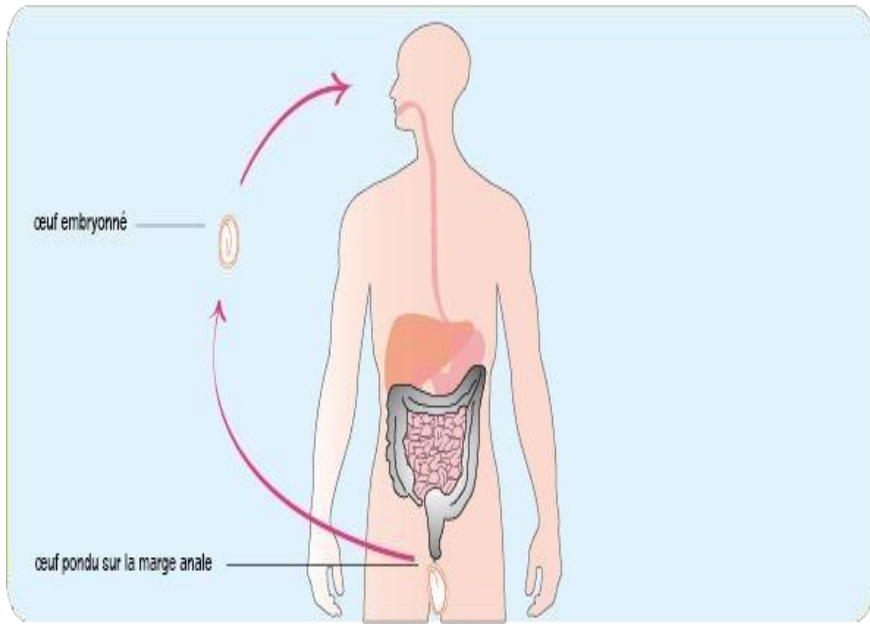


Fig 15: Lifecycle of Pin worm

2.4.Clinic

Pinworm disease is often asymptomatic. The essential sign, if it is symptomatic, is anal pruritus, especially in the evening and at night. It leads to scratching lesions. Abdominal pain, sometimes diarrhea with soft, mucus-coated stools, sometimes streaked with blood, may accompany the pruritus. Character changes have been described in children (vermin syndrome).

Complications include genital and urinary manifestations in girls, such as vulvovaginitis and urinary tract infections with E. coli. Subacute appendicitis with intra-luminal pinworms is not uncommon.

Diagnosis

It is parasitological:

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

Treatment

Benzimidazoles are used: VERMOX® or FLUVERMAL® 100 mg/ 1 day, ZENTEL® 400 mg/1 day. Alternative: pyrantel pamoate (COMBANTRIN®) 11 mg/kg/1 dose pryvinium emboate (POVANYL®) 5 mg/d/3 days, with the disadvantage of red stools. Whichever medication is prescribed, a 2nd course of treatment should be administered on D15. In case of recurrence, treat the whole community with VERMOX® or FLUVERMAL®, 2 tablets at 100 mg for 3 days, then 1 tablet every 15 days

for 3 months.

Prevention

The key is to combat direct transmission (auto-infection): it consists of regular hand-washing and nail-cleaning, wearing closed pyjamas at night, and caring for the child's bedding, underwear and objects (especially toys). The fight against faecal peril is of secondary importance.

3. Species *Trichiuris trichiura* (Trichocephalosis)

3.1. General

Trichocephalosis is caused by a nematode, *Trichiuris trichiura*. It is a cosmopolitan intestinal parasitosis which is usually asymptomatic, but which, in the event of massive infection, can lead to serious symptoms in young children.

3.2. Epidemiology

Adult worms live in the colon. Their filiform anterior end is embedded in the intestinal mucosa, especially in the cecum, due to the relative stercoral stagnation at this level. They feed on blood. Eggs are eliminated in the stool and embryonate in the environment. Humans are contaminated by ingesting embryonated eggs with food or by hands contaminated with soil. In the small intestine, the embryo gives rise to a larva that settles 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 (over 1000 worms) lead to anemia.

According to the WHO, between 500 and 800 million people are affected. Underprivileged populations, and in particular children aged 2 to 7 living in unhygienic conditions, are the main sufferers.

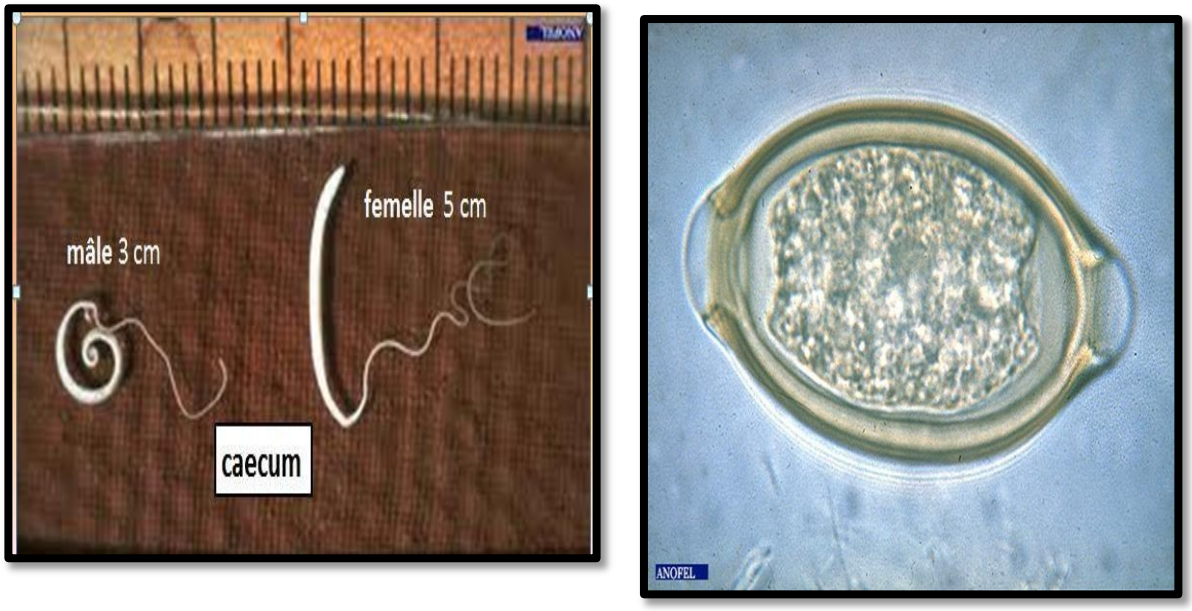


Fig 16. Morphology of nemathelminth, *Trichiuris trichiura* (Trichocephalosis)

3.3.Clinic

There are 2 phases:

- **The invasion phase** is always asymptomatic, with blood hypereosinophilia as the only stigma.
- **A state or intestinal phase** in which clinical expression depends on the parasite load:
- **Asymptomatic**, the most common form, **Symptomatic** or **trichocephalosis disease**, uncommon: it occurs mainly children or in the elderly. immunocompromised adults. Two forms can be distinguished:
- the minor form is characterized by a dyspeptic syndrome with nausea and flatulence, often associated with constipation, weight loss and a "vermin syndrome",
- the major form is **massive infantile trichocephalosis,** linked to

massive involvement of the colon, from the cecum to the rectum. It affects children aged 2 to 7, and produces a number of clinical pictures: trichocephalic enteritis with abdominal pain and diarrhea leading to dehydration; trichocephalic appendicitis; trichocephalic rectocolitis producing a dysenteric syndrome with tenesmus and epithelium. Rectoscopy makes the diagnosis, showing numerous worms embedded in a hyperhemic mucosa with hemorrhagic staining and sometimes ulcerations. Rectocolitis may be complicated by profuse rectal haemorrhages and rectal prolapse lined with white filaments (adult worms) embedded in a haemorrhagic mucosa. This severe form is accompanied by hypochromic, microcytic, hyposideremic anemia.

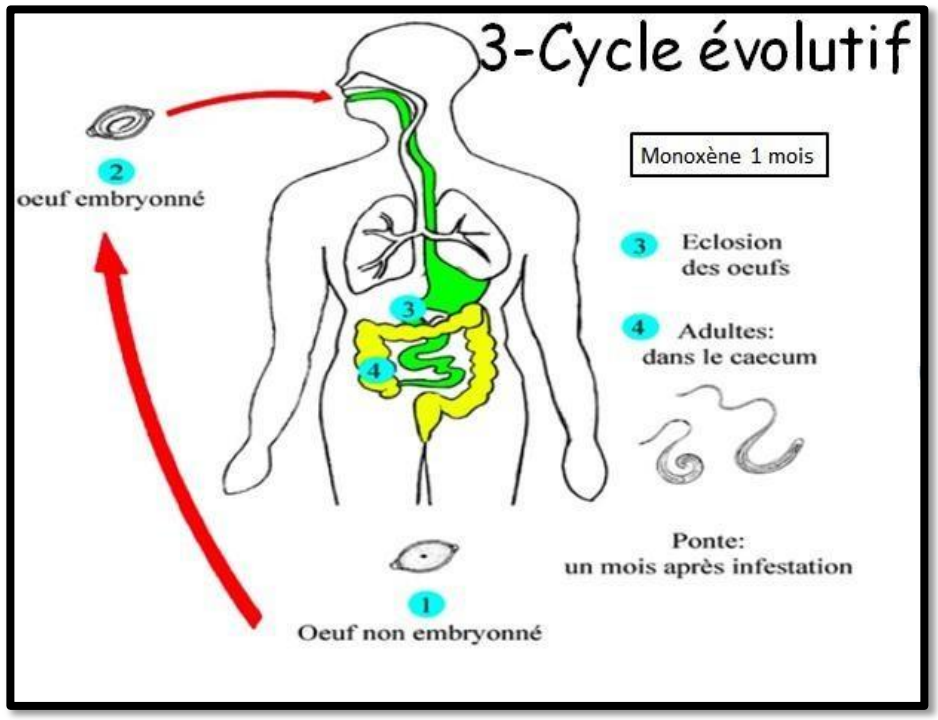


Fig 17. Life cycle of *Trichiuris trichiura* (Trichocephalosis)

Diagnosis

Diagnosis is parasitological: characteristic ovoid eggs with translucent mucous plugs at both poles. They are surrounded by a thick, dark-brown shell. They measure 50 µm by 22 µm. Their "little lemon" appearance makes them easily recognizable under the microscope. Diagnosis is also endoscopic, with adult worms found in the rectum and/or cecum.

Treatment Benzimidazoles are used: mebendazole or flubendazole 200 mg/d x 3 days for all ages, or albendazole 400 mg/d x 1 day for adults and children over 2 years of age, and 200 mg/d x 1 day for children under 2 years of age. A second course of treatment is prescribed on D15. Nitazoxamide is an alternative treatment.

Prevention: Fecal peril disease, prophylaxis is identical to that for ascariasis.

Trematodes zoonoses

1- Fasciola genus

Study of *Fasciola hepatica*, commonly known as the liver fluke. Disease: Distomatosis or fasciolosis is a zoonosis caused by a trematode, *Fasciola hepatica*, localized in the adult state in the bile ducts of many animals.

many herbivores and occasionally of man. Contamination occurs through consumption of raw plants (watercress, dandelion, etc.). Classical symptoms include hepato-biliary signs associated with hypereosinophilia. Significant economic losses can be recorded in pastoral regions.

1.1.Classification

Kingdom: Metazoa

Phylum: Platyhelminthes

Class: Trematoda

Subclass: Digenea

Order: Prosostomata

Suborder: Paramphistomata - Distomian group

Family: Fasciolidae

Genus: Fasciola

Species:*Fasciola hepatica*

1.2. **Morphology**

Adult: is a small leaf-shaped pinkish flatworm, 2 to 3 cm long and about 1 cm at its widest point. It is hermaphroditic. It has two suckers at its front end, enabling it to attach itself to the epithelium of its host's bile ducts. They are hematophagous. The cuticle is thick and covered with spines. Digestive caeca are branched.

Location: in the bile ducts of many herbivores and occasionally humans.

Egg: ovoid, elongated, rugby ball-shaped. It is 140 µm long and 75 µm wide. It is capped and not laid.

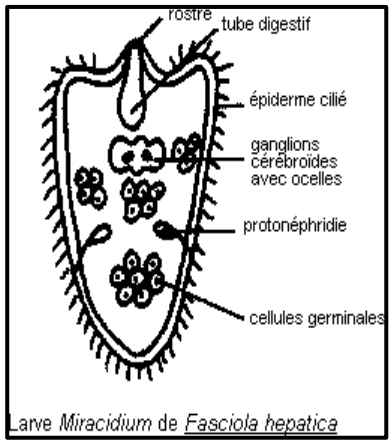
The miracidium: It has a ciliated epithelium, making it suitable for swimming. There are two cerebroid ganglia, two dorsal ocelli and two protonphridia. It has a rudimentary, non-functional digestive tract, masses of cells that have remained embryonic and germ cells. Its lifespan is 8 hours.

Sporocyst: This is the phase following miracidium enlargement. are no sense organs. The general cavities divide, each giving rise to a new type of larva. Each sporocyst produces 5 to 8 redia.

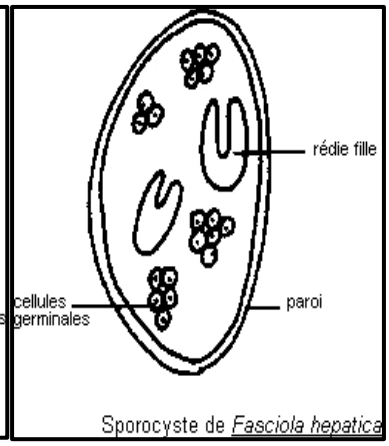
Redia: each redia measures between 1 and 3 mm, and is an elongated sac with a mouth, a muscular pharynx, a simple digestive tract and an egg- laying orifice at the front. It still contains germ cells.

The cercaria: It has the same organization as the adult fluke: two suckers, a two-branched digestive tract, an excretory apparatus, cerebroid ganglia, but no differentiated genitalia. The tail is muscular, and the larva has numerous cystogenic glands. The life span of the free-living cercaria is just a few hours. The cercarial body measures between 0.25 and 0.35 mm.

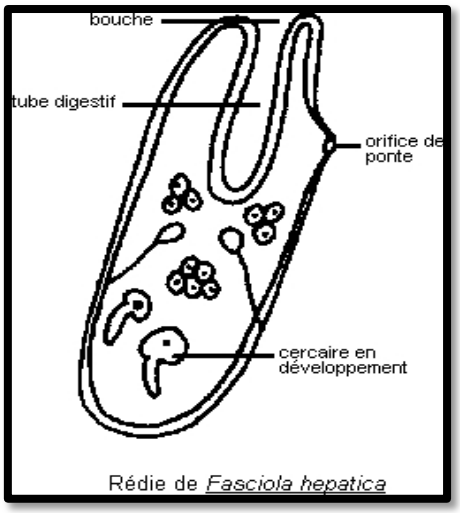
Metacercaria: this is an encysted, infesting form attached to an aquatic plant. It has a diameter of around 0.2 mm.



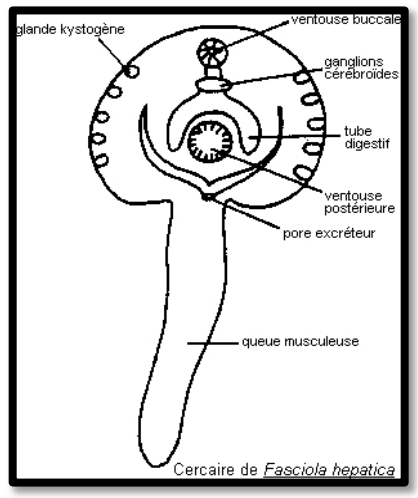
Larve *Miracidium* de *Fasciola hepatica*



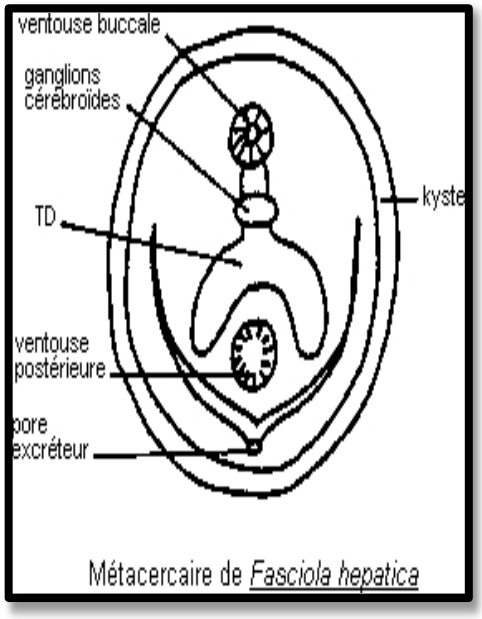
Sporocyste de *Fasciola hepatica*

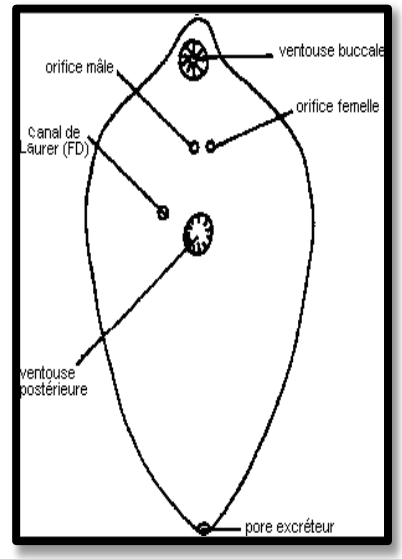


Rédie de *Fasciola hepatica*



Cercaire de *Fasciola hepatica*





Adult *Fasciola hepatica*

Fig 17. **Morphology of different *Fasciola* forms**

1.3.**Evolutionary cycle** .

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

The adult (approx. 3 cm) lives coiled 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 reach the water, they continue their evolution. The larva they engender escapes by lifting the polar operculum and swims vigorously thanks to its ciliated integument: it's called **miracidium**. It is attracted by almost all species of **Limnea** (**pulmonate gastropod molluscs**), principally ***Limnea trunculata***. It penetrates the mollusc's lung, where it transforms into a **sporocyst** through degeneration of all organs except the integuments (which

lose their cilia) and reproductive cells. In this sac, the germ cells come into play, forming larvae or **redia** with a rough digestive tract and clusters of germ cells. When the sporocyst bursts, the redia are released and migrate into the hepato-pancreas of their host. At low temperatures, they rise to other redia, the **daughter redia**. If the temperature rises, they give rise to a new type of larva, the **cercaria**, in which a young trematode with suckers, digestive tract and locomotor tail is emerging. The cercaria emerges from the limnea, working its way through the viscera (the mollusc's development time under optimal conditions (20-22°C) is 40 days). After a short period of free living, it attaches itself to the underside of a **semi-aquatic plant** (near the surface of the water), loses its tail and secretes a thick cystic envelope (enabling it to withstand the external environment for several weeks), thus becoming the infesting **metacercaria**. The definitive host becomes infected by ingesting the plants to which the metacercariae are attached. The cystic wall is digested in the duodenum; the young fluke crosses the liver parenchyma, creating lesions, before settling in the bile ducts (10 to 20 days after infection), where it becomes an adult 3 months after infection. Adult flukes live for around 3 to 5 years.

Noting that contamination is often seasonal, beginning at the end of the summer or autumn. Rainy summers, which are favorable to snails, increase the risk of contamination.

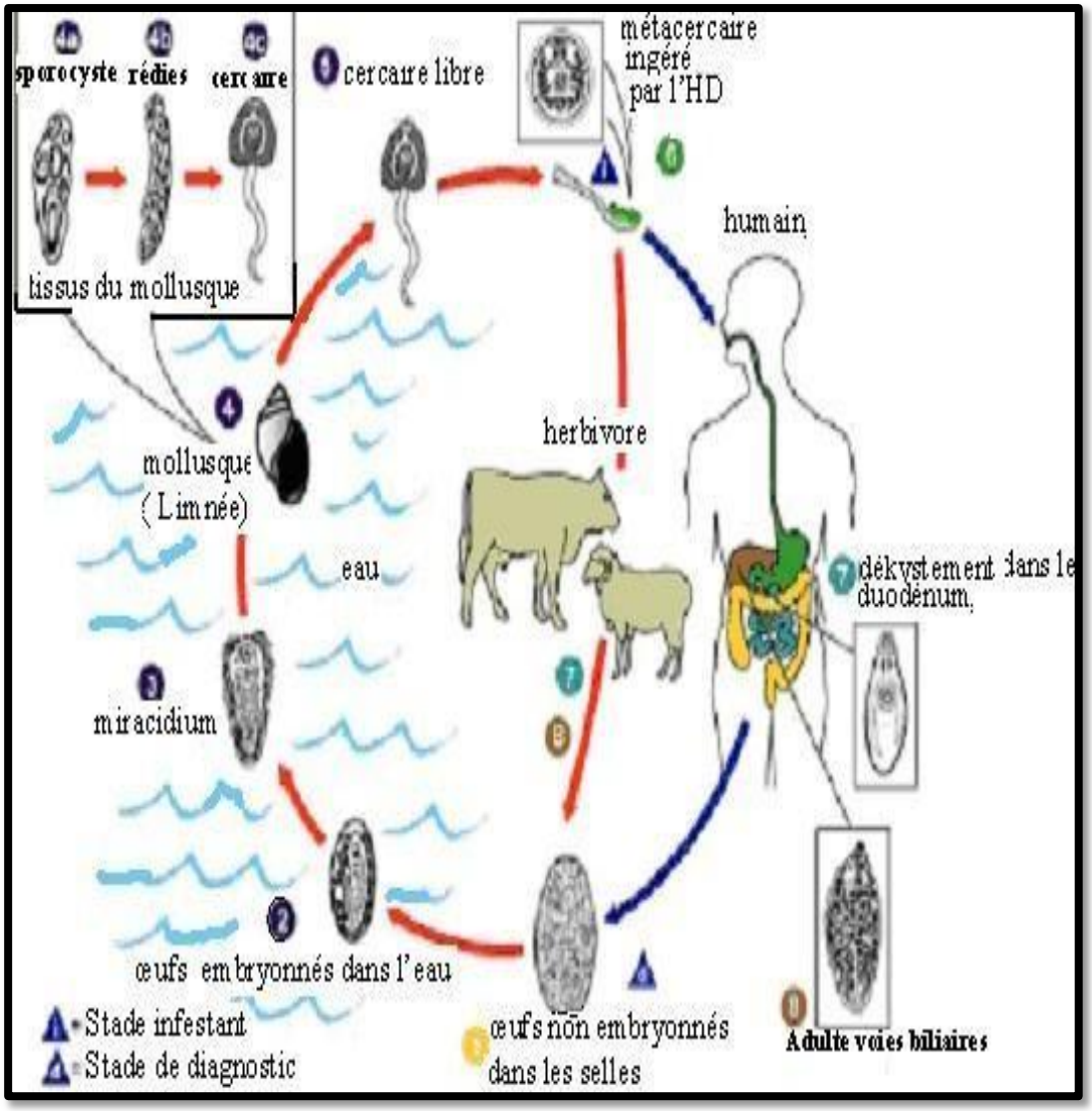


Fig 18. Life cycle of *Fasciola hepatica*

1.4.**Ecological changes:** control is currently focused on mollusc vectors, the most vulnerable point in the epidemiological chain. The use of molluscicides is a technique that is likely to systematic application. Some molluscicides do not spare fish, an important source of food. Ecological methods can be used, such as periodically draining irrigation

canals and destroying the vegetation on which molluscs feed. The use of molluscs that compete 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 water, even for very short and very partial immersions. Swimming should only be allowed in seawater or in a duly controlled swimming pool.

2.Genus Schistosoma

The metacercal stage does not exist. Species of this genus live in the circulatory system of mammals. The sexes are separate and they are hematophagous. Several parasitize humans. Adults live in the veins. The male houses the female, who is very threadlike, in a groove on his ventral side (gynecophore canal). The two suckers are very close together and are located in the anterior part of the animal's body. There is no pharynx.

Bilharziasis or schistosomiasis are parasitic diseases caused by trematodes, flat worms with separate sexes, hematophagous, living in the adult stage in the circulatory system of mammals and evolving in the larval stage in freshwater molluscs. There are 200 million cases of bilharziasis in the world, and five species are pathogenic to humans and endemic on three continents

2.2. Classification

Phylum: Platyhelminthes

Class: Trematoda

Subclass: Digenea

Order: Prosostomata

Suborder: Strigaeta

Family: Schistosomatidae

Genus: Schistosoma

Species: 5 species that can parasitize humans:

Schistosoma haematobium: causes urogenital bilharziasis

Schistosoma mansoni: causes intestinal and hepatosplenic bilharziasis

Schistosoma intercalatum: causes rectal bilharziasis

Schistosoma japonicum: causes arteriovenous bilharziasis

Schistosoma mekongi: causes arteriovenous bilharziasis

2.3. Study of *Schistosoma haematobium*

2.3.1. Morphology

We distinguish between the different stages, except for the redia and metacercaria stages (see photos).

Adult: present in the definitive host (man); sexes are separate. The 2 suckers are in a very anterior position. The integument is warty and spiny, making it easier to attach the animal to blood vessels. Male: 10 to 15 mm long and 0.7 to 1 mm wide, with a foliaceous appearance; the lateral edges are folded into a gutter forming the gynecophore canal. The genital pore is located under the ventral sucker, where 4 to 6 testes are found. Female: 15 to 20 mm long and 0.1 mm wide. She is cylindrical in shape. The ovary is located in the middle of the animal's body; the number of eggs visible in the uterus is 20 to 30.

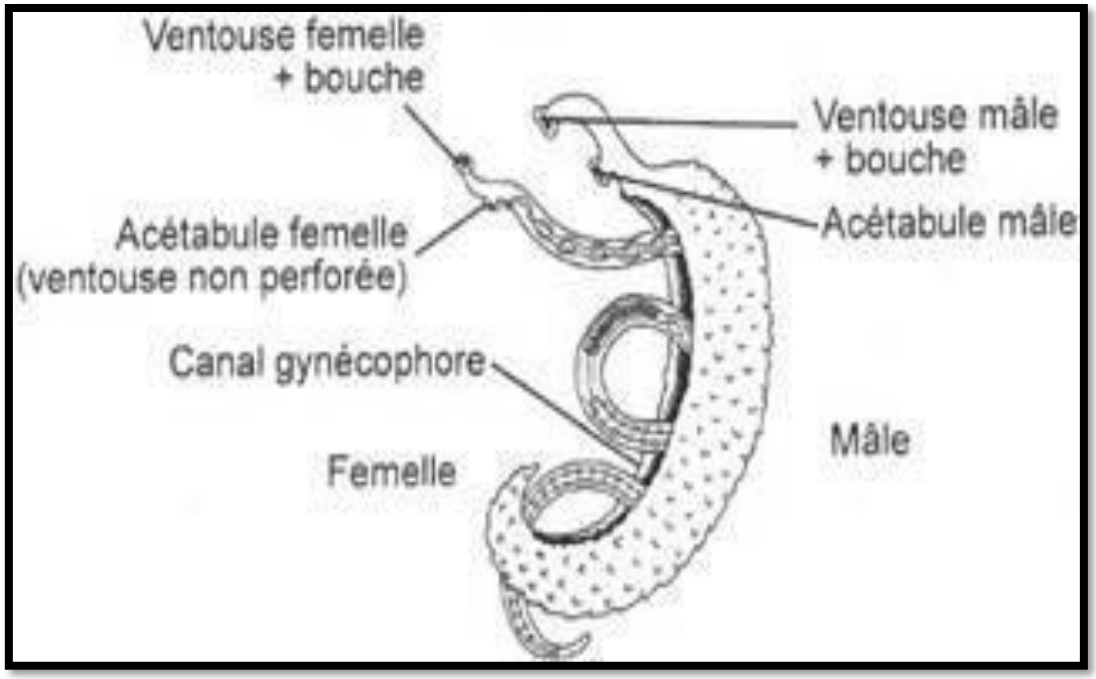
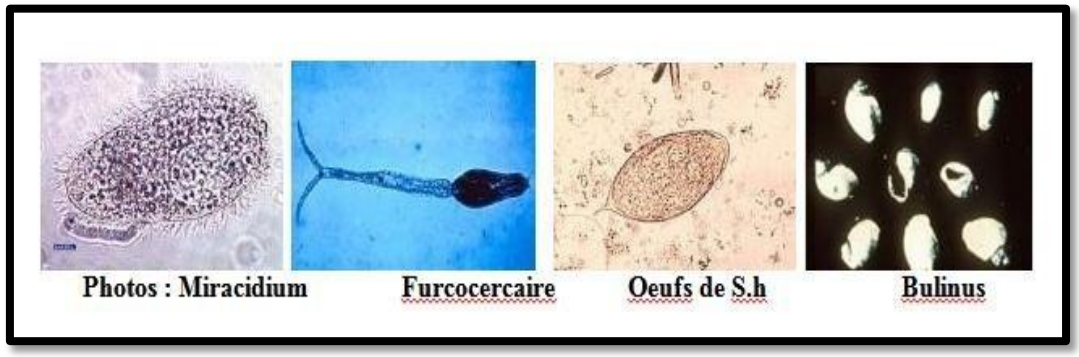


Fig 19. Morphology of *Schistosoma haematobium*

Eggs: egg-shaped, clear, with a 15 µm terminal spur. Egg size averages 140 µm long by 60 µm wide. It should be noted that the eggs are not embryonated when laid by the female, but when exteriorized with urine. It is these eggs that determine the disease. Adults live up to 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 (human) form.



2.3.2. Evolutionary cycle

The cycle is indirect. **HD** = man, **HI** = Bulin, 1 cm high freshwater conoid mollusc (genus *Bulinus*); **R.P=** man.

Adult worms are found mated in the large hepatic and mesenteric venous trunks, where fertilized females ascend the network of vesical capillaries and lay non-embryonated eggs. Using their spurs and lytic enzymes, the eggs leave the capillary and break through the tissue until they fall into the bladder lumen. The eggs are expelled into the environment by urination (S.h.) and if conditions are favorable (pH close to neutral and temperature between 18°C and 33°C), on contact with fresh water, they release a ciliated larval form: the miracidium (short-lived: a few hours) which must swim in search of the mollusc specific to the schistosome species: genus *Bulinus* for S.h. In the mollusc's hepatopancreas, at the right temperature (30°C), larval forms give rise to stage I and II sporocysts. The evolution

larval development in molluscs takes 1 month. The ultimate form larval evolution emerges from the mollusk: the cercaria, which measures 0.5 mm, has a bifid "tail" (furcocercaria). Through polyembryony, one miracidium produces thousands of cercariae (10,000). Furcocercaria circulates in water, ready to penetrate any immersed part of the human body via the

transcutaneous route in just a few minutes (oral contamination is very rare). The survival time of furcocercaria is short (24 to 72 hours), and it is by chemotaxis that they are attracted to and penetrate the integuments of the definitive host (by the action of enzymes secreted by the head of the furcocercaria; duration of transcutaneous passage: a few minutes). As soon as the furcocercariae are attached, they separate from their "tail" and the anterior part, or schistosomula, is carried by the lymphatic system into the general circulation. From the 48th hour, and for several days, they are in the pulmonary capillaries, then reach the heart, and via 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 upstream against the current. The fertilized females then separate from the males and, according to a particular tropism, enter the fine ramifications of the bladder, where they deposit their eggs.

2.3.3. Symptomatology

Apart from the irritating action of cercariae penetrating the skin and the toxic phenomena caused by the migration of schistosomules and adults, it is essentially the parasite eggs that are responsible for the anatomical lesions and consequently the clinical disorders observed.

Invasion

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

Toxemic phase

It coincides with the migration and maturation of schistosomules in the bloodstream and intrahepatic portal vessels. It may be marked by general malaise asthenia, fever, headache, anorexia (safari fever) accompanied by

anaphylactic symptoms: pruritus, arthralgia, myalgia, urticaria...

Status

This is when the females lay their eggs. In the case of urogenital Bilharzia, the main symptom is hematuria. Hematuria is painless and unpredictable. It may be microscopic and discovered by chance, or macroscopic, discrete and terminal, or abundant and complete with clots.

Complication phase

It corresponds to egg retention. In the case of *S. haematobium*, the entire urinary tract may be affected: urethral fistula, urethral stenosis, ureterohydronephrosis, superinfection (cystitis, pyelonephritis, pyonephrosis, etc.), bladder lithiasis, glomerulonephritis. The genital systems of both sexes can be affected: urethritis, epididymitis, spermato-cystitis, prostatitis, salpingitis, endometritis, vaginitis, cervicometritis can lead to impotence and sterility. The prognosis of urogenital bilharziasis is mainly renal.

2.3.4. Epidemiology:

The disease is linked to human urinary peril, bulins and furcocercariae in fresh and warm water. Human contamination occurs through transcutaneous penetration. The disease is endemic to tropical regions: Africa (North Africa, Nile Valley, South Africa), the Near and Middle East. Approximately 100 million people are infected.

Diagnosis

Orientation diagnosis :

- Epidemiological: stay in a bilharzia-endemic zone
- Clinical: urinary hematuria
- Biologicalhypereosinophilia

Diagnostic methods will vary throughout the schistosome cycle:

- During invasion phase: the host's reaction leads significant

hypereosinophilia (rarely diagnosed at this stage).

- During the maturation phase :

Direct method: eggs are emitted and may be found in the urine; hence The detection of eggs provides indisputable proof of parasitosis. The best test is to examine the sedimentation pellet of 24-hour urine, after eliminating any chemical substances (mainly phosphates and blood) that may interfere with the reading. This 24-hour urine should collected, if possible, after a premicturition physical effort (walking, climbing stairs, pelvic gymnastics, jumping, etc.), which improves the sensitivity of the examination.

Indirect methods

These are serological techniques

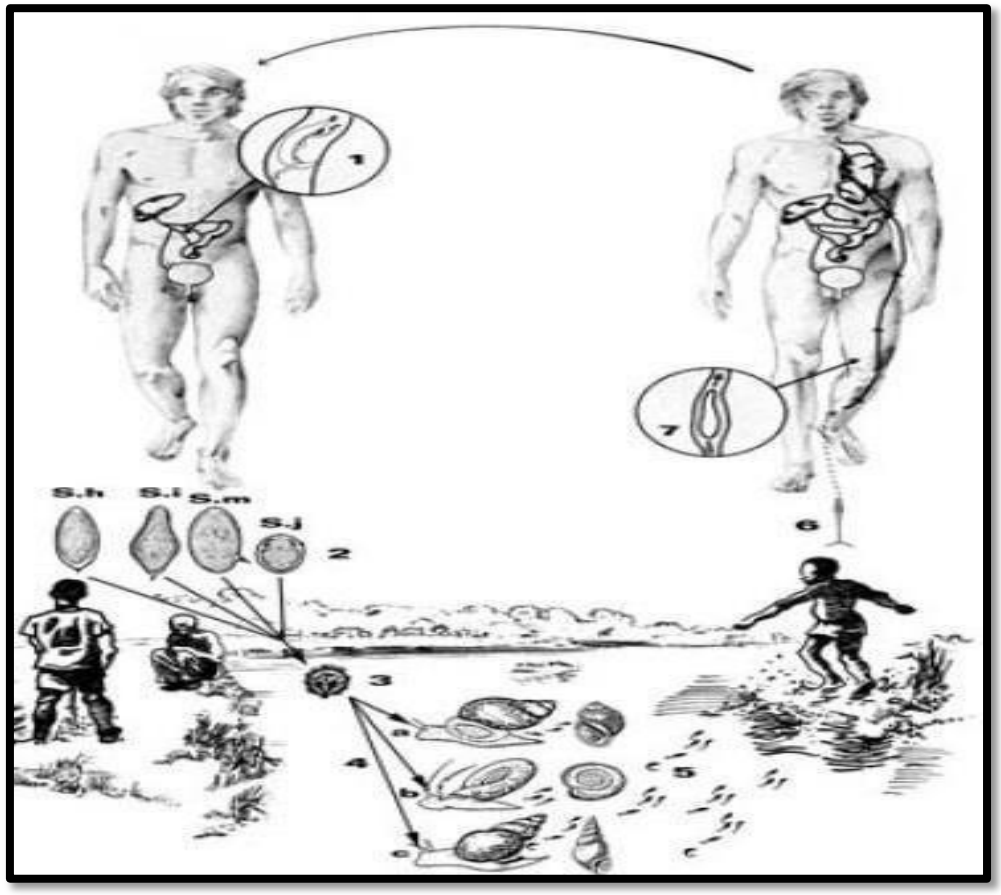


Fig 20. Schistosome life cycle

1. Adults live in the abdominal arteriovenous plexuses 2. eggs areliminated into the environment through urine: *S. haematobium* (S. haematobium, S. haematobium, S. haematobium)

Treatment

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

Prophylaxis

General prophylaxis: This is based on :

Health education and prevention of contamination of water bodies by urine, but individual protection against parasitic water remains a function of improved living standards.

Cestodes zoonoses

1. Genus Diphyllobothrium

1.1.Morphology: Diphyllobothrium is a tapeworm (class Cestodes, order Diphyllobothriidae, family Diphyllobothriidae). which can live for several years in the digestive tract of humans. It is responsible for a parasitic digestive infection called diphyllobothriosis.

Fig 21. Species *Diphyllobothrium latum*

1.2. Life cycle: The parasite's life cycle includes a definitive host

These include man (and other fish-eating mammals) and at least two intermediate hosts: a planktonic crustacean and one or more freshwater fish. Under favorable environmental conditions, eggs (45 x65 µm) emitted into freshwater with the fecal matter of the final host, complete their maturation in 8 to 12 days, then hatch and release a ciliated embryo, the coracidium.

This is ingested by a microscopic crustacean of the Cyclops or Eudiaptomus genus and transforms into a larva (known as a procercoid) within the general cavity. When a carnivorous fish ingests this planktonic crustacean, the larva transforms into a second type of larva (called a plerocercoid**),** several millimeters long. This larva encysts in the musculature or viscera of the fish. Man and other fish-eating mammals then become contaminated by ingesting the raw or undercooked flesh of these freshwater fish. Once in the intestine of the definitive host, the plerocercoid larva grows several centimetres per day, and the first eggs are shed with the feces, around one month after infestation. Several species of this parasite are pathogenic to humans, but only D. latum can be contracted from freshwater fish in mainland France. However, cases of D. nihonkaiense (Pacific species) have been observed in consumers of salmon (Onchorynchus sp.) imported from the Pacific (Canada).

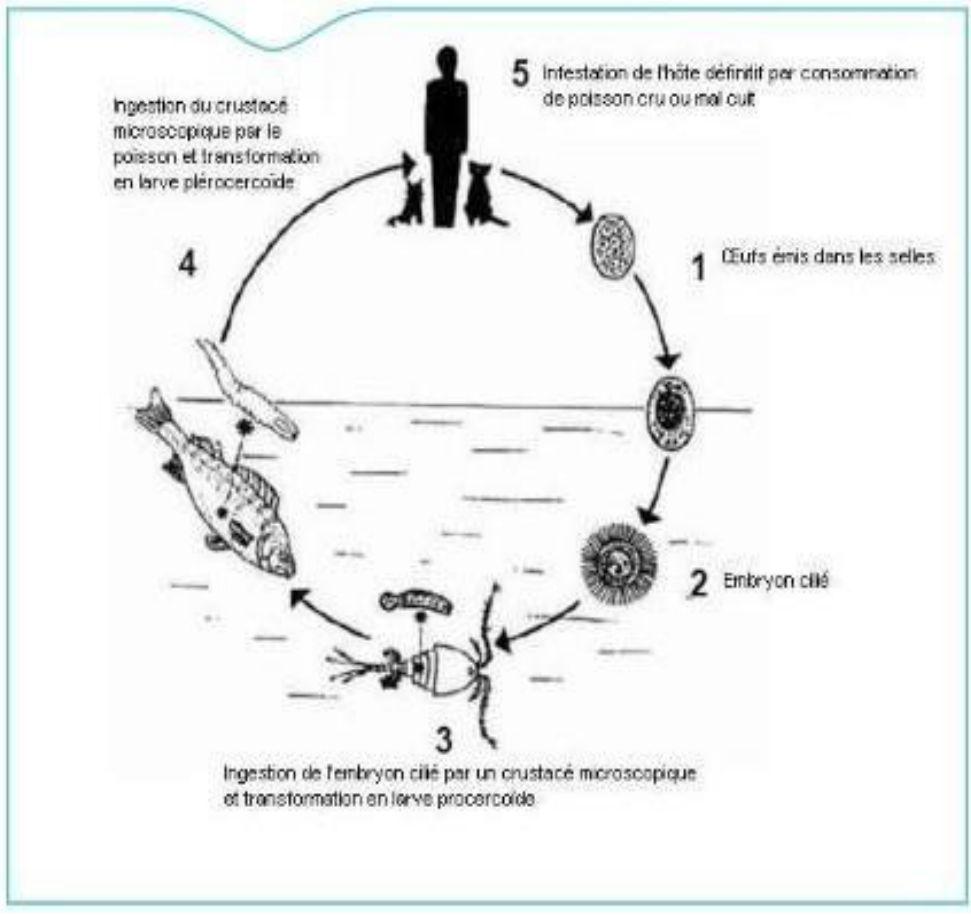


Fig 22. Life cycle of *Diphyllobothrium latum*

1.3. Sources of danger

The source of the danger is carnivorous freshwater fish or anadromous (i.e. living in both fresh and salt water). The reservoir consists of the parasite's definitive hosts (humans, cats, dogs, foxes, etc.) and intermediate hosts (carnivorous fish).

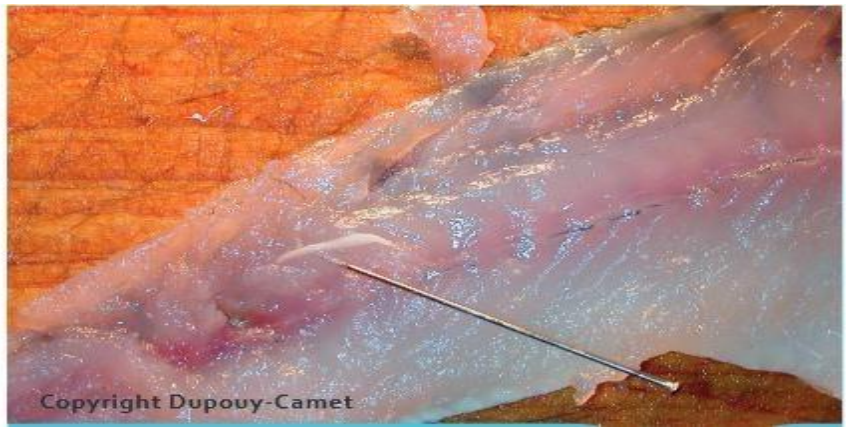


Fig 23. Plerocercoid larva on the surface a perch net

1.4.Transmission routes

This parasite affects piscivorous mammals and fish, and is therefore to be avoided. the origin of a zoonosis4. Human contamination occurs exclusively through ingestion of raw or undercooked fish flesh or eggs. Eggs released with human faeces are not directly contaminating.

1.5.Epidemiology

is no surveillance system for diphyllobothriosis in France. and Europe (apart from Poland and the Baltic states). Sources of danger

The source of the danger is carnivorous freshwater or anadromous fish (i.e. living in both fresh and salt water). The reservoir consists of the parasite's definitive hosts (humans, cats, dogs, foxes, etc.) and intermediate hosts (carnivorous fish).

Diphyllobothriosis is a cosmopolitan parasitosis, still present in Western Europe. It is on the wane in the Baltic and Scandinavian countries, which were the parasite's historical hotbeds. On the other hand, it seems to be emerging in French- and Italian-speaking areas of peri-alpine lakes, where

professional fishing is often practiced.

Household hygiene

Recommendations for consumers

- Parasite inactivation is ensured by :
- thorough cooking (60°C for ten minutes or 65°C for one minute). Pink cooking at edge is insufficient to inactivate any larvae that may be present.
- freezing (-20°C for 24 hours) fish in a freezer domestic.

- Wild fish should be eaten after cooking or freezing (-20°C for 24 hours).

2. Genus *Echinococcus*

2.1.Classification

Phylum: Platyhelminthes

Class: Cestoda,

Order: Cyclophyllidae

Family: *Taeniidae*

Genus : *Echinococcus*

Species: *E. granulosus*

E. multilocularis

Subspecies *E.granulosus granulosus ; E.granulosus equinus ; E.granulosus borealis; E.granulosus canadensis*

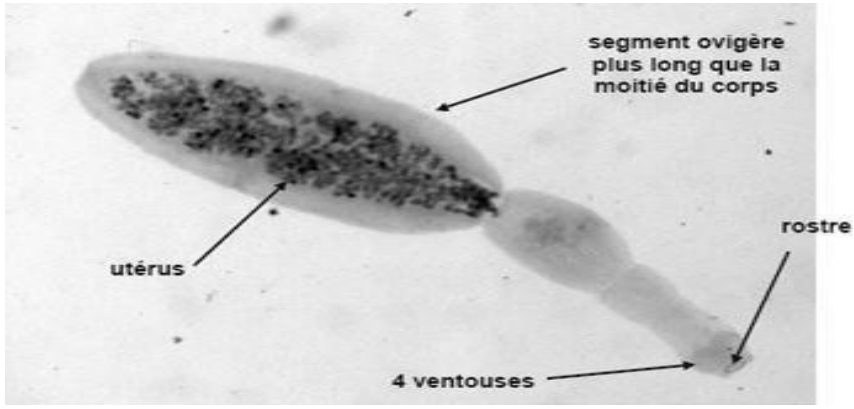
2.2.*E.granulosus granulosus* study

2.3.1. Morphology

The **adult** form is found in canids (small intestine). It can measure from 4 to 8 millimeters in length. The scolex has 4 suckers and a rostrum with a double

crown of hooks. The strobile consists 3 to 5 proglottids. The first two proglottis are maturing; the third, longer than half the body, formed genitalia (ovigerous); the last two contain fertilized eggs. Each cucurbit contains 300 to 800 eggs.

The compound vesicular **larva** (hydatid) develops in humans, cattle, sheep, camelids and goats, causing **hydatid echinococcosis** (hydatid cyst). The vesicles are numerous and voluminous. Hydatid larvae develop in the liver and lungs of sheep, cattle, camels, goats and humans.



a

b

Fig 24. Photos of an adult and a Hydatid cyst of *E. granulosus*

2.3.2. **Evolutionary cycle**

This tapeworm, whose reproductive cycle includes multiplication phases in the larval stage, has a heteroexene cycle (indirect cycle). **HD**= canidae (adults are numerous and live for several months in the digestive tract). The exteriorization of the wall segments is done with the faeces; **HI** = herbivores including man, the larva is located in the viscera (survival of the larva for a few years). The larva does not emerge.

Each cucurbit contains 300 to 800 eggs, which are released with the dog's excrement and then ingested by herbivores. Humans can become accidentally infected through contaminated raw vegetables, or by touching a dog carrying the parasite (anal pruritus).

The worm passes through the intestinal wall into the herbivore's liver, where it differentiates into a ball 20 centimetres in diameter: the hydatid stage. This stage develops slowly: 16 months in sheep and cattle; 18 months to 30 years in humans. In the hydatid, scolexes bud from the inner membrane (proliga), reaching up to 400,000 scolexes per cubic centimeter.

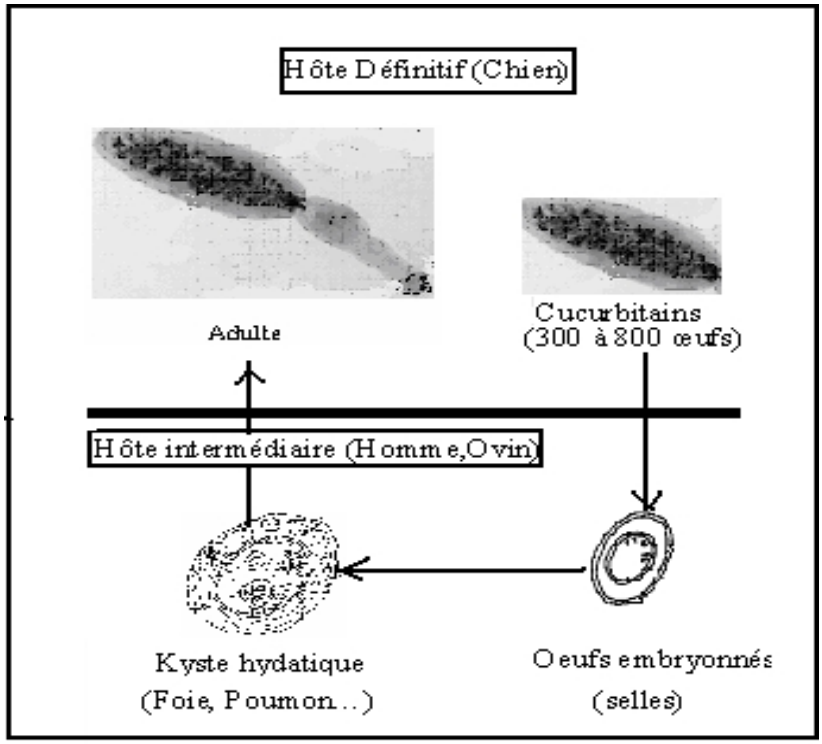


Fig 25. Echinococcus tapeworm (*E.granulosus*) life cycle

2.3.3. Epidemiology

Hydatidosis, also known as hydatid echinococcosis or hydatid cyst, is a zoonosis. Hydatidosis is a cosmopolitan parasite. It is widespread in regions where sheep farming coexists (sheep : main intermediate host) and dogs (the dog: definitive host and reservoir of the parasite), mainly in South America, Australia and the Mediterranean basin, notably the Maghreb. In Algeria, the hydatid index is 7 per 100,000 inhabitants. Human contamination occurs through ingestion of embryophores (contact with a carrier dog) or water and/or food contaminated with embryophores. Embryophores are highly 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).

2.3.4. Symptomatology

In dogs: asymptomatic well tolerated.In humans:

Primary Echinococcosis

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

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

Liver (60 to of cases): , hepatomegaly, pain **Lung** (20 to 30

Kidney, spleen, nervous system, bone... **Secondary** peritoneal **echinococcosis** is observed following spontaneous or induced larval rupture, where the scolexes released by the rupture form vesicles and re- form hydatid cysts, which can then appear anywhere in the body.

Progression is slow and spontaneous recovery is rare.

Diagnosis

Clinical diagnosis

Hepatic hydatidosis: The latency period can last up to fifteen years. Clinical examination may reveal painless hepatomegaly and hypersensitivity reactions (urticaria, angioedema). The diagnosis may also be evoked by abdominal imaging.

Pulmonary hydatidosis: The clinical latency period is shorter. Single or multiple, rounded, opaque or hydroaeric lung parenchymal opacities reveal the diagnosis on a chest X-ray, sometimes performed routinely. Coughing, dyspnea and hemoptysis complete the picture. Biological diagnosis

Liver biopsy should be avoided (risk of secondary echinococcosis through dissemination of protoscolex during puncture). Hypereosinophilia in this helminthiasis is inconstant, and can be detected particularly in the event of cyst rupture. Serological

reactions (ELISA, immunoprecipitation, indirect immunofluorescence) help to orientate the diagnosis, but latent cysts sometimes remain silent. A definitive diagnosis is sometimes made by parasitological examination of puncture fluid, despite the ban on puncture, surgically extracted cysts or vomitus (sputum following rupture of a cyst in a bronchus). This reveals the adult worm's head (or scolex), or the scolex hooks.

Treatment

In humans, surgery is required to remove the hydatid cyst en bloc, or hydatidectomy. During this procedure, any risk of swarming must be avoided by not rupturing the cyst wall. In inoperable patients, or to reduce the risk of secondary echinococcosis during surgery, ESKAZOLE® (albendazole) may be used as an adjunctive treatment. The efficacy of treatment can monitored by serological tests every three months.

Prophylaxis

General prophylaxis: based on veterinary supervision sheep flocks and dogs (deworming of domestic dogs, elimination of stray dogs, slaughterhouse regulations). Individual prophylaxis: humans must protect themselves through individual measures (hygiene measures when in close proximity to dogs: washing hands, avoiding food contact with dogs).

Health education and information play a very important role.

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